The Functions and Initial Reinforcement of Non-Suicidal Self-Injury: A Startle Modulation Evaluation

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Abstract

Although non-suicidal self-injury (NSSI) is a pernicious and increasingly prevalent behavior, why people start and continue to engage in NSSI still is poorly understood. To elucidate these issues, the present study utilized a sample of 73 undergraduates (33 control; 24 affect dysregulation; 16 NSSI) and employed psychophysiological measures of affect (startle-alone reactivity) and quality of information processing (prepulse inhibition), and experimental methods involving a NSSI-proxy to mimic the NSSI process. Consistent with theory, it was predicted that the NSSI group would display cognitive-affective regulation after the NSSI-proxies whereas the control group would display dysregulation after the NSSI-proxy. Additionally, consistent with theory about initial reinforcement of NSSI, it was predicted that the affect dysregulation group would display dysregulation to the first, but regulation to the second NSSI-proxy. Results supported hypotheses, providing the best evidence yet for why people start and continue to engage in NSSI.

Dedication

To the days between March 10, 2005 and January 23, 2007.

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Special thanks to Dr. Terry Blumenthal for teaching me a little bit about science and startle, Dr. Jonathan Abramowitz for the equipment and allowing me to completely take over his lab space for a year or so, Dr. Gabriel Dichter for his insightful comments, and Dr. Mitch Prinstein for his introduction to the field of NSSI and support for a strange project.

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List of Abbreviations

AD: Affect dysregulation

ANR: Automatic negative reinforcement

APR: Automatic positive reinforcement

CPT: Cold pressor task

dB: Decibel

FASM: Functional assessment of self-mutilation

NSSI: Non-suicidal self-injury

PPI: Prepulse inhibition

SUDS: Subjective units of distress

Introduction

NSSI and Its Proposed Functions

Non-suicidal self-injury (NSSI) is defined as the direct and intentional destruction of one's own body tissue in the absence of suicidal intent (Nock et al., 2006; Nock & Prinstein, 2004). In contrast to the stereotypic self-injury that is characteristic of some individuals with mental retardation and the severe self-injury (e.g. autocastration, limb amputation) that sometimes occurs during episodes of psychosis, NSSI typically is defined as non-stereotypic and moderate in severity (e.g. skin-cutting and burning). Reports of the prevalence of NSSI vary according to the population studied and severity of NSSI behaviors meeting inclusion criteria; however, some general trends have emerged: there appears to be a prevalence of approximately 4% in the general United States population (Briere & Gil, 1998); 14% in college (Gratz, 2001) and community adolescent populations (Favazza et al., 1989; Ross & Heath, 2002); 21% in adult clinical populations (Briere & Gil, 1998); and 40-60% in adolescent clinical populations (Darche, 1990; DiClemente, Ponton, & Hartley, 1991).

Given that NSSI is associated strongly with several forms of psychopathology (Haw et al., 2001; Klonsky et al., 2003; Nock et al., 2006; Stanley et al., 2001) and there are relatively high prevalence rates of NSSI in both clinical and nonclinical populations (4-60%; Briere & Gil, 1998; Darche, 1990; DiClemente et al., 1991; Favazza et al., 1989; Gratz, 2001), it is alarming that the reasons that people engage in NSSI (i.e., the functions

of NSSI) are not well-understood. In a recent review of 18 published studies on the functions of NSSI, Klonsky (2007) determined that this literature was nearly unanimous in finding that (a) acute negative affect precedes NSSI, and (b) after NSSI there is a sense of relief and a decrease in negative affect. As such, Klonsky (2007) concluded that NSSI primarily serves an affect regulation function (more precisely, an automatic negative reinforcement function [ANR], Nock & Prinstein, 2004), with a few other NSSI functions receiving relatively moderate support (e.g., automatic positive reinforcement, social reinforcement, self-punishment). Although the extant NSSI function literature is informative, it is comprised primarily of studies that utilized correlational designs and self-report data. Correspondingly, these studies have a limited capacity to determine causality and are vulnerable to self-report and social-desirability biases, which are particularly salient threats given the socially unacceptable nature of NSSI. Consequently, conclusions drawn from these studies regarding the functions of NSSI remain tentative.

Laboratory Investigations of NSSI Functions

A few studies have eschewed some of the aforementioned limitations by utilizing psychophysiological measures and/or experimental designs that employ painful stimuli or imagery tasks that act as NSSI-proxies (i.e., "laboratory studies"). Similar to correlational and self-report studies, results of these laboratory studies have been interpreted as providing support for the hypothesis that ANR is the primary function of NSSI (Klonsky, 2007). In the first published laboratory NSSI study, Russ et al. (1992) used the cold pressor task (CPT) as a NSSI-proxy and found that, relative to non-patient controls, a sample of 11 patients diagnosed with borderline personality disorder (BPD) and with a history of NSSI demonstrated a significant post-CPT decrease in self-report

measures of negative affect. In a similar study, Schmahl et al. (2006) examined the neural correlates of pain in a sample of BPD patients with a history of NSSI. Using thermode-generated heat stimuli as a NSSI-proxy and measuring brain activity with blood oxygen level-dependent functional magnetic resonance imaging (BOLD fMRI), Schmahl et al. (2006) found that, during administration of painful heat stimuli, a sample of BPD patients showed greater dorsolateral prefrontal cortex (DLPFC) activity and decreased amygdala and anterior cingulate cortex (ACC) activity relative to non-patient controls. The authors concluded that this DLPFC-ACC-Amygdala pattern represents an antinociceptive mechanism that works by down-regulating the emotional components of pain in BPD patients with a history of NSSI. Taking a somewhat different approach, Haines et al. (1995) measured physiological arousal (e.g., heart rate, skin conductance) during a NSSI imagery task and found that incarcerated men with a history of NSSI, relative to prison and non-prison controls, displayed increased physiological activity leading up to the NSSI-phase of the task and decreased physiological activity during and after the NSSI-phase of the task. Importantly, this pattern was not observed during a control task (e.g., imagining an accidental injury). Generalizing these results, this same group replicated these findings in a psychiatric sample (Brain et al., 1998) and Welch et al. (2008) replicated the finding of decreased skin conductance after NSSI imagery in a sample of participants diagnosed with BPD. However, Welch et al. (2008) failed to replicate the finding of decreased skin conductance *during* NSSI imagery and, furthermore, failed to extend these findings using respiratory sinus arrhythmia, an index of parasympathetic nervous system activity.

These laboratory studies represent innovative and important examinations of the functions of NSSI and have been interpreted as providing crucial psychophysiological support for an ANR function; nonetheless, these studies also have several limitations. First, although there is substantial evidence suggesting that acute negative affect precedes self-injury (Klonsky, 2007), there has yet to be a laboratory NSSI study that has induced negative affect before administering the NSSI-proxy. As such, to observe the functions of NSSI with the greatest accuracy and ecological validity, NSSI-proxies should be administered in the context of acute negative affect.

Second, the NSSI groups of these studies primarily have consisted of either BPD patients or incarcerated men. Although the prevalence of NSSI is known to be especially high in these two populations, they represent relatively severe forms of psychopathology and, thus, the functions of NSSI observed in these groups may not be isomorphic with the functions of NSSI in less impaired populations. Consequently, basing knowledge of the functions of NSSI on these two severely impaired populations may lead to a distortion of the more general functions of NSSI. A laboratory examination of the functions of NSSI in a broad nonclinical population accordingly may represent a more accurate and generalizable investigation of the functions of NSSI. Third, although affect dysregulation is often associated with NSSI (Klonsky, 2007), none of these studies has employed a comparison group with demonstrated affect dysregulation levels that are comparable to those of the NSSI group. Including such a control group would serve to test the possibility that affect dysregulation, and not a history of NSSI, is the most important factor in determining one's response to a NSSI-proxy.

