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Los Angeles

# Social Pain and the Onset of Social Anxiety Disorder

A dissertation submitted in partial satisfaction of the

Requirements for the degree Doctor of Philosophy in Psychology

by

Richard Thomas LeBeau

2014

ABSTRACT OF THE DISSERTATION

Social Pain and the Onset of Social Anxiety Disorder

by

Richard Thomas LeBeau

University of California, Los Angeles, 2014

Professor Michelle Craske, Chair

The widely accepted theory regarding the etiology of social anxiety disorder (SAD) is rooted in learning theory. In line with Pavlovian conditioning models, the fear and arousal experienced in response to aversive social experiences are believed to generalize, thus producing conditioned fear responses to formally neutral stimuli. Although there is overwhelming evidence for the role of conditioning in the onset of anxiety disorders, there is a paucity of research regarding fear conditioning to aversive social experiences. The present studies aimed to address key questions regarding this significant gap in the literature. The first two studies aimed to develop a socially relevant unconditioned stimulus (US) that is theoretically grounded, sufficiently potent to elicit fear responding, and feasible for use in conditioning paradigms. Study 1 examined the unconditioned response (UR) to this social US by measuring subjective and physiological reactions to a range of intensities of the stimulus and comparing these reactions to those of a well-established physical stimulus. Results suggested that the social US elicited significant subjective and physiological fear responses, but that increasing responding did not occur at increasing levels of intensity and SAD severity did not moderate reactions to the social US. Study 2 aimed to enhance the social US from Study 1 and test its ability to produce a conditioned fear response in a

differential classical conditioning paradigm. The paradigm allowed for the comparison of acquisition, extinction, and return of fear to conditioned stimuli paired with the social US, a physical US, and a control stimulus. Preliminary results from a small pilot sample suggest that fear conditioning to stimuli associated with the social US occurred as hypothesized. Study 3 moves out of the laboratory by examining how social pain and SAD symptoms are related over time in a longitudinal sample of adolescents. Results replicated prior research by finding that chronic stress in an individuals' social group prospectively predicted SAD severity one year later, but did not support the specific relationship between social stress and SAD that etiological model of SAD hypothesizes. The limitations and implications of these studies are discussed and directions for future research are outlined.

The dissertation of Richard Thomas LeBeau is approved.

Naomi I. Eisenberger

Aaron P. Blaisdell

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BIOGRAPHICAL SKETCH

**Richard T. LeBeau, M.A., C.Phil.**

**Education**

*Ph.D. Candidate in Psychology (2008 – Present);* University of California, Los Angeles, CA

- Master of Arts degree conferred on April 24, 2010
- Advanced to candidacy on June 5, 2012
- GPA (current): 3.97
- Minor: Social Psychology

*Bachelor of Arts Degree (2002 – 2006);* Colgate University, Hamilton, NY

- Overall GPA: 3.84
- Concentrations: Psychology and English (with an emphasis in Creative Writing)
- Graduated Summa Cum Laude and with High Honors in Psychology

**Research Grants and Fellowships**

Ruth L. Kirschstein National Research Service Award for Individual Predoctoral Fellows (F31)

- 1F31MH096413-01: "Fear Conditioning and the Onset of Social Anxiety Disorder," National Institute of Mental Health (2011 – 2013)

UCLA Graduate Research Mentorship Award (2011 – 2012)
UCLA Summer Graduate Research Mentorship Program Award (2009, 2010)
UCLA University Fellowship (2008-2009)

**Peer-Reviewed Publications**

1) **LeBeau, R. T.,** Bogels, S., Moller, E., & Craske, M. G. (In Press). Integrating dimensional assessment and categorical diagnosis in DSM-5: The benefits and challenges of the paradigm shift for the anxiety disorders. *Psychopathology Review.*
2) **LeBeau, R. T.,** Mischel, E. R., Resnick, H. S., Kilpatrick, D. G., Friedman, M. J., & Craske, M. G. (In Press). Dimensional assessment of DSM-5 posttraumatic stress disorder. *Psychiatry Research.*
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## General Introduction

*Social Pain*

The need to belong is considered fundamental for a wide variety of species, including (and perhaps especially) humans (Baumeister & Leary, 1995). Humans are biologically driven to form and maintain positive, significant, lasting relationships and as such threats and injuries to an individual's social relationships are met with significant distress. Throughout their lives, humans experience a variety of painful events that threaten their sense of belonging, including being the target of humiliation and insults, being excluded from a group, and the unwanted end of close relationships through discord and death. Social pain is the negative emotional state that is associated with actual or potential damage to one's sense of social connection or social value. Social pain is associated with a wide array of emotional responses, but is most commonly described as "hurt feelings" (MacDonald, 2009). Such feelings are thought to be an evolutionary adaptation seen across many species in order to ensure the maintenance of social bonds that are essential for survival (Panskepp, 2011). Interestingly, the use of physical pain words to describe the subjective experience of social pain is ubiquitous (e.g., "hurt feelings," "broken heart") and nearly universal, occurring in countless cultures and languages throughout the world (MacDonald & Leary, 2005).

The biological system through which social pain exerts its effects in many species is believed to have built off the pre-existing physical pain system. There is indeed evidence of significant overlap between the two systems (see Eisenberger, 2012 for a thorough review). Animal research has demonstrated that repeated social defeat leads to the same biological and behavioral effects as receiving inescapable shocks (Mineka & Zinbarg, 1996). The administration of opioid receptor agonists (such as morphine) that have long been used to

alleviate physical pain have been found to substantially reduced distress associated with maternal separation in many species (Panskepp, 1998). Neuroimaging research in humans demonstrates that areas central to physical pain responses, such as the dorsal anterior cingulate cortex (dACC) and anterior insula (AI), are also central to social pain responses in humans (Eisenberger, 2011). A double blind, placebo-controlled study of Tylenol (an analgesic drug that alleviates physical pain) demonstrated the drug's ability to reduce the degree of social pain in response to daily stressors as well as dACC and AI activation in response to social exclusion in a laboratory (DeWaal et al., 2010).

A growing body of research suggests that in addition to being subjectively unpleasant, social pain has deleterious effects on the health of humans. Laboratory studies tend to examine this by using social evaluative threat (SET) paradigms, in which individuals are exposed to potential or actual negative judgment from others. SET is accompanied by specific physiological responses, including negative alterations in neuroendocrine, cardiovascular, and immune system functioning (e.g., Dickerson, 2008). A review of 208 acute psychological stressor studies found that the effect size for SETs on cortisol increase was nearly three times that of stressors lacking a social evaluative component ($d$ = 0.67 vs. $d$ = 0.21; Dickerson & Kemeny, 2004). Studies in which the SETs were coupled with uncontrollability demonstrated an even larger effect size ($d$ = 0.92). Similar elevations to SETs have been found with regard to cardiovascular markers, although the effects seem less specific to SET compared to the effects of cortisol (Gruenewald, Dickerson, & Kemeny, 2007). A meta-analysis found that SETs reliably alter certain immune parameters, including an increase in proinflammatory cytokines (Segerstrom & Miller, 2004). Studies have also found that although the affective response to SETs are in many ways similar to

non-social threat scenarios, the prominence of self-consciousness appears to be a unique feature of SETs (e.g. Gruenewald et al., 2007; Dickerson, 2008).

Research has also demonstrated that chronic experiences of social pain outside of the laboratory can have long-term negative impacts on physiology and health. A meta-analysis concluded that chronic stressors that are social in nature are related to elevated daytime HPA axis activity (elevated cortisol levels; Miller, Chen, & Zhou, 2007). Loneliness, which is often experienced as social pain, shows similar cortisol effects (e.g., Pressman et al., 2005). It is widely believed that overexposure to stress hormones over time results in a cumulative toll on the body known as allostatic load, which in turn elevates risk to a variety of diseases (e.g., McEwen, 1998).

The findings from the social pain literature converge to support the hypothesis that experiences of social pain are not only subjectively distressing but also have negative health outcomes that are objective and measurable. The vast majority of this research has been done in areas such as social psychology, health psychology, and neuroscience, and has received relatively little attention in clinical psychology. This is unfortunate, considering that the social pain literature has potentially enormous implications for understanding the etiology, maintenance, and treatment for many psychopathologies.

*Fear of Social Pain and Clinical Anxiety*

Just as humans are predisposed to fear and avoid physical pain, they are also predisposed to fear and avoid social pain. Such fear of social pain has long been noted in humans. For example, a great deal of evidence shows that human beings are predisposed to fear other humans displaying anger toward them. Evolutionary biologists have noted the structural similarities

between responses to threatening animals and angry facial expressions and have suggested that social fears in humans can be traced back to the predator defense systems of early mammals (Ohman, 1986). Interestingly, rapid fear acquisition and slow extinction has been found for angry faces directed at participants, but not for faces displaying other emotions and not for angry faces looking away from the participant (Ohman & Dimberg, 1978; Dimberg & Ohman, 1983). These effects are seen not only in anxious individuals, but healthy controls as well, suggesting the common and perhaps adaptive fear response to stimuli that are likely to engender social pain.

It is also well established in the literature that a tendency to fear and avoid social pain can become highly problematic for individuals, leading to clinically significant pathology. Several decades ago, Beck and colleagues (1974) observed that anxiety disorders tend to be rooted in one of two types of concerns: concerns about the possibility of physical harm and concerns about the possibility of social harm. The latter fear is a hallmark of Social Anxiety Disorder (SAD), a disorder characterized by "a marked fear or anxiety about one or more social situations in which the individual is exposed to possible scrutiny by others" (DSM-5; APA, 2013). In line with this definition, SAD appears to be more theoretically connected to social pain experiences in which the relationship is threatened or lost because the self is devalued (e.g., rejection, negative evaluation) as compared to experiences in which the relationship is lost but the self is not implicated (e.g., death of a loved one). Such a distinction is often made when conceptualizing different types of social pain experiences (Eisenberger, 2012).

SAD is a highly prevalent and disabling condition. The replication of the National Comorbidity Survey reported a 12-month prevalence rate of 7.1% and a lifetime prevalence rate of 12.1% in the United States (Ruscio et al., 2008). SAD is the third most common psychiatric disorder, following major depression and alcohol abuse (Schneier, 2006). The disorder also

begins very young, with a mean age of onset in adolescence and many cases beginning by age seven (Chavira & Stein, 2005). SAD is accompanied by significant psychiatric comorbidity, with an estimated 69 to 81% of individuals with SAD meeting criteria for another mental disorder at some point in their life (Fehm & Wittchen, 2004). Individuals with SAD are more likely to abuse substances and more likely to commit suicide than individuals without SAD (Baldwin & Buis, 2004). Decreases in occupational, academic, and family functioning have also been associated with the disorder, with individuals with SAD more likely to be unemployed and unmarried than individuals without SAD (Baldwin & Buis, 2004). SAD is also a chronic condition for many individuals, as previous research has found that over half of individuals diagnosed with SAD at one time point will still have significant symptoms five years later (Beard, Weisberg, & Keller, 2010).

Although much research has been conducted on SAD in the past three decades, the mechanisms through which the disorder develops remain in need of further research (Bogels et al., 2010). Many etiological models of SAD support a diathesis-stress paradigm, such that an individual has a certain predisposition to the development of the disorder, which helps determines the amount of stress needed to trigger the onset of the disorder (e.g., Schmidt, Polak, & Spooner, 2005). Such models focus on several distinct areas of contribution to the onset of SAD, such as genetic and temperamental factors, cognitive aspects, parent-child interactions, and adverse environments (Brook & Schmidt, 2008). A review of environmental factors that have demonstrated relevance to etiological models of SAD suggests that many social pain experiences have been empirically linked to the onset of SAD. These experiences include separation from parents, exposure to sexual and physical abuse, and teasing and bullying at the hands of peers (Brook & Schmidt, 2008).

Several other lines of research support the link between social pain and SAD. Constructs related to social pain have also been found to be predictive of SAD, including high trait behavioral inhibition (e.g., Schwartz, Snidman, & Kagan, 1999) and low levels of perceived social support (e.g., Moak & Agrawal, 2010). Individuals who are high in rejection sensitivity (a personality construct that describes the tendency of an individual to anxiously expect, readily perceive, and intensely react to experiences of social rejection) are more likely to experience mood and anxiety disorders than those without this trait (e.g., Downey, Mougios, Ayduk, London, & Shoda, 2004; Tops, Riese, Oldehinkel, Rijsdijk, & Ormel, 2007). Furthermore, retrospective self-report data shows that individuals tend to conceptualize experiences of social pain as leading to the onset and worsening of their social anxiety symptoms (Ost, 1985).

Although there is considerable evidence for the role of social pain experiences in the development of SAD, little is known about the precise manner in which social pain experiences impact SAD onset. One possibility is that social pain experiences are aversive events that produce conditioned fear, which becomes overgeneralized and leads to clinically significant distress and impairment for individuals predisposed to develop anxiety disorders. This hypothesis has been fairly prevalent in the literature over the past few decades (e.g., Ost & Hugdahl, 1981; Mineka & Zinbarg, 2006) and is the core theory underlying cognitive behavioral therapy for SAD, a highly effective and widely used intervention for SAD (Hofmann & Smits, 2008). Although there is overwhelming evidence for the role of conditioning in anxiety disorders (Craske et al., 2009) there is a paucity of research regarding the role of conditioned fear to a social pain experience in the onset of SAD. The present studies aim to address key questions regarding this significant gap in the literature.

*Overview of the Present Research*

In order to demonstrate that fear conditioning to stimuli associated with social pain experiences plays an important role in the onset and maintenance of SAD, three key hypotheses need to be tested. First, it must be established that social pain experiences produce significant subjective and objective fear responses. Study 1 addresses this issue by comparing unconditioned responses (UR) to social pain and physical pain across a range of intensity. Second, it needs to be shown that such responses are sufficiently aversive to generalize to associated stimuli that were formally neutral (thus, that social pain is capable of producing conditioned fear responses). Study 2 utilizes a differential conditioning paradigm to examine the degree to which a social pain stimulus elicits conditioned fear compared to a physical pain stimulus and a neutral stimulus. Third, a unique relationship between social pain experiences and SAD symptomatology must be demonstrated (such that social pain events predict SAD more strongly than distressing but socially irrelevant events and that social pain events more strongly predict SAD than other disorders not rooted in social fears). Study 3 addresses this by examining the relationship between acute and chronic experiences of social pain and the onset and worsening of SAD symptomatology one year later in a large sample of adolescents assessed longitudinally.

## Study 1: Exploring the Unconditioned Response to Social Pain

### Introduction

Decades of research supports the theory that the conditional fear acquisition and extinction process is analogous to the onset of fear-related disorders and the treatment of such disorders with exposure-based therapy (e.g., Bouton, Mineka, & Barlow, 2001). In such paradigms, an initially neutral conditional stimulus (CS) does not elicit a fear response when presented alone. During fear acquisition, the CS is paired with an unconditional stimulus (US), an aversive stimulus that naturally elicits a fear 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. The CR is then extinguished by presenting the CS repeatedly without the US. Although this process alone cannot explain the complex cognitive processes that occur in fear-based psychopathologies, it is believed to be a vital component of the secondary, higher order conditioning processes that comprise such complex cognitive functions (Blaisdell, 2009). However, very few studies have examined classical conditioning to negative social stimuli.

A few studies have evaluated whether individuals with SAD were more likely than healthy controls to form aversive associations in such experimental paradigms, but each used CSs and USs that were socially irrelevant (e.g., noxious odors, painful pressure; Herman, Ziegler, Birbaumer, & Flor, 2002; Schneider et al., 1999; Veit et al., 2002). These studies found no evidence for elevated rates of conditioning in individuals with SAD to these socially irrelevant stimuli relative to healthy controls. These findings, however, may be due to the discordance between the participants' fears and the stimuli used in the experiments. It is likely

that socially relevant stimuli would be more salient to individuals with SAD and as such are more likely to result in elevated conditioning.

Studies have investigated this hypothesis by examining conditioned fear responses to a socially relevant CS, but are limited by their use of nonclinical samples and their use of physically painful USs (e.g., Dimberg, 1990; Dimberg & Christmanson, 1991; Dimberg & Thunberg, 2007; Esteves, Parra, Dimberg, & Ohman, 1994). In such research, a social pain cue (e.g., an angry face) is paired with a physically painful stimulus (e.g., an electric shock) and compared to a neutral social cue (e.g., a neutral face) that is also paired with a shock. Although such paradigms are effective at producing conditioned fear to the neutral face paired with the electric shock, these effects often fail to generalize to fear of other such faces. This is clearly inconsistent with models of SAD in which the overgeneralization of social fears is a core feature (Mineka & Zinbarg, 1996). One possible reason is that, again, the US in these studies is a socially irrelevant stimulus. The use of a US that is more consistent with individuals with SAD's locus of fear (e.g., a verbal insult paired with a threatening face) may provide a more ecologically valid US and thus be more likely to elicit conditional responding.

Only four conditioning studies have utilized a socially painful US and each had notable limitations. The first used a novel paradigm in which a fearful face accompanied by a loud scream served as the US (Lau et al., 2008). This US was selected given ethical concerns about administering electric shock or negative feedback to anxious adolescents, who comprised the sample of the study. The US was effective at producing conditional fear responses, but has potential limitations as a socially painful US. First, having a participant view and hear the reaction of a fearful person is likely to operate in a different manner than a US, which communicates threat directly to the participant. This 'fearful individual' US may serve more as

an interesting extension of vicarious conditioning models in that it signals that there is potential danger in the environment and that fear is an appropriate response. Second, the US is limited by the fact that it involves a loud, unpleasant sound and thus may have evoke a response akin to those evoked by physically painful stimuli. Furthermore, this study looked only at subjective markers of conditioning and did not use objective physiological measures of arousal, which are not subject to the same biases as self-report and are generally preferred in conditioning research.

The second study examined responses to a socially painful US with demonstrated among adults with SAD (Lissek, 2008). Critical facial expressions combined with negative comments (e.g., "I don't like you!") served as the US, which were paired with a neutral facial expression (CSneg). Participants' subjective arousal and fear-potentiated startle responses to CSneg were compared to their responses to two other sets of neutral faces, one which was paired with a neutral face and a neutral verbal comment (CSneu) and one which was paired with a happy face giving a positive verbal comment (CSpos). Results revealed conditioned startle potentiation (a commonly used indicator of elevated fear responding) and self-reported fear elevation to CSneg in individuals with SAD but not age- and gender-matched healthy controls. This study introduced a novel, ecologically valid paradigm and highlighted the importance of examining USs relevant to social pain when examining conditioning to social fears.

