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Nonsuicidal self-injury and diminished pain perception: the role of emotion dysregulation

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Abstract

Nonsuicidal self-injury (NSSI) is the deliberate destruction of one's own body tissue in the absence of suicidal intent (e.g., cutting or burning the skin). Previous studies have found that people with a history of NSSI display diminished pain perception. However, it remains unclear why this effect occurs. In the present study, we used a sample of participants with (n = 25) and without (n = 47) a history of NSSI to test the hypothesis that emotion dysregulation partially explains why NSSI is associated with diminished pain perception. Pain perception was quantified as pain threshold, pain tolerance, and pain intensity ratings assessed during the cold pressor task. Nonsuicidal self-injury was associated with increased emotion dysregulation and diminished pain perception. Results showed that emotion dysregulation was correlated with diminished pain perception within both groups, demonstrating that this association exists regardless of NSSI history. Results also specified that emotion dysregulation partially accounted for the association between NSSI and pain tolerance but not other pain variables. Overall, results were consistent with the hypothesis that emotion dysregulation may increase NSSI risk in part by increasing the willingness to experience the pain involved in self-injury. Studies are needed to more directly investigate this hypothesis.

1. Introduction

Nonsuicidal self-injury (NSSI) is self-inflicted tissue damage that is intentional, direct, socially unacceptable, and without suicidal intent (e.g., cutting or burning the skin; [1]). NSSI is surprisingly common, with 1% to 4% of adults and 13% to 23% of adolescents reporting that they have engaged in NSSI at least once in their lives [2]. Rates in clinical samples are even higher, with some studies finding that 40% to 61% of adolescent psychiatric inpatients engage in NSSI [3,4]. In addition to being a health risk behavior itself [4], NSSI also may increase the risk of suicidal self-injury [5,6]. Although knowledge about NSSI has increased greatly for the last decade, many aspects of this behavior remain poorly understood.

Most people who engage in NSSI report that they do it because it regulates their emotions [7]. Initial laboratory studies using psychophysiological measures and experimental pain induction procedures (e.g., cold pressor task, thermode-generated heat) have supported these self-report findings [8-10]. Interestingly, some of these laboratory studies have found that pain generally regulates affect in both NSSI and non-NSSI groups (although there are some additional unique effects in the NSSI groups; [8,9]). This suggests that pain may naturally regulate affect. Basic research on pain supports this hypothesis and specifies that pain *offset* (i.e., the removal of pain)—rather than pain itself—may increase positive emotion (e.g., [11,12]) and decrease negative emotion (e.g., [13,14]). Other research suggests that this effect may not require the total offset of pain to occur; instead, any reduction in the intensity of a painful stimulus may generate this effect (e.g., [15]). Although it may regulate emotions, pain offset has the obvious downside of first requiring pain. Pain itself has been defined as the unpleasant sensory and emotional experience caused by actual or potential tissue damage [16]. The pain involved in self-injury helps to explain why most people do not engage in NSSI. But how are people who engage in NSSI able to overcome the instinct to avoid pain?

A partial answer to this question might be that people who engage in NSSI have diminished pain perception. This would make NSSI behaviors such as cutting or burning less aversive, thereby increasing the likelihood of such behaviors. Consistent with this idea, several studies have shown that

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NSSI participants diagnosed with borderline personality disorder have diminished pain perception [9,10,17,18]. Hooley and colleagues [19] obtained similar results in a community sample of NSSI participants. These studies demonstrate that NSSI is associated with diminished pain perception, but it remains unclear *why* this effect occurs. Addressing this question may provide insight into how people are able to overcome the pain involved in self-injury and increase knowledge about who is at risk for NSSI.

Although there are many potential explanations for this (e.g., fearlessness, naturally diminished pain perception, a history of painful and provocative events), one promising possibility is that emotion dysregulation is partially responsible for diminished pain perception in NSSI participants. Various lines of evidence indirectly support this hypothesis by showing that emotion dysregulation, diminished pain perception, and NSSI are all interrelated. First, several disorders associated with emotion dysregulation are also associated with decreased pain perception, including borderline personality disorder, posttraumatic stress disorder, and major depressive disorder [20]. Second, NSSI is associated with these disorders [21] and emotion dysregulation [22,23]. Third, most NSSI pain perception findings have occurred in samples of patients diagnosed with borderline personality disorder [9,10,17,18], which features emotion dysregulation as a core symptom. In a community sample of NSSI participants, Hooley and colleagues [19] found that NSSI and pain endurance were positively correlated with neuroticism, which is related to emotion dysregulation (cf. [24]). These lines of evidence strongly suggest that emotion dysregulation plays a role in the NSSI pain association, but this evidence remains preliminary and indirect.