Fourth and finally, although these studies have demonstrated that a NSSI-proxy leads to decreased autonomic arousal (Brain et al., 1998; Haines et al., 1995; Welch et al., 2008), and decreased amygdala/ACC and increased DLPFC activity (Schmahl et al., 2006), the psychophysiological measures in these studies are limited in their ability to investigate the valence-specific functions of NSSI. Specifically, although autonomic measures can reliably discriminate between levels of arousal, they are less able to discriminate between affective valences because both increased positive and negative affect are associated with increased arousal (Andreassi, 2007). Likewise, some researchers have suggested that because of the temporal dynamics of affect and the substantial overlap of the neutral substrates of positive and negative affect, BOLD fMRI also may primarily index arousal instead of valence and, moreover, may be limited in its ability to measure phasic affective states (Burgdorf & Pankseep, 2006; Hamann et al., 2002; Kim & Hamann, 2007). Accordingly, there has yet to be a direct psychophysiological examination of the hypothesis that NSSI leads to a reduction in negatively valenced affect (i.e., the ANR function).

To address these issues, the present study investigated the functions of NSSI in a nonclinical population (divided into three groups: NSSI, affect dysregulation, control) by inducing acute negative affect before the administration of a NSSI-proxy and indexing changes in negative affect with a specific psychophysiological index of negative affect: startle reactivity. The acoustic startle response is a defensive reflex that occurs in response to a sufficiently sudden and intense sound (Blumenthal et al., 2005). The startle response is mediated by a nucleus in the brain stem, the nucleus reticularis pontis caudalis (nRPC), which receives modulatory input from the respective areas of the brain

associated with positive and negative affect (i.e., the limbic areas; Koch & Fendt, 2003). As the startle response is a defensive reaction and, thus, has a negative valence, stimuli that match this negative valence cause phasic increases in startle reactivity relative to neutral stimuli (Bradley, Cuthbert, & Lang, 1999; Cook, 1999; Grillon et al., 1996, 1998). Correspondingly, stimuli that do not match this negative valence cause phasic decreases in startle reactivity relative to neutral stimuli (Bradley et al., 1999; Cook, 1999). Accordingly, startle reactivity would appear to be uniquely suited for the investigation of valence-related functions of NSSI. For someone who engages in NSSI, startle reactivity should be increased during a stressor task and, if NSSI serves an ANR function, startle reactivity should be decreased following the administration of a NSSI-proxy. Individuals who do not engage in NSSI should display a similar pattern, with the exception that their startle reactivity should be increased following a NSSI-proxy. Such an investigation would have the potential to provide the strongest empirical evidence yet for the ANR function of NSSI, establish startle reactivity as an effective measure of the functions of NSSI, and imply that NSSI normalizes activity (post-stressor) in the specific limbic-brainstem circuits that are known to regulate startle reactivity (see Koch & Fendt, 2003).

NSSI and Information Processing

As reviewed above, NSSI has been shown to occur in the context of acute affect dysregulation and is believed to function primarily as an affect regulation mechanism (Klonsky, 2007). Consequently, many recent investigations of the functions of NSSI have focused directly on the relationship between NSSI and affect. Although such investigations are critical to understanding the functions of NSSI, they overlook other

potentially important factors such as the association between NSSI and the cognitive concomitants of affect dysregulation and regulation. Specifically, affect dysregulation may disrupt the reception, integration, storage, retrieval, and/or output of information (i.e., information processing) by composing a neural system that is biased towards affect-relevant information and action, thereby decreasing problem-solving skills and leading to more maladaptive behaviors such as NSSI. Indeed, neuroimaging research indicates that increased arousal is associated with a simultaneous increase in limbic and decrease in prefrontal activity, suggesting that affect- and problem-focused coping are in opposition to one another (Brown et al., 2006; Gushnard & Raichle, 2001). Correspondingly, as Haines and Williams (1997) point out, it may be that individuals who engage in NSSI rely on affect-focused coping to the detriment of their problem-solving skills and, moreover, that this disparity may be particularly severe during states of acute negative affect. Supporting this possibility, Nock and Mendes (2008) demonstrated that participants with a history of NSSI display greater affect dysregulation and poorer problem-solving skills after acute stress induction. Unfortunately, there have been no published studies that have taken the extra step of investigating the effect of NSSI on information processing, in part because there are few direct, effective, and economical psychophysiological measures of information processing.

Prepulse inhibition (PPI) of the acoustic startle response, however, represents such a measure of information processing (see Braff & Geyer, 1990; Franklin & Blumenthal, in preparation; Graham, 1975; Swerdlow et al., 2000, 2001). PPI occurs when a stimulus (i.e., the prepulse) is presented 30-500ms before a startle-eliciting stimulus and causes decreased startle reactivity to the startle stimulus relative to non-

prepulse trials (Blumenthal, 1999). Presumably, the presence of the prepulse causes a decrease in the reactivity to the startle stimulus because, while the brain is processing the prepulse, it cannot simultaneously process the startle stimulus to the same degree as it is able to when there is no prepulse (Braff & Geyer, 1990; Graham, 1975). Thus, decreased PPI is indicative of decreased information processing. Supporting this notion, clinical populations posited to have difficulty processing information display PPI deficits, indicating that the prepulse (and thus, information) is not as well-processed by these populations (e.g., Braff et al., 2001; Castellanos et al., 1996; Duncan et al., 2006; Franklin et al., 2007a, 2009; Franklin et al., under review-a; Hazlett et al., 2007; Grillon et al., 1996; Hoenig et al., 2005; Kumari et al., 2005; McAlonan et al., 2002). Given that NSSI and affect dysregulation are associated with many of these PPI-deficient psychopathologies (Nock et al., 2006), I propose that these two groups will evidence significantly decreased PPI relative to a control group (i.e., a non-NSSI, non-affect dysregulation group) at baseline.

Although PPI almost always is employed as a trait measure and only indexed at baseline, Grillon & Davis (1997) indirectly demonstrated that stress decreases PPI, and neurophysiological studies suggest that PPI may vary with arousal level (see Koch and Fendt, 2003; Swerdlow et al., 2000, 2001). Specifically, studies have demonstrated that increased activity in the limbic areas and striatal areas, and decreased activity in the frontal areas, leads to an inhibition of the area in the midbrain that mediates PPI, the pedunculopontine tegmental nucleus, resulting in decreased PPI (Swerdlow et al., 2001). Such activity is similar to the neurological concomitants of increased arousal (see Brown et al., 2006; Gushnard & Raichle, 2001). Implied in this association is the possibility that

decreased arousal should be associated with increased PPI. Although this hypothesis rarely has been tested, it does receive support from Duley et al. (2007) who found that, whereas a high trait anxiety group had decreased PPI relative to a low anxiety group at baseline, this difference disappeared after the groups underwent 30 minutes of exercise. Further bolstering the possibility of a state-level NSSI-PPI association, Nock and Mendes' (2008) findings of increased affect dysregulation and decreased problem solving/social information processing in participants with a history of NSSI after acute stress induction may imply that such participants also would display decreased PPI. Moreover, the findings of Schmahl et al. (2006) provide some neurological support for the possibility that NSSI may regulate PPI as the increased frontal and decreased limbic activity that they found as a consequence of a NSSI-proxy is consistent with the neural substrates of increased PPI (Swerdlow et al., 2001). Accordingly, as hypothesized by Haines and Williams (1997), it may be that NSSI serves to regulate both affect and cognition.

Consistent with the aforementioned examination of the NSSI functions with startle reactivity, participants with a history of NSSI should display decreased PPI (indicating poorer information processing) during a stressor task and a return to baseline levels of PPI after the administration of a NSSI-proxy. In contrast, although non-NSSI groups also should display decreased PPI during a stressor task, they should display further decreased, not increased, PPI after a NSSI-proxy. This latter finding would be consistent with Leitner (1989), who found that rats evidenced significantly decreased PPI after undergoing a cold water swim test. Overall, these PPI patterns would be the first direct evidence of a cognitive regulation function of NSSI and, furthermore, would imply

that NSSI serves to regulate some portion the complex neural circuitry that undergirds PPI.

Investigating the Initial Reinforcement of NSSI

Although NSSI necessarily is assumed to be reinforcing to those who continually engage in such behaviors, there has yet to be an empirical investigation of how these behaviors are initially reinforced. Based on the aforementioned literature, it is reasonable to assume that through social, incidental, accidental, or other means, one learns of and engages in NSSI, and through automatic, social, or other reinforcement, continues to engage in NSSI. It follows that if one has no history of NSSI, but undergoes acute stress followed by a self-administered painful stimulus, then one may learn that self-administered pain results in the regulation of affect and cognition and, consequently, may be more likely to engage in NSSI in the future. Additionally, it may be that the greater the negative affect experienced before this initial painful stimulus, the more reinforcing the NSSI may become.