Despite the theoretical strengths of the socially relevant US developed by Lissek (2008), participants experienced a much higher degree of habituation to the associated CS than typically occurs when using a physical pain US. It is possible that this rapid habituation is typical for social pain stimuli, but it is also possible that this is an artifact of the study methodology. One notable limitation is that the only physiological measure used was fear potentiated startle. It is possible that simply using other physiological measures that are less susceptible to rapid

habituation (e.g., skin conductance responses; Vansteenwegen, Crombez, Baeyens, & Eelen, 1998) would correct this problem. However, it is also possible that there are ways in which the socially relevant US could be enhanced. One option is to use angry adult male face instead of a young female face, as evolutionary theorists have posited that angry male faces are most in line with the predator defense system and as such they may evoke a greater fear response (e.g., Ohman, 1986). A second option is to use a social pain US that is more direct and personally relevant to the participant (e.g., negative verbal feedback delivered specifically to participants regarding their performance on a task). The use of a personally relevant US would bring the fear conditioning paradigms more in line with SET paradigms, which have been used extensively in the social pain literature. In SET paradigms, the participant typically believes that they are at risk for being negatively evaluated, which contrasts with these fear conditioning paradigms, in which the participants watch an individual make a critical facial expression and a comment that they have no reason to believe is directed at them personally. Personal relevance of the social US is also important to comparing its effects with that of a physically painful US, which is inherently personally relevant.

Hassad, Lissek, Pine, and Lau (2011) conducted a follow-up to Lissek et al. (2008), in which healthy controls aged 12 to 15 were shown neutral faces paired with a happy face and a positive comment (CSpos), a neutral face and a neutral comment (CSneu), and an angry face with a written insult (CSneg). Salience of the stimuli was enhanced in two key ways. First, CSs were matched to the adolescents' age and gender. Second, the face-comment pairings were described as being part of an interaction between the three children depicted rather than as being directed at the participant. Although this is an improvement over the previous paradigm in that it has more ecological validity, it is still limited by its lack of personal relevance to the participant.

Results indicated significant decrease in pleasantness ratings and increase in scariness ratings to CSneg following acquisition, but the study did not include physiological measures of conditioning.

Tinoco-Gonzalez and colleagues (2014) attempted to replicate the findings of Lissek et al. (2008) in a series of two studies. They utilized the identical social US present in the prior study as well as the same dependent variables (fear potentiated startle and subjective ratings of valence and arousal). The authors found no evidence of physiological conditioning to the social US during the acquisition phase for any of the groups in either study. The authors found evidence of conditioning to the social US when analyzing the subjective ratings, but found no significant differences between individuals with SAD and healthy controls (Study 1) or individuals with subthreshold social anxiety and healthy controls (Study 2). Although discouraging, the authors' conclusion that "fear conditioning is not enhanced in social anxiety and that alternative mechanisms may play a role in understanding social anxiety" may be premature. It is possible that what is needed is an improved experimental paradigm. Such a paradigm would utilize a social US that is personally relevant and examines a range of physiological measures.

Interestingly, a highly effective and specific US for studying social pain has been developed in mice. In the social fear conditioning (SFC) paradigm developed by Toth, Neumann, and Slattery (2012), mice are exposed to repeated, uncontrollable foot shocks while exploring unfamiliar con-specifics. This aversive experience leads to a short-term and long-term reduction in social behavior in the mice that experience it. The effect of the paradigm is not only robust, but also specific, as it leads solely to reduced social behavior and not reduced exploration of novel items, reduced locomotion, general anxiety, or depressed behavior. Such specificity

distinguishes this paradigm from the many others that have been developed to induce social anxiety responses in animals, such as social isolation, social instability, social defeat, conditioned defeat, and maternal separation (for a review, see Toth and Neumann, 2013). Unfortunately, this paradigm does not translate easily to human research – administering physical pain to individuals as they perform in vivo social interactions is ethically dubious and logistically impractical. The paradigm also suffers from the conflation of physical and social pain in that the mice may only be avoiding social behavior out of fear of physical pain. However, the effectiveness of this paradigm provides further support for the importance of personal relevance and ecological validity in creating an effective social US.

The few studies that have examined a socially relevant US relied primarily on subjective measures of fear arousal, with the exception of two studies that included fear-potentiated startle (Lissek et al., 2008; Tinoco-Gonzalez, 2014). As such, the appropriate physiological measurements to use when assessing response to a socially relevant US is unknown. Some evidence can be gleaned from a study by McTeague and colleagues (2009), which examined the physiological responses to visualizing feared scenarios in participants with SAD and demographically matched controls. Participants were seated in a quiet, dimly lit room and instructed to vividly imagine the events described by the audio recordings, which included social threat scenarios (e.g., social confrontation, public speaking failure), survival threats, neutral events, and idiosyncratic scenarios created for each participant based on their greatest area of fear. Participants with SAD demonstrated elevated fear-potentiated startle, skin conductance reactivity, and heart rate acceleration compared to controls for the idiosyncratic and social fear scenarios, but the groups did not differ in response to survival threat or neutral scenarios on any of these measures. Interestingly, no difference was found between participants with SAD and

healthy controls with regard to corrugator tension (tension in the muscles of the upper face). The authors hypothesized that this finding might be due to the fact that not only can facial expressivity be modulated by top-down control, but also that it often is during episodes in which social pain either occurs or is threatened. These findings are generally consistent with responses to physical pain USs and thus provide some evidence that physiological responses to social pain may be similar to those of physical pain. However, the present study is limited by its use of imagery as opposed to visual or auditory stimuli and as such does not directly inform the question of what the appropriate physiological measurements are to capture response to a socially painful US.

In contrast to the findings of McTeague et al. (2009), there is at least one prominent theory that suggests that the observed fear response to a socially painful stimulus markedly differs from those typically seen to a physically painful stimulus. Ohman (1986) suggests that the Social Submissiveness System (SSS) may be activated in response to a socially painful US whereas the Predatory Defense System (PDS) is activated by a physically painful US. Whereas the PDS is activated by cues suggesting the presence of predators and functions to promote escape or avoidance of predators, the SSS is activated by a dominant, threatening opponent and serves to avert attack from dominant members of one's own species. The PDS produces vigorous escape and avoidance behaviors and utilizes the sympathetic nervous system, producing elevated heart rate, shortness of breath, and many other symptoms characteristic of an acute fear response. The SSS, on the other hand, produces a much less significant response by the sympathetic nervous system and is characterized by submissive gestures, such as blushing and lowering of the head. Thus, if the SSS is being activated by a socially painful US, the typically utilized

objective markers of sympathetic nervous system arousal (such as increases in skin conductance response, heart rate, and fear-potentiated startle) may not be appropriately suited.

One potential marker that may be uniquely activated by the SSS is the blushing response, which is a hallmark of embarrassment, a common feature of SAD. Both fear of blushing and actual blushing behavior are reliably present in many individuals with SAD, but the two do not always correlate highly (Gerlach, Wilhelm, Gruber, & Roth, 2001; Mulken, Bogels, de Jong, & Louwers, 2001). In other words, how much one fears that they will blush in a given situation and perceives that they actually are blushing is not particularly predictive of actual blushing response. Previous research has found evidence that individuals with SAD are more likely to blush than healthy controls during certain tasks and that individuals with SAD who also fear blushing show greater sympathetic nervous system activation than individuals with SAD who do not fear blushing (Gerlach et al., 2001). Actual blushing response can be measured by changes in facial blood volume, which there are several well-established methods of collecting (for a review, see Gerlach & Cooper, 2013). Thus, including measurements of blushing response in studies examining the effects of a social pain US may provide important information. In light of the dearth of research in this area, it is important that one of the first steps is examining what UR profile typically occurs in response to social USs. This can best be done through comparison of the well-established UR profile of physical USs.

In the present study, the UR to a range of intensity of a novel, personally relevant social pain US were examined in individuals both high and low in SAD symptomatology. Participants were exposed to social pain USs and physical pain USs in a counterbalanced, within-subjects design and objective and subjective responses to the stimuli were recorded. We hypothesized that the social US will elicit significant subjective and physiological responses. We expect these

responses to be moderated by SAD such that participants with high levels of SAD will demonstrate significantly greater responding to the social US than those low in SAD. Similarly, we hypothesize that participants will demonstrate increasing subjective and physiological responding to increasing levels of the social US intensity. We also expect SAD to moderate this relationship, such that participants with high levels of SAD will show greater responding to increasing intensity levels of the social US than those low in SAD. As physical and social USs have yet to be evaluated in the same study, we make no a priori hypotheses about the comparative strength in responding to the social US and physical US. However, based on the theoretical literature described above, we do hypothesize that participants' responses to the social US will involve less sympathetic nervous system activation and greater blushing response than responses to the physical US.

## Method

**Participants.** Forty-five individuals participated in the present study. Participants were recruited from undergraduate psychology courses and the broader community via advertisements. Upon completion of the protocol, participants received either course credit or $20 in cash. Study participants were 60% female and 40% male. The racial makeup of the sample was 47% White/Caucasian, 47% Asian/Asian-American, 4% Black/African-American, and 2% Native American/Pacific Islander. Hispanic/Latino ethnicity was endorsed by 20% of the sample. The sample was comprised mostly of college-aged individuals ($M$ = 22.2, $SD$ = 7.9, *range* = 18 – 56). Participants were only excluded from the present study if they had significant difficulties with hearing or vision, diagnosed with serious cardiac or respiratory conditions, were currently pregnant, or had been instructed by their physician to stay away from stressful situations.

**Objective Measures.** Autonomic activity was recorded continuously using a non-invasive ambulatory monitoring device (BioPac MP150; http://www.biopac.com/data-acquisition-analysis-system-mp150-NDT-Mac; for more information on this system, see Pflanzer, 1999). This device has demonstrated successful measurement of skin conductance responses (SCRs) and fear-potentiated startle (FPS) in previous fear and anxiety research (e.g., Lipp, 2006). Baseline physiology measures were collected for five minutes prior to the initiation of experimental procedures. Data was cleaned, inspected, and analyzed using AcqKnowledge software (http://www.biopac.com/acqknowledge-data-acquisition-analysis-software-win.)

Skin conductance signals were transmitted using two electrodes attached to the ring and middle fingers of the non-dominant hand. Skin conductance amplitude was recorded to the nearest microsiemen (μS). SCR was determined by taking the difference between the mean skin conductance level 2 seconds prior to US onset and the maximum SCL in the 6 seconds following US onset. Non-responders were classified as those participants who did not have at least one SCR value above 0.01 μS amplitude change, and their SCR data was not used as in other studies (e.g., Hughdahl, 1995).

Fear-Potentiated Startle (FPS) was measured by exposing the subject to a startle probe (which consisted of a burst of 105 decibel white noise presented for 50ms; Williams, Elfner, & Howse, 1978) and recording of the resultant change in electromyography, or electrical activity of the muscles. Eye blink was recorded from two 6-mm electrodes placed under the right eye. Two startle probes were presented during each inter-trial interval (ITI). The first occurred 15 to 35 seconds after US offset. The second probe occurred 10 seconds after the first. FPS responses were calculated by taking the difference between the mean of EMG level for the 200

milliseconds (ms) prior to startle probe onset and the maximum EMG level in the 20-150 ms following startle probe offset.

Blushing response (BR) was measured using a photoplethysmograph (PPG), which detects changes in infrared reflectance resulting from varying blood flow to the face. This was done via a matched infrared emitter and photo diode that was attached to each participant's forehead. Placement on the forehead is based on prior research, which concluded that such placement yields the same results as cheek placement and is much less sensitive to facial movement (Cooper & Gerlach, 2013). BRs were calculated by taking the difference between the mean blood flow for the 10 seconds prior to US onset and the peak blood flow in the 30 seconds following US offset. This comparatively long window of measurement is based on prior research that suggests that the blushing response may take significantly longer to reach its peak than other measures of autonomic activity (Cooper & Gerlach, 2013).

**Self-Report Measures.** All participants completed the Social Phobia Inventory (SPIN; Connor et al., 2000), a 17-item questionnaire that has demonstrated strong validity and reliability in the assessment of the spectrum of SAD symptomatology in adolescents (Johnson, Inderbitzen-Nolan, & Anderson, 2006) and adults (Antony, Coons, McCabe, Ashbaugh, & Swinson, 2006). A cutoff score of 19 has been established as effectively distinguishing clinical from non-clinical populations (Connor et al., 2000). Participants also completed the Rejection Sensitivity Questionnaire (RSQ; Downey & Feldman, 1996), an 18-item measure assessing the general tendency to perceive and expect rejection in interpersonal relationships. The Blushing Propensity Scale (BPS; Leary & Meadows, 1991) asked participants to rate how often they feel they blush in each of 14 circumscribed social situations. This measure will allow the relationship between subjective and objective blushing responses to be examined. Trait anxiety was measured by the

State Trait Anxiety Inventory (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983), a 20-item measure assessing cognitions and emotions associated with anxiety. Depression symptoms were assessed using the Beck Depression Inventory (BDI-II*;* Beck, Steer, & Brown, 1996) a widely used 21-item questionnaire that assesses symptoms of negative mood. In the present study, we omitted 1 item that assessed current suicidality due to IRB concerns about having research assistants who were not clinically trained administering the questionnaires. Thus we used a modified 20-item version. Finally, mindfulness was measured using the Mindful Attention Awareness Scale (Brown & Ryan, 2003), a well-validated 15-item self-report measure that examines individual's tendency to engage in mindful awareness on a routine basis.

Participants rated the unpleasantness of each US they received during the experiment on a Subjective Units of Unpleasantness Scale specifically designed for this study. The scale was based on the Subjective Units of Distress Scale (Wolpe, 1969) and asks individuals to rate how unpleasant each stimulus was on a scale ranging from 0 ("not unpleasant at all") to 100 ("the most unpleasant thing I have ever experienced"). Anchors were also provided for 25 ("mildly unpleasant"), 50 ("moderately unpleasant"), and 75 ("very unpleasant"). The word 'unpleasantness' was chosen because it was determined by the researchers to be a descriptor that would not be biased toward either US type. See Appendix A for the Subjective Units of Unpleasantness Scale that was presented to participants.

**Experimenter Rating of Believability.** Due to the hypothesized importance of personal relevance in increasing the salience of the US, experimenters made ratings of how much the participant appeared to believe that there really was another individual evaluating them and that the feedback they received during the experimental phase was actually based on their performance on the interview task. The experimenter's rating was based on a combination of the

participant's responses to open-ended questioning about their experience of the study prior to debriefing and the participant's explicit self-report of how much they believed the manipulation following debriefing. The experimenter responsible for running each participant rated the participant on a Likert-type scale ranging from 1 ("not convinced at all") to 10 ("completely convinced").

**Procedure.** Upon arriving at the lab, participants were taken through the informed consent process, completed self-report measures, and were oriented to the set-up of the laboratory by a member of the research team, which was comprised of highly trained Bachelor's level research assistants. Participants were informed that the aim of the study was to examine individual's emotional and biological reactions to evaluating others and being evaluated by others. Electrodes monitoring SCR, FPS, and BR were then attached and participants underwent a 5-minute baseline period in which baseline physiological data for the participants was collected.

Participants then underwent an interview task. They were told they would be interviewed for 5 minutes by the experimenter and that this interaction would be recorded on a webcam attached to the participant's computer monitor. They were informed that an individual in an adjacent room would then rate this recording and provide feedback about the participant's performance, which would be presented to them later in the experiment. Participants then watched a 5-minute video in which a young woman goes through the interview task, in which she responded to a series of questions. Questions including those that asked about interests ("What is your favorite hobby?"), goals ("What do you see yourself doing in five years?"), and current events ("What do you feel are the biggest challenges facing the United States today?"). Participants were then instructed to select up to 5 adjectives from the same list of positive,

neutral/ambiguous, and negative adjectives that were pilot tested earlier. Afterward, participants took part in the same interview task.

The experimenter then informed the participant that they had transmitted the file to the raters in the adjacent room. Participants were told that various adjectives that the rater selected to describe them would be shown to the participant later in the experiment along with a picture of the rater's face. This combination of the image of the rater's negative facial expression and the auditory negative adjective comprised the social US. Participants were then provided examples of all stimuli that would be used in the experiment, including one low intensity muscle stimulation, one example of neutral feedback, and one startle probe.

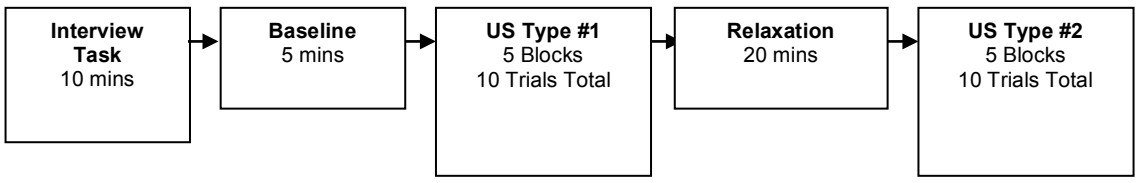
In the experimental phase, individuals experienced two distinct blocks of stimuli. In the physical US block, they were administered 10 muscle stimulations. These stimulations are designed to be annoying to the participant, but not painful, and have been established in prior research with undergraduates to be safe and effective at eliciting a fear response (e.g., Lipp, 2006). Five levels of increasingly intense muscle stimulation levels were administered that varied along two dimensions – stimulus duration and voltage. Two trials of each level were presented. See Table 1 for the muscle stimulation properties at each level of intensity.

**Table 1. Muscle stimulation properties at each level of intensity**

| | **Voltage** | **Duration** |
|---|---|---|
| **Level 1** | 15 v | 50 ms |
| **Level 2** | 25 v | 100 ms |
| **Level 3** | 35 v | 150 ms |
| **Level 4** | 45 v | 200 ms |
| **Level 5** | 55 v | 250 ms |

In the social pain US block, participants were administered 10 pieces of feedback from the interview task. Five levels of increasingly negative feedback were administered that also varied along two dimensions - the negativity of the adjective and the clarity of the image (more information about the development of the social US is provided below). Two trials of each level were presented. In both blocks, the stimuli were presented between 60 and 90 seconds apart. During this inter-trial interval the startle probe was presented (see description of FPS above). Participants underwent random assignment to determine which US block they would experience first and which of three pre-established stimulus presentation orders they would experience. These orders were created randomly with the caveat that two presentations of the same intensity level were never presented consecutively. In between the two blocks, participants were disconnected from the psychophysiology equipment and instructed to take a 20-minute in order to give them a chance to emotionally and physiologically return to baseline prior to initiating the second block. After each US presentation in the study, the participant provided ratings of unpleasantness on the scale described above. See Figure 1 for a flowchart of study procedures.

**Figure 1. Flowchart of procedures for Study 1**




Following completion of both US blocks participants were thoroughly debriefed. This included full disclosure that the negative feedback received during the experiment had nothing to do with their actual performance on the interview task. The experimenter also explained to the participants that such deception and negative feedback are necessary in social pain research in

order to increase ecological validity. Participants were reminded that the UCLA North General Institutional Review Board (IRB#11-003136) and the National Institute of Mental Health (1F31MH096413-01) approved all study procedures. All participants were provided with local low-cost mental health resources at the conclusion of the study.