Despite this initial support, there are also reasons to doubt this hypothesis. In particular, there is little direct evidence showing that a specific measure of emotion dysregulation is associated with diminished pain perception. In fact, Anestis and colleagues [25] examined 2 components of emotion dysregulation and found that one (negative urgency) was not associated with pain tolerance and that another (lack of distress tolerance) was associated with *decreased* pain tolerance (i.e., heightened pain perception). In addition, Gratz and colleagues [26] found that NSSI was only associated with increased pain tolerance under conditions of acute interpersonal stress (however, it should be noted that other studies found this association under nonstress conditions; e.g., [10,18,19]). This indicates that situational factors related to stress may play a more important role than emotion dysregulation itself.

These findings demonstrate that 2 important questions require further examination: (1) is emotion dysregulation itself associated with decreased pain perception? and (2) does emotion dysregulation partially account for why NSSI is associated with decreased pain perception? The primary purpose of the present study was to investigate these 2 issues. To examine the first issue, we tested the association between a multidimensional measure of emotion dysregulation and pain perception in a nonclinical sample that included a group of people who had engaged in NSSI in the previous year (n = 25) and a group of people who had never engaged in NSSI (n = 47). Pain perception was operationalized as 4 pain measures: pain threshold, pain intensity ratings at the threshold point, pain tolerance, and pain intensity ratings at the tolerance point. We predicted that emotion dysregulation would be associated with diminished pain perception in the NSSI group. We hypothesized that this same association would exist in the non-NSSI group. This latter finding would demonstrate that this association can exist independently of NSSI and may partially account for why emotion dysregulation is a risk factor for NSSI (although this specific risk factor hypothesis cannot be directly examined within the present cross-sectional design). We also explored the association between pain perception and various facets of emotion dysregulation to determine if some facets were more strongly correlated with pain perception than others (cf. [25]).

To examine the role of emotion dysregulation in the concurrent association between NSSI and pain perception, we investigated whether emotion dysregulation accounts for variance within the NSSI pain perception association. To test this, we used a new statistical technique that simultaneously considers multiple contributors to variance within a given association between 2 variables [27]. Importantly, significant effects within these models represent unique effects that do not overlap with other potential contributors to variance. This provided the opportunity to test the ability of emotion dysregulation to account for variance within the NSSI pain perception association uniquely from other variables (e.g., other pain variables, stress ratings). For example, it is possible that emotion dysregulation diminishes pain perception during NSSI because people high in emotion dysregulation have more intense responses to acute stressors. This might generate more stress-induced analgesia, thereby reducing pain perception. To test this, we induced stress before the pain induction task (cf. [26]) and measured subjective units of distress immediately before pain induction. If acute stress levels primarily explain diminished pain perception, then emotion dysregulation should not account for significant variance when subjective units of distress are entered into the model. It should be noted that the present study is cross-sectional, and thus, these analyses cannot speak to temporal relations. Nevertheless, these results have the potential to provide new insights into the interrelationships among NSSI, emotion dysregulation, and pain perception. Such insights can advance knowledge about how people overcome the pain involved in NSSI and who is at risk for developing and maintaining NSSI.

2. Methods

2.1. Participants

Participants were 72 young adults: 52 women, 20 men; 80.55% European American, 8.33% Asian American, 8.33%

African American, and 2.78% Hispanic American; ages ranged from 18 to 29 (mean, 19.09; SD, 1.32). Participants were recruited from 2 sources: (1) introductory psychology classes that included a research participation option (n = 46) and (2) campuswide email advertisements that offered payment of $20 for participation in the study (n = 26). 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 a summer college orientation program (n = 17) or (*b*) responded affirmatively to a campuswide 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). Analyses indicated that sex, ethnicity, and age did not significantly differ by group and were not significantly associated with NSSI, pain variables, or proposed mediators. Participants completed informed consent forms at the beginning of the study; all materials, measures, methods, and procedures were approved by the university institutional review board.

On the basis of their responses to the NSSI assessment measure described later, participants were sorted into either the NSSI group (n = 25) or the control group (n = 47). Participants were only included in the NSSI group if they endorsed at least one "severe" NSSI behavior—cutting (64%, n = 16), burning (24%, n = 6), or scraping the skin (24%, n = 6).

