CHANGES IN NEGATIVE AFFECT FOLLOWING PAIN (VS. NONPAINFUL) STIMULATION IN INDIVIDUALS WITH AND WITHOUT A HISTORY OF NONSUICIDAL SELF-INJURY

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Changes in Negative Affect Following Pain (vs. Nonpainful) Stimulation in Individuals

With and Without a History of Nonsuicidal Self-injury

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The Supervisory Committee certifies that this ***disquisition*** complies with North Dakota State University's regulations and meets the accepted standards for the degree of

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ABSTRACT

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Theoretical models of nonsuicidal self-injury (NSSI; i.e., purposeful destruction of body tissue without suicidal intent) suggest that individuals engage in NSSI in order to regulate intense emotions. However, empirical support for these models is limited. This study attempted to address previous limitations by comparing the emotional response to a mood induction and pain (vs. nonpainful) heat stimulation in individuals with history of NSSI (vs. no history of NSSI) following a negative mood induction. It was predicted that individuals with a history of NSSI would have a larger increase in negative emotion following the mood induction, and individuals with a history of NSSI who are exposed to a painful stimuli would have the largest decrease in negative emotions compared to the other three groups. Though the first hypothesis was not supported, the second hypothesis received partial support. Clinical implications and future research directions are discussed.

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INTRODUCTION

Nonsuicidal self-injury (NSSI) is defined as self-inflicted damage to one's own body tissue that is performed in the absence of suicidal intent and cultural acceptance (Klonsky, 2009). The onset of NSSI is usually during adolescence (Kumar, Pepe, & Steer, 2004). Lifetime prevalence rates range from 11 to 39% (Gratz, Conrad, & Roemer, 2002; Heath, Toste, Nedecheva, & Charlebois, 2008) and the one year prevalence is approximately 7.3% (Whitlock, Eckenrode, & Silverman, 2006). NSSI is a behavior that is difficult to understand because it lies in opposition to the common principle of approach/maximize pleasure and avoid/minimize pain (Freud, 1929; Gray, 1982; Kahneman & Tversky, 1979; Mowrer, 1960).

Theoretical work suggests that individuals who engage in NSSI have increased responses to emotional stimuli (Nock, 2009) and empirical work supports the idea that NSSI and physical pain may improve emotional states (e.g., Hollin & Derbyshire, 2009; Muehlenkamp et al., 2010). Still, there are gaps in the current literature. For example, evidence is mixed as to whether or not self-injurers have increased responses to emotional stimuli. Also, currently it is unclear if painful stimulation is necessary to regulate emotion or whether any tactile stimulation is adequate. The primary aim of this study was to investigate the influence of physical pain on negative emotional states for individuals who engage in NSSI. To do this, I compared individuals with a recent history of NSSI to individuals with no NSSI history on their emotional response to a mood induction and subsequent painful (versus nonpainful) heat stimulation.

Nonsuicidal Self-injury as Affect Regulation

There are multiple models of NSSI (e.g., Chapman, Gratz, Brown, 2006; Nock, 2009), as well as models of dysregulated behavior in general that may be applied to NSSI (e.g., Selby & Joiner, 2009). Across models, it is proposed that individuals who engage in NSSI experience intense emotions that they have difficulty regulating. NSSI, then, is seen as a dysfunctional strategy that is used to regulate emotion (e.g., Linehan, 1993). More specifically, most models suggest NSSI serves to reduce negative affect (NA; Klonsky, 2007).

For example, the emotional cascade theory of borderline personality disorder (BPD; Selby & Joiner, 2009) posits that individuals who engage in dysregulated behaviors, including NSSI, experience intense negative affect (NA). This is purportedly due to an emotional vulnerability, where they have a heightened sensitivity to emotional stimuli, a tendency to experience intense emotions, and have difficulty returning to baseline. The theory proposes that dysregulated behaviors serve to distract an individual from rumination and intense feelings of NA. Furthermore, the model suggests that less intense methods of distraction (e.g., a cold shower) are unsuccessful in regulating intense emotions, and only strong sensations (e.g., physical pain, sexual stimulation) are able to distract from high NA states.