**Development of the Social US.** The social fear conditioning paradigm used in the present study combined a social US similar to that used in Lissek et al. (2008) with a manipulation designed to enhance its personal relevance that was based on a task deigned by Leary, Haupt, Strasser, and Chokel (1998) and modified by Eisenberger, Jarcho, Leiberman, and Naliboff (2006). As described above, we hypothesized that using a rejecting male face would enhance the salience of the US. In order to limit variability in US duration, we decided to use adjectives (e.g., boring) rather than full phrases (e.g., "I don't like you") for the verbal feedback. The images and adjectives were selected through a multistep process. Images were taken from a large database of videos that were created for a prior study in the laboratory (see Niles et al., 2013 for more details). Individuals in the videos included both volunteers and professional actors in their twenties and thirties. The videos consisted of the individual saying a series of statements that ranged from highly negative ("Nobody likes you") to neutral ("I need a pencil") to highly positive ("I really like you"). After reading these statements, individuals were asked to make a series of facial expressions that they felt captured specific negative, neutral, and positive emotions. Videos ranged from 10 to 20 minutes long.

First, two researchers isolated images from the videos of eight male actors that they felt represented clear and distinct examples of negative, neutral, and positive emotional states. This process resulted in approximately 20 images for each actor. Second, the pool of actors was cut down to four by selecting the actors who had the highest quality and broadest range of negative

facial expressions. Third, seven images were selected for each of these four actors – four negative, two neutral, and one positive. More negative facial expressions were included than neutral or positive facial expressions due to the fact that finding evocative negative images was the most important to the present research. Although no positive images were included in the final studies, they were included in pilot testing to provide a greater degree of variability in ratings.

Finally, the 28 images (7 for each of the 4 actors) were uploaded to an internet-based data collection system (Survey Monkey) and the valence, arousal, and degree of social pain associated with the pictures were rated by a sample of convenience. After each picture, participants were asked: 1) "How negative is this facial expression?", 2) "How distressed would you feel if somebody made this facial expression at you?", and 3) "How rejected would you feel if somebody made this facial expression at you?" Participants were asked to rate their responses on a 1 to 7 Likert-type rating scale ranging from "Not [negative, distressed, rejected] at all" to "Extremely [negative, distressed, rejected]."

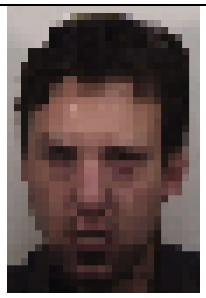
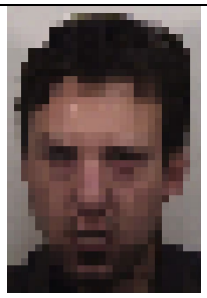
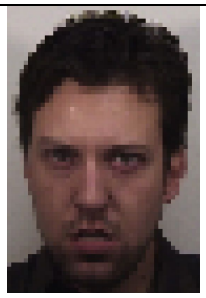
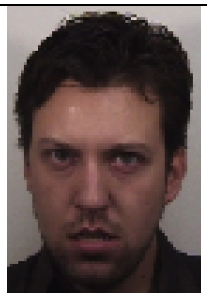
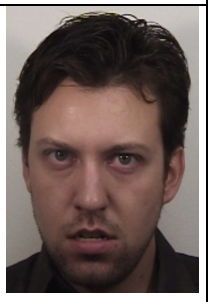
In addition to the images, 40 adjectives (26 negative, 8 neutral/ambiguous, and 6 positive) commonly used to describe the personality and performance of individuals were generated by the researchers and pilot tested. After each adjective, participants were asked: 1) "How negative is this word?", 2) "How distressed would you feel if somebody used this word to describe you?", and 3) "How rejected would you feel if somebody used this word to describe you?" Participants were asked to rate their responses on a 1 to 7 Likert-type rating scale ranging from "Not [negative, distressed, rejected] at all" to "Extremely [negative, distressed, rejected]."

Forty-nine undergraduate and graduate students at UCLA rated the images and adjectives. The sample was 74% female, racially and ethnically diverse (50% Caucasian, 26% Asian-American, 10% Hispanic/Latino, 8% Middle Eastern, 6% other race not specified, 4% multiracial,

2% African-American), and college-aged ($M$ = 23.6, $SD$ = 3.7, *range* = 19 – 36). Ratings of negativity, distress, and rejection were highly inter-correlated (all $r$s > 0.60), with the relationship between ratings of distress and rejection being particularly strong (all $r$s < 0.80). Thus, we decided to look only at distress ratings.

We selected the image with the highest distress rating ($M$ = 4.90, $SD$ = 1.62) and subjected it to various levels of pixilation using the iPhoto program. Level 1 was almost entirely pixilated (thus mostly obscuring the image) and the pixilation gradually decreased until the image was completely clear in Level 5. We selected five adjectives that had associated distress ratings ranging from mildly to extremely distressing and that were considered by the researchers to be particularly relevant to performance on the interview task. The adjectives selected were "nervous" ($M$ = 3.53, $SD$ = 1.43); "naïve" ($M$ = 4.02, $SD$ = 1.84), "boring" ($M$ = 4.64, $SD$ = 1.69), "selfish" ($M$ = 5.80, $SD$ = 1.34), and "unlikeable" ($M$ = 6.06, $SD$ = 1.27). See Figure 2 for the social USs at each of the five levels.

**Table 2: Social US properties at each level of intensity.**

| | **Level 1** | **Level 2** | **Level 3** | **Level 4** | **Level 5** |
|---|---|---|---|---|---|
| Image |  |  |  |  |  |
| Adjective | Nervous | Naïve | Boring | Selfish | Unlikeable |

**Statistical Analyses.** All analyses were conducted using SPSS v21. Scores on the SPIN, BPS, and BDI-II were calculated by totaling the items. Scores on the STAI and MAAS were

calculated by reverse coding the necessary items and then totaling all items. Scores on the RSQ were determined by multiplying the anticipated distress and perceived likelihood of each of the 18 scenarios and then dividing the total score by 18, thus creating an average score for each scenario that took distress and likelihood into account simultaneously. Descriptive statistics were run for all of the self-report scales and the relationship among the scales was determined via Pearson correlations (*r*s). The reliability of subjective ratings was determined by calculating a Pearson correlation (*r*) for each pair of stimulus presentations that comprised each level (e.g., trial 1 and trial 2 for level 5 of the social US).

SCRs were range-corrected via a three-step method. First, the largest SCR to any US was determined for each participant. Second, the SCR to each US was divided by the largest SCR. Third, the square-root of this value was taken. Outliers at each time point for all psychophysiology measures were transformed through Winsorizing (Wilcox & Keselman, 2003). In order to control for the effects of inter-individual variability in baseline EMG and PPG levels, each FPS and BR was converted to a *z* score based on the mean and standard deviation of all responses for the respective measure. Analyses were conducted with the raw data and the *z*-transformed data for FPS and BR. As no differences were found between the two sets of analyses and the raw data is easier to interpret with respect to our analyses, we present the raw data in the results section.

The relationship between stimulus intensity and reactivity was determined using repeated measures ANOVAs. For the subjective unpleasantness analyses, the within-subjects factor was intensity level and five levels were defined using the mean unpleasantness rating for the two trials. If Mauchly's Test of Sphericity was violated ($p > 0.05$), a Greenhouse-Geisser correction was used. Tests of within-subjects effects were examined to determine the main effect of intensity

and tests of within-subjects contrasts were examined to determine whether a linear model provided the best fit for the data. To test the moderating effects of SAD symptoms, the repeated measures ANOVAs were rerun with total score on the SPIN as a covariate. The main effect for the SPIN and the resultant interaction term (intensity x SPIN) were then inspected for statistical significance. Several additional analyses were run in which total scores on the other self-report scales, manipulation believability score, gender, race, and ethnicity were entered as covariates in order to determine whether any of these variables served as moderators of the relationship between intensity levels and subjective and physiological responding. In order to examine the overall difference in subjective and physiological reactivity to the two types of USs, we calculated an average score of responses to the social US and physical US for each index (i.e., subjective unpleasantness, SCR, FPS, and BR) and subjected the mean values for each index to a paired samples *t*-test.

## Results

**Self-report scales.** A wide range of scores was observed across self-report scales in the sample. Scores on the SPIN exceeded the established clinical cut-off for 67% of the sample, thus suggesting that a significant portion of the present sample had clinically significant SAD symptoms. As expected, the three self-report scales most relevant to SAD (SPIN, RSQ, and BPS) were significantly inter-correlated (all $r$s > 0.50). See Table 3 for descriptive statistics of the scales and Table 4 for the correlations between the scales.

**Table 3. Descriptive statistics for self-report scales in Study 1**

| | *M* | *SD* | *Range* |
|---|---|---|---|
| **Social Phobia Inventory (SPIN)** | 28.2 | 16.1 | 1 – 64 |
| **Rejection Sensitivity Questionnaire (RSQ)** | 11.7 | 4.7 | 2 – 25 |
| **Blushing Propensity Scale (BPS)** | 40.3 | 11.0 | 17 – 62 |
| **State Trait Anxiety Inventory (STAI)** | 43.6 | 11.9 | 22 – 66 |
| **Beck Depression Inventory-II (BDI-II)** | 7.7 | 8.0 | 0 – 31 |
| **Mindful Attention Awareness Scale (MAAS)** | 61.2 | 12.5 | 29 – 90 |

**Table 4. Inter-correlations of self-report scales in Study 1**

| | **SPIN** | **RSQ** | **BPS** | **STAI** | **BDI-II** | **MAAS** |
|---|---|---|---|---|---|---|
| **SPIN** | 1 | | | | | |
| **RSQ** | 0.61** | 1 | | | | |
| **BPS** | 0.61** | 0.54** | 1 | | | |
| **STAI** | 0.45** | 0.35* | 0.14 | 1 | | |
| **BDI-II** | 0.25 | 0.18 | -0.13 | 0.78** | 1 | |
| **MAAS** | -0.42** | -0.27 | -0.28 | -0.61** | -0.45** | 1 |

**Note:** ** = $p < 0.01$; * = $p < 0.05$; all values without notation = $p > 0.05$

**Believability of manipulation.** Believability data was gathered for 36 participants (80% of the sample). The mean believability rating was quite low but scores were distributed across the range of possible scores ($M = 4.3$, $SD = 3.1$, *Range* = 1 – 10). Experimenters characterized participants as follows: 6 (17%) completely believed the manipulation with little to no skepticism (believability rating of 10), 12 (33%) mostly believed the manipulation but were somewhat

skeptical (believability rating of 5 – 9), 7 (19%) somewhat believed the manipulation but were very skeptical (believability rating of 2 – 4), and 11 (31%) did not believe the manipulation at all (believability rating of 1). Believability rating did not correlate with any self-report scale with the exception of the BPS ($r = 0.34$, $p < 0.05$), suggesting that participants who were more concerned about blushing were also more likely to believe the manipulation.

**Subjective ratings of unpleasantness.** Following each US presentation, participants provided an unpleasantness rating. Ratings across both trials of each level were highly reliable for both US types (see Table 5). When averaged across all stimulus presentations in each block, subjective reactions to the social US did not differ from subjective reactions to the physical US (social US: $M = 34.4$, $SD = 20.1$; physical US: 41.8, $SD = 15.4$; $t(35) = -1.7$, $p = 0.11$). A repeated measures ANOVA with a Greehouse-Geisser correction determined that unpleasantness ratings significantly differed by intensity level for both the social US ($F(3.1,132)=19.7$, $p < 0.001$) and physical US ($F(1.4,51.1)=82.5$, $p < 0.001$). Relationships between intensity level and unpleasantness rating were best characterized by a linear relationship for both the social US ($F(1,42)=44.8$, $p < 0.001$) and the physical US ($F(1,36)=100.5$, $p < 0.001$). Examination of the F values and visual inspection of the means (see Figure 2) suggests that this linear function was more pronounced for the physical US than the social US. When entered as a covariate, SPIN score did not moderate the unpleasantness ratings for the social US (main effect of SPIN: $F(1,41)=0.04$, $p = 0.85$; interaction of SPIN and intensity: $F(4,164)=0.05$, $p = 0.99$) or the physical US (main effect of SPIN: $F(1,35)=0.8$, $p = 0.34$; interaction of SPIN and intensity: $F(4,140)=0.11$, $p = 0.98$). Further analyses revealed that no other variables moderated the unpleasantness ratings, including self-report scales, demographic factors, and experimenter rating of believability of the manipulation. Discomfort during the physical US phase resulted in 3

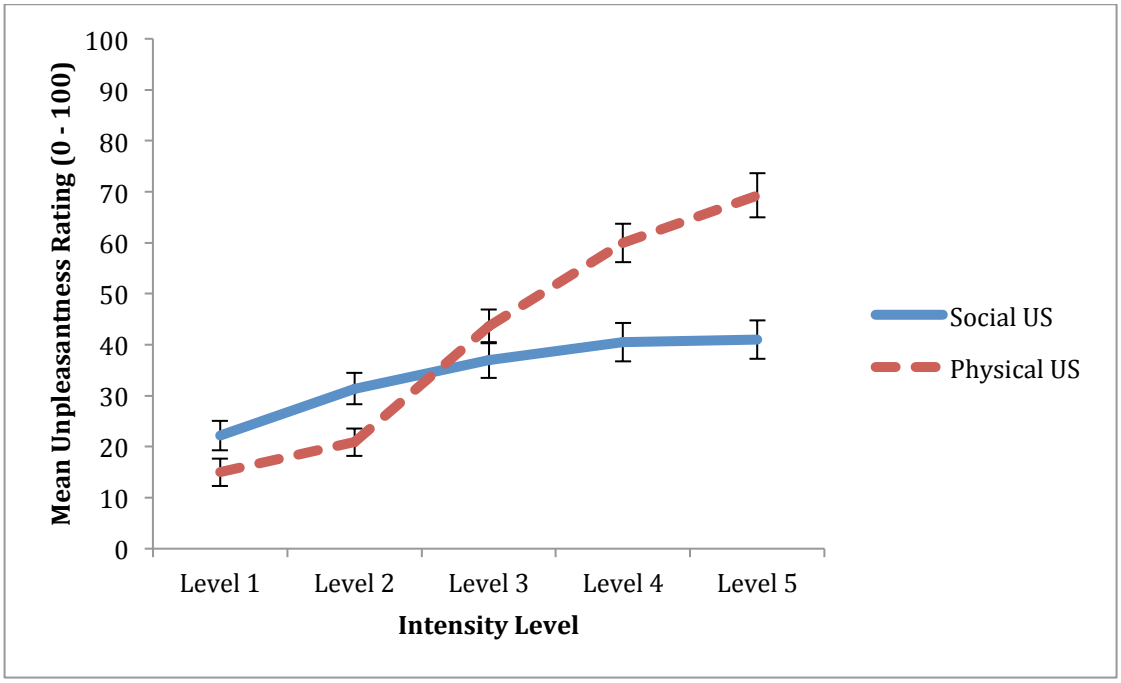
participants (7%) asking to terminate the experiment early. No participants asked to terminate the experiment early during the social US phase.

**Table 5: Reliability of unpleasantness ratings at each level of intensity for both stimuli**

| | Level 1 | Level 2 | Level 3 | Level 4 | Level 5 |
|---|---|---|---|---|---|
| **Social US** | 0.70 | 0.84 | 0.75 | 0.77 | 0.82 |
| **Physical US** | 0.77 | 0.72 | 0.68 | 0.80 | 0.92 |

*Note:* Values represent Pearson Correlations (*r*) between the unpleasantness ratings made for both presentations of each level of each stimulus. All correlations are significant at $p < .001$.

**Figure 2: Unpleasantness ratings for social and physical USs by levels of intensity**

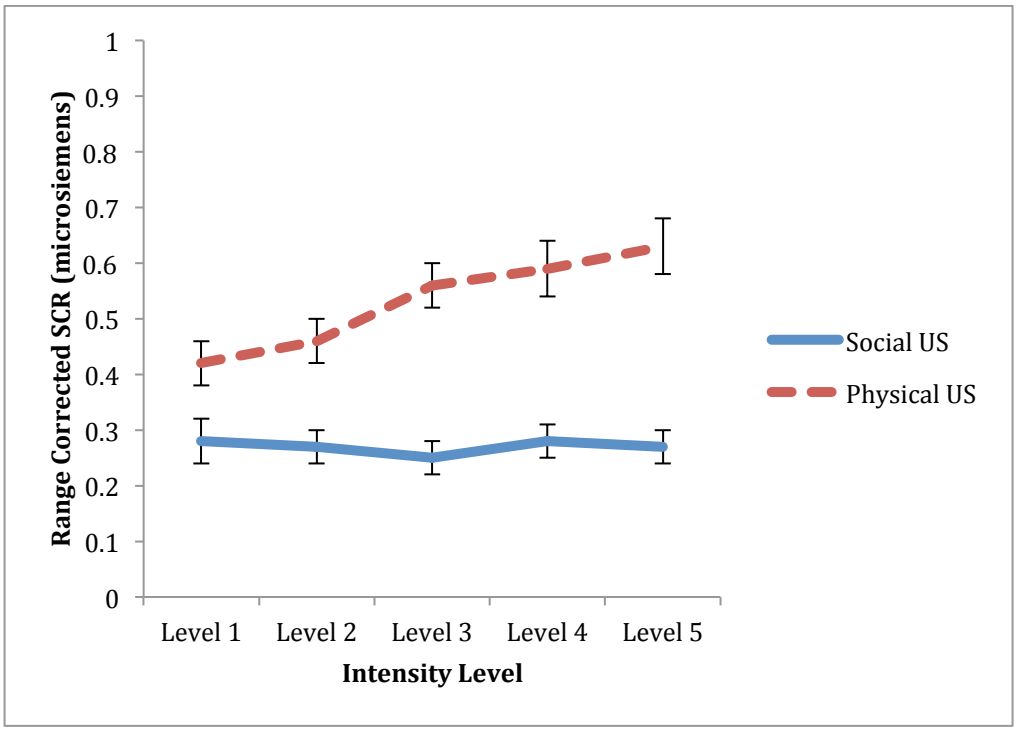



**Note:** Error bars represent the standard error of the mean.

**Physiological reactivity.** Range-corrected SCR was calculated for each stimulus presentation. When averaged across all stimulus presentations in each block, SCRs to the two stimuli types differed at a statistically significant level with the physical US eliciting a greater SCR response than the social US (physical US: $M = 0.62$, $SD = 0.19$; social US: $M = 0.3$, $SD =$

0.18; $t(39)=-9.0$, $p < 0.001$). A repeated measures ANOVA with a Greenhouse-Geiser correction revealed that SCRs significantly differed by intensity level for the physical US ($F(2.4,91.9)=10.7$, $p < 0.001$) but not for the social US ($F(3.2,142.6)=0.3$, $p = 0.87$). The relationship between intensity level and SCR for the physical US was best characterized by a linear relationship ($F(1,38)=19.2$, $p < 0.001$). See Figure 3 for means and standard errors by intensity level. When entered as a covariate, SPIN score did not moderate the significant relationship between SCRs and intensity level for the physical USs (main effect of SPIN: $F(1,37)=3.0$, $p = 0.09$; interaction of SPIN and intensity: $F(2.4,88.7)=0.35$, $p = 0.74$). Further analyses revealed that no other variables moderated the SCR – intensity relationship, including self-report scales, demographic factors, and experimenter rating of believability of the manipulation.