Consistent with this reasoning, individuals with both a high degree of affect dysregulation and no history of NSSI (versus individuals with a history of NSSI or with a low degree of affect dysregulation) should display a significant increase in cognitive-affective regulation in response to the second administration (i.e., Set B; see Figure 1) of a NSSI-proxy during acute negative affect relative to an earlier administration (i.e., Set A). In other words, the degree of increased cognitive-affective regulation between Set A and Set B should be moderated by (a) the degree of cognitive-affective dysregulation before previous painful stimuli and (b) prior experience with NSSI. Such an investigation would represent a highly inferential examination of one possible pathway

through which NSSI may be initially reinforced and may initiate a line of research that has strong implications for NSSI prevention.

Based on the aforementioned model, if participants undergo Sets A and B: (1) relative to an NSSI group, participants with relatively low levels of dysregulation should demonstrate significantly increased startle-alone reactivity and decreased PPI between stress and NSSI-proxy conditions in both Sets A and B NSSI-proxy because the effects of the painful stimulus are not sufficiently reinforcing for this group; and (2) relative to an NSSI group, participants with relatively high levels of dysregulation, but who do not have a history of NSSI, should demonstrate significantly increased startle-alone reactivity and decreased PPI between stress and NSSI-proxy conditions in Set A, but not in Set B, because they should begin to learn that pain facilitates cognitive-affective regulation. In other words, if repeated stress-pain pairings represent one form of the initial reinforcement of NSSI, then NSSI-naïve individuals should begin to respond similarly to NSSI individuals across repeated stress-pain parings, particularly if participants are high in affect dysregulation.

Summary of Hypotheses

The present study will investigate the functions and initial reinforcement of NSSI using three methods: startle-alone reactivity, PPI, and self-reported subjective units of distress (SUDS). For startle-alone reactivity, whereas non-NSSI groups should display an increase in reactivity after a NSSI-proxy, the NSSI group should display decreased reactivity in these conditions. For PPI: (1) relative to the control group, the NSSI and affect dysregulation (AD) groups will display significantly decreased PPI at baseline; (2) although each group should display decreased PPI during the stressor task, consistent

with Nock and Mendes (2008), the NSSI and AD groups should display a greater decrease than the control group in this condition; and (3) whereas the AD and control group should evidence further decreases in PPI after a NSSI-proxy (Leitner, 2008), the NSSI group should display an increase in PPI that approaches baseline levels. Regarding initial reinforcement, the presentations of a second stressor and NSSI-proxy (i.e., Set B) should result in the same patterns as predicted above, with the exception that the AD group should show less of a decrease in PPI after the second NSSI-proxy. For SUDS, consistent with findings of discordance between self-report and psychophysiological measures (e.g., Patrick et al., 1993), relative to baseline, SUDS should increase during all stressors and NSSI-proxies for all groups.

Methods

Participants

Participants were 87 undergraduates; however, the final sample consisted of 73 participants (51 females, 22 males; 62 Caucasian, 11 Other) as other participants were disqualified due to excessive EMG noise (n = 10; all due to a faulty electrode during the same week), antipsychotic medications (n = 1), and startle-alone/PPI values that were greater than 3 SDs away from the overall means (n = 4). Disqualified participants did not significantly differ from included participants on any self-report measure (all *p*s > .05). Participants were recruited from two sources: (1) introductory psychology classes that included a research participation option (n = 38); and (2) campus-wide email advertisements that offered payment of $20 for participation in the study (n = 35). Participants recruited from email advertisements were either (a) recruited based on their high affect dysregulation or NSSI scores on our screening questionnaire that was

administered as part of a separate experiment during the Carolina Testing and Orientation Session (CTOPS; N = 26); or (b) responded affirmatively to a campus-wide email that asked, "In the last year, have you purposefully injured yourself without intending to die (e.g. cutting or burning your skin) more than 6 times?" (N = 9). Participants were sorted into three groups based on their responses on NSSI and affect dysregulation questionnaires: low affect dysregulation, no history of NSSI (Control Group; n = 33); high affect dysregulation, no history of NSSI (Affect Dysregulation [AD] Group; n = 24); history of NSSI (NSSI Group; n = 16). Participants scoring greater than 1 SD (i.e., >15) above the mean on the affect dysregulation screener were placed into the AD group; participants scoring 15 or below were placed in the Control group. Chi-square analyses revealed that gender and ethnicity did not differ by group (all *p*s >.05) and univariate analyses showed that there were no gender or ethnicity effects on psychophysiological variables (all *p*s >.05).

Procedure

Screening. Participants recruited from introductory psychology classes entered the study without being screened; subsequently, these participants filled out a screening questionnaire and were sorted into one of the three groups. Pending their responses on the screening questionnaire administered during the experiment, these participants were sorted into one of the three groups.

Experimental Session. Participants were seated individually in a sound-attenuated room where they read and signed an informed consent form. Subsequent to this, participants filled out the battery of questionnaires. After completing these measures, participants were prepared to undergo the startle portion of the experiment: (1) the skin

on the left temple and below the left eye was cleaned with a cotton swab dipped in rubbing alcohol; (2) surface recording electrodes filled with Synapse conducting paste were then placed on the cleaned areas: one electrode was attached to the skin overlaying the orbicularis oculi muscle directly below the pupil, but below the lower eyelid, and another electrode was placed approximately 15mm (center to center) lateral to and slightly higher than the other electrode, and the ground electrode was placed on the skin overlaying the left temple; and (3) headphones then were comfortably placed on the participant (cf., Blumenthal et al., 2006; Franklin et al., 2009).

Background noise then was turned on (and remained on throughout the session) and participants were given three minutes to acclimate to it before any other stimuli were presented. Following standard procedure, three habituation trials of a 100dB(A) startle stimulus then were presented (these trials are not included in the analyses). Next, two sets of trial blocks were presented (see Figure 1): Set A was composed of three blocks (i.e., baseline, first stressor, first cold pressor) and Set B was composed of two blocks (i.e., second stressor and second cold pressor). Each block was composed of 12 trials, with each trial containing a 100dB(A) startle stimulus and half of the trials also containing an 85dB(A) prepulse, resulting in 6 startle-alone and 6 PPI trials per condition. Within blocks, trial order varied randomly. Each block was followed by a three minute period during which the participant either: (1) rested and was given instructions for a speech task; or (2) underwent the cold pressor task (CPT). SUDS was assessed at five points during the experiment: (1) before the baseline block; (2) immediately after the first stress block; (3) immediately after the first CPT; (4) immediately after the second stress block; and (5) immediately after the second CPT.

Block 1 was the baseline block. Following this, participants rested for three minutes and then were given instructions for one of their two speech tasks (speech prompt order was counterbalanced across participants). Participants then were given four minutes to prepare for this speech, during which time Block 2 (i.e., the Stress1 block) was presented. Next, the CPT was administered (hand order was counterbalanced across participants); the CPT lasted for a maximum of two minutes (preparation for, and execution of, the CPT last approximately 4 minutes for each participant). Immediately upon completion of the CPT, Block 3 (the CPT1 block) was administered. Participants then rested for three minutes and, subsequently, were given instructions for their second speech task. Block 4 (the Stress2 block) was presented during the four minute preparation interval for this speech. Finally, the CPT was administered again (to the opposite hand), immediately after which Block 5 (the CPT2 block) was presented. Participants then were debriefed, compensated, and allowed to leave.

Stimuli

Acoustic Stimuli. All stimuli settings were set according to recommendations from parametrical PPI studies (Blumenthal et al., 2005; Blumenthal & Franklin, 2009; Franklin et al., 2007). Startle stimuli were 100dB(A) broadband noises (20Hz – 20KHz), with a 50ms duration and a rise/fall time of <1ms. Prepulses were 85dB(A) broadband noises, each with a 40ms duration and a rise/fall time of <1ms. The stimulus onset asynchrony (prepulse to startle stimulus) for each trial was 120ms. Background noise was a continuous 70dB(A) broadband noise present during the entire testing session. Intertrial intervals varied randomly from 14 to 23s. All stimuli were generated by Adobe Audition, presented by Superlab, and delivered to the participants through Sennheiser

PX200 headphones. Stimulus intensities were calibrated with steady-state signals presented through the headphones and measured with a sound level meter.