Emotional Vulnerability and Nonsuicidal Self-injury

Compared to research on the emotion regulation effects of pain in NSSI, very little work has tested the prediction that individuals who engage in NSSI are more emotionally vulnerable. Some support has been found in group differences in personality traits. For example, individuals with a history of NSSI score higher on traits such as neuroticism (Brown, 2009) and difficulties regulating emotion (Gratz & Roemer, 2004) than individuals with no NSSI history. Also, Nock, Wedig, Holmberg, and Hooley (2008) found that individuals who engaged in NSSI in the last year had higher levels of self-report emotional reactivity than individuals who had engaged in NSSI more than one year ago. Support for this hypothesis using laboratory paradigms has been mixed. Nock and Mendes (2008) found that adolescent self-injurers had larger skin conductance responses to a distressing card sorting task compared to controls. However, Gratz et al. (2010) found no group differences in increase in NA following a distressing mood induction. Therefore, it is currently unclear if individuals who engage in NSSI are more reactive to emotional stimuli.

Pain as Affect Regulation

A growing body of research has found support for the prediction that NSSI and pain can decrease the experience of NA. One series of studies exposed individuals with a history of NSSI to imagery scripts of NSSI incidents (Brain, Haines, & Williams, 2002; Haines, Williams, Brain, & Wilson, 1995; Welch, Linehan, Sylvers, Chittams, & Rizvi, 2008). These studies suggest that NA and heart rate increase in the moments leading up to the incident and decrease following the imagined NSSI incident. This pattern of responses also differs from responses to control scripts (e.g., accidental injury). One major limitation to these studies is that no physical stimulus is presented. Therefore, it is unclear if these results would replicate with the inclusion of the physical pain experienced during actual NSSI.

Other studies using undergraduate samples without a history of NSSI suggest that the experience of pain may decrease NA (e.g., Hollin & Derbyshire, 2009). For example, Bresin, Gordon, Bender, Gordon, and Joiner (2010) measured positive affect (PA) and NA before and after the experience of pain in two samples of college undergraduates. Results showed that both PA and NA decreased following the experience of pain. For NA, this effect was also moderated by emotional reactivity (a construct similar to emotional vulnerability; Nock et al., 2008) such that individuals higher in emotional reactivity had larger decreases in NA following the experience of pain. This may suggest that those high in emotional reactivity, a correlate of NSSI, may find the experience of pain more rewarding than individuals low in emotional reactivity. One major limitation of this study is that there was no manipulation of emotion. It is possible that at high levels of NA, pain may exacerbate NA. Also, this study included individuals without a history of NSSI which potentially limits generalizability of the results to self-injuring populations.

These limitations were addressed in a study by Franklin, Hessel, Aaron, Arthus, Heilbron, and Prinstein (2010). In this study, affective modulation of the startle reflex was measured at baseline, during anticipation of a stressful speech, and after a cold pressor task. There were three groups in the study: individuals with a history of NSSI, individuals with no history of NSSI who were matched on the difficulties in emotion regulation scale (Gratz & Roemer, 2004) with the NSSI group, and individuals with no history of NSSI not matched on emotion dysregulation. There was also a group of control participants who were not exposed to the cold pressor task. The results showed that, regardless of group, participants displayed a reduction in the startle reflex after exposure to the cold pressor task, indicating that, regardless of NSSI status, the experience of pain may reduce NA. One limitation of this study is the small sample in the NSSI group ($n = 16$), which may have limited statistical power. It is possible that larger samples would yield different results. Another limitation is the lack of a control condition that consists of nonpainful tactile stimulation. Based on this study, it is unclear whether an intense experience, such as pain, is necessary for decreasing NA (as the emotional cascade model of BPD would predict; Selby & Joiner, 2009) or if any tactile stimulation would also decrease NA.

A recent study by Niedtfeld et al. (2010) attempted to examine the difference between painful and nonpainful stimulation. In this study, a group of individuals with BPD (80% with a history of NSSI) and healthy controls were exposed to negative and neutral emotion eliciting slides for three seconds. Then, depending on the trial, participants were exposed to either painful stimuli or warm nonpainful stimuli along with the slide for nine seconds. The participants' brain activity was recorded using functional magnetic resonance imaging. Consistent with previous findings (Schmahl et al., 2006), the authors predicted that individuals with BPD, as compared to controls, would show a deactivation in the amygdala (an area associated with NA; Davidson & Irwin, 1999) in response to pain but not to nonpainful heat stimulation. Inconsistent with predictions, both groups showed deactivation in the amygdala during pain and nonpainful heat stimuli, though this effect was larger for individuals with BPD than controls. These results may suggest that any sensory stimulation, not just painful stimulation, may be effective in reducing NA.