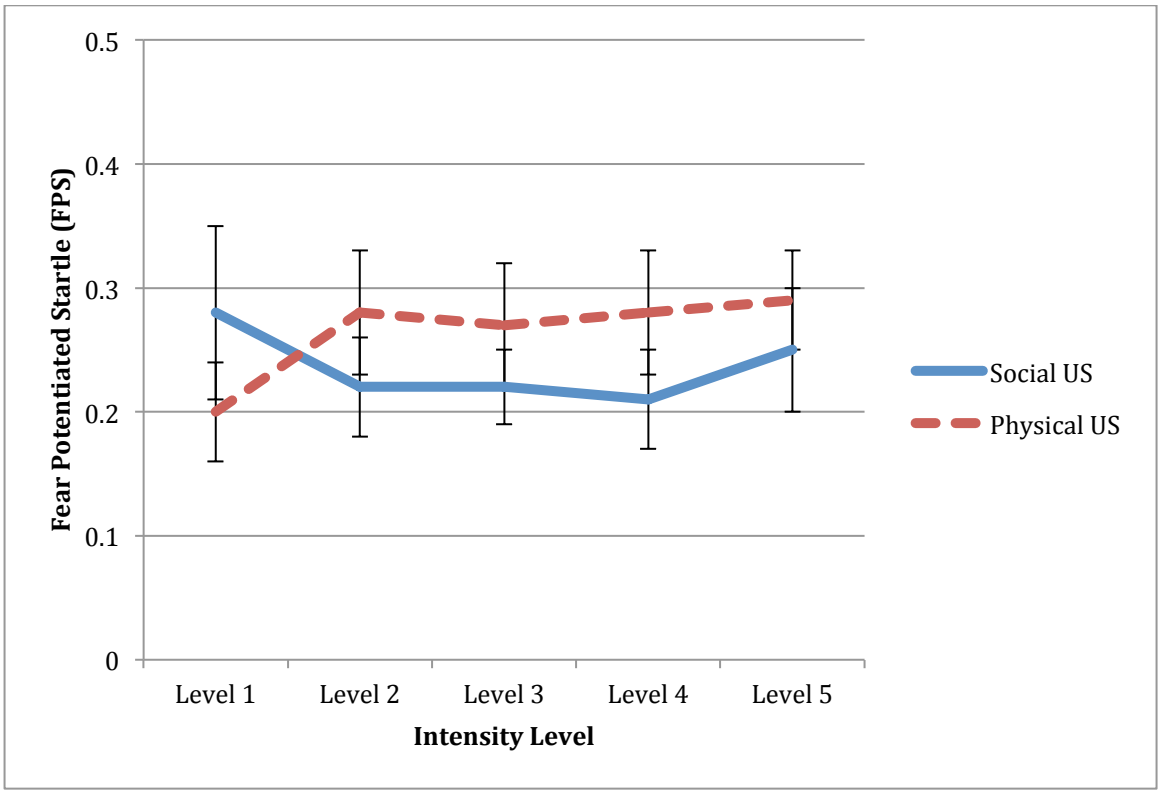
**Figure 3: Skin Conductance Response (SCR) for social and physical USs by levels of intensity**




**Note:** Error bars represent the standard error of the mean.

FPS was calculated for each stimulus presentation. When averaged across all stimulus presentations in each block, FPS to the two stimulus types did not differ at a statistically significant level (social US: $M = 0.36$, $SD = 0.25$; physical US: 0.28, $SD = 0.28$; $t(36)=-0.6$, $p = 0.57$). A repeated measures ANOVA with a Greenhouse-Geiser correction revealed that FPS did not significantly differ by intensity level for the either US type (social US: $F(1.8,64.9)=1.0$, $p = 0.40$; physical US: $F(2.8,104.7)=1.3$, $p = 0.26$). See Figure 4 for means and standard errors of FPS by intensity level.

**Figure 4: Fear potentiated startle (FPS) for social and physical USs by levels of intensity**

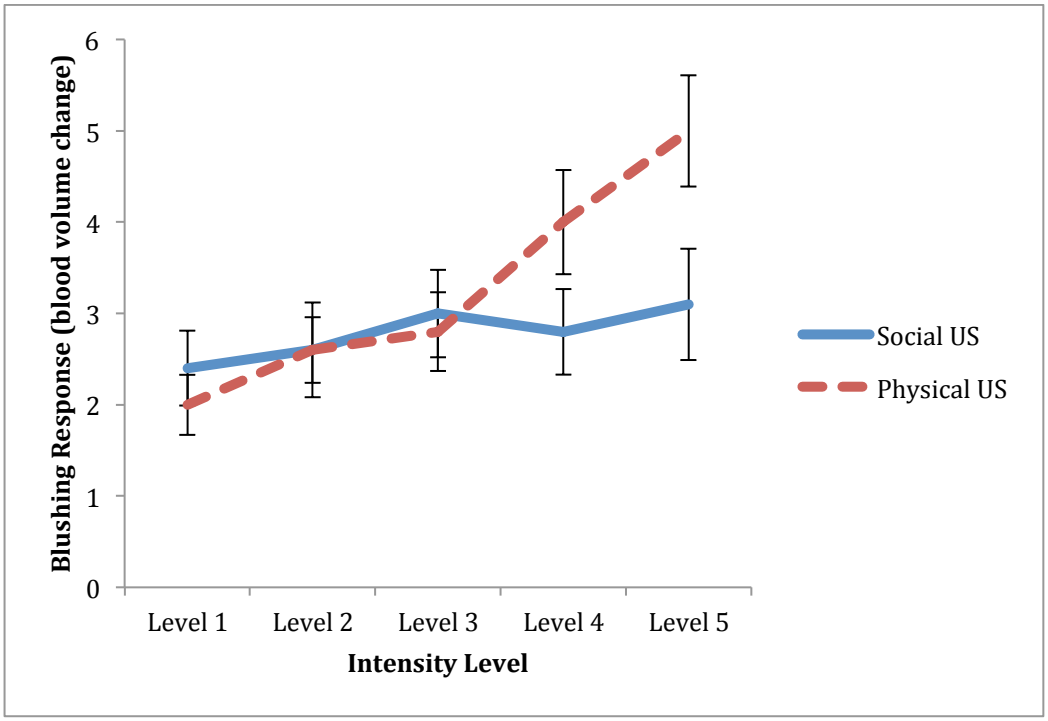



**Note:** Error bars represent the standard error of the mean.

BR was calculated for each stimulus presentation. When averaged across all stimulus presentations in each block, BR to the two stimulus types did not differ at a statistically significant level (social US: $M = 3.0$, $SD = 2.9$; physical US: 3.3, $SD = 2.6$; $t(36)=-0.6$, $p = 0.52$). A repeated measures ANOVA with a Greenhouse-Geiser correction revealed that BR differed by

intensity level for the physical US ($F(2.9,102.8)=9.1$, $p < 0.001$) but not for the social US ($F(2.7,97.2)=1.5$, $p = 0.20$). The relationship between intensity level and BR for the physical US was best characterized by a linear relationship ($F(1,35)=20.6$, $p < 0.001$). See Figure 3 for means and standard errors of BR by intensity level. When entered as a covariate, SPIN score did not moderate the significant relationship between BR and intensity level for the physical US (main effect of SPIN: $F(1,34)=0.1$, $p = 0.71$; interaction of SPIN and intensity: $F(3.6,107.1)=1.2$, $p = 0.29$). Further analyses revealed that no other variables moderated the BR, including self-report scales, demographic factors, and experimenter rating of believability of the manipulation.

**Figure 5: Blushing response (BR) for social and physical USs by levels of intensity.**




**Note:** Error bars represent the standard error of the mean.

## Discussion

The development of an effective socially relevant US is essential to conducting the research necessary to improve our etiological models of SAD. The present study examined subjective and physiological responding to a social US that was developed to address key limitations of those used in prior studies. Participants with a wide range of SAD symptoms were exposed to varying levels of intensity of the social US as well as varying levels of intensity of a physical stimulus traditionally utilized in conditioning research (i.e., bicep muscle stimulation). Such a design allows for the examination of key theoretical assumptions regarding the role of fear conditioning in SAD onset. Paramount among these questions is whether increasing the intensity of a social US leads to enhanced subjective and physiological responding as is typically seen with a physical US and if so, if this relationship is specific to individuals with high levels of SAD.

Results revealed that both the social US and physical US elicited significant subjective and physiological responding. Although SCRs were significantly larger in response to physical USs than social USs, mean responses to the two stimuli types did not differ for subjective unpleasantness, FPS, or BR. These findings suggest that the social US utilized in the present study is salient enough to elicit emotional and physiological arousal, which is consistent with the findings of Lissek et al. (2008). It expands upon this prior research by demonstrating significant SCRs and BRs to a social US, which have been hypothesized but never tested in prior literature.

Consistent with hypotheses, a linear function of subjective responding to the increasing stimulus intensity levels was observed for the social US. Thus, participants reported increasing levels of intensity as more unpleasant. While significant, the effects were small and the linear function demonstrated by the social US was much less pronounced than the linear function demonstrated for the physical US. Contrary to hypotheses, a linear function of responding to the

increasing stimulus intensity levels was not observed for the social US on any of the physiological measures. Thus, increasing the intensity of the social US did not lead to significantly greater SCR, FPS, or BR. The findings regarding the linear function of physiological responding to the physical US intensity levels were mixed. As is well established in the literature, increasing physical US intensity led to greater SCRs. The same was true for BR, marking the first time this relationship has been demonstrated in the literature. However, the expected linear function was not observed for FPS in response to the physical US.

Three plausible explanations exist for the lack of significant findings regarding the linear function for physiological responses to increasing intensity of the social US. The first explanation is that the dimensions on which we varied the social US (i.e., clarity of the rejecting face, negativity of the adjective) are not the key dimensions on which the social US should be varied. Perhaps there are other dimensions (e.g., the negativity of the facial expression, the tone of voice in which the feedback is delivered) that would elicit increased responding. The second explanation is that responses to a social US may be dichotomous in nature, such that once an individual perceives rejection s/he finds it similarly distressing regardless of the intensity of the rejection. Perhaps it is merely the perception that another person does not like an individual that drives the subjective and physiological responses, particularly in the absence of an explanation for rejection or negative evaluation. If this is the case, increasing levels of clarity of the facial expression and the negativity of the adjective would not necessarily be expected to elicit increased responding. The third explanation is that the inter-individual variability in responding to social rejection is so substantial that reliable and distinct levels of intensity are not easily established. Perhaps the specific facial expressions and comments that are particularly distressing are idiosyncratic in nature, such that a lower level of the social US may have been more

distressing to a subset of participants than a higher level, thus obscuring the data. This possibility seems more plausible for the social US than the physical US, as few participants are likely to find a lower voltage delivered for a shorter period of time less distressing than a higher voltage delivered for a longer period of time. Of course, these three explanations are not mutually exclusive and may all be playing a role. Each should be examined in future research.

Contrary to our hypotheses, SAD symptom severity did not moderate subjective or physiological responding to the social US. The fact that participants high in SAD symptoms did not react more strongly to the social US than participants low in SAD suggests that although it may be effective at eliciting subjective and physiological responses, the social US may not be specifically distressing to individuals with SAD. This is discouraging considering that the development of a social US was pursued due to concern that the physical USs typically utilized in conditioning research are not relevant to individuals with SAD and that more robust findings would likely result from paradigms utilizing a socially relevant US.

The lack of significant findings regarding SAD severity as a moderator of responses to the social US may be due to several factors. The possibility that social USs are not more salient for individuals with social anxiety than individuals without social anxiety cannot be discounted (although it makes little theoretical sense). Alternatively, these findings may be due to the use of a non-clinical sample. Perhaps many of the individuals with high SPIN scores in the present sample do not actually have clinically significant SAD symptoms and responding to the social US is enhanced only in individuals with an established case of the disorder. Future research could examine this possibility by replicating the design of the present study in a sample of treatment-seeking patients who have received a formal diagnosis of SAD. It should be noted that responding to the physical US was also not moderated by any of the measures assessing negative affect,

which is inconsistent with the prior literature and suggests that there may be features of the present sample that are influencing these results.

Contrary to hypotheses, blushing was not a unique indicator of response to the social US. Blushing responses did not differ significantly between the social and physical US. However, it should be noted that elevated blushing responses were found in response to both sets of stimuli and as such blushing appears to be a useful physiological measure in conditioning research. Evidence for lower sympathetic nervous system activation in response to the social US than for the physical US is mixed. Although significantly smaller SCRs were elicited by the social US compared to the physical US as hypothesized, this same pattern was not true for FPS. The findings regarding FPS may be due in part to the study design, which administered the startle probes in the ITI between the random presentations of stimuli. We conceptualized the FPS response in the ITI following the US to be indicative of the response to the prior US, but results may have been impacted by the somewhat lengthy duration between US offset and the first startle probe (15 to 35 seconds) or the participants' anticipation of the upcoming US.

In addition to the potential limitations already reviewed (e.g., the limited dimensions the social US was varied on, the use of a non-clinical sample), the overall lack of believability of the manipulation is a significant limitation that likely affected the present findings. Only 17% of participants completely believed the manipulation, with others citing confusion or disbelief regarding certain aspects of the experimenter's explanation. As previously discussed, the personal relevance of the social US is likely to be key to eliciting a strong subjective and physiological response. If the majority of participants truly did not believe another individual was actually negatively evaluating their performance, responding to the social US was likely blunted and vulnerable to rapid habituation. We attempted to address this issue by examining experimenter

ratings of participant believability as a moderator of responding to the social US, but these results were not significant. This may be due in part to the fact that these ratings were highly subjective and only collected for a subset of the sample. Future research should identify methodological changes that may enhance the believability of the manipulation and utilize more sophisticated and objective measures of participant believability.

Despite these limitations, the present study advanced the study of social fear conditioning and addresses several significant gaps in the literature. This investigation marks the first study to examine the unconditioned response to a social US. Although the four studies discussed earlier have examined fear conditioning to a social US, no study has ever examined the UR to a social US. The present design also marks the first time social and physical USs have been examined in the same study. This design allows for comparing and contrasting the UR of a social US with a physical US, the UR of which is much better understood. As previous studies have used subjective responses and FPS as dependent variables, the addition of SCR and BR provided an opportunity for a more detailed understanding of physiological responses to a social US. Perhaps most notably, the social US developed in the present study marks the first personally relevant social US suitable for use in human fear conditioning research. The US merges the precise methodology of the prior conditioning studies with the ecological validity of the social evaluative threat (SET) paradigms in a manner that can continue to be refined and enhanced.

Ultimately, the present findings generate more questions than they do provide answers. However, given the nascent stage of research regarding social fear conditioning this was expected and should be seen as an opportunity to generate future research. The present study needs to be replicated in a clinical sample of patients with SAD in order to determine if the lack of significant findings regarding SAD symptoms and enhanced responding to the social US found in the present

study is robust or a product of the present sample. The social US used in the present study should be refined for enhanced believability and utilized in a fear conditioning paradigm. This will help determine whether the social US is salient enough to produce conditional responding and will provide an important replication and extension of the prior studies in this area. Exploring such questions is vital to improving our understanding of the etiology of a prevalent and disabling condition.

## Study 2

## Classical Conditioning with a Social Pain Stimulus: A Pilot Study

### Introduction

As previously reviewed, the fear acquisition and extinction process that occurs in Pavlovian conditioning paradigms is widely considered to be analogous to the onset of fear-related disorders and the treatment of such disorders with exposure-based therapy (e.g., Bouton, Mineka, & Barlow, 2001). Such paradigms traditionally involve three phases: 1) a brief habituation phase, in which participants become familiar with the CSs; 2) an acquisition phase, in which the CS is paired with the US; and 3) an extinction phase, in which the CS is repeatedly presented without the US. This paradigm allows for the examination of how quickly and strongly fear to the CS is learned as well as how long it takes this excitatory relationship between the US and CS to be replaced by an inhibitory relationship, such that the participant realizes the two are no longer paired (for a review of key issues in the extinction of human fear conditioning, see Hermans, Craske, Mineka, & Lovibond, 2006). Researchers are increasingly noting the importance of adding a fourth phase in which the individual returns several days to one week after the initial procedure and is again exposed to the CSs (e.g., Rescorla, 2000). This phase is called extinction retest and evaluates whether the inhibitory relationship between the US-CS that should develop by the end of the extinction phase of the initial procedure is maintained over time or whether the excitatory US-CS relationship returns. This so-called spontaneous recovery of fear is hypothesized to be a key reason for the return of symptoms following exposure-based treatment (Bouton, 1993; Craske et al., 2008).

Despite the prominence of etiological theories of SAD that suggest the key role of a conditioning process similar to the one described above, few studies have evaluated conditioning

to socially relevant USs. A few studies have evaluated whether individuals with SAD were more likely than healthy controls to form aversive associations in such experimental paradigms, but these studies found no evidence for elevated rates of conditioning in individuals with SAD (Herman et al., 2002, Schneider et al., 1999, Veit et al., 2002). This may be because each study used CSs and USs that were socially irrelevant. Several additional studies have examined conditioning to socially relevant stimuli (e.g., Dimberg, 1990; Dimberg & Christmanson, 1991; Dimberg & Thunberg, 2007; Esteves et al., 1994). Unfortunately most of these studies used physical USs and were not conducted with socially anxious samples. These studies have had some success with eliciting conditioned fear responses to the socially relevant CSs, but these effects often fail to generalize to fear of other faces. This is clearly inconsistent with models of SAD in which the overgeneralization of social fears is a core feature (Mineka & Zinbarg, 1996).

In light of these findings, many hypothesized that in order for enhanced conditioning to occur in socially anxious patients, a US relevant to social fears was needed. Four studies have been conducted that utilized social USs in fear conditioning paradigms in adolescents and adults (Lau et al., 2008; Lissek, 2008; Haddad, et al., 2011; Tinoco-Gonzalez et al., 2014). The results of these studies were mixed. Subjective fear conditioning occurred in each study, but a physiological measure was only used in two studies (Lissek, 2008; Tinoco-Gonzalez et al., 2014). Lissek et al. (2008) found rapid habituation with regard to the FPS response and Tinoco-Gonzalez and colleague's (2014) attempt to replicate their findings resulted in no evidence of FPS responses. The lack of robust findings in these studies may be due to the lack of personal relevance of the US used in each study and the use of only one measure of physiological responding (see Study 1 for a detailed description of these issues).