Speech Tasks. Participants in this study were given two speech tasks. For one, the topic was "give a speech about why you should be picked to be on a reality show about people your age," and for the other, the topic was "give a speech about whether or not you believe it is right for the government to execute people." Participants were given four minutes to prepare for, and one minute to deliver, each speech. Participants performed their speeches in front of a video camera and a monitor that displayed their live image. Additionally, participants were told that their speech would be recorded and subsequently evaluated by a group of their peers as part of a study that examined how well they articulated their speech and the persuasiveness of their argument. Each participant was asked to deliver both speeches and speech order was counterbalanced across participants.

The Cold Pressor Task (CPT). The CPT is perhaps the most widely used form of experimental pain induction in psychological studies (e.g., Russ et al., 1992; Hagelberg et al., 2002). For this task, a cooler containing a 2°C (as indexed by a thermometer) mixture of crushed ice and water was placed on a stool next to the participant. A water circulator was placed in the cooler to prevent the water near the participant's hand from warming up. The participant was given instructions to place their hand (up to the wrist) into the water and to inform the researcher when (a) they first feel pain and (b) when the pain becomes intolerable; additionally, participants were told to rate their pain on a 0-10 scale at these two points, with 1 being absolutely no pain and 10 being the most pain that they had ever experienced. As soon as the participant's hand was submerged, the timer

began. Participants were allowed to pull their hand out of the water whenever they pleased, and were allowed to keep their hand in the water for a maximum of two minutes. Participants underwent the CPT twice, alternating hand used for the CPT within session, with hand order being counterbalanced across participants.

Psychophysiological Measures

Measures and settings were in accord with current methodological standards (Blumenthal et al., 2005). Eyeblink EMG responses were measured from the orbicularis oculi muscle with In Vivo Metric surface recording electrodes (Ag/AgCl, 11mm outer diameter, 4mm inner diameter contact surface) placed below the left eye. EMG activity of this muscle was amplified with a Biopac EMG amplifier and sampled (1000Hz) by a Biopac MP150 workstation which stored four versions of the EMG input: raw unfiltered EMG, filtered EMG in a passband of 28-500Hz, a rectification of the filtered EMG signal, and a rectified and smoothed (five sample boxcar filter) derivation of the filtered signal. The analyzed data was based on the smoothed EMG signal.

Self-Report Measures

Screening Questionnaire. This measure was designed to screen for the presence of both NSSI and affect dysregulation. NSSI was screened with the item, "In this past year, how often have you harmed or hurt your body on purpose (for example, cutting or burning your skin, hitting yourself, etc.)." Affect dysregulation was screened with 6 items from the Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004); these items had the highest factor loadings on each of the 6 factors of the DERS: Nonacceptance ("when I'm upset, I feel guilty for feeling that way"); Goals ("when I'm upset, I have difficulty concentrating"); Impulse ("when I'm upset, I lose control over my

behaviors"); Awareness ("I am attentive to my feelings" [reverse scored]); Strategies ("when I'm upset, I believe I'll end up feeling very depressed"); and Clarity ("I have difficulty making sense of my feelings"). All items are scored on a 1 ("almost never") to 5 ("almost always") Likert scale. The complete DERS shows high internal consistency, good test-retest reliability, adequate construct and predictive validity, and is positively correlated with NSSI in both men and women (see Gratz & Roemer, 2004).

Functional Assessment of Self-Mutilation (FASM; Lloyd, Kelly, & Hope, 1997). The FASM is a 33-item measure that assesses type, frequency, and functions of NSSI (e.g., "[Have you ever] cut or carved your skin"; "[did you do this] to feel relaxed"). The FASM was utilized as a follow-up to the NSSI screening question in order to assure that the participants' NSSI behaviors fell into the 'severe NSSI' category. Specifically, although the FASM asks about such behaviors as lip-biting and wound-picking, only participants endorsing severe NSSI behaviors such as skin cutting and burning were included in the NSSI group (see Table 1). Although hitting is sometimes a severe behavior, follow-up interviews suggested that no participants who indicated 'hitting' as their most severe NSSI behavior engaged in severe hitting (i.e., causing tissue damage or bruising) or viewed their behavior as a form of self-injury. Accordingly, these participants were sorted into non-NSSI groups based on their affect dysregulation scores; however, it should be noted that results were essentially similar when these participants were removed from analyses and when these participants were placed in the NSSI group.

Subjective Units of Distress Scale (SUDS). Participants were asked to "rate their level discomfort on a scale of 0 to 100, with 0 indicating the most relaxed you have ever

felt in your life, 50 being quite distressed but still functioning adequately, and 100 signifying the most distressed that you have ever felt in your life."

General Health Questionnaire. A general health questionnaire also was administered to monitor participants' use of psychoactive medications (n =3) and tobacco (n = 6). Chi-square analyses indicated that neither medication nor tobacco use differed by group (*p*s >.05) and univariate analyses showed that neither of these variables had a significant effect on psychophysiological variables (*p*s >.05). Participants also were screened for current hearing-related illnesses (e.g., sinus infections, ruptured eardrums); however, no participants indicated any such illnesses.

Data Analyses

Startle-Alone Data. Blink response magnitude was calculated for each stimulus condition (see Blumenthal et al., 2005). Response magnitude was the average of the difference between peak and onset voltage of the smoothed EMG, within a window of 20–150 ms after stimulus onset for all trials; trials on which no response was found were assigned a value of 0. Startle-alone means were calculated for all five conditions. A 3x5 (Group x Condition) mixed ANOVA was conducted to test for the predicted interaction effect. Pending a significant interaction effect, four 3x2 (Group x Selected Conditions) mixed ANOVAs were planned and, pending significant interaction effects, follow-up independent-samples t-tests of adjacent-condition change scores were conducted (i.e., change score = later condition mean – earlier condition mean). In an absence of

significant interaction effects, main effects were examined. Note that due to a failure to respond in some conditions, some participants are not included in all analyses.

PPI Data. PPI was calculated as the proportion of the difference from control (i.e. [prepulse condition − control condition]/control condition) for response magnitude, as recommended by Blumenthal et al. (2004). Similar to startle-alone analyses, PPI means were calculated for all five conditions. Again similar to startle-alone analyses, a 3x5 (Group x Condition) mixed ANOVA was conducted to test for the predicted interaction effect and, pending a significant interaction effect, four 3x2 (Group x Selected Conditions) mixed ANOVAs were planned. If these latter interaction effects were shown to be significant, follow-up independent-samples t-tests of adjacent condition change scores were conducted (i.e., change score = later condition mean – earlier condition mean). Additionally, a one-way between-groups ANOVA was conducted to test the main effect of group on PPI in the baseline condition. Again, note that due to a failure to respond in some conditions, some participants are not included in all analyses.

SUDS. Mean SUDS were calculated for all five conditions. A 3x5 (Group x Condition) mixed ANOVA was conducted to test for a possible interaction effect and the predicted main effect of condition. Pending a significant main effect of condition, a follow-up contrast test was conducted to specify the nature of this effect.

Results

Questionnaire Measures

FASM. Descriptive information from the FASM is displayed in Table 1. Although minor NSSI behaviors (e.g., picking at skin, biting one's lip) were common in

all groups, severe NSSI behaviors (e.g., cutting, burning) were only indicative of the NSSI group.

Affect Dysregulation Screener. A one-way ANOVA examining the effect of group on screening scores showed that there was a significant effect of group (F[2, 72] = 50.73, p<.001), with a posthoc Tukey's HSD test showing that both the AD (M = 19.54; SD =2.52) and the NSSI (M = 18.44; SD = 4.17) groups had significantly higher scores (p<.001) than the Control group (M = 12.78; SD = 2.35), but were not significantly different from one another (p = .42).

SUDS

The 3x5 mixed ANOVA revealed that there was no interaction effect of Group and Condition on SUDS (F[8, 268] = .781, p = .64), no main effect of Group on SUDS (F[2, 67] = 1.86, p = .16), but there was a significant main effect of Condition on SUDS (F[4, 268] = 37.05, p<.001). A follow-up contrast test showed that this latter effect was a significant quartic trend (F[1, 67] = 6.78, p<.01), with SUDS increased across the first three conditions, decreased in the second stress condition, and increased again during the second CPT condition. These results demonstrate that participants' SUDS, regardless of group, were increased during all stressful and painful tasks relative to baseline (see Figure 2).