One limitation of this study is the within-subject nature of the design. Participants were exposed to a total of 80 images and only had 6-10 seconds between trials. It is possible that participants may not have returned to baseline before the next trial began. Also, repeated exposure to painful and nonpainful stimuli from trial to trial may have lead to carryover effects. A second limitation is that this study was designed to examine the effect of pain on emotion in individuals with BPD, not self-injurers per se. Though NSSI is one possible symptom of BPD, it is possible to meet criteria for BPD without engaging in NSSI (American Psychiatric Association, 2000). Moreover, when controlling for NSSI many individuals no longer meet criteria for BPD (Herpetz, Sass, & Favazza, 1997). It is possible that the relationship between pain and emotion may differ for those who engage in NSSI but do not necessarily meet criteria for BPD.

Current Study

There is a growing body of research to suggest that individuals who engage in NSSI have heightened levels of emotional vulnerability, and that pain can reduce the experience of NA. Nevertheless, these studies have limitations. First, there is mixed support that self-injurers have more intense reactions to emotional stimuli. Second, few studies have compared pain to other types of sensory stimulation (e.g., nonpainful heat stimuli). Therefore, it is unclear whether pain is necessary to reduce NA or whether any sensory stimulation is effective. Finally, studies have used a variety of samples ranging from college students without a history of NSSI (e.g., Bresin et al., 2010) to individuals with BPD but not necessarily a history of NSSI (e.g., Niedtfeld et al., 2010). Therefore, it is unclear exactly how individuals with a recent history of NSSI differ in their emotional response to pain from those with no NSSI history.

The current study attempts to address limitations of previous studies. Individuals with a history of NSSI in the last year and without a history of NSSI were recruited. Participants completed a mood induction where they wrote about a time when they were not living up to an important personal attribute (e.g., being intelligent). Following the mood induction, participants were either exposed to painful heat stimulation or nonpainful heat stimulation (based on participant ratings; detailed below). Finally, participants gave self-report levels of affect at three times during the study (Time 1: before any mood induction; Time 2: following the mood induction; Time 3: following the painful/non painful induction).

There are two main hypotheses for the study. First, based on theoretical models (e.g., Chapman et al., 2006) and previous research (Nock & Mendes, 2008) it was predicted that the NSSI group would have a larger increase in NA following the mood induction (Time 1 to Time 2) compared to the Non-NSSI group. Second, a significant three-way interaction between group, condition, and time was predicted. Based on models of dysregulated behavior (e.g., Selby & Joiner, 2009) and previous research (Bresin et al., 2010), it was predicted that the individuals with a history of NSSI who were in the pain condition would have a larger decrease in NA than the other three groups. Similar analyses were conducted with PA, but were exploratory in nature.

METHOD

Participants

Over the course of two semesters, 1,569 undergraduates were screened using the Deliberate Self-Harm Inventory (DSHI; Gratz, 2001) for participation in this study. Of these, 130 participants (8%) indicated that they had engaged in NSSI in the past 12 months, 246 participants (15%) indicated engaging in NSSI more than 12 months ago, and the remaining participants (77%) reported no history of NSSI.

NSSI Group: Of the130 participants who indicated NSSI in the last 12 months, 48 (28 Female) were recruited for the NSSI group. The mean age was 19.20, (*SD* = 2.72). The ethnic breakdown was 95% Caucasian and 4% Black/African American. The average time since the last NSSI episode was 5.33 months (*SD* = 4.01). The most common method of NSSI was cutting (49%), followed by scratching (38%) and burning (29%). Participants reported an average of 2.88 methods (*SD* = 1.84). The median number of NSSI incidents was 13 (range = 1-1000).

Non-NSSI Group: For the control group, 47 (27 Females) participants without a history of NSSI were recruited (*M* age =19.29; *SD* = 2.47). The ethnic breakdown was 95% Caucasian, 2% Black/African American, and 2% Asian or Pacific Islander.

Materials

Pain Stimuli: All pain stimuli were produced by a Thermal Sensory Analyzer (TSA; Medoc TSA 2001; Ramat-Yishai, Israel), a computerized device for the quantitative assessment of heat- and cold-induced pain. All stimuli were presented inside the left arm. Participants were exposed to pain stimuli twice during the session. Early in the experiment, participants were exposed to and rated a series of temperatures (40^{o} C, 43^{o} C, 45^{o} C, 48^{o} C,

50^{o} C). On each trial, the thermode began at 35°C. The temperature level increased at a rate of 4^{o} C per second until the target temperature was reached. Once the target temperature was reached, the thermode maintained the temperature for seven seconds.