2.2. Procedure

Participants first filled out questionnaires assessing NSSI and affect dysregulation. During the experimental portion of the study, participants completed a speech task. At the end of the speech task, we assessed participants' subjective units of distress. Immediately after this, we administered the cold pressor task, a painful task that involves placing a hand in cold water. During the cold pressor task, we measured participants' pain threshold, tolerance, and pain intensity ratings. We provide more information about these tasks and measures in the following paragraphs.

2.3. Questionnaires

2.3.1. Screening questionnaire

This questionnaire was designed to briefly measure NSSI and affect dysregulation levels. Nonsuicidal self-injury was screened with the item, "In this past year, how often have you harmed or hurt your body on purpose (e.g., cutting or burning your skin, hitting yourself)." Affect dysregulation was screened with the 6 items from the Difficulties in Emotion Regulation Scale [28] that had the highest factor loadings on its 6 factors: 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.

2.3.2. Difficulties in Emotion Regulation Scale

The complete Difficulties in Emotion Regulation Scale is a 36-item questionnaire that shows high internal consistency, good test-retest reliability, adequate construct and predictive validity, and is positively correlated with NSSI in both men and women [28]. Cronbach α for the 6 subscales were between .80 and .89, with the overall α being .92 (cf. [28]). This scale was used to measure overall emotion dysregulation and 6 dimensions of emotion dysregulation (see previous paragraph).

2.3.3. Functional Assessment of Self-Mutilation [29]

The Functional Assessment of Self-Mutilation is a 33-item measure that assesses method, frequency, and functions of NSSI (e.g., "[Have you ever] cut or carved your skin"; "[did you do this] to feel relaxed"). The Functional Assessment of Self-Mutilation was used as a follow-up to the NSSI screening question to gather more information about specific behaviors.

2.3.4. Subjective units of distress scale

After the speech task, participants were asked to "rate your level of 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." This measure was used to quantify stress immediately before the cold pressor task.

2.4. Experimental tasks

2.4.1. Speech tasks

Participants in this study were given 1 of 2 speech tasks to induce acute stress before pain induction. 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." Speech topic was alternated across participants. Analyses indicated that speech topic did not influence any pain variables (*P*s > .05). Participants were given 4 minutes to prepare for, and 1 minute to deliver, each speech. Participants performed their speeches in front of a video camera and a monitor that displayed their live image. In addition, 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.

2.4.2. The cold pressor task

The cold pressor is one of the most widely used forms of experimental pain induction in psychological studies (e.g., [17,18,26,30]). For this task, a cooler containing a 2°C (as

indexed with 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. When instructed, participants submerged their hand (up to the wrist) in the water. Hand order alternated across participants, with half of the participants submerging their dominant and half submerging their nondominant hand. Analyses indicated that hand order did not influence pain variables (*P*s > .05).

Whereas previous studies examined pain ratings during the cold pressor task across a set amount of time (e.g., [17]), we followed the methods of studies that have used the cold pressor task to investigate pain threshold, tolerance, and intensity (e.g., [30]). Specifically, we instructed participants to inform the researcher when (*a*) he or she first felt pain and (*b*) when the pain became intolerable. Pain threshold was quantified as the time elapsed until participants indicated that they first felt pain. Pain tolerance was quantified as the time elapsed until participants pulled their hand out of the water, indicating that they could no longer withstand the pain. Other studies have measured pain endurance instead of pain tolerance (e.g., [19]). Pain endurance is calculated as the time elapsed between when the participant first indicates pain and when the participant indicates that the pain is too painful to tolerate (i.e., pain tolerance minus pain threshold). We note that results were very similar for analyses of the present data when pain endurance was used rather than pain threshold.

Elapsed time was measured with 2 stopwatches: one that was used to measure pain threshold and another that was used to measure pain tolerance (researchers later recorded the values on these stopwatches). As soon as the participants submerged their hands in the water, both timers began. At both the pain threshold and tolerance points, participants were instructed to announce the painfulness of the water on a scale of 1 to 10, with "1" indicating barely perceptible pain and "10" indicating the most intense pain imaginable. A researcher in an adjacent room (who could hear the participants) recorded these pain intensity values. Participants were allowed to pull their hand out of the water whenever they felt it necessary and were allowed to keep their hand in the water for a maximum of 2 minutes. We used this 2-minute limit to reduce outliers: pilot data indicated that participants rarely reached 2 minutes but that when they did, they would often continue indefinitely because of a numbed hand.