Startle-Alone

The 3x5 mixed ANOVA indicated that there was no interaction effect of Group and Condition on startle-alone reactivity (F[8, 236] = .39 , p = .93) and no main effect of Group (F[2, 59] = .16, p = .85); however, there was a significant main effect of condition (F[4, 236] = 34.95, p<.001), with subsequent contrast analyses showing that this was a

significant quartic trend (F[1, 59] = 14.46, p<.001), such that reactivity decreased across the first three conditions, increased during the second stress condition, and decreased again during the second cold pressor condition. For all groups, it appears that startle habituation was prominent for the first three conditions, stress increased reactivity during the Stress2 condition, and reactivity was lowest during both CPT conditions (see Figure 3).

PPI

The one-way between-groups ANOVA revealed that there was a significant main effect of Group on PPI in the Baseline condition (F[2, 69] = 4.17, p<.05), with PPI was lower in the Control group relative to the AD and NSSI groups (see Figure 4). Follow-up independent-samples t-tests indicated that both the AD (t[52] = 1.85, p<.05) and NSSI groups (t[45] = 3.60, p<.01) were significantly different from the Control group, but not from one another (t[37] = .84, p = .20).

The 3x5 mixed ANOVA showed that there was a significant interaction effect of Group and Condition on PPI (F[8, 224] = 2.92, p<.01), with contrast analyses demonstrating that this was a significant quartic effect (F[2, 56] = 7.70, p<.001), with the effect of Group on PPI varying across each set of adjacent Conditions (see Figure 4). Follow-up 3x2 mixed ANOVAs conducted on selected conditions did not indicate an interaction effect for the Baseline-Stress1 set (F[2, 66] = .21, p = .81) or the Baseline-Stress2 set, but they did reveal significant interaction effects for the Stress1-CPT1 (F[2, 64] = 5.64, p<.01) and Stress2-CPT2 sets (F[2, 60] = 3.23, p<.05). Although there was not an interaction effect for the Baseline-Stress1 set, there was a trend for a main effect of Condition (F[1, 66] = 3.78, p = .06), with PPI being lower in the Stress1 block relative to

the Baseline block. Similarly, although there was not a significant interaction effect of the Baseline-Stress2 set, there was a significant main effect of Condition (F[1, 64] = 4.39, p<.05), with PPI being lower in the Stress2 block relative to the Baseline block.

Within the three significant interaction effects noted above, follow-up independent-samples t-tests on change scores between adjacent conditions revealed: (1) no significant differences in change scores between the Control and AD groups in any conditions (all ps >.05); (2) significant differences in change scores between the Control and NSSI groups in the Stress1-CPT1 (t[42] = 3.02, p<.01) and Stress2-CPT2 sets (t[42] = 2.76, p<.01), with PPI increasing for the NSSI group and decreasing for the Control groups between these conditions; and (3) following a similar pattern, a significant difference in change scores between the AD and NSSI groups in the Stress1-CPT1 (t[34] = 2.67, p<.05), but not the Stress2-CPT2 set (t[31] = 1.71, p = .10), with PPI increasing for the NSSI group (in both sets) and the AD group in the Stress2-CPT2 set, and decreasing for the AD group in the Stress1-CPT1 set (see Figure 4).

Taken together, these PPI results reveal that: (1) both the AD and NSSI groups display PPI deficits at baseline; (2) stress decreases PPI for all groups; (3) whereas pain generally leads to further decreased PPI for the non-NSSI groups, it leads to increased PPI for the NSSI group; and (4) although, relative to the NSSI group, the AD group evidenced a significantly greater decrease in PPI between the Stress1 and CPT1 conditions, this difference was not significant between the Stress2 and CPT2 conditions (see Figure 4).

Discussion

The present study investigated the functions and initial reinforcement of NSSI in the context of an experimental NSSI-proxy paradigm with psychophysiological measures of affect and information processing, and a self-report measure of distress. Results partially supported hypotheses: (1) PPI results both supported the cognitive-affective regulation function of NSSI and provided a preliminary demonstration of a possible mechanism of the initial reinforcement of NSSI; and (2) consistent with previous reports of a discordance between self-report and psychophysiological measures (e.g., Patrick et al., 1993), SUDS results largely were in opposition to the startle results as there were no group differences in SUDS. However, startle-alone results were less consistent with hypotheses: although, as predicted, the NSSI group evidenced a decrease in reactivity during both cold pressor conditions, the non-NSSI groups displayed this same pattern. Additionally, although results generally supported the prediction that stress would disrupt PPI, this disruption was not greater for the non-control groups. Overall, these results strongly contribute to both basic and applied clinical science: (1) they suggest that PPI can be effectively employed to index phasic changes in information processing; (2) they represent the first empirical evidence of a cognitive regulation function of NSSI; (3) provide the strongest evidence yet for the ANR function of NSSI; (4) demonstrate a possible mechanism through which NSSI may be initially reinforced; and (5) elucidate the neurobiological mechanisms of the functions and initial reinforcement of NSSI.

Baseline PPI

Consistent with predictions, both the NSSI and AD groups displayed significantly less PPI at baseline than the Control group, suggesting that individuals who are high in affect dysregulation and/or engage in NSSI have poorer trait information processing. This result is consistent with clinical PPI studies that have demonstrated decreased PPI in association with psychopathologies that commonly feature NSSI, including anxiety disorders (Franklin et al., 2009; Grillon et al., 1996; Ludewig et al., 2005) and antisocial and borderline personality disorder symptomatologies (Franklin et al., under review-a; Kumari et al., 2005). As the NSSI and AD groups did not have significantly different levels of PPI or affect dysregulation, it may be that both of these groups displayed significantly lower PPI relative to the control group primarily due to increased affect dysregulation. Although decreased PPI has been associated with some psychopathologies that feature affect dysregulation (e.g., Franklin et al., 2009, under review; Grillon et al., 1996; Ludewig et al., 2005), and neurophysiological evidence suggests that affect dysregulation should be associated with decreased PPI (Swerdlow et al., 2001), this is the first direct evidence of this association. Indeed, this association may be robust given that all participants were nonclinical college students and the AD and Control groups were partitioned with a relatively low-powered split of their affect dysregulation scores.

PPI and Stress

The prediction that, relative to the control group, the PPI of non-control groups would be significantly more disrupted by stressor tasks received no support; in contrast, the hypothesis that stress would significantly decrease PPI (i.e., a main effect of stress) received moderate support. Although each group evidenced decreased PPI in the Stress1

relative to the Baseline block, this decrease only represented a nonsignificant trend; nonetheless, there was a significant decrease in PPI in the Stress2 block relative to the Baseline block. Overall, when combined with indirect evidence (Grillon & Davis, 1997) and neurophysiological suggestions (Swerdlow et al., 2001) of the disruptive effect of stress on PPI, the present results confirm that PPI is sensitive to phasic changes in stress such that stress decreases PPI. Moreover, inasmuch as the CPT is a stressful stimulus, the present study provides even stronger support for this hypothesis as PPI was lowest for the non-NSSI groups during the CPT blocks.

PPI and Pain

Consistent with hypotheses, the NSSI group displayed an increase in PPI between the Stress1 and CPT1 blocks whereas the non-NSSI groups showed a decrease in PPI between these blocks. This pattern was similar between the Stress2 and CPT2 blocks, with the exception that there was only a nonsignificant trend for change score differences between the NSSI and AD groups (this latter finding is discussed further below). Overall, these results indicate that, whereas pain disrupts the information processing of individuals who do not engage in NSSI (cf., Leitner, 1989), it substantially improves the information processing of individuals who have a history of NSSI – even beyond baseline levels. As such, these findings represent the first demonstration of the cognitive regulation function of NSSI; moreover, because PPI also varies to some degree with arousal level (i.e., PPI decreases with stress, increases with relief of stress; see present results; Duley et al., 2007; Grillon & Davis, 1997; Swerdlow et al., 2001) and due to the interrelated nature of cognition and affect (e.g., Brown et al., 2006), these results also provide indirect support the affect regulation function of NSSI.