Following each trial, participants made a verbal rating of stimulus intensity on a scale ranging from 0 (*no heat sensation*) to 100 (*intolerable heat pain*) with 50 (*heat pain threshold*) as a midpoint (Tran Wang, Tandon, Hernandez-Garcia, & Casey, 2010). The order of the temperatures was held constant. In order to prevent habituation, there was a 30 second time block between trials (Tran et al., 2010). If the participant felt they could not withstand a temperature, they were instructed to click a mouse button which decreased the temperature at a rate of 10^{o}C per second until the adaptation temperature was reached.

Following the mood induction, participants were exposed to the temperature they previously rated nearest to 20 (nonpainful stimulation) or the temperature they rated nearest to 60 (painful stimulation) based on random assignment. Cut scores were chosen to be consistent with Niedtfeld et al. (2010). In the case of ties, higher temperatures were used. No temperatures were used if the participant did not previously complete the entire seven second trial. Procedures were the same as earlier in the study, aside from the fact that each participant was only exposed to one temperature.

Mood Induction: As stimuli for the mood induction, participants completed the computerized selves interview (Shah & Higgins, 2001). First, participants listed four "attributes of the person you would ideally like to be" and four "attributes of the person you feel you should be". After providing all eight attributes, participants rated each attribute on how well it applies to them (Apply), how likely they are to possess it in the future (Future), how far they are from possessing it (Far), and how important it is for them to possess (Important). All ratings were made on a 7-point Likert scale (1 = *not at all*, 7= *extremely*), aside from Apply which uses a 5-point Likert scale (1 = *does not describe me at all*, 5 = *completely describes me*).

A discrepancy score was calculated to decipher the attribute each participant rated most important and currently furthest from (i.e., Far + Important). In the case of ties, attributes entered into the computer earlier were used. Then, participants were asked to "choose a specific time in your life in which you were supposed to act with or be *attribute* and were not". Participants were encouraged to pick a situation that caused great stress and made them "upset, mad, nervous, sad, or fearful and in that moment you felt as if you could not do much to change it". After choosing a situation, participants rated the situation on a scale to 1 (*not stressful at all*) to 10 (*the most stress I have experienced*). Finally, participants were given 10 minutes to describe the situation they chose in detail including: where they were, who was there, what they were doing, and the body sensations they experienced. Participants were asked to try to generate the same sensations and feelings that they felt in the situation while they were writing. This procedure has been successful in increasing NA in other studies from our lab.

<u>Screening Measure:</u> All potential participants completed the DSHI (Gratz, 2001) on a secure website prior to participating in the study. The DSHI is a 17-item questionnaire that assesses the lifetime history of a variety of NSSI methods. First, participants indicate if they have ever intentionally (i.e., on purpose) injured areas of their body without suicidal intent using a specific method (e.g., cutting). If participants respond yes, they answer follow up questions (e.g., When was the last time you did this?). Participants who indicated they had engaged in any form of NSSI within the last 12 months were sent an

email about participation in the study. The time frame of NSSI in the last year was chosen to be consistent with previous studies (e.g., Gratz et al., 2011; Nock & Banaji, 2007). Also, participants who reported no history of NSSI in their lifetime were contacted about participation in the study.

Covariate Measure –Borderline Personality Features: To control for group differences in psychopathology, I measured BPD symptoms, depression symptoms, and suicidal ideation. BPD symptoms were assessed using the McLean Screening Instrument for Borderline Personality Disorder (MSI-BPD; Zanarini et al., 2003). The MSI-BPD is a 10-item self report measure of BPD symptoms. Participants respond true or false to each item (e.g., "Have chronically felt empty?") with respect to the last few years. True responses were summed to create one MSI-BPD score ($\alpha = .80$).

Covariate Measure- Depression: Depression was assessed using the Beck Depression Inventory II (BDI-II; Beck, Steer, & Brown, 1996). The BDI-II is a 21-item self-report measure of depression. For each item, participants endorse one option (e.g., 0 = *I get as much pleasure as I ever did from the things I enjoy*, 1= *I don't enjoy things as much as I used to enjoy*, 2 = *I get very little pleasure from the things I used to enjoy*, 3 = *I can't get any pleasure from the things I used to enjoy*) reflecting their symptoms "over the last two weeks including today". Items were summed to create a depression score ($\alpha = .91$). One participant who scored higher than 28 (consistent with severe depression) was excluded from the study in order to minimize risk of discomfort.