2.5. Data analytic plan

First, we examined correlations among pain variables, emotion dysregulation variables, and subjective units of distress within the total sample. We then examined these correlations separately within the NSSI and control groups. Because we hypothesized that these variables would be positively correlated, these analyses were 1-tailed. Second, we used analyses of variance to examine hypotheses that the NSSI group would display higher levels of emotion dysregulation, diminished pain perception, and increased subjective units of distress.

Third, we examined the ability of variables to account for variance within the NSSI pain perception association with a new statistical technique called a multiple mediation model [27]. Because of the cross-sectional nature of the data, it is important to note that these analyses tested whether specific variables accounted for variance within the NSSI pain perception association (i.e., statistical mediation) but cannot provide direct evidence for any temporal conclusions (i.e., theoretical mediation). We followed the recommendations, models, and methods described by Preacher and Hayes [27] to test mediation hypotheses with bootstrapping. We used bootstrapping because simulations comparing it with traditional mediation methods (e.g., causal steps method, the Sobel Test) show that bootstrapping is the most powerful, most effective for small sample sizes, and least vulnerable to type I error [27]. In addition, bootstrapping does not assume normal distributions for any variable—an assumption that is commonly violated when other methods are used, especially in smaller samples [27]. Essentially, bootstrapping is a nonparametric resampling procedure that calculates indirect effects (i.e., mediation effects) each time the data are resampled. After the data have been resampled thousands of times, an empirical approximation of the indirect effects can be calculated as a confidence interval, which can then be adjusted for bias [27]. Following recommendations, we resampled the data 5000 times [27]. Preacher and Hayes [27] described a new method for simultaneously testing multiple mediators. This technique allows for a test of the combined effects of all proposed mediators (i.e., the total indirect effect) and controls for collinearity among variables and mediation effects. This means that significant mediation effects are unique from one another. Beta weights provide an index of the magnitude of the indirect effect size.

We provide the model tested in the present study in Fig. 1. Similar to the basic idea of traditional mediation methods, "A" paths represent the association between NSSI group (i.e., NSSI or non-NSSI) and mediator variables. The "B" paths represent the association between mediator variables and the specific outcome variable (note that separate tests were conducted for pain threshold and pain tolerance) controlling for "A." The "C" path represents the total effect of NSSI on the specific outcome variable, and "C-" path represents "C" after controlling for indirect effects. Indirect effects (i.e., mediation effects) are defined as A × B. We tested these models using regression (to calculate statistics for specific paths) and bootstrapping (to generate a confidence interval for the mediation effects). These analyses yielded significance tests of specific paths and confidence intervals for mediation effects.

Pending significant NSSI group differences with pain variables, we conducted mediation analyses to determine the specific mediators between NSSI and each pain variable. For each analysis, we entered in affect dysregulation, subjective units of distress, and all other pain variables

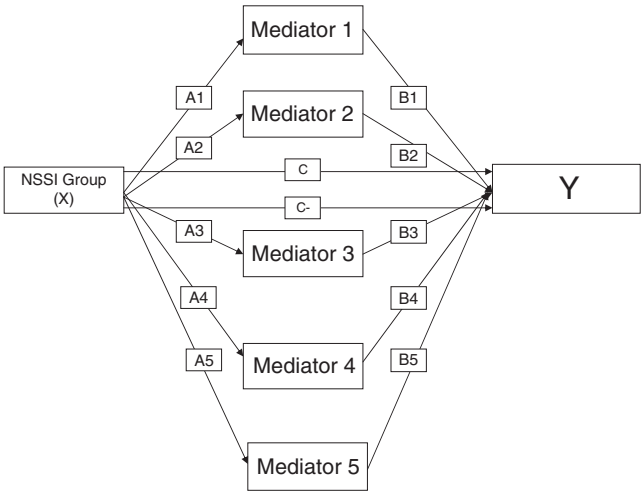

Fig. 1. General multiple mediation model for the effect of NSSI group on pain variables.

as potential mediators. If affect dysregulation was a significant mediator for a particular analysis (e.g., NSSI and pain tolerance), we then ran a second analysis on the association that included the 6 subscales of the Difficulties in Emotion Regulation Scale as potential mediators. The purpose of this was to determine if specific facets of emotion dysregulation accounted for variance within NSSI pain perception associations.