Startle-Alone Results

Inconsistent with hypotheses and seemingly inconsistent with PPI results, the startle-alone findings did not reveal any significant group differences. Despite the standard inclusion of three habituation trials before the baseline block, perhaps the most salient aspect of these results is that the first three conditions appear to be confounded with the phenomenon of startle habituation (see Figure 3). Because startle rarely has been employed to measure differences in reactivity to more tonic stimuli (e.g., a stressful situation versus an unpleasant picture), and because there is a paucity of information about the specific nature of startle habituation (Franklin et al., under review-b), the degree to which startle habituation would impact these results was unclear at the outset of this study. Fortunately, the Set B conditions appear to be free of this contamination: startle reactivity increases for all groups during the Stress2 block, during the CPT2 block, decreases back to levels observed during the CPT1 block. Although the NSSI group followed its predicted pattern of displaying its lowest reactivity in the cold pressor conditions, inconsistent with predictions, the non-NSSI groups also showed this pattern (see Figure 3).

Initially, these results might be interpreted as providing no support for the affect regulation function of NSSI; nonetheless, a closer examination in conjunction with the PPI results reveals that these findings may not only provide support for an affect regulation function, but also may specify that NSSI primarily functions to decrease negative affect rather than to increase positive affect (i.e., the ANR versus APR function; both are commonly endorsed functions on self-report measures, see Klonsky, 2007). Startle reactivity decreases in the context of three things: (1) habituation; (2) decreased

negative affect; and (3) increased positive affect (Bradley, Cuthbert, & Lang, 1999); however, PPI decreases in the context of two things, only one of which corresponds with startle reactivity decreases: (1) increased positive affect (e.g., manic states, cocaine administration; Braff et al., 2001; Swerdlow et al., 2001); and (2) increased negative affect (present results; Grillon & Davis, 1997; Leitner, 1989; Swerdlow et al., 2001). In other words, startle-alone reactivity decreases as affect becomes less negative/more positive and PPI decreases as arousal increases. Because the non-NSSI groups displayed both decreased startle-alone reactivity and decreased PPI in the cold pressor conditions (with the exception of the AD group after the second CPT, discussed below), the most likely explanation for this pattern is that these groups experienced positive affect, or at least its neurological concomitants, after undergoing the CPTs. This explanation would be consistent with evidence that pain normally instantiates the release of dopamine, which decreases both PPI and startle-alone reactivity, possibly through opioid-mediated mechanisms (Bortolato et al., 2005; Martin-Iverson & Else, 2000; Sills et al., 2001; Swerdlow et al., 1991; Wan et al., 1996). Supporting this possibility, Tavernor et al. (2000) found that startle reactivity was reduced during the CPT in a sample of 12 nonclinical male participants. Similarly, Leitner's (1989) study, which demonstrated decreased PPI in rats after a cold water swim test, showed that these rats displayed a concomitant decrease in startle-alone reactivity. Given that the NSSI group displayed a decrease in startle-alone reactivity and an increase PPI after the CPTs, the most likely explanation is that this pattern is due to decreased negative affect in response to pain. Accordingly, despite displaying similar startle-alone reactivity to non-NSSI groups after the CPTs, examining this data with the PPI data converges on the conclusion that NSSI

primarily functions to reduce negative affect. Nonetheless, in the absence of a control group to account for the specific contribution of habituation to these results (i.e., a group that undergoes the same experimental procedure with the exception of the CPTs), these startle-alone conclusions remain tentative.

Initial Reinforcement

In addition to supporting the cognitive-affective regulation function of NSSI, the present study provides preliminary support for one possible mechanism of the initial reinforcement of NSSI: repeated stress-pain parings. Furthermore, results indicated that individuals higher in affect dysregulation may be more susceptible to this initial reinforcement: as noted above, whereas the NSSI group displayed a significantly greater PPI increase between the first stressor and cold pressor conditions relative to both non-NSSI groups, there was no such difference between the NSSI and AD groups during Set B. In fact, whereas both non-NSSI groups evidenced decreased PPI between the first stressor and cold pressor, and the control group displays a similar decrease during Set B, the AD group shows a slight PPI increase in Set B (see Figure 4), implying that the AD group experienced the second CPT as slightly regulating. Although the startle-alone results do not provide direct support for initial reinforcement, they are not inconsistent with this possibility: it may be that the AD group has decreased reactivity in the first cold pressor condition due to increased positive affect and decreased reactivity in the second cold pressor condition due to decreased negative affect (or some combination of increased positive and decreased negative). Despite being suggestive of initial

reinforcement, it is important to note that the present study does not conclusively demonstrate initial reinforcement. However, these preliminary results do provide a foundation from which future studies aimed more specifically at elucidating initial reinforcement can be conducted.

Tentative Explanation of Pain-Relevant Results

Although the results of the present study, in conjunction with the NSSI literature, help to provide a description of the NSSI process, there has been a limited discussion in the literature as to why this process may occur psychophysiologically. Drawing on the pain and PPI literatures, it is possible to construct a tentative model to explain the present results which suggest that pain may be regulating for some and dysregulating for others.

To construct such a model, the overlapping neural correlates of reinforcing behaviors, pain, and PPI must be explicated. All known reinforcing behaviors result in the release of dopamine in the nucleus accumbens, also known as the primary pleasure center of the brain, which causes the release of endogenous opioids (Nicola et al., 2005; Robinson and Berridge, 2000; Volkow et al., 2002). Recently, however, this dopamine-opioid circuit also has been shown to be activated by both stress and pain (Thierry et al., 1976; Horvitz, 2000; Pruessner et al., 2004), leading some to posit that this circuit is activated by salient stimuli regardless of valence (Scott et al., 2006). Nonetheless, as this circuit is activated by tonic, but not phasic, stress and pain (Scott et al., 2006), it may be that this circuit is activated as an antinociceptive mechanism that serves to mollify the effects of stress and pain. Supporting this possibility, increased baseline dopaminergic tone is associated with increased pain threshold and tolerance (Hagelberg et al., 2002), ostensibly because this tonic dopaminergic activity represents a more chronically active

antinociceptive system. Interestingly, increased dopaminergic tone also is associated with decreased trait PPI. Taken together, this evidence may explain why the groups in the present study with lower baseline PPI (i.e., the non-control groups) displayed greater pain tolerances ($F[1, 69] = 4.20$, $p<.05$) than the control group and, additionally, why stress and pain generally serve to reduce PPI.

Unfortunately, this evidence does not explain why the NSSI group displayed significantly increased PPI during the CPT conditions or, likewise, why the AD group appeared to demonstrate PPI regulation during the CPT2 condition. Nevertheless, another phenomenon from the pain literature does seem to adequately explain this pattern: placebo analgesia. In short, this phenomenon occurs when one expects that a normally painful stimulus will not be painful or, in fact, may be pleasurable in some way (Goffaux et al., 2007). This phenomenon is associated with increased DLPFC, medial prefrontal cortex, and midbrain activity, among other regions (Goffaux et al., 2007). It is thought that this increase in frontal activity leads to an increase in midbrain activity which, in turn, leads to the activation of the descending nociceptive inhibitory circuit (DNIC), thereby inhibiting pain in the peripheral nervous system (Goffaux et al., 2007). Interestingly, BPD patients with a history of NSSI evidenced similar neurological activity in the study by Schmahl et al. (2006) and, moreover, this activity is very similar to the neurobiological correlates of increased PPI (cf., Swerdlow et al., 2001). Lending further credence to this possibility, the fact that NSSI imagery task studies (Brain et al., 1998; Haines et al., 1995; Welch et al., 2008) have yielded results that are largely consistent with painful NSSI-proxy studies implies that a top-down mechanism (such as placebo

analgesia), that is independent of the actual administration of pain, may strongly influence one's response to NSSI.

Accordingly, whereas the dopamine-opioid pain circuit may explain the majority of the present PPI findings, placebo analgesia appears to explain the PPI patterns of the NSSI group during the CPT conditions and the AD group during the CPT2 condition. It may be that the NSSI group, by virtue of their experience with self-injury, expected (likely unconsciously) the CPT to lead to pleasurable effects (i.e., antinociceptive activity generated by the dopamine-opioid circuit) and consequently demonstrated placebo analgesia. Similarly, it may be that, after experiencing the pleasurable effects of the first CPT, the AD group developed placebo analgesia, albeit to a much smaller degree than the NSSI group. This latter finding might be expected given that placebo analgesia is more likely to occur in individuals with increased negative affect (Wasan et al., 2006), can be conditioned (Colloca & Benedetti, 2006), and the opponent process theory suggests that this phenomenon should be more likely as more painful stimuli are delivered (Joiner, 2005). This model represents the first detailed account of the neurobiological processes that may undergird NSSI (beyond hypotheses that opioids may be involved); however, this model accordingly remains largely untested. Nonetheless, it is hoped that this model may provide a foundation from which a greater understanding of NSSI can be gleaned.