Covariate Measure- Suicidal Ideation: Current suicidal ideation was assessed using the Beck Scale for Suicidal Ideation (BSS; Beck & Steer, 1991). The BSS is a 21-item self-report measure of suicidal symptoms. For each item, a score of 0-2 is assigned by each item (e.g., 0 = *I have a strong will to live,* 1 = *I have a weak wish to live,* 2 = *I have no wish to live*) to describe how the participant has felt in the last week including today. Items 1-19 were summed to make a current suicidal ideation score (α = .80). Items 20 and 21 assess past attempts and were not used in the ideation score.

Dependent Measure: PA and NA were measured by a 10 item version of the Positive and Negative Affective schedule (PANAS; Watson, Clark, & Tellegen, 1988) created by Thompson, 2007. Five items (upset, hostile, ashamed, nervous, afraid) measured NA, and five items (alert, inspired, determined, attentive, active) measured PA. Participants rated each item on a 5-point Likert scale (1 = *not at all*, 5= *extremely*) with the instructions of "indicate the extent to which you feel AT THIS MOMENT". Participants completed the PANAS three times during the study: Time 1 (before mood induction), Time 2 (following the mood induction), and Time 3 (following the painful/non painful induction). Internal consistencies ranged from .77-.78 for NA and .72 -.88 for PA.

Procedure

Upon arrival to the laboratory, participants provided consent and completed the BDI and BSS. Then, participants rated the intensity of the warm temperature stimuli. Next, participants completed a pre-induction measure of affect (i.e. Time 1 NA and PA). Then, participants completed the mood induction and a Time 2 NA and PA assessment. Immediately following the mood induction, participants were randomly selected to be exposed to either a nonpainful stimulation or a painful stimulation and subsequently reported their affect (i.e., Time 3 NA and PA). Participants then completed the MSI-BPD. Finally, participants were thanked for their time and excused. Participants completed the study for course credit or 10 dollars.

RESULTS

Preliminary Analysis

Of the 48 participants in NSSI group, 29 were in the nonpainful stimulation condition and 19 were in the painful stimulation condition. Of the 47 participants in the Non-NSSI group, 23 were in the nonpainful condition and 24 were in the painful condition. The distribution of males and females did not differ among conditions for the NSSI group, χ^2 (1) = 1.32, p =.24, or the Non-NSSI group, χ^2 (1) = .21, p =.64.

The NSSI group had higher scores on the MSI-BPD (M =4.14, SD = 2.80) than the Non-NSSI group (M = 1.52, SD =1.62) and this difference was significant, t (92) =5.59, $p <$ .001, d = 1.12. The NSSI group also had significantly higher BDI scores (M = 11.16, SD = 8.31) than the Non-NSSI group (M = 3.12, SD = 5.29), t (94) = 5.66, $p < .001$, d = 1.15. Finally, the NSSI group (M = 1.75, SD = 3.25) had significantly higher scores on the BSS than the Non-NSSI group (M = .14, SD = 1.01), t (94) = 3.26, $p < .001$, d = .66. These findings are consistent with previous research (Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006). Groups did not significantly differ in the stress rating of their event for the mood induction, t (94) = .87, p = .38, d = .18. Across groups, the average rating was 6.75 (SD = 1.77) on a 10-point scale.

As predicted, participants in the nonpainful condition rated the intensity of the stimuli at the second exposure lower (M = 26.21, SD = 16.35) than participants in the painful condition (M = 55.83, SD = 21.15) and this difference was significant, t (92) = -7.66, $p < .001$, d = 1.58. There was no effect of NSSI group on intensity ratings of heat stimuli and group did not interact with condition (p's > .18). In the nonpainful condition, 80% of the participants were exposed to a temperature of 42°C or lower. Conversely, in the painful condition, 82% of the participants were exposed to a temperature greater tha 48°C.