3. Results

3.1. Zero-order correlations

Across groups, all 4 pain perception variables were correlated with multiple emotion regulation variables (Table 1). Correlations between pain tolerance and emotion dysregulation variables were particularly strong; correlations with pain intensity ratings at threshold were relatively weak (Table 1). Taken together, these correlations provide direct evidence that emotion dysregulation is associated with diminished pain perception. These associations largely held when examined within the NSSI group, although reduced power decreased the number of correlations reaching significance (Table 2). In contrast to combined results, correlations between pain intensity at threshold and emotion dysregulation were strong and significant within the NSSI group (Table 2). In the control group, significant correlations between pain perception and emotion dysregulation were primarily confined to associations with pain tolerance (Table 3). These latter results demonstrate that the association between emotion dysregulation and diminished pain perception can exist independent of NSSI history. There were no consistent correlations between subjective units of distress and any other variables.

3.2. Group differences

As shown in Table 4, there were several significant group differences on pain and emotion dysregulation variables. These were large group differences for all pain variables (*d*s = .67-.81), with the exception of pain intensity ratings at the threshold point (*d* = .34). Specifically, the NSSI group displayed increased pain tolerance and threshold and decreased pain intensity ratings. Similarly, there were generally moderate to large group differences for emotion dysregulation variables (*d*s = .40-1.04; Table 4). These differences are consistent with previous findings showing that NSSI is associated with diminished pain perception and increased emotion dysregulation (e.g., [19,22,23]). Groups displayed nearly identical subjective units of distress before the cold pressor task (Table 4).

Table 1
Zero-order correlations between pain and emotion dysregulation variables across both groups

	1	2	3	4	5	6	7	8	9	10	11	12
1. Threshold	–											
2. Tolerance	0.55***	–										
3. Intensity at threshold	−0.28**	−0.49***	–									
4. Intensity at tolerance	−0.47**	−0.45***	0.72***	–								
5. SUDS	0.00	−0.04	0.18	0.16	–							
6. DERS—total score	0.32**	0.42**	−0.18	−0.20*	0.13	–						
7. DERS—nonacceptance	0.20	0.29**	−0.11	−0.16*	0.08	0.87***	–					
8. DERS—goals	0.23*	0.29**	0.04	−0.06	0.09	0.71***	0.58***	–				
9. DERS—impulse	0.14	0.18	0.00	−0.14	0.06	0.59***	0.56***	0.51***	–			
10. DERS—awareness	0.25*	0.31**	0.38***	−0.24*	0.05	0.27**	0.14	−0.17	0.12	–		
11. DERS—clarity	0.12	0.36**	−0.30**	−0.20*	0.30**	0.63***	0.48***	0.26*	0.32**	0.50***	–	
12. DERS—strategies	0.39***	0.40***	0.08	−0.22*	0.04	0.85***	0.71***	0.68***	0.59***	0.11	0.44***	–

SUDS indicates subjective units of distress assessed immediately after the stressful task and immediately before the cold pressor pain task; DERS, Difficulties in Emotion Regulation Scale.

* $P < .05$.

** $P < .01$.

*** $P < .001$.

Table 2
Zero-order correlations between pain and emotion dysregulation within the NSSI group

	1	2	3	4	5	6	7	8	9	10	11	12
1. Threshold	–											
2. Tolerance	0.57**	–										
3. Intensity at threshold	−0.40*	−0.57**	–									
4. Intensity at tolerance	−0.57**	−0.46*	0.72***	–								
5. SUDS	−0.04	−0.06	0.03	0.20	–							
6. DERS—total score	0.31	0.41*	−0.53**	−0.38*	0.01	–						
7. DERS—nonacceptance	0.24	0.21	−0.36*	−0.37*	−0.13	0.86***	–					
8. DERS—goals	0.28	0.31	−0.01	−0.01	0.00	0.50**	0.47***	–				
9. DERS—impulse	0.04	0.16	−0.41*	−0.20	−0.24	0.46**	0.56***	0.55***	–			
10. DERS—awareness	0.19	0.33	−0.51**	−0.33*	0.02	0.27**	0.07	−0.34*	0.20	–		
11. DERS—clarity	0.10	0.33	−0.61***	−0.37*	0.33	0.54**	0.35*	0.10	0.36*	0.60***	–	
12. DERS—strategies	0.31	0.27	−0.13	−0.16	−0.12	0.72***	0.71***	0.60***	0.52**	0.02	0.26	–

SUDS indicates subjective units of distress assessed immediately after the stressful task and immediately before the cold pressor pain task; DERS, Difficulties in Emotion Regulation Scale.

* $P < .05$.