Limitations and Future Directions

Although the present study provides the strongest evidence yet for a cognitive-affective regulation function of NSSI and, moreover, represents the first experimental foray into the study of the initial reinforcement of NSSI, it should be interpreted in accord with its limitations.

First, the sample of the present study included only college students, which may limit the generalization of the present findings to a more clinically severe sample. Nonetheless, as rates of NSSI are relatively high in nonclinical samples (Gratz, 2001) and employing nonclinical samples allows for the avoidance of some problematic factors associated with clinical samples (e.g., medications, hospitalizations, etc.), the present findings may provide a more general account of the functions of NSSI than previous studies that utilized clinical samples. Additionally, bolstering generalizability to clinical samples, the NSSI group only included individuals who reported severe NSSI behaviors (i.e., cutting, burning, scraping, etc.). Future studies, however, would benefit from replicating the present study in and larger and more clinically severe sample.

Second, it is unclear how much ecological validity the experimental paradigm of the present study possesses. Although the present paradigm (i.e., baseline, stress, pain) is more ecologically valid than any previous study due to the induction of acute negative affect before the administration of the NSSI-proxy, the degree to which this stress induction and NSSI-proxy approximate one's actual NSSI process is difficult to estimate. In particular, measures were taken after, not during, the administration of the CPTs, so one may argue that the cessation of pain, rather than response to pain itself, was measured in the present study. Practically, it would have been difficult to measure psychophysiological variables during the CPTs due to the possibility of electrocution and the fact that some participants only remained in the CPT for a few seconds (it takes 4 minutes to deliver a block of startle stimuli). It is likely that NSSI is primarily reinforced by the effects of pain (e.g., endorphins, relief), not directly by the sensation of pain itself; however, these two factors are not independent of one another. To that end, the CPT was

chosen as the NSSI-proxy because: (1) of all of the forms of experimental pain, the CPT is most associated with the affective components of pain (Hagelberg et al., 2002); and (2) it was reasoned that the CPT would provide a close approximation to actual NSSI as CPT pain continues after removal from the CPT because (a) the hand remains painfully cold for several seconds and (b) the process of the thawing of the hand is itself painful. Thus, it may be argued that the CPT provides a valid approximation of many NSSI behaviors in that delivers both acute pain (cf., cutting of the skin) and more tonic, though less intense, pain (cf., burning sensation due to cutting the skin). As such, taking measurements soon after the completion of the CPT would seem to adequately index the effects of engaging in NSSI. In any case, given that the present findings are consistent with self-report information about the process of NSSI, it would seem that the present paradigm is sufficiently valid for the examination of the functions of NSSI. Future studies would nevertheless benefit from both taking measurements continuously throughout an NSSI paradigm and employing in vivo methods to investigate the functions of NSSI.

Third, the present study was limited in its ability to conclusively distinguish between an ANR and APR function of NSSI. Although, as discussed above, the present findings converge on an ANR function, until habituation is ruled out as the cause of the across-groups decrease in startle-alone reactivity during the CPT conditions, this conclusion cannot be confidently drawn. Indeed, the present results also demonstrate that future studies should include many more (approximately 30) non-experimental habituation trials before attempting to index changes in startle reactivity due to tonic stress and pain. An additional implication of these findings is that startle-alone reactivity may not be the most appropriate test to distinguish between the ANR/APR functions;

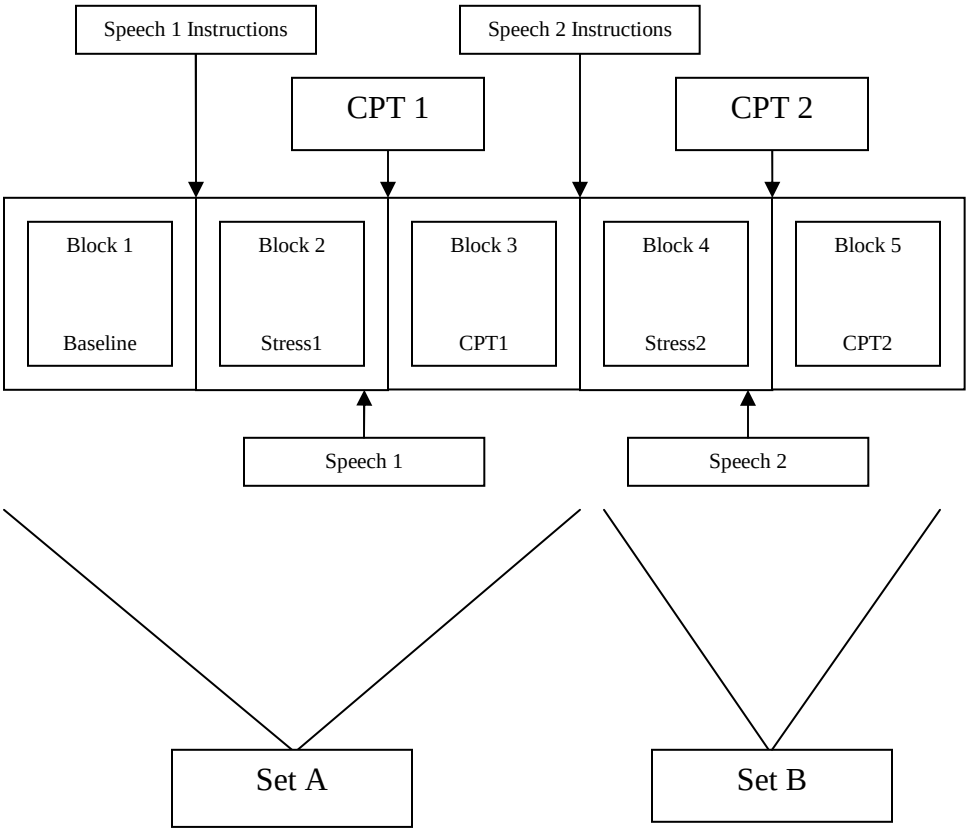
however, another startle modulation paradigm – affective-valence startle modulation – would seem to be well-suited for such an investigation.

Fourth and finally, despite providing preliminary support for the possibility that repeated stress-pain pairings represent one mechanism of the initial reinforcement of NSSI, the present study was not able to conclusively support this hypothesis. Nevertheless, the present study does take the important step of suggesting that such investigations are feasible. Future studies should expand this paradigm by measuring changes in reactivity to pain across several NSSI-proxy administrations and, additionally, such studies may benefit from examining possible social influences on initial reinforcement of NSSI (e.g., altering one's perception of what the experience of the NSSI-proxy will be like before administering it).

Conclusion

The present study has important implications for both basic and applied research. In terms of basic research, the present findings indicate that: (1) PPI can be effectively employed to measure state changes in information processing; (2) PPI is disrupted by both stress and pain; and (3) to examine startle-alone changes due to stress and pain, several (e.g., 30) habituation trials should be delivered. In terms of applied research, the present results suggest that: (1) affect dysregulation and NSSI are associated with decreased PPI at baseline; (2) whereas pain disrupts the information processing of non-NSSI individuals, it improves the information processing of NSSI individuals beyond baseline; and (3) repeated stress-pain pairings may be a mechanism of the initial reinforcement of NSSI and, additionally, individuals high in affect dysregulation may be more susceptible to this initial reinforcement.

Figure 1. *Diagram of Experimental Design*.



Notes. CPT stands for cold pressor task; there were three minutes between each block of stimuli (this interval was filled with either rest/instructions or speech/CPT).