Hypothesis One

To test the first hypothesis, the data wer submitted to a group (NSSI vs. Non-NSSI) by time (Time 1 vs. Time 2) mixed model analysis of variance (ANOVA), with group as a between-subject factor, time as a within-subject factor, and NA as the dependent variable. There was not a significant main effect of group, $F(1, 92) = 1.38, p = .24$, partial $\eta^2 = .01$. There was a significant main effect of time, $F(1, 92) = 186.87, p < .001$, partial $\eta^2 = .51$. The main effect of time indicated that the mood induction was successful in significantly increasing NA across groups. More importantly, there was a significant group by time interaction, $F(1, 92) = 8.28, p < .01$, partial $\eta^2 = .04$. Contrary to my predictions, individuals without a history of NSSI had a significantly larger increase in NA following the mood induction (i.e., Time 2 NA – Time1 NA; $M = 1.64$, $SD = .95$) compared to individuals with a history of NSSI, ($M = 1.17$, $SD = .95$), $t(92) = 2.53, p < .05, d = .49$. Groups did not significantly differ at Time 1, $t(92) = 1.43, p = .15, d = .26$, but at Time 2 the Non-NSSI group had significantly higher NA, $t(92) = 2.68, p < .01$ $d = .43$.

To examine if controlling for borderline features, suicidal ideation, and depression changed the relationship between group and mood induction, MSI-BPD, BSS and BDI scores were added as covariates to the previous ANOVA model. The group by time interaction was no longer significant, $F(1, 92) = .14, p = .70$, partial $\eta^2 = .00$. Moreover, there was a significant MSI-BPD by time interaction, $F(1, 92) = 5.37, p = .05$, partial $\eta^2 = .03$. Neither the BSS by time or BDI by time interactions were significant. Consistent with the above results, higher scores on the MSI-BPD were related to significantly lower levels of change in NA from Time 1 to Time 2 (i.e., Time 2 NA – Time1 NA), $r = -.41, p < .001$. These results may suggest that borderline features may account for group differences in reactivity to the mood induction.

Hypothesis Two

To test the second hypothesis, NA scores were submitted to a group (NSSI vs. Non-NSSI) by condition (painful stimulation vs. nonpainful stimulation) by time (Time 2 vs. Time 3) mixed model ANOVA, with group and condition as between-subject factors and time as a within-subject factor. There was not a main effect of group or condition, and both the group by condition and time by condition interactions were not significant (all p's > .05). There was a significant main effect of time, $F(1, 88) = 270.39, p < .001$, partial $\eta^2 = .55$, and time by group interaction, $F(1, 88) = 4.95, p < .05$, partial $\eta^2 = .02$. Finally, consistent with my prediction, the three way interaction was significant, $F(1, 88) = 5.90, p < .05$, partial $\eta^2 = .02$.

To follow up this interaction, I first created a change score by subtracting Time 2 NA from Time 3 NA, so that negative values indicated a decrease in NA. Then, I ran pair-wise comparisons between the cells (see Figure 1). Consistent with my hypothesis, individuals in the NSSI group in the No pain condition had the smallest change in NA ($M = -.94$, $SD = .74$), and this was significantly smaller than all other groups (p's < .05). Inconsistent with my predictions, the other three cells were not significantly different (p's > .35). In spite of the lack of significant group differences, individuals in the Non-NSSI/nonpainful stimulation cell had the largest change in NA ($M = -1.75$, $SD = .94$), followed by NSSI/pain ($M = -1.64$, $SD = .79$) and the NSSI nonpainful stimulation ($M = -1.53$, $SD = .86$). Controlling for MSI-BPD, BSS and BDI did not change these results.