The aim of the present study is to evaluate the ability of the social US developed in Study 1 to elicit conditional fear responding to formerly neutral stimuli in a small pilot sample. The study builds upon the prior research in several key ways. First, it uses a personally relevant social US that should theoretically produce greater distress than the social USs utilized in previous research. Second, a differential conditioning paradigm was used that provided the opportunity to compare the conditional responding elicited by the social US to a well-established physical US as well as a neutral stimulus that was unlikely to elicit an unconditioned fear response. Third, the study measures multiple indices of responding, both subjective (US expectancy, valence and arousal ratings) and objective (evaluation of SCR, BR, and FPS). Fourth, participants were be recruited across a wide spectrum of SAD symptomatology, allowing for a more nuanced examination of how SAD symptoms are related to responses to the social US. Fifth, the study includes an extinction retest to evaluate whether the reduction of fear that individuals experience during the extinction phase is maintained one week later.

Based on the results of Study 1 and prior studies using social USs (Lau et al., 2008; Lissek, 2008; Haddad, et al., 2011; Tinoco-Gonzalez et al., 2014), we generated the following hypotheses. We expect the social US to elicit conditional fear responding, such that participants will experience greater objective and subjective fear response to the CS associated with the social US compared to the CS associated with the neutral stimulus across acquisition, extinction, and extinction retest. Despite the lack of significant moderating effects of SAD severity in Study 1, we hypothesized that individuals with significant SAD symptoms will experience greater responding to the CS associated with the social US than individuals low in SAD symptoms. As the results of Study 1 provided mixed findings about the relative strength of responding across the social and physical US types and this is the first study to examine a differential conditioning

paradigm using the two US types, no hypotheses were made about responding to the CS associated with the social US compared to the CS associated with the physical US.

## Method

**Participants.** Ten individuals participated in the present pilot study. Recruitment methods were identical to Study 1. Upon completion of the protocol, participants received either course credit or $30 in cash. Study participants were 60% female and 40% male. The racial makeup of the sample was 90% White/Caucasian and 10% Asian/Asian-American. Hispanic/Latino ethnicity was endorsed by 30% of the sample. The sample was comprised mostly of college-aged individuals ($M$ = 23.0, $SD$ = 12.0, *range* = 18 – 57). The same exclusion criteria applied as in Study 1.

**Measures.** Participants in Study 2 completed the same six self-report scales completed by participants in Study 1 (SPIN, RSQ, BPS, STAI, BDI-II, MAAS). An additional scale was added to Study 2 to examine fear of physical sensations due to the theoretical relevance of this construct to the physical US. To measure this, we used the well-validated physical components subscale of the Anxiety Sensitivity Index-3 (Taylor et al., 2007).

The same physiological measures used in Study 1 were used in Study 2. SCRs were measured by subtracting the mean skin conductance level of the 2 seconds prior to CS onset from the maximum skin conductance level of the 6 seconds post-CS onset. Similarly, BRs were measured by subtracting the mean PPG level of the 2 seconds prior to CS onset from the maximum PPG level of the 6 seconds post-CS onset. FPS was measured by subtracting the mean EMG level of the 200 ms prior to startle probe onset from the maximum EMG level in the 20 ms – 150 ms window following startle probe offset. As in Study 1, startle probes were 50ms presentations of 105 decibel white noise presented through headphones. One startle probe was

presented during each CS presentation (between 5.5 and 7 seconds post-onset) and during each ITI (between 15 and 25 seconds after CS offset).

Study 2 also utilized self-report measures of conditioning. Participants made valence and arousal ratings in response to each CS at 4 time points: 1) post-acquistion/pre-extinction (Visit 1), 2) post-acquisition (Visit 1), 3) pre-extinction retest (Visit 2), and 4) post-extinction retest (Visit 2). Valence was rated on a Liker-type scale ranging from 1 (not at all unpleasant) to 7 (extremely unpleasant). Arousal was rated on a similar scale (1 = not at all fearful; 7 = extremely fearful). Online US expectancy ratings were made for the social US and the physical US upon each CS presentation. See Appendix B for the valence and arousal rating forms provided to participants. Ratings were made via two continuous, sliding dials (one assessing physical US expectancy and one assessing social US expectancy) placed conveniently in front of them (Lovibond, Davis, & O'Flaherty, 2000). Participants were asked to rate how certain they were that each US would appear at the end of each trial on the respective dial. Each dial had three anchors: completely certain the US will occur, uncertain whether the US will occur, and completely certain the US will *not* occur. There was significant room in between each of the three anchor points so that participants could provide nuanced expectancy ratings.

**Stimulus selection for Study 2.** The CSs used in the present study were selected from the pool of images pilot tested in Study 1 (see section "Development of the Social US" for detailed discussion of methodology). We selected the image for each of the four actors that had the lowest distress rating among those classified as a neutral facial expression by the researchers. As the present study only utilizes three CSs, the three images with the lowest distress ratings were selected. The ratings for the three selected images were as follows. The first image (CSsoc), which was paired with the social US, had a mean distress rating of 2.25 (*SD* = 1.30). The second

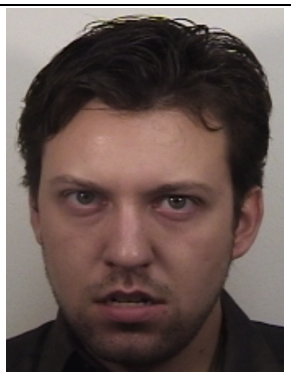
image (CSphy), which was paired with the physical US, had a mean distress rating of 1.96 (*SD* = 1.21). The third image (CSneu), which was paired with the neutral US, had a mean distress rating of 1.86 (*SD* = 1.04). On the rating scale used during pilot testing, a score of 1 corresponded to "not at all distressing." Thus, it is unlikely that any of the CSs selected elicited emotional distress prior to being paired with the US.

The social and physical USs utilized in the present study were based on the findings from Study 1. As this study is specifically interesting in looking at the ability of the social US to elicit conditional fear responding, we selected the highest level of intensity of the social US, which was also the level that had the highest unpleasantness rating. We then examined the various levels of the physical US to determine a level that was as subjectively unpleasant as the highest level of the social US so that the USs utilized in the present study were matched on intensity level. The physical US with the closest mean distress rating was the level 3 physical stimulus (a 35v stimulation administered for 150ms). A paired sample *t*-test confirmed that the mean unpleasantness ratings for the selected stimuli did not differ significantly in Study 1 (social US: *M* = 40.3, *SD* = 25.7; physical US: 43.9, *SD* = 20.3; $t(35)=-0.64$, $p = 0.53$).

In addition to comparing the conditioning capacity of the social US to the physical US, we also sought to compare the social US to a neutral US that was not hypothesized to elicit any distress. In effect, this US served as a control. In order to match it as closely as possible to the social US, we decided to use an image of a male face and the audio of a male voice. For the stimulus presented at CSneu offset, we retained the image of CSneu and paired it with an audio recording of a male saying the word "monitor." A male with a distinct voice from the male who spoke the negative feedback for the social US recorded the audio. The word was selected due to

the fact that it was task-relevant and not emotionally evocative, and thus served as a good control. See Figure 6 for the CS and US pairs used in the present study.

**Figure 6. Stimuli used for CSs and USs in Study 2.**

| | **Social** | **Physical** | **Neutral** |
|---|---|---|---|
| **CS image** |  |  |  |
| **US image** |  | --- |  |
| **US audio** | "Unlikeable" | --- | "Monitor" |
| **US stimulation** | --- | 35v for 150 ms | --- |

**Enhancement of the manipulation for the social US.** Due to the low believability ratings reported for the manipulation in Study 1, several minor changes were made to the protocol. These changes addressed comments made by Study 1 participants during debriefing about what parts of the manipulation they felt were particularly unrealistic, illogical, or confusing. These changes included 1) providing greater context by explaining who the other

raters were and how they were communicating with the experimenter, 2) having the participants record the word they selected for the individual they were rating so they could see how the audio file presented to them later was supposedly acquired, 3) substantially limiting what the participant could see of the experimenter station and participant computer files, and 4) increasing the duration between the interview task and the initiation of the experimental phase so that the negative feedback from the rater could be realistically integrated into the present experiment.

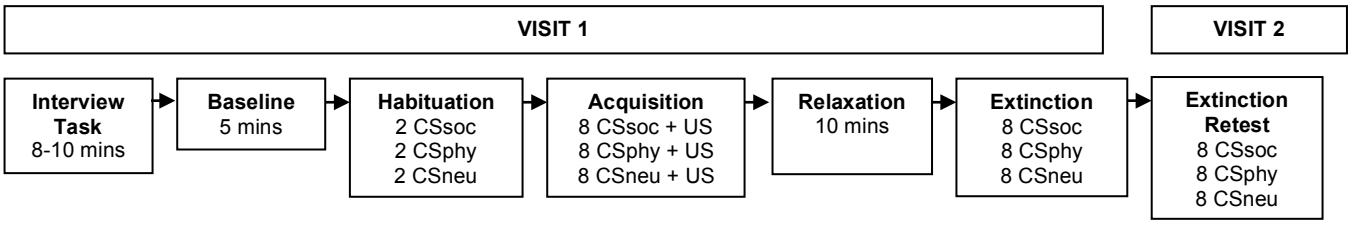
**Procedure.** The procedures for the provision of informed consent, completion of the self-report scales, electrode placement, and testing of physiological equipment was identical to Study 1. Following these procedures, participants underwent a 5 minute *baseline* phase of sitting quietly, allowing for measurement of baseline physiological levels. The interview task then followed. Next, samples of the stimuli were presented to the participants as in Study 1. A *habituation* phase then followed, which consisted of 2 presentations each of the 3 male faces that later became the CSs when paired with the US in the following phase. The *acquisition* phase immediately followed. This phase consisted of 24 trials: 8 presentations of one neutral face that was paired with the rejecting facial expression and negative feedback (CSsoc), 8 presentations of another neutral face that was paired with the bicep muscle stimulation (CSphy), and 8 presentations of the final neutral face that was paired with a neutral comments (CSneu). Each CS was presented for 8 seconds and was immediately followed by the onset of the respective US. Inter-trial intervals ranged from 25-35 seconds.

Participants then underwent a 5-minute *relaxation* phase where they sat quietly. Following this, they completed the *extinction* phase. During this phase they received 24 more trials (8 CSsoc, 8 CSphy, and 8 CSneu), in which the CSs were presented without their respective USs. Participants then were asked a few questions about their subjective experience of

the experiment, disconnected from the physiology equipment, and reminded of their next appointment.

On the second visit, which occurred 7 +/- 3 days after the first visit, participants were connected to the physiology equipment upon arrival. They underwent a second 5-minute *baseline* phase to ensure that the equipment was recording properly and to establish the participant's resting physiology. Participants then underwent the *extinction re-test* phase, which featured identical procedures to the extinction phase. After extinction re-test was complete, participants were disconnected from the physiology equipment. They were then asked several questions about their subjective experience of the study and were thoroughly debriefed, using the same procedures as in Study 1.

**Figure 7. Flowchart of procedures for Study 2**




**Statistical Analyses.** All analyses were conducted using SPSS v21. Scores on the self-report scales were calculated using the identical procedure as Study 1. For the ASI-3, the items that comprise the physical components subscale were totaled. Descriptive statistics were run for all of the self-report scales and the relationship among the scales was determined via Pearson correlations (*r*s).

Due to the small size of the pilot sample, we were not able to run the required analyses to thoroughly test our hypotheses. However, we were able to examine whether SCR and BR differed between the CS types (social, physical, and neutral). We examined this by calculating the mean

response of the 8 trials for each CS type in each phase and the values to paired sample *t*-tests. We were also able to examine whether subjective responses differed to each CS type at post-acquisition, post-extinction, pre-extinction retest, and post-extinction retest. We subjected the ratings at each time point to paired-samples *t*-tests. EMG and expectancy analyses were not calculated for the pilot sample.

## Results

**Self-report scales.** A wide range of scores was observed across self-report scales in the sample. Scores on the SPIN exceeded the established clinical cut-off for 50% of the pilot sample, thus suggesting that a significant portion of the pilot sample had clinically significant SAD symptoms. The physical components subscale of the Anxiety Sensitivity Index (which was added for Study 2 to measure specific fear of physical sensations) correlated particularly strongly with both trait anxiety and rejection sensitivity ($r > 0.9$). See Table 5 for descriptive statistics of the scales and Table 6 for the correlations between the scales

**Table 6. Descriptive statistics for self-report scales in Study 2**

| | ***M*** | ***SD*** | ***Range*** |
|---|---|---|---|
| **Social Phobia Inventory (SPIN)** | 19.5 | 11.5 | 5 – 33 |
| **Rejection Sensitivity Questionnaire (RSQ)** | 9.2 | 3.6 | 5 – 15 |
| **Blushing Propensity Scale (BPS)** | 37.2 | 12.4 | 17 – 58 |
| **State Trait Anxiety Inventory (STAI)** | 38.3 | 10.7 | 26 – 63 |
| **Beck Depression Inventory-II (BDI-II)** | 6.5 | 6.0 | 0 – 21 |
| **Mindful Attention Awareness Scale (MAAS)** | 60.7 | 9.3 | 43 – 78 |
| **Anxiety Sensitivity Index – 3 (ASI3): Physical Components Subscale** | 9.0 | 4.6 | 6 – 17 |

**Table 7. Inter-correlations of self-report scales in Study 2**

| | SPIN | RSQ | BPS | STAI | BDI-II | MAAS | ASI-PC |
|---|---|---|---|---|---|---|---|
| **SPIN** | 1 | | | | | | |
| **RSQ** | 0.49 | 1 | | | | | |
| **BPS** | 0.88** | 0.26 | 1 | | | | |
| **STAI** | 0.60^ | 0.44 | 0.53 | 1 | | | |
| **BDI-II** | 0.49 | 0.42 | 0.40 | 0.94** | 1 | | |
| **MAAS** | -0.42 | -0.24 | -0.45 | -0.56^ | -0.31 | 1 | |
| **ASI-PC** | 0.79 | 0.91* | 0.50 | 0.94* | 0.79 | -0.88^ | 1 |

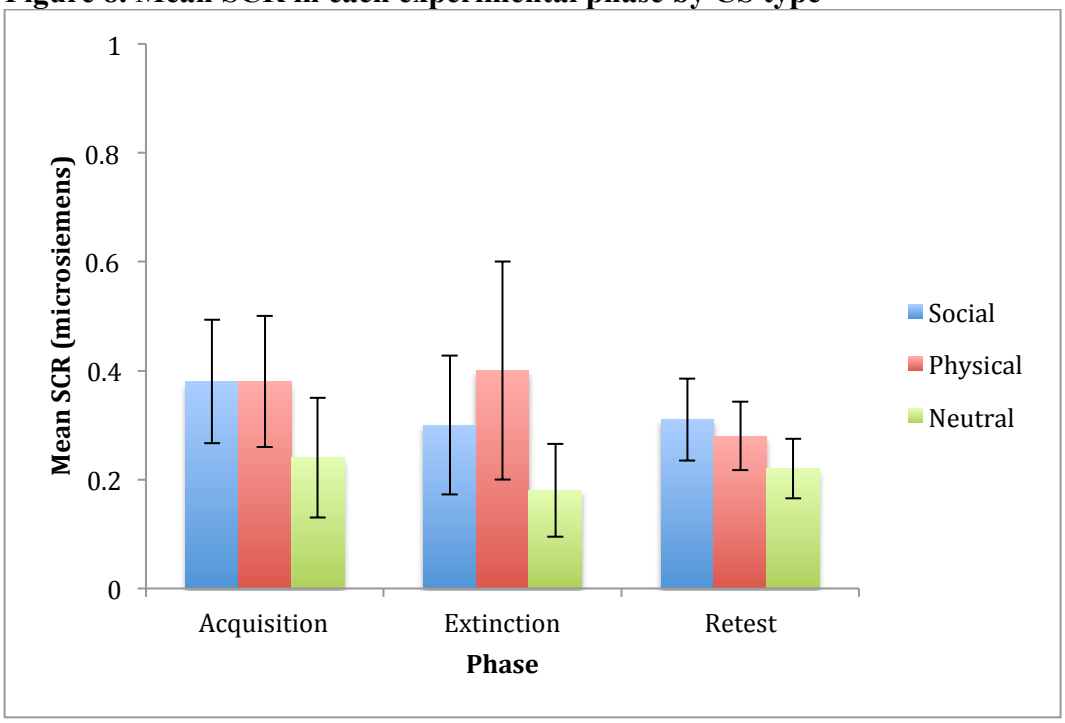
**Note:** ** = $p < 0.01$; * = $p < 0.05$; ^ = $p < 0.10$; all values without notation = $p > 0.10$

**Believability of manipulation.** The average believability rating for participants in the pilot sample was 7.3 ($SD = 2.1$) on the 0 – 10 scale described in Study 1. This indicates a significant improvement over the believability of the manipulation in Study 1 ($M = 4.3$).

**Physiological responding.** SCRs to each CS type were averaged across phases. Visual inspection of the means suggest that mean SCRs to the physical and social USs appear to be higher than mean SCRs to the neutral US (which was intended to serve as a control). Paired sample *t*-tests were run between mean SCRs to 1) the social CS and the physical CS and 2) the social CS and the neutral CS for each phase. The only significant finding was that mean SCR to the social CS was significantly greater than mean SCR to the neutral CS during the extinction retest phase ($t(2) = 5.1$, $p < 0.05$). See Figure 8 for mean SCR to each CS type by phase.