** $P < .01$.

*** $P < .001$.

3.3. Mediation analyses

3.3.1. Pain threshold

Regression analyses showed that NSSI status (i.e., group) was significantly associated with pain tolerance, pain intensity at tolerance, and emotion dysregulation (i.e., "A" paths in Table 5). Analyses also indicated that pain tolerance and pain intensity at tolerance were significantly associated with pain threshold after controlling for "A" paths (i.e., "B" paths in Table 5). As shown in Table 5, the total effect of NSSI and proposed mediators on pain threshold was significant (i.e., the "C" path is Table 5). The direct effect of NSSI on pain threshold was not significant (i.e., the "C-" in Table 5), indicating that the combined effect of the proposed mediators reduced the magnitude of this association. Bootstrapping analyses confirmed this combined mediation effect (Table 6). These analyses further specified that pain tolerance and pain intensity at tolerance were the only significant, unique mediators of the association between NSSI and pain threshold (Table 6).

3.3.2. Pain tolerance

Regression analyses revealed that NSSI status was significantly associated with pain threshold, pain intensity at tolerance, and emotion dysregulation (Table 5). These analyses also showed that pain threshold, pain intensity at threshold, and emotion dysregulation were significantly associated with pain tolerance after controlling for "A" paths (Table 5). The total effect of NSSI and the proposed mediators on pain tolerance was significant, but the direct effect of NSSI on pain tolerance was not significant after controlling for proposed mediators (Table 5). As shown in Table 6, bootstrapping analyses confirmed this combined mediation effect. These analyses also specified that pain threshold and emotion dysregulation were significant

Table 3
Zero-order correlations between pain and emotion dysregulation variables within the control group

	1	2	3	4	5	6	7	8	9	10	11	12
1. Threshold	–											
2. Tolerance	0.36**	–										
3. Intensity at threshold	−0.07	−0.41**	–									
4. Intensity at tolerance	−0.13	−0.31*	0.72***	–								
5. SUDS	0.04	−0.02	0.28*	0.14	–							
6. DERS—total score	0.15	0.31*	0.08	0.14	0.22	–						
7. DERS—nonacceptance	0.02	0.26*	0.08	−0.15	0.25*	0.87***	–					
8. DERS—goals	0.08	0.18	0.11	0.09	0.09	0.76***	0.59***	–				
9. DERS—impulse	0.03	−0.03	0.29*	0.21	0.21	0.63***	0.50***	0.45***	–			
10. DERS—awareness	0.28*	0.19	−0.24	−0.04	0.09	0.20	0.11	−0.18	0.12	–		
11. DERS—clarity	−0.02	0.31*	−0.06	0.13	0.30*	0.64***	0.51***	0.28*	0.18	0.37**	–	
12. DERS—strategies	0.28*	0.34**	0.05	0.04	0.19	0.90***	0.68***	0.70***	0.55***	0.07	0.49***	–

SUDS indicates subjective units of distress assessed immediately after the stressful task and immediately before the cold pressor pain task; DERS, Difficulties in Emotion Regulation Scale.

* $P < .05$.

** $P < .01$.

*** $P < .001$.

Table 4
Group differences in pain perception, emotion dysregulation, and subjective units of distress

Variable	Control group mean (SD)	NSSI group mean (SD)	*F*	Cohen's *d*
Pain threshold	8.59 (4.51)	15.64 (12.89)	11.39***	0.81
Pain tolerance	34.94 (22.26)	54.13 (35.45)	7.81**	0.67
Pain intensity at threshold	5.26 (1.59)	4.67 (1.76)	2.00	0.34
Pain intensity at tolerance	8.32 (1.37)	7.04 (1.81)	11.32***	0.81
SUDS	40.83 (18.98)	40.36 (21.95)	0.01	0.02
DERS—total score	72.73 (17.68)	90.54 (20.88)	14.24***	0.91
DERS—nonacceptance	12.68 (5.63)	15.92 (6.03)	5.00*	0.54
DERS—goals	14.49 (4.45)	16.88 (3.77)	5.06*	0.54
DERS—impulse	8.47 (2.88)	11.17 (4.86)	8.61**	0.71
DERS—awareness	11.25 (2.48)	12.50 (3.83)	2.74	0.40
DERS—clarity	10.53 (3.14)	12.21 (3.80)	3.89*	0.47
DERS—strategies	15.34 (5.67)	22.04 (7.33)	18.11***	1.02

SUDS indicates subjective units of distress assessed immediately after the stressful task and immediately before the cold pressor pain task; DERS, Difficulties in Emotion Regulation Scale.
* $P < .05$.
** $P < .01$.
*** $P < .001$.

unique mediators of the association between NSSI and pain tolerance.