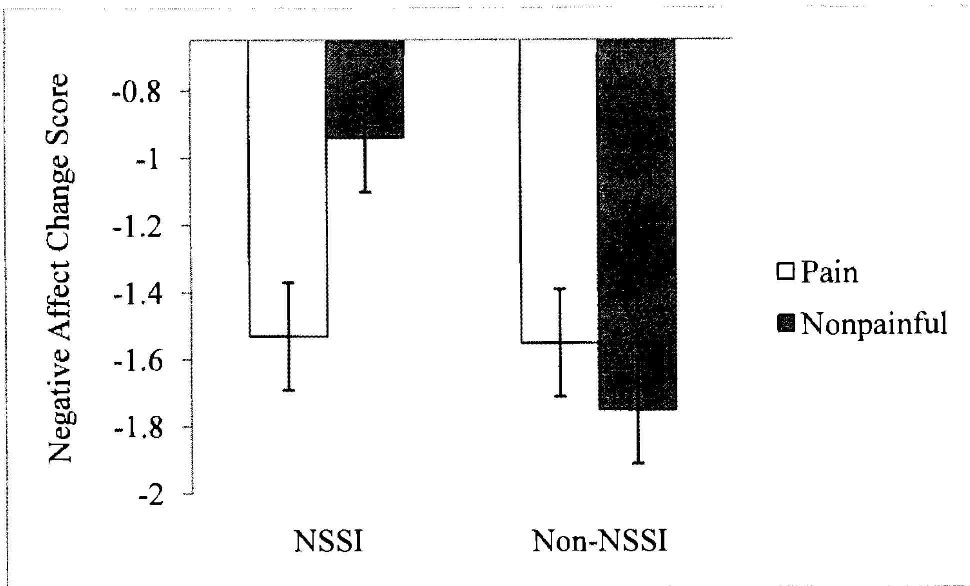

Figure 1. Change in Negative Affect Scores from Time 2 to Time 3 as a function of stimulation type and nonsuicidal self-injury status (bars reflect standard error of the mean).

Positive Affect

For exploratory purposes, I ran a group (NSSI vs. Non-NSSI) by condition (painful stimulation vs. nonpainful stimulation) by time (Time 1 vs. Time 2 vs. Time 3) mixed model ANOVA, with PA as the dependent measure. This omnibus test was used since there were no a priori hypotheses. There was not a main effect of group or condition, or a group by condition interaction (p's > .05). However, there was a main effect of time, F (2, 176) = 78.58, $p < .001$, partial $\eta^2 = .48$. Averaged across group and condition, there was a significant decrease in PA from Time 1 to Time 2, t (92) = -10.05, d = -1.03 and a significant increase from Time 2 to Time 3, t (92) = 9.01, d = .93. Neither the time by condition nor the three way interaction was significant (p's > .05). There was a significant group by time interaction, F (2, 176) = 5.55, $p < .001$, partial $\eta^2 = .03$. However, when controlling for MSI-BPD, BDI, and BSS the group by time interaction was no longer significant and time did not interact with any psychopathology measures. The main effect of time was still significant when covarying for psychopathology.

DISCUSSION

The primary aim of this study was to examine how individuals with a recent history of NSSI differ from individuals without a history of NSSI in their emotional response to a mood induction and painful versus nonpainful heat stimulation. Based on previous research and theoretical models two predictions were made. First, it was predicted that individuals with a history of NSSI would have a stronger reaction to the mood induction compared to those with no history of NSSI. Second, it was predicted that for individuals with a history of NSSI, pain would lead to a larger reduction in NA compared to nonpainful stimulation. I found mixed support for these hypotheses.

The first hypothesis was not supported. Individuals with a history of NSSI had smaller reactions to the mood induction compared to individuals with no NSSI history. This finding is inconsistent with previous work showing individuals who engage in NSSI report elevated trait levels of emotional reactivity (Nock et al., 2008) and increased skin conductance during a distressing task (Nock & Mendes, 2008), but is consistent with Gratz et al. (2011). Follow up analyses indicated that this effect may be due to BPD symptoms, since when controlling for BPD symptoms there was no longer a significant group by time interaction and a very similar pattern was found in a significant BPD symptom by time interaction.

Previous work has found mixed results for increased emotional reactivity in individuals with BPD (See Rosenthal, Gratz, Kosson, Cheavens, Lejuez, & Lynch, 2008 for a review). My results in this study are similar to Herpertz et al. (1999) who found that compared to controls, individuals with BPD had decreased skin conductance response when exposed to emotion-eliciting slides. It should be noted that the current study only tested certain aspects of emotional vulnerability. More support has been found for other components of emotional vulnerability in BPD such as high baseline NA (e.g., Kuo, & Linehan, 2009). These results stand in contrast to predominant theories of BPD (e.g., Linehan, 1993), but are consistent with theories of other disorders such as the context insensitivity theory of depression (Rottenberg, 2005), which suggests that depressed mood states lead to a bias of inaction. Therefore, individuals with high levels of NA react less to both positive and negative stimuli. Future work is necessary to fully understand the relationship between NSSI, BPD, and emotional vulnerability.

The second hypothesis was partially supported. Consistent with my prediction, for individuals with a history of NSSI, painful sensations compared to nonpainful sensations lead to a larger decrease in NA. Inconsistent with my prediction, compared to controls that experienced either painful or nonpainful stimulation, the experience of pain for self-injurers did not lead to larger reductions in NA. This effect appears to be specific to NA, since similar analysis on PA found that groups and conditions did not differ on PA change.