Figure 2. *Group SUDS by condition (with 95% error bars).*

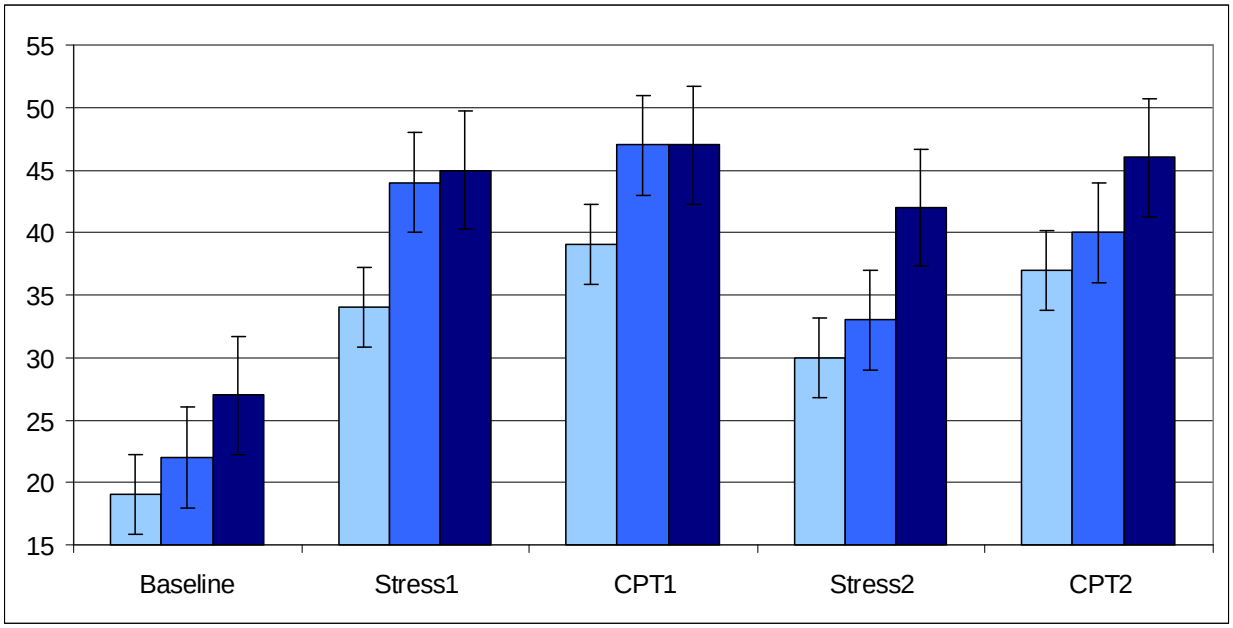


Figure 3. *Group startle-alone reactivity by condition (with 95% error bars).*

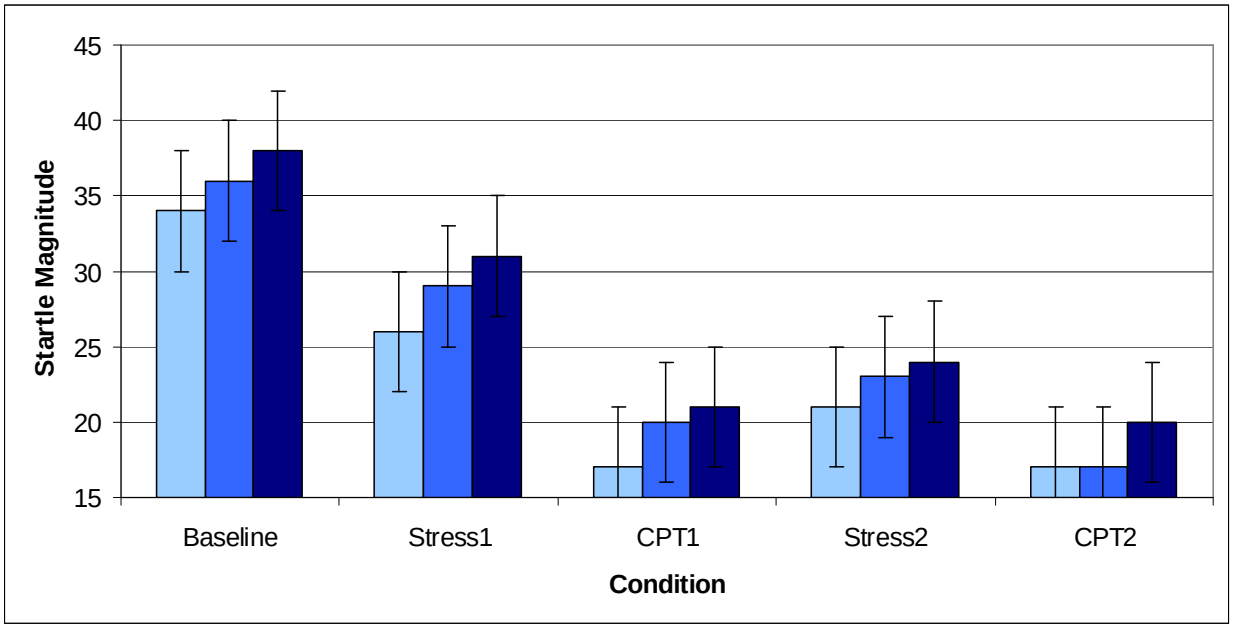


Figure 4. *Group PPI by condition (with 95% error bars).*

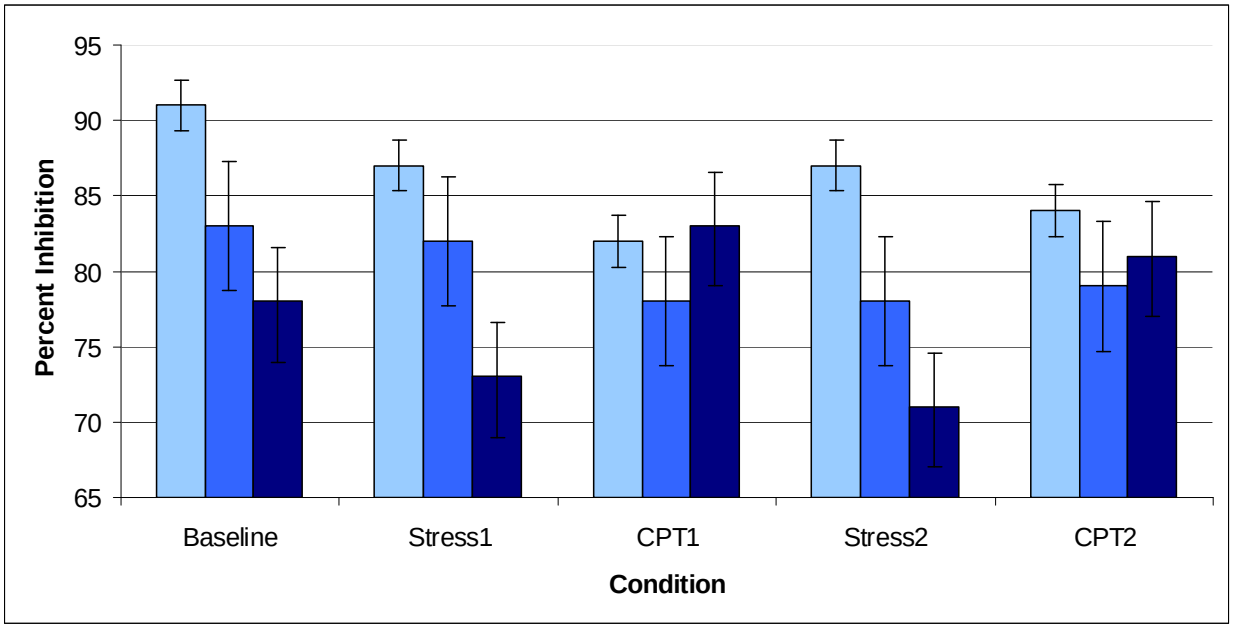


Table 1. *FASM Frequency Data (%).*

Group	Cut	Burn	Scrape	Insert	Tat	Hair	Hit	Pick	Bite	None	N
Control	0	0	0	0	0	3	9	9	9	66	33
AD	0	0	0	0	0	0	16.7	50	25	37.5	24
NSSI	62.5	25	25	18.8	12.5	31.4	37.5	56.3	43.8	0	16

Notes. Cut = cutting the skin; Burn = burning the skin; Scrape = scraping the skin; Insert = inserting objects under the fingernails/skin; Tat = giving oneself a tattoo; Hair = pulling hair; Hit = hitting oneself; Pick = picking the skin or wounds; Bite = biting the self (e.g., lip); None = did not endorse any FASM items.

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