3.3.3. Pain intensity at threshold

Because the association between NSSI status and pain intensity at threshold was not significant, we did not conduct mediation analyses for this association.

3.3.4. Pain intensity at tolerance

Regression analyses showed that NSSI status was significantly associated with pain threshold, pain tolerance, and emotion dysregulation (Table 7). After controlling for "A" paths, pain threshold and pain intensity at threshold were significantly associated with pain intensity at tolerance. Total and direct effects were both significant, indicating that the combined effect of the proposed mediators was small. Bootstrapping analyses were consistent with these findings, indicating that the combined effect of the mediators was not significant (Table 6). However, bootstrapping analyses did reveal that pain threshold was a significant mediator of the association between NSSI and pain intensity at tolerance. Specific mediators can be significant even when the combined effect of several mediators is not [27]. This indicates that other proposed mediators diluted the effect of pain threshold within the combined analysis.

3.3.5. Pain tolerance and facets of emotion dysregulation

Emotion dysregulation only significantly mediated the association between NSSI and pain tolerance. Therefore, we only conducted follow-up analyses on specific facets of emotion dysregulation for the association between NSSI and pain tolerance. Regression analyses indicated that NSSI status was significantly associated with the nonacceptance, goals, impulse, and strategy subscales of the Difficulties in Emotion Regulation Scale (Table 7). No subscales were significantly associated with pain tolerance after controlling for "A" paths. The total effect was significant, but the direct effect was not. This suggested that the combined effect of the subscales had an effect on the association between NSSI and pain tolerance. Bootstrapping analyses not only confirmed this combined mediation effect (Table 6) but also showed that no subscales were significant mediators of the association between NSSI and pain tolerance.

Table 5
Regression results for paths in the multiple mediation model for pain threshold and tolerance

Path	Beta (SE)	*t*
Pain threshold		
NSSI to mediators ("A" paths)		
Pain tolerance	18.61 (7.00)	2.66**
Pain intensity at threshold	−0.52 (0.42)	−1.22
Pain intensity at tolerance	−1.15 (0.39)	−2.94**
Subjective units of distress	0.21 (5.13)	0.04
Emotion dysregulation	15.18 (4.40)	3.45***
Mediators to pain threshold ("B" paths)		
Pain tolerance	0.13 (0.04)	3.48***
Pain intensity at threshold	1.29 (0.77)	1.67
Pain intensity at tolerance	−2.20 (0.80)	−2.76**
Subjective units of distress	0.01 (0.04)	0.14
Emotion dysregulation	0.04 (0.05)	0.63
Effect of NSSI on pain threshold ("C" paths)		
Total effect (C)	7.06 (2.09)	3.37***
Direct effect (C′)	2.28 (2.07)	1.10
Pain tolerance		
NSSI to mediators ("A" paths)		
Pain threshold	7.06 (2.09)	3.37**
Pain intensity at threshold	−0.52 (0.42)	−1.22
Pain intensity at tolerance	1.15 (.40)	2.94**
Subjective units of distress	0.21 (5.13)	0.04
Emotion dysregulation	15.18 (4.40)	3.45***
Mediators to pain tolerance ("B" paths)		
Pain threshold	1.25 (.36)	3.48***
Pain intensity at threshold	−6.64 (2.32)	−2.86**
Pain intensity at tolerance	1.14 (2.62)	0.44
Subjective units of distress	0.01 (0.13)	0.02
Emotion dysregulation	0.33 (0.16)	2.07*
Effect of NSSI on pain tolerance ("C" paths)		
Total effect (C)	18.61 (7.00)	2.66**
Direct effect (C′)	2.64 (6.50)	0.41

Emotion dysregulation was quantified as the total score of the Difficulties in Emotion Regulation Scale.
* $P < .05$.
** $P < .01$.
*** $P < .001$.

4. Discussion

In the present study, we investigated emotion dysregulation as a potential partial explanation for why people with a history of NSSI display diminished pain perception. Results showed that emotion dysregulation is generally associated with diminished pain perception, even in people without a