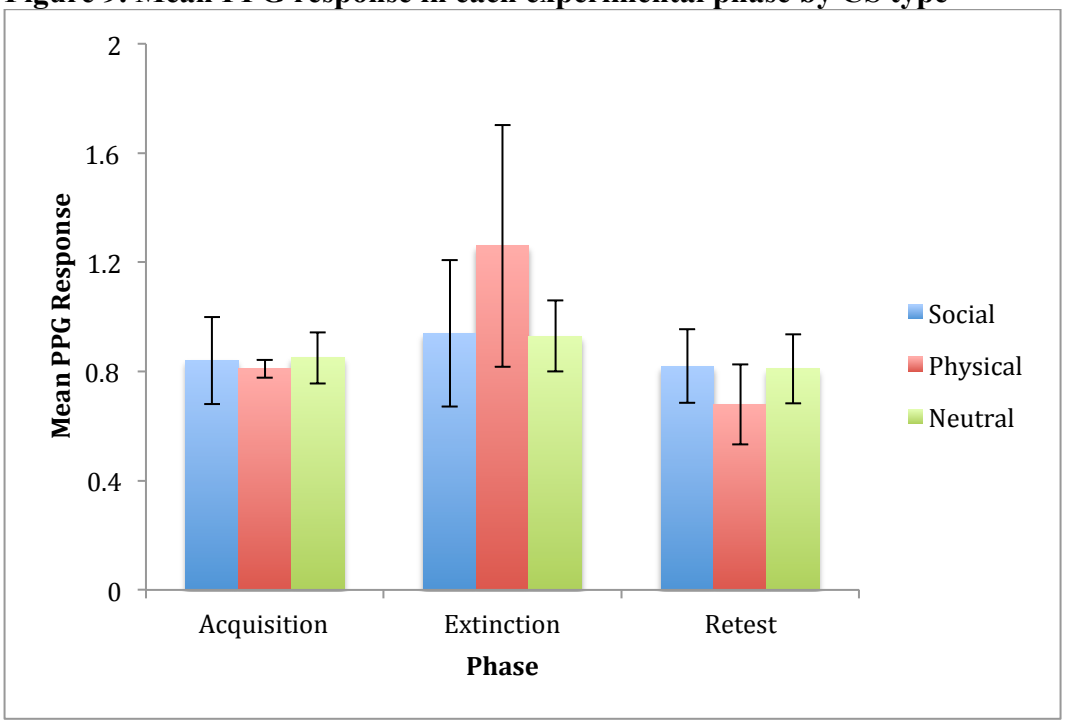
BR to each CS type was averaged across phases. Visual inspection of the means suggest that BRs did not differ between the CS types in any of the phases. Paired sample *t*-tests confirmed this (all *p*s > 0.05). See Figure 9 for mean BRs to each CS type by phase.

**Figure 8. Mean SCR in each experimental phase by CS type**




**Note:** Error bars represent standard errors of the mean.

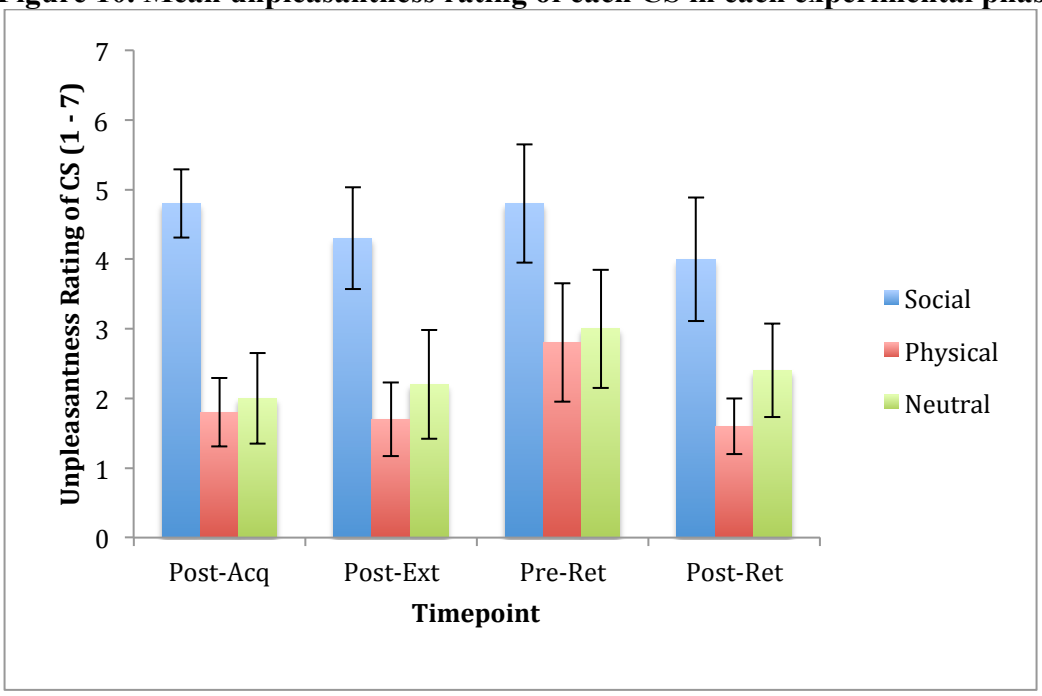
**Figure 9. Mean PPG response in each experimental phase by CS type**




**Note:** Error bars represent standard errors of the mean.

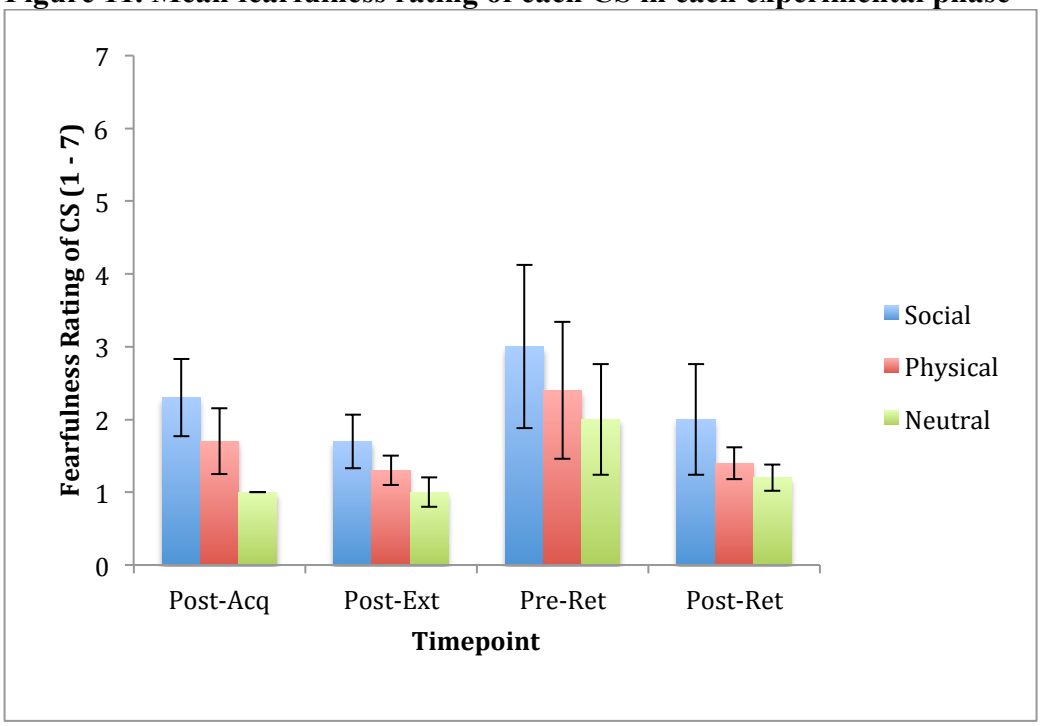
The subjective unpleasantness and fearfulness of each CS were assessed for each time point. Visual inspection of the data suggests that unpleasantness and fearfulness ratings to the social CS were higher than for the physical and neutral CS across all phases. Paired sample *t*-tests confirmed that several of these contrasts were statistically significant. The unpleasantness of the social CS was rated significantly higher than the physical CS and neutral CS at post-acquisition (social CS-physical CS: $t(5)=3.7$, $p < 0.05$; social CS-neutral CS: $t(5)=4.7$, $p < 0.01$), post-extinction (social CS-physical CS: $t(5)=3.0$, $p < 0.05$; social CS-neutral CS: $t(5)=4.5$, $p < 0.01$), and post-extinction retest (social CS-physical CS: $t(4)=3.0$, $p < 0.05$; social CS-neutral CS: $t(4)=3.1$, $p < 0.05$). There was a statistical trend in the same direction at pre-extinction retest (social CS-physical CS: $t(4)=2.4$, $p = 0.08$; social CS-neutral CS: $t(4) = 2.7$, $p = 0.05$). The findings were less pronounced for fearfulness ratings with the only significant contrast being that the participants were more fearful of the social CS than the neutral CS post-acquisition ($t(5)=2.7$, $p < 0.05$). See Figures 10 and 11 for unpleasantness and fearfulness ratings for each CS by phase.

**Figure 10. Mean unpleasantness rating of each CS in each experimental phase**




**Note:** Error bars represent standard errors of the mean.

**Figure 11. Mean fearfulness rating of each CS in each experimental phase**




**Note:** Error bars represent standard errors of the mean.

## Discussion

In Study 2, we compared a social US, a physical US, and a neutral stimulus in a differential classical conditioning paradigm. We sought to examine whether the social US we developed, which elicited significant subjective and physiological fear responding in the prior study, was sufficiently aversive to produce a conditioned fear response to formerly neutral stimuli. The present results reflect a pilot sample comprised of the first 10 participants who enrolled in the ongoing study. Due to the extremely small *n*, full statistical modeling could not be conducted and all results should be considered preliminary and exploratory.

The sample included a wide range of scores on various self-report measures of psychopathology. Participants were equally represented below and above the established clinical cut-off for our SAD screening measure, suggesting that the study is successfully recruiting individuals across a wide spectrum of SAD symptomatology. All participants who completed the first part of the study returned for the second, indicating good retention. The changes made to the study protocol that were intended to increase the personal relevance of the social US appear to have been effective, as evidenced by substantially higher believability ratings than those found in Study 1. We hope that by enhancing the personal relevance of the social US it will make it more salient and thus more likely to produce conditional fear.

Few notable differences were found in SCRs to the different CS types throughout the study. SCRs to the social CS appeared to be higher in general and in fact were significantly higher than SCRs to the neutral CS during retest. Thus, there is preliminary evidence for elevated conditioning to the CS paired with the social US relative to the CS paired with the control stimulus. No differences in BR to the three CS types were observed in the present study. These limited findings are not surprising due to the small sample size, especially when the substantial

inter-individual variability in physiological responding is considered.

Subjective reactions to the CSs showed significant elevated unpleasantness ratings to the social CS relevant to the physical CS and the neutral CS after the acquisition, extinction, and extinction retest phases. The differences between the CS types with regard to participant ratings of fearfulness to the CS were not as pronounced as they were for unpleasantness, with the only statistically significant difference occurring between fearfulness to the social CS and the neutral CS after acquisition. These results suggest that the elevated negative responding participants are displaying toward the social CS may be more driven by negative valence than elevated arousal. Thus, while participants are experiencing the social US negatively and are learning the social CS-social US contingency, they are not reporting a particularly strong subjective fear response.

In addition to the very small sample size, which prevents firm conclusions from being drawn from the present findings, two key limitations to the study that should be noted. First, the CS being used in the present study (a neutral face) is more meaningfully related to the social pain US than to the physical pain US. Based on the theory of CS-US belongingness (first demonstrated by Garcia and Koelling, 1966) this may mean that the social CS has an advantage over the physical US in terms of acquiring conditional fear responses. This possibility should be examined in future research. Second, the assessment of the believability of the manipulation utilized in the study, which is paramount to the understanding of the personal relevance of the stimulus, was complicated by the study design. Directly assessing participant believability at the end of the first visit would likely introduce significant doubt to the participants who had believed the manipulation, thus affecting their responses during the second visit. However, assessing participant believability at the second visit only introduces significant retrospective biases.

Despite these limitations, there are several strengths to the design of the present study. It

marks the first time that a social US has been evaluated alongside a physical US and a control stimulus in a classical conditioning paradigm. Thus, the results regarding the potency of the social US can be more effectively evaluated here than in prior research. The design includes a retest phase that allows for the evaluation of return of fear to the social CS relative to the physical and neutral CSs. Return of fear to the stimuli associated with a social US has been rarely addressed in the literature. The present study also uses a much wider range of measures than prior research examining the effects of a social US, including multiple measures for subjective (expectancy ratings, valence and arousal ratings) and physiological (SCR, BR, FPS) responding. This will provide a more comprehensive understanding of the nature of the CR produced by the social US.

Although the collection of further data will allow us to run the statistical models necessary to address many key questions regarding classical conditioning to a social US, follow-up studies are needed regardless of the ultimate findings. Directions for future research include 1) continuing to enhance the personal relevance of the social US, 2) evaluating the comparative strength of social and physical USs in a population of individuals diagnosed with SAD, and 3) testing the ability of a social US to produce conditional fear responding to a socially irrelevant CS. These are important issues to address as we try to elucidate the social pain – SAD relationship, but many additional opportunities exist outside of conditioning research. In Study 3, the clinical applicability of the conditioning model examined in Study 1 and Study 2 is examined in a large, longitudinal sample.

## Study 3

## The Relationship between Social Pain Experiences and Social Anxiety Disorder Symptoms One Year Later

### Introduction

The widely accepted etiological theory of SAD posits that the disorder is rooted in significant fear responses to aversive, socially relevant events that then become generalized to formerly non-threatening stimuli that are associated with the aversive event (e.g., Mineka & Zinbarg, 2006). By definition, SAD is a disorder characterized by significant distress related to these so-called conditioned stimuli and persistent avoidance of situations in which they may be encountered. Although conditioning does not account for the entire process of SAD development (biological predispositions, temperament, and cultural contexts often play key roles), the importance of conditioning to the development of anxiety disorders is supported by decades of empirical research and forms the theoretical basis for cognitive behavioral interventions for anxiety disorders, which have strong empirical support and are widely utilized (see Hofmann and Smits, 2008 for a review of their efficacy). However, the unique relationship between fear conditioning to stimuli associated with socially relevant aversive experiences and symptoms of SAD has yet to be adequately explored in the literature. The demonstration of such a unique relationship is important to provide empirical support for this etiological theory that seeks to answer the question of why an individual predisposed to develop anxiety might develop SAD versus pathological anxiety focused on unpleasant bodily sensations (Panic Disorder), specific objects or situations (Specific Phobia), or bad outcomes happening across a broad range of domains (Generalized Anxiety Disorder).

Previous research has isolated several environmental factors that are reliably associated with SAD onset (for a review, see Brook & Schmidt, 2008). Due to the early age of onset of SAD,

the literature regarding environmental factors related to its etiology has focused mainly on the period of childhood and adolescence. Many of these experiences are related to social pain, including separation from parents, exposure to sexual and physical abuse by caregivers, and teasing and bullying at the hands of peers. However, the review also notes that there are several adverse events not directly linked to the individual's social environment empirically linked to SAD (e.g, low socioeconomic status, health problems in childhood) and that the socially relevant adverse events tend to be related to the experience of psychopathology in general, not just SAD. Furthermore, the authors point out significant limitations to the studies reviewed, particularly the cross-sectional nature of the vast majority of studies and lack of standardized assessment of adverse events.

Although the majority of studies examining the adverse mental health outcomes associated with social pain events tend to use broadband internalizing symptoms as their dependent measure (for a review, see Reijntjes, Kamphius, Prinzie, & Telch, 2010), a few studies have been conducted that examine relationship between social pain events in adolescence and SAD symptoms. Noting that the majority of research linking adverse events and SAD are focused on physical and sexual abuse, Iffland and colleagues (2012) sought to clarify the role of emotional maltreatment in SAD symptoms by also examining the contribution of emotional abuse and neglect by caretakers in childhood and emotional victimization by peers in childhood and adolescence. They found that although physical and sexual maltreatment were significantly associated with SAD symptoms, the relationship between emotional maltreatment and SAD symptoms was much stronger and in fact statistically mediated the relationships found between physical and sexual maltreatment and SAD. This investigation highlights the potential importance of social pain at the hands of caregivers and peers in SAD onset, but the authors also note that

such emotional maltreatment was also a broad risk factor for psychopathology in general and was not unique to SAD. The study also had significant methodological limitations, including self-reports of past maltreatment and current SAD.

La Greca and Harrison (2005) looked broadly at peer relationship quality in the role of SAD development and found that relational victimization and negative interactions in best friendship predicted high SAD symptoms. Further analyses revealed that peer affiliations, positive qualities in best friendships, and the presence of a dating relationship were protective against SAD symptoms. However, these aspects of peer relationship also predicted self-reported depression and the strength of the associations between these qualities and SAD and these qualities and depression were not compared. This well-designed study had several strengths, including its consideration of multiple social domains (both positive and negative), gender, and ethnicity, but it did not test for the specificity of the relationship and it used a cross-sectional design that precluded causal inferences. Further work by these authors examined various types of peer victimization (overt, relational, and reputational) prospectively and found that all types of victimization predicted SAD symptoms two months later. Additionally, they found that relational victimization (operationalized here by items assessing exclusion and rejection) was especially important to the prediction of SAD symptoms (Siegel, La Greca, and Harrison, 2009). Storch, Masia-Warner, Crisp, and Klein (2005) found a similar pattern of results, noting that relational but not overt victimization predicted SAD symptoms one year later.

Retrospective data regarding past social pain and current SAD symptoms have also been collected. McCabe, Antony, Summerfeldt, Liss, and Swinson (2003) found that 92% of participants with SAD reported severe bullying and teasing in childhood, compared to 50% of participants with Obsessive Compulsive Disorder and 36% of participants with Panic Disorder.

This provides compelling evidence that individuals with SAD perceive a greater history of peer victimization than individuals with other anxiety disorders. Roth, Coles, and Heimberg (2002) reported similar findings in a non-clinical sample, finding that self-reported history with teasing was significantly associated with SAD but not other anxiety disorders. Although both of these studies provide support for the notion that individuals reporting SAD symptoms perceive greater histories of peer victimization than those who do not, the retrospective self-report nature of the studies leave open the possibility that these findings are driven by memory biases that are substantially influenced by the cognitive distortions and attentional biases inherent in SAD. Additionally, the data did not establish time course. Thus, it is possible that the bullying and teasing followed SAD onset rather than played a significant causal role.

The present study examines the specificity of the relationship between social pain and SAD in a sample of 627 adolescents participating in the Northwestern-UCLA Youth Emotion Project (YEP; see Zinbarg et al., 2010 for an in depth overview of the study). The study improves upon the majority of previous research in this area in three key ways. First, it is longitudinal in nature. Second, it utilizes ratings of life stress and psychopathology that are made by trained and objective raters using standardized instruments. Third, the objective ratings of life stress and psychopathology include domains outside of social pain and SAD, thus allowing for the comparison of the SAD-social pain relationship to theoretically distinct relationships.

In line with the widely accepted etiological model of SAD described above, we make the following hypotheses: 1) Higher levels of social stress will be a stronger predictor of SAD severity one year later than higher levels of non-social stress; 2) Higher levels of social stress will be a stronger predictor of SAD severity one year later than symptoms of other anxiety

disorders one year later; and 3) Acute social stress will be a stronger predictor of SAD severity one year later than chronic social stress.