These results are in line with previous research showing that physical pain leads to a reduction in NA in healthy controls (e.g., Bresin et al., 2010). More importantly, these results are consistent with Franklin et al. (2010), who found a reduction in startle reflex following a cold pressor task, but no differences between individuals with a history of NSSI and controls. Finally, these results stand in contrast to Niedtfeld et al. (2010) who found no interaction between group and stimulation type. However, as mentioned above, this study included individuals with BPD, not exclusively individuals with recent NSSI. Also, the current study used only one mood induction, and a between-subject manipulation of painful versus nonpainful stimuli potentially eliminating carry over effects from previous trials. Finally, this study measured experiential aspects of emotion as opposed to brain activity, which may have led to divergent results.

With respect to the second hypothesis, these results are consistent with the emotional cascade theory (Selby & Joiner, 2009), which suggests that emotionally dysregulated individuals engage in maladaptive behaviors to distract themselves from NA. Furthermore, the emotional cascade theory posits that intense sensations (e.g., pain) are necessary to distract from high levels of NA. My results show that individuals who engage in NSSI may have more incentive to engage in NSSI to regulate emotion compared to other forms of stimulation (e.g., cold shower), which may not reduce NA as effectively. Nevertheless, the emotional cascade theory suggests intense stimulation is necessary for intense emotional responses. Since my first hypothesis was not supported, the second hypothesis only provides partial support for this theory.

The results of the second hypothesis are also in line with opioid dysregulation theories of NSSI (Bandelow, Schmahl, Falkai, & Wedekind, 2010; Thompson, Symons, Delaney, & England, 1995) which suggest that individuals who engage in NSSI may have a dysregulated opioid system that leads them to experience decreased pleasure and increased dysphoria and depersonalization. Consequently, these individuals are proposed to engage in NSSI, among other dysregulated behaviors, as an attempt to regulate opioid levels in the body. This may suggest that, for individuals without a history of NSSI, NA change did not differ between the painful and nonpainful conditions because their opioid system responds similarly in both situations. In contrast, individuals who have a history of NSSI may need more intense stimulation to regulate their opioid system in a similar manner to people without a history of NSSI. However, since opioids were not measured or manipulated, this study is an imperfect test of these theories. Future work may benefit by examining the role of opioids in the reduction of NA following the experience of pain.

Treatment Implications

The results of this study may have clinical implications. Clinical interventions may be focused on encouraging clients to use less destructive ways to induce pain (e.g., hold an ice cube, snap a rubber band) to regulate affect. The results of this study provide some empirical support for therapeutic methods used in some current treatment models for NSSI such as Dialectical Behavior Therapy (Linehan, 1993). Also, teaching individuals alternative, healthy emotion regulation skills may reduce the need for NSSI.

Limitations and Future Directions

This study should be considered in light of its limitations. First, there were limitations of the sample. The sample was a highly homogeneous sample of college age students. It is possible that different results would be found in more diverse samples. Second, the NSSI group and Non-NSSI group differed on psychopathology symptoms. Although we statistically controlled for some of these variables, future research would benefit by using control groups matched on possible confounding variables (e.g., BPD symptoms) to draw clearer conclusions. Third, my NSSI group included individuals with a wide range of psychopathology and NSSI severity. Therefore, it is unclear how well these results would generalize to more severe clinical samples. Finally, since this study did not include a group of individuals with a less recent history of NSSI (e.g., > 1 year ago). It is unclear if individuals who have engaged in NSSI recently differ in emotional response to pain from those who have engaged in NSSI ever.

This study should also be considered in light of its strengths. First, compared to previous research, this study had a relatively large sample of individuals who engaged in NSSI recently. Also, I was able to recruit an equal amount of males and females for the NSSI group, where previous research has used primarily female samples. Therefore, these results are not drawn by group differences in gender. Second, this study provided a strong test of whether pain functions differently from nonpainful stimulation in individuals with a recent history of NSSI compared to those with no history of NSSI. Third, compared to previous research, the mood induction was one that may be more related to real world triggers to NSSI. Finally, this study used a method of pain induction (i.e., heat) that is more similar to actual NSSI (e.g., burning) than some previous studies which used no pain induction (e.g., Brain et al., 2002) or a cold pressor (Franklin et al., 2010).

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