## Method

**Participants.** Participants in YEP were recruited from two ethnically and socio-economically diverse high schools: one in suburban Chicago and one in suburban Los Angeles. Three consecutive classes of juniors were recruited at each school beginning in Fall 2002. A total of 1,976 students provided assent and parental consent. These students were administered the screening measure, a 23-item version of the Neuroticism scale of the revised Eysenck Personality Questionnaire (EPQ-R-N; Eyseck & Eysenck, 1975). Based on their total scores of the EPQ-R-N, students were categorized as low-, medium-, and high-risk to develop anxiety and unipolar depression. Using a method designed to oversample high-risk students and maintain gender balance, 1,269 of the screened students were invited to participate in a longitudinal study. High-risk students were oversampled in order to increase the number of anxiety and unipolar depression cases that would emerge throughout the study. Of those invited to participate, 668 completed the assent and parental consent procedures. Of those, 627 completed the baseline assessment.

Study participants ($n$ = 627) included 432 females and 195 males (68.9% vs. 31.1%). The oversampling of females was not directly intended. The unequal ratio resulted from more females being invited to participate due to having generally higher neuroticism scores than males and females being more likely to provide consent and assent and complete the baseline assessment. The racial composition of the sample was as follows: Caucasian, $n$ = 302 (48%); Hispanic/Latin American, $n$ = 96 (15.3%); African American, $n$ = 82 (13.1%); multiracial, $n$ = 82 (13.1%); other race not specified, $n$ = 34 (5.4%); Asian American, $n$ = 27 (4.3%); and Pacific Islander, $n$ = 4

(0.6%). At the time of enrollment in the study, participants had a mean (*M*) age of 16.9 (*SD* = 0.4, range = 15 – 18). Risk status of the total sample based on scores on the EPQ-R-N is as follows: high-risk, *n* = 368 (58.7%); medium-risk, *n* = 145 (23.1%); and low-risk, *n* = 114 (18.2%). The sample included roughly equal numbers of participants from each site (UCLA: 51% vs. Northwestern: 49%).

**Measures.** Diagnosis of Axis I disorders were made using the Structured Clinical Interview for the Diagnostic and Statistical Manual for Mental Disorders–IV (SCID; First, Spitzer, Gibbons, & Williams, 2002). SCID interviewers were graduate students, postdoctoral fellows, and bachelor's level research assistants. Training included approximately 60 hours of didactics, matching gold standard ratings, role-playing, and live observations. Each diagnosis was presented at a supervision and consensus meeting led by doctoral-level supervisors. To maintain consistency across sites, difficult cases were presented at weekly teleconferences that were periodically attended by supervisors from the other site.

After completing each SCID interview, the interviewers not only assigned categorical DSM diagnoses but also rated the severity of each current diagnosis in the past month using the Di Nardo and Barlow (1988) 0 to 8 clinical severity rating (CSR) scale. Scores of 4 and above indicate clinically significant severity, impairment, or distress in the past month, scores of 3 indicate possible caseness, and ≤ 2 indicates some symptoms of the disorder but no case. CSRs were confirmed by licensed psychologists who supervised the interviewers. The inter-rater Pearson *r* for CSR ratings ranged from 0.74 for major depressive disorder (MDD) and specific phobia-natural subtype to 0.97 for obsessive compulsive disorder (OCD) and post-traumatic stress disorder (PTSD). Reliability for diagnoses was assessed by having trained interviewers observe live SCIDs on 69 cases. Kappas were good when aggregated across all disorders (kappa = 0.82)

and at least acceptable for individual disorders that occurred frequently enough to be examined independently: Generalized Anxiety Disorder (GAD) = 0.85, PTSD = 0.85, MDD = 0.83, and SAD = 0.65.

Ratings of social pain experiences were made using the Life Stress Interview (LSI; Hammen et al., 1987), a semi-structured interview that assesses chronic life stress and episodic life stress. The chronic life stress portion assessed the level of ongoing objective stress experienced by the participant in 10 domains over the past year. This version of the LSI contains chronic life stress domains relevant to an adolescent population, including four interpersonal domains (close friendship, social life, romantic relationships, and family) and six non-interpersonal domains (neighborhood, school, work, finances, personal health, and health of close family members).

To determine chronic life stress scores, the interviewer used suggested general probes to elicit relevant objective information. Subjective impressions offered by the participant were not probed for and were disregarded if offered. Ratings ranged in half-point intervals from 1 (ideal circumstances) to 5 (most stressful circumstances), with specific behavioral anchors for each point on the scale. Training of interviewers involved approximately 30 hours of didactics, matching gold standard ratings, role-plays, and live observations. Reliability at Time 1 was assessed by rating 76 inter-site and intra-site audio-recorded interviews. Intraclass correlation coefficients (ICCs) ranged from 0.58 for health-other to 0.92 for neighborhood. Averaged across all domains, the ICC was 0.70.

Episodic events were probed for within each chronic life stress domain. Interviewers obtained details concerning the description and date of the event, the degree, duration, and impact of its consequences, the participant's prior experience with the event, and availability of social

support. This information was later presented by the interviewer to an independent team of two raters who evaluated the event on its level of contextual threat. Any subjective impressions the participants offered about the stressfulness of an event, as well as any diagnostic information about the participant, were not presented to the raters. Contextual threat was assessed by objectively rating how much impact a particular episodic event would have for the average person in those exact circumstances. Ratings for episodic events were made on a 1–5 scale: 1 (minimal or no threat), 2 (mild threat), 3 (moderate threat), 4 (marked impact with many consequences), and 5 (severe and catastrophic negative impact). Reliability at Time 1 was assessed by rating 208 audio recordings of life events across sites. The ICC was 0.82.

Events were categorized into one of 77 categories using the Paykel Life Events Scale (Paykel et al., 1975), which was modified for use with the present population. In addition to a contextual threat rating for episodic events, raters assessed the dependence of the event, with 1 denoting complete independence and 5 signifying that the event was completely caused by the respondent. These ratings of dependence are outside the scope of the current study. See Appendix C for the modified version of the Paykel Events Scale.

**Procedure.** Mass testing and initial assessments were completed in person. Participants had the option of completing follow-up interview assessments via phone. Participants were financially reimbursed for each assessment they completed. In addition to the measures described here, several other assessments were completed, including a battery of self-report measures assessing various aspects of personality and various symptoms. Subsets of participants also took part in additional assessments, such as peer ratings, genetic samples, and behavioral tasks. Detailed descriptions of the procedures of this study have been reported elsewhere (e.g., Uliaszek et al., 2011; Zinbarg et al., 2010).

**Statistical Analyses.** All analyses were conducted using STATA v13.0. Path analyses were run using structural equation modeling (SEM; for reviews of this method, see Brown & Cudeck, 1993, Kline, 1998, and Ullman, 2001). The models were set up such that acute and chronic stress scores served as time-lagged predictors of disorder CSR ratings at the following time point. The models utilized asymptotically distribution free (ADF) interval estimation, a method for dealing with data that is not normally distributed. This method was selected due to the fact that SAD severity scores were highly positively skewed due to the large number of individuals with no symptoms of the disorder.

In each model several variables conceptualized as potential confounds were entered as covariates. These included gender, race/ethnicity, SAD severity at Time 1, and the presence of comorbid mood disorders at Time 1. Race/ethnicity was recoded into a binary variable in which 0 was "Caucasian" and 1 was "Non-Caucasian." This was done due to the relatively low sample sizes of specific minority groups in the sample. The presence of comorbid mood disorders was determined by the presence of a CSR greater than 0 for Major Depressive Disorder or Dysthymic Disorder at Time 1 (0 = "mood disorder present," 1 = "mood disorder not present").

Ten domains of chronic stress and 77 categories of acute stress were generated by the LSI and thus were available for use in the first model, which compared whether chronic and acute social stress predicts SAD severity one year later more strongly than chronic and acute non-social stress. The decision of which chronic and acute stress types to select was made as follows. First, the authors reviewed all chronic and acute stress types and isolated the ones that were most theoretically relevant to social stress (e.g., chronic social life stress, end of friendship, argument with romantic partner) and those that were most theoretically distinct from social stress (e.g., chronic financial problems, traffic accident, health problems). Second, the prevalence of the

isolated acute stress variables were analyzed and those that occurred relatively infrequently during the first time point were discarded. Third, the mean severity of the chronic stress variables and the remaining acute variables were calculated. For the small percentage of individuals who had multiple instances of the same acute life event (e.g., they ended two significant dating relationships in one year), the mean severity of these events was taken. Fourth, both chronic and acute stress variables, each socially relevant stress type and each socially irrelevant stress types were compared using paired samples *t*-tests. Finally, pairs of socially relevant and socially irrelevant stressors that did not statistically differ were identified. These pairs were selected use in the first model.

For the second model, which examined whether social stress more strongly predicted SAD severity than the severity of other psychopathology one year later, we needed to isolate a diagnostic category to use as a comparison for SAD. The disorder had to be theoretically unrelated to social evaluative concerns, have similar severity ratings to SAD, and be prevalent enough in the sample for us to conduct our statistical models. Panic disorder (PD), generalized anxiety disorder (GAD), and specific phobia (SP) met the first two conditions but had relatively low prevalence rates in the sample. Thus, we decided to make a composite variable in which the maximum CSR rating among the three disorders was selected and entered into the model. Severity of the respective disorder at Time 1 was entered as an additional covariate in the second model.

To test Hypothesis 1, we examined a model in which acute and chronic social stressors and non-social stressors (e.g., financial stress) at Time 1 predicted SAD CSR at Time 2. The standardized beta weights associated with each type of stress were examined to determine if it was a significant predictor of SAD symptomatology at the following time point. In cases where

the models were confirmed, the beta weights were statistically compared using *z*-statistics. To test Hypothesis 2, we examined a model in which social stress at Time 1 predicted SAD CSR and CSR for a socially irrelevant disorder at Time 2. The standardized beta weights associated with social stress and the two disorders were examined to determine if social stress significantly predicted each of the two disorder variables at the following time point. In cases where the models were confirmed, the beta weights were statistically compared using *z*-statistics.

The data was available in the present study to conduct cross-lag analyses and to include time points beyond the first two years of the study. However, these analyses were not conducted for theoretical and logistical reasons. A cross-lag, or bi-directional, analysis was not conducted because the question of whether SAD severity predicts stress at the next time point is beyond the scope of the present study, despite potentially having interesting implications for the stress generation literature (e.g., Hammen, 2005). Models predicting time points beyond Time 2 were not conducted due to concern that the further study attrition following Time 2 would reduce the statistical power of the model. See Table 7 for the number of total participants and number of participants with any SAD symptoms at each time point.

**Table 8. SAD prevalence and total sample size at each time point**

| | **Time 1** | **Time 2** | **Time 3** | **Time 4** | **Time 5** | **Time 6** | **Time 7** |
|---|---|---|---|---|---|---|---|
| Total *n* | 626 | 491 | 423 | 425 | 422 | 423 | 425 |
| SAD *n*<br>(As % of total *n*) | 192<br>(31%) | 75<br>(15%) | 66<br>(16%) | 55<br>(13%) | 54<br>(13%) | 45<br>(11%) | 48<br>(11%) |
| Net change in *n* | | -135 | -68 | +2 | -3 | +1 | +2 |
| Net change in SAD cases | | -117 | -9 | -11 | -1 | -9 | +3 |

## Results

**Descriptive statistics.** At T1, 193 participants (30.7% of the total Time 1 sample) had CSR scores of greater than 0 for SAD whereas 339 (54.1%) had a CSR score of greater than 0 for PD, GAD, or SP. The severity ratings for SAD cases ($M = 2.7$, $SD = 1.4$) and PD, GAD, or SP cases ($M = 2.5$, SD = 1.2) did not significantly differ at Time 1 ($t(125) = 1.96$, $p > 0.05$). At T3, 75 participants (15% of the total Time 2 sample) had CSR scores of greater than 0 for SAD whereas 203 (41%) had CSR scores of greater than 0 for PD, GAD, or SP. The severity ratings for SAD cases ($M = 2.4$, $SD = 1.3$) and PD, GAD, or SP cases ($M = 2.2$, $SD = 1.2$) did not significantly differ at Time 3 ($t(33) = 0.23$, $p > 0.05$).

Descriptive statistics were calculated at Time 1 for the three socially relevant chronic stress domains (social life, romantic relationship, and close friendship) and the two socially irrelevant chronic stress domains (finances and self-health). Each socially relevant chronic stress domain was compared to each socially irrelevant chronic stress domain using paired sample *t*-tests. Two pairs of socially relevant – socially irrelevant chronic stress domains were found that did not differ statistically. The first was close friendship and financial stress; the second was close friendship and self-health. The former pair was chosen as financial hardship of an individual's family was hypothesized to have less implications for one's close relationships than declines in one's own physical health. However, due to the fact that social group stress has the strongest theoretical relevance to social pain and was rated only slightly more severe than the socially irrelevant stress domains (Cohen's $ds < 0.3$), an alternative model comparing social group stress and the socially irrelevant stress domain was also run. See Table 9 for descriptive statistics for the chronic stress variables of interest at Time 1.

**Table 9. Descriptive statistics for chronic stress variables on the Life Stress Interview at Time 1**

| | M | SD | Range |
|---|---|---|---|
| Social group[a] | 2.4 | 0.7 | 1 – 5 |
| Romantic relationship[a] | 2.4 | 0.6 | 1 – 4.5 |
| Close friendship[bc] | 2.1 | 0.6 | 1 – 5 |
| Finances[b] | 2.1 | 0.6 | 1 – 4.5 |
| Self-health[c] | 2.1 | 0.5 | 1 – 4 |

Note: Variables containing the same superscript are not statistically different.

Descriptive statistics were also calculated at Time 1 for the three socially relevant acute stress domains (end of friendship, argument with close friend, and end of dating relationship) and the three socially irrelevant acute stress domains (traffic accident, moderate financial difficulties, and minor personal physical illness, injury, or accident). The only socially relevant and socially irrelevant acute events that did not differ statistically were end of friendship event and moderate financial difficulty. Thus, this comparison was selected for use in the model. See Table 10 for descriptive statistics for the acute stress variables of interest at Time 1.

**Table 10. Descriptive statistics for acute stress variables on the LSI at Time 1**

| | M | SD | Range |
|---|---|---|---|
| End of friendship[a] | 0.1 | 0.5 | 0 – 3 |
| Argument with close friend[b] | 0.5 | 0.8 | 0 – 3 |
| End of dating relationship[b] | 0.5 | 0.8 | 0 – 3.5 |
| Traffic accident[c] | 0.3 | 0.7 | 0 – 3.5 |
| Financial difficulties[a] | 0.1 | 0.4 | 0 – 3.5 |
| Physical illness or injury[c] | 0.2 | 0.7 | 0 – 3 |

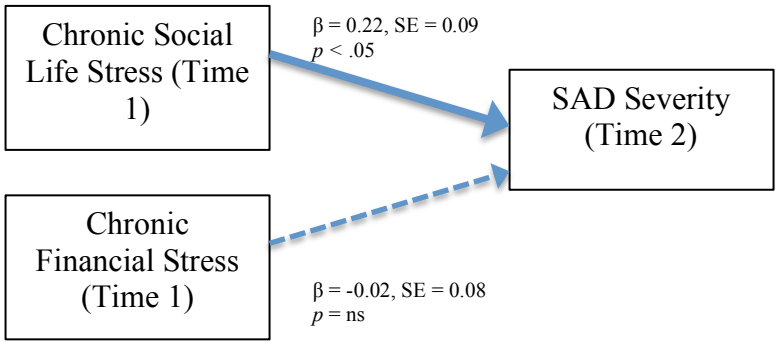
Notes: Variables containing the same superscript are not statistically different. The low means are due to the fact that any participant who did not report an event with this code was coded 0, thus the mean conflates prevalence and severity.

**Model #1: Social stress vs. non-social stress predicting SAD**

For the first model, chronic social and non-social stressors were used as predictors of SAD severity. Specifically, chronic stress in close friendships and chronic stress related to finances at Time 1 were entered as predictors of SAD severity at Time 2. Contrary to hypotheses, chronic close friendship stress at Time 1 did not predict SAD severity at Time 2 ($\beta = 0.05$, SE = 0.08, $p$ = ns). Chronic financial stress at Time 1 also did not predict SAD severity at Time 2 ($\beta = 0.01$, SE = 0.08, $p$ = ns). In the alternative model, chronic stress in the participants' social group replaced chronic stress in the participants' close friendships. Consistent with hypotheses, chronic social group stress was a significant predictor of SAD severity at Time 2. However, when the beta weights for chronic social life stress and chronic financial stress were compared statistically, their difference did not achieve statistical significance despite trending in the hypothesized direction ($z = 1.71$, $p = 0.09$). See Figure 1 for the model. Of the covariates, SAD severity at

Time 1 (β = 0.24, SE = 0.04, $p < .001$) and race/ethnicity (β = 0.18, SE = 0.09, $p < .05$) also significantly predicted SAD severity at Time 2.

**Figure 12. Path analysis of chronic social stress vs. non-social stress predicting SAD**



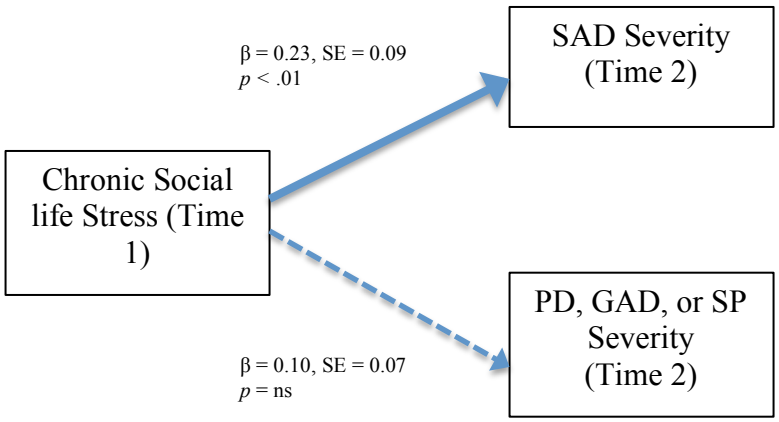

For the second model, acute stressors were used to predict SAD severity at the next time point. Specifically, the mean severity of end of friendship events and moderate financial difficulties at Time 1 were entered as predictors of SAD severity at the second time point. Contrary to hypotheses, end of friendship events at Time 1 did not predict SAD severity at Time 2 (β = 0.11, SE = 0.11, $p$ = ns). Chronic financial stress at Time 1 also did not predict SAD severity at Time 2 (β = 0.11, SE = 0.10, $p$ = ns).

**Model #2: Social stress predicting SAD vs. other anxiety disorders**

For the third model, chronic social stressors were used to predict SAD severity and the severity of anxiety disorders not centered on social evaluative concerns. Specifically, chronic stress in the participants' close friendships at Time 1 were entered as predictors of SAD at Time 2 and as predictors of PD, GAD, or SP severity at Time 2. Contrary to hypotheses, chronic stress in close friendship predicted PD, GAD, or SP severity at Time 2 (β = 0.24, SE = 0.07, $p < .001$)

but did not predict SAD severity at Time 2 (β = 0.05, SE = 0.08, *p* = ns). In the alternative model, chronic stress in the participants' social group at Time 1 replaced chronic stress in the participants' close friendships as the predictor. Consistent with hypotheses, chronic stress in the participants' social life at Time 1 was a significant predictor of SAD severity at Time 2 but not of PD, GAD, or SP severity at Time 2. However, when the beta weights for chronic social life stress predicting the two domains were compared statistically, their difference did not achieve statistical significance (*z* = 1.25, *p* = 0.21). See Figure 2 for the model. Of the covariates, SAD severity at Time 1 (β = 0.24, SE = 0.04, *p* < .001) and race/ethnicity (β = 0.17, SE = 0.09, *p* < .05) also significantly predicted SAD severity at Time 2.

**Figure 13. Path analysis of social stress predicting SAD vs. other anxiety disorders**




For the fourth model, acute social stress was used to predict SAD severity and the severity of anxiety disorders not centered on social evaluative concerns. Specifically, end of friendship events at Time 1 were entered as predictors of SAD at Time 2 and as predictors of PD, GAD, or SP severity at Time 2. Contrary to hypotheses, end of friendship events did not predict SAD severity at Time 2 (β = 0.10, SE = 0.10, *p* = ns). It also did not predict PD, GAD, or SP

severity at Time 2 (β = 0.08, SE = 0.08, $p$ = ns). When the other two acute stressors that were identified as socially relevant were entered into the model, the only significant finding was that end of dating relationships predicted SAD severity (β = -0.11, SE = 0.04, $p < .05$). However, this relationship was in the opposite direction of what was hypothesized, such that the greater the stress associated with ending dating relationships the lower the SAD severity.

## Discussion

The present study explored the relationship between social pain and SAD symptoms in a large sample of adolescents. There are many strengths of the current study that represent significant methodological improvements over the majority of the research previously conducted in this area. First, the longitudinal design allows for causal inferences to be made. Second, the use of objective reports of life stress and psychopathology used in the present study reduce the likelihood that the data is being substantially influenced by the various biases often presented in self-report. Third, the broad assessment of stressful life domains and psychopathology allow for a comparison of socially relevant and socially irrelevant life stress as well as SAD and psychopathology not focused on social evaluative fears. Fourth, the inclusion of both chronic and acute stressors allows for a more nuanced conceptualization of the social pain – SAD relationship than was present in the prior literature.

The most notable findings were that 1) chronic social group stress significantly predicted SAD severity one year later but chronic stress in a non-social domain (i.e., finances) did not and 2) chronic social life stress significantly predicted SAD severity but not the severity of anxiety disorders not based in social evaluative concerns (PD, GAD, and SP). However, it should be noted that when the coefficients of the hypothesized pathway and the alternative pathway were compared for each model, they did not differ at a statistically significant level ($p < 0.05$), thus

precluding full support for the hypotheses. As $p < 0.05$ has been deemed a key threshold for "significance" the fact that one pathway exceeds this threshold and the other does not suggests that the difference between the two coefficients may be meaningful (albeit not statistically significant). These findings do provide evidence of a prospective relationship between social group stress and later SAD severity even if the comparison of the coefficients preclude us from concluding that the relationship is unique. Given the lack of longitudinal research addressing this relationship, the identification of a prospective relationship between social group stress and SAD severity alone is notable.

The significant relationship between chronic social group stress and later SAD symptoms provides support for the conditioning-based etiological model of the disorder. In this domain, raters are assessing factors such as how many friends and social acquaintances an individual has, whether they spend time wishing their social life were different, and whether they feel included and well liked by their social group. Individuals with high stress ratings in this area are more likely to be repeatedly exposed to aversive social events in which social fear conditioning is likely to occur. At the time that the stressors were being measured in the present study participants were approximately 17 years old and in high school and thus likely to be in social circles in which they were routinely exposed to events such as rejection, ostracism, bullying, and teasing, which are hallmarks of social pain and have been linked to SAD in prior research (e.g., La Greca & Harrison, 2005).

It is of note that the findings regarding chronic social life stress did not hold true for the other chronic social stress variables selected for analysis in the present study. A key reason for this may be that the domains of close friendship and ending of a dating relationship are imprecise proxies for social pain events. Although they represent domains in which aversive social

experiences (and thus social fear conditioning) are likely to occur, they are confounded by the fact that the participants who are having problems in their friendships and dating relationships are those that are more likely to be making friends and dating (and thus less likely to be socially anxious; e.g., Baldwin and Buis, 2004). The same issue is germane to the three socially relevant acute stress events selected for inclusion in the model – end of friendship, serious argument with friend, and end of dating relationship.

The fact that the acute events coded in the LSI are imprecise for the measurement of social pain is perhaps unsurprising given that measuring social pain is not the intention of the LSI. In fact, by design the LSI is seeking to capture an objective assessment of the stressful conditions in an individual is in across multiple domains of functioning. When the participant offers subjective responses, the raters are instructed to disregard them. This aspect of the LSI may render it an inadequate measure of social pain events, given research that has found that may be an individual's reactivity to peer victimization, not the frequency and severity of the peer victimization, that significantly predict SAD symptoms (Levinson, Langer, & Rodebaugh, 2013). An ideal measure of social pain events would retain the precision and structure of the LSI while assessing reactivity to events and specifically probing for and coding events theoretically related to social pain. Such a measure is needed for methodologically sound research.

In addition to the issues inherent in the LSI as a measure of social pain events, there are two other limitations of the present research that must be noted. First, we did not analyze the retrospective history of SAD and other psychopathology collected upon enrollment in the study. As the mean age of onset of SAD occurs in early adolescence (Chavira & Stein, 2005), some individuals likely developed SAD prior to the start of the study. Thus, in addition to capturing cases in which social pain experiences trigger the first onset of SAD symptomatology, the data

also captured cases in which SAD symptomatology began before the study started and social pain experiences served only to exacerbate or renew pre-existing symptoms. Second, there was significant attrition in the sample between the first and second time points and thus the subsample with SAD symptoms at Time 2 is relatively small for conducting complex statistical models with multiple covariates.

Although the present study does not present a definitive statement on the role of social pain in the onset and maintenance of SAD, it fills an important gap in the literature and represents an important step in the right direction for this line of research. In order to improve our etiological models and treatment of SAD, the mechanisms through which the disorder develops and maintains need to be better understood. Much research is being conducted regarding the biological contributions to the disorder, including brain imaging, genetic markers, and stress hormones. However, much less research is examining the conditioning-based model of SAD development. This discrepancy persists despite the fact that this model is widely accepted in the scientific and clinical communities (e.g., Mineka and Zinbarg, 2006). A significant increase in theoretically grounded and methodologically sound studies examining the role of conditioning and other learning processes in SAD is needed. This needs to occur at every level, from animal models of social fear conditioning in the laboratory to the large-scale longitudinal studies exploring these variables as they evolve naturally.

## General Discussion

The series of three studies described herein aimed to address three key questions related to the relationship between social pain and the onset and maintenance of SAD symptoms. Study 1 examined the UR to a social US by measuring subjective and physiological reactions to a range of intensities of the stimulus. Study 2 enhances the social US from Study 1 and tested its ability to produce a conditioned fear response in a classical conditioning paradigm. Study 3 moved out of the laboratory by examining how social pain and SAD symptoms are related over time in a longitudinal sample of adolescents.

Results from Study 1 suggested that while the social US elicited subjective and physiological fear responding from the sample, it did not show a linear function (such that increased intensity leads to increased responding) nor was responding significantly influenced by participants' SAD severity as hypothesized. The results from a small pilot sample in Study 2 provided very preliminary evidence that the social US developed here is capable of eliciting conditional fear responding. Findings from Study 3 replicated prior research showing that chronic stressors in one's social life prospectively predict SAD symptoms, but did not compellingly demonstrate that the relationship was unique. Overall, results from the three studies suggested that social pain likely plays an important in the etiology of SAD, but the precise nature of this relationship remains unclear.

It is surprising that although the widely espoused etiological theory of SAD is deeply rooted in the science of fear conditioning, little research has tested the core hypotheses of the theory in a methodologically sound manner. This discrepancy could be due to several factors. First, research regarding SAD is relatively young. The vast majority of research has been conducted in the past two decades. Second, the significant advances that are occurring in the

understanding of how individuals respond to socially painful experiences is occurring largely outside of clinical psychology in the area of social cognitive neuroscience. Thus, limited communication and collaboration between these areas could be playing a role in the lack of research being conducted in this area. A third reason is the logistical difficulty of integrating social stimuli into the established classical conditioning paradigms. Typically, social evaluative threat is induced in a laboratory setting using paradigms such as the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993). This paradigm is highly ecologically valid and the negative feedback is highly personally relevant, but ultimately it induces social stress not social pain and it does not lend itself well to a classical conditioning paradigm, which requires a controlled environment and precise timing. The social USs developed in prior research likely suffered from low ecological validity and personal relevance precisely for these logistical reasons. The present studies aimed to merge the many strengths of the previous social USs and enhance them by adopting features from more ecologically valid paradigms. Although we believe our social US improves upon those previously utilized in conditioning research in key ways, it introduces a significant burden on the experimenter. In addition to the substantial amount of time the manipulation takes to carry out, it also requires the experimenter to engage with the participant in a more complex manner than is typically the case during conditioning studies.

The procedural complexities inherent in incorporating a more ecologically valid and personally relevant CS would undoubtedly be worth it if it were firmly established that such methods are essential to evaluate social fear conditioning. However, the extant research does not yet demonstrate this. The key question that needs to be explored is whether the lack of robust findings in this area is due primarily to methodological weaknesses of the studies or is reflective of an inaccurate – or at least incomplete – theoretical model. Perhaps there are other factors that

are playing a more prominent role in SAD development than enhanced acquisition, impaired extinction, and return of fear. It is possible that other features of SAD, such as distortions in the processing of social information (Rapee & Heimberg, 1997) and attentional bias to threat (Mogg, Philippot, & Bradley, 2004) play an important role that is not appropriately incorporated into current conditioning paradigms. In light of the increasing movement in the field of psychopathology research away from traditional diagnostic categories and toward cross-cutting symptom domains (e.g., Insel et al., 2010), it should also be considered whether we should be focusing on broader mechanisms.

Although it remains possible that the hypotheses generated by the conditioning-based etiological theory of SAD is flawed, it is important to remember that research regarding the mechanisms through which individuals acquire conditioned fear responses to aversive social experiences is in its infancy – or at least its early adolescence. As such, it seems more likely that what is needed is not a revolution in our etiological theory but rather refining existing paradigms and developing innovative methodologies. Such innovation is likely to come from the work being conducted in social cognitive neuroscience and as such increased cross-pollination between social cognitive neuroscientists and researchers in the field of learning and behavior seems like an important goal.

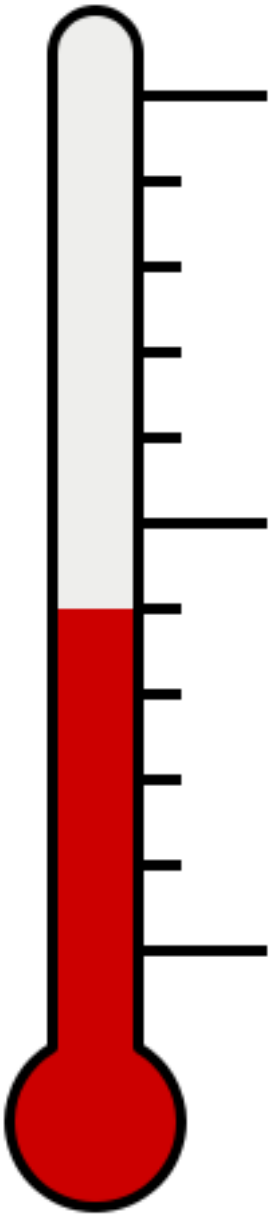
Interdisciplinary collaboration is also of paramount importance to improving clinical research regarding the relationship between social pain and SAD. A review of the literature clearly demonstrates that there is a dearth of theoretically grounded and well-controlled longitudinal studies that examine the role of environmental factors in the onset and maintenance of SAD. The majority of research being conducted use cross-sectional designs, rely on self-report measures, and examine a broad swath of loosely defined stressors. Study 3 marked a significant

improvement over prior studies by using a longitudinal design, objective and structured ratings of psychopathology and stress, and the testing of models rooted firmly in theory. However, the study was limited by the fact that the measures utilized in the study were not specifically designed to assess social pain but rather to make an *objective* assessment of the stress the participant is under. This is especially problematic given that it is likely that the experience of social pain is in fact highly *subjective*. Thus, it follows that not only do specific measures for assessing social pain events need to be developed and validated, but that they should also include information about how the individual interpreted and responded to the event.

The long overdue union of social neuroscience, fear conditioning, and SAD research will likely benefit research in all three areas. Increasingly examining responses to social pain in individuals with SAD (who exemplify pathological responding to such events) rather than primarily relying on healthy controls will likely lead to the challenging of existing paradigms and the development of new theories for social neuroscientists and learning theorists. Meanwhile, increasing the theoretical basis and methodological rigor of the clinical research being conducted on SAD is likely to lead to more accurate etiological models of SAD, improved assessment and classification, and enhanced treatments. Although the present findings do not provide direct implications for treatment, improvement in the understanding of how social fears and other dysfunctional responses to social pain develop is vital to designing more specific and effective treatments and prevention efforts.

**Appendix A:**
**Subjective Units of Unpleasantness Thermometer (Study 1)**

*On the following scale, how unpleasant was the stimulus you just received?*



**100:** The most unpleasant thing I have ever experienced

**75:** It is very unpleasant

**50:** It is moderately unpleasant

**25:** It is mildly unpleasant

**0:** Not unpleasant at all

**Appendix B:**
**Valence and Arousal Rating Forms for Conditioned Stimuli (Study 2)**

**Directions:** Please circle the number that corresponds to how you react to the faces after Phase ____.



How unpleasant do you now find this face to be?

1 2 3 4 5 6 7
Not at all Somewhat Extremely

How fearful are you of this face now?

1 2 3 4 5 6 7
Not at all Somewhat Extremely

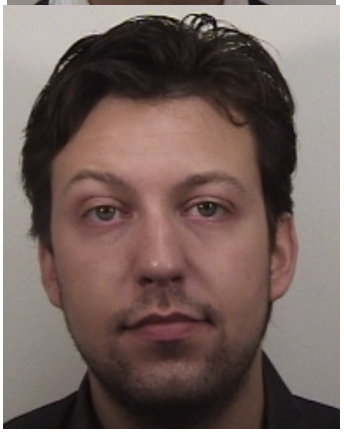


How unpleasant do you now find this face to be?

1 2 3 4 5 6 7
Not at all Somewhat Extremely

How fearful are you of this face now?

1 2 3 4 5 6 7
Not at all Somewhat Extremely



How unpleasant do you now find this face to be?

1 2 3 4 5 6 7
Not at all Somewhat Extremely

How fearful are you of this face now?

1 2 3 4 5 6 7
Not at all Somewhat Extremely

## Appendix C:
## Modified Paykel List of Life Events (Study 3)

Instructions: *The Paykel list may be used: a) to structure queries about topic areas especially if not also administering the Chronic Stress Interview and b) to provide event code numbers. Note that we added new items (>69) as encountered in a particular population with some frequency. You may find another list that works just as well for your purposes.*

WORK

01. Start working for first time ever.
02. Change to a new line of work, or get a new job.
03. Substantial change in work conditions, e.g., transfer, new boss/colleagues, change in duties or responsibilities.
04. Substantial change in work hours.
05. Onset of troubles or disagreement with boss or coworkers.
06. Promotion (must have salary change).
07. Demotion (must have salary change).
08. Fired or laid off.
09. Unemployed for one month or more (do not code if job was intended to be temporary).
10. Failure of business (code for owners and management).
70. Quit job.

EDUCATION

11. Begin full time or half time education.
12. Change schools.
13. Ceases full time education (graduation or drop out).
14. Important academic failure.
15. Trouble with professor.
16. Prepare for, or take important exam (include thesis, do not include routine exams unless they are the first ever taken in college or exams taken when one is in danger of academic failure.
77. Graduation (rate 1.5 generically).

FINANCIAL

17. Moderate financial difficulties (needing second job or more income).
18. Major financial difficulties (e.g., bankruptcy, very heavy debts, defaulting on bills, loans).
19. Substantial improvement in finances (unconditional improvements, not loans).

HEALTH

20. Minor personal physical illness, injury or accident.
21. Major personal physical illness, operation, injury or accident.
22. Physical or emotional illness, injury or accident to close family member, friend, romantic partner (not leading to death).
23. Wanted pregnancy.
24. Unwanted pregnancy.
25. Miscarriage, stillbirth or abortion.
26. Birth of child.
27. Assault, rape (including date rape).

BEREAVEMENT

28. Death of a close friend or family member.
29. Death of an acquaintance or distant family member.
30. Death of own child.
31. Death of spouse or romantic partner.
32. Loss or robbery of objects or personal or actual value. Victim of a crime.
33. Termination of therapy.

MIGRATION

34. Move out of home for first time.
35. Move within same city.
36. Move to another city or state.
37. Move to another country.
76. Move back home with parents.

COURTSHIP AND COHABITATION

38. Become engaged.
39. Break engagement.
40. Begin new dating relationship.
41. End dating relationship.
42. Serious argument or difficulties with romantic partner.
43. Begin cohabitating.
71. Resume dating relationship.
72. Affair with married person or with person in a relationship.
73. Problems related to affair.

LEGAL

44. Minor violation not leading to court appearance (e.g. traffic ticket, academic violations).
45. More important violation leading to court appearance.
46. Jail sentence.
47. Law suit with legal action.
48. Legal problems of close family member.
49. Traffic accident.

FAMILY AND SOCIAL

50. Birth of child to family member or close friend.
51. Adoption of child by family member or close friend.
52. New person (other than new baby) moves into the household.
53. Serious argument or problem with family member.
54. Marked improvement in relationship with family member.
55. Serious argument or problem with friend.
56. Marked improvement in relationship with friend (not meeting a new friend).
57. Start new friendship.
58. End friendship.
59. Separation from significant person (e.g., family member, close friend, romantic partner, therapist).
60. Marital problems of close family members.

MARITAL

61. Marriage.
62. Serious argument or problem with spouse.
63. Marital separation not due to argument (e.g., in hospital, out of town; any circumstance where couple is separated and does not want to be).
64. Marital separation due to argument.
65. Extramarital affair by either partner or extra-relationship affair.
66. Marked improvement in relationship with spouse.
67. Marital reconciliation after separation.
68. Divorce.

OTHER

69. Generic other.
74. Natural disaster.
75. Victim of crime.

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