Table 6
Bootstrapping of indirect effects in the multiple mediation model

Proposed mediators (A × B paths)	Beta (SE)	BCa 95% confidence interval lower	upper
Pain threshold			
Pain tolerance	2.40 (1.61)	0.40	7.45[a]
Pain intensity at threshold	−0.67 (0.83)	−3.54	0.21
Pain intensity at tolerance	2.52 (1.62)	0.52	8.00[a]
Subjective units of distress	0.01 (0.24)	−0.61	0.45
Emotion dysregulation	0.51 (0.81)	−0.87	2.52
Combined effect	4.77 (2.39)	1.32	11.52[a]
Pain tolerance			
Pain threshold	8.80 (4.67)	2.13	21.55[a]
Pain intensity at threshold	3.43 (3.60)	−1.54	13.37
Pain intensity at tolerance	−1.31 (4.34)	−12.64	6.16
Subjective units of distress	0.00 (0.72)	−1.17	1.91
Emotion dysregulation	5.06 (3.03)	0.65	13.77[a]
Combined effect	15.97 (6.88)	4.07	31.34[a]
Pain intensity at tolerance			
Pain threshold	−0.35 (0.19)	−0.92	−0.08[a]
Pain tolerance	0.05 (0.16)	−0.26	0.40
Pain intensity at threshold	−0.32 (0.29)	−0.95	0.18
Subjective units of distress	0.00 (0.04)	−0.10	0.08
Emotion dysregulation	0.09 (0.11)	−0.09	0.38
Combined effect	−0.53 (0.36)	−1.32	0.12
Facets of emotion dysregulation for pain tolerance analyses			
Nonacceptance	−0.46 (2.72)	−7.36	4.36
Goals	3.67 (3.22)	−0.51	14.32
Impulse	−3.18 (3.29)	−11.92	1.81
Awareness	3.02 (2.54)	−0.19	11.69
Clarity	1.41 (2.60)	−2.31	8.63
Strategies	6.29 (6.10)	−4.69	19.82
Combined effect	10.75 (4.97)	2.73	23.28[a]

BCa indicates bias corrected and accelerated, as recommended by Preacher and Hayes (2008) to reduce bias in bootstrapping analyses. Data were resampled 5000 times, as recommended by Preacher and Hays (2008). Facets of emotion dysregulation were quantified as the 6 subscales of the Difficulties in Emotion Regulation Scale.

[a] 95% confidence interval did not include 0, indicating that this effect is significantly different from 0.

Table 7
Regression results for paths in the multiple mediation model for pain intensity at tolerance and facets of emotion dysregulation for pain tolerance analyses

Path	Beta (SE)	*t*
Pain intensity at tolerance		
NSSI to mediators ("A" paths)		
Pain threshold	7.06 (2.09)	3.37**
Pain tolerance	18.61 (7.00)	2.66**
Pain intensity at threshold	−0.52 (0.42)	1.22
Subjective units of distress	0.21 (5.13)	0.04
Emotion dysregulation	15.18 (4.40)	3.45***
Mediators to pain threshold ("B" paths)		
Pain threshold	−0.05 (0.02)	−2.76**
Pain tolerance	0.01 (0.01)	0.44
Pain intensity at threshold	0.62 (0.09)	7.03***
Subjective units of distress	0.01 (0.01)	0.33
Emotion dysregulation	0.01 (0.01)	0.73
Effect of NSSI on pain threshold ("C" paths)		
Total effect (C)	−1.15 (0.39)	−2.94**
Direct effect (C′)	−0.62 (0.30)	−2.03*
Facets of emotion dysregulation for pain tolerance analyses		
NSSI to mediators ("A" paths)		
Nonacceptance	3.13 (1.49)	2.10*
Goals	2.54 (1.07)	2.34*
Impulse	2.76 (0.95)	2.92**
Awareness	1.26 (0.77)	1.63
Clarity	1.55 (0.86)	1.80
Strategy	6.80 (1.62)	4.19***
Mediators to pain tolerance ("B" paths)		
Nonacceptance	−0.15 (0.81)	−0.18
Goals	1.44 (1.10)	1.31
Impulse	−1.15 (1.04)	−1.10
Awareness	2.39 (1.30)	1.85
Clarity	0.91 (1.22)	0.75
Strategy	0.92 (0.79)	1.17
Effect of NSSI on pain tolerance ("C" paths)		
Total effect (C)	20.45 (7.03)	2.91*
Direct effect (C′)	9.69 (7.63)	1.27

Facets of emotion dysregulation were quantified as the 6 subscales of the Difficulties in Emotion Regulation Scale.

* $P < .05$.

** $P < .01$.

*** $P < .001$.

history of NSSI. Analyses specified that emotion dysregulation partially accounted for group differences in pain tolerance but did not explain group differences in pain threshold or pain intensity ratings. These effects appeared to be due to trait emotion dysregulation because subjective units of distress assessed immediately before pain induction were not consistently correlated with pain perception or emotion dysregulation. Later, we discuss these findings in greater detail.

Although several lines of indirect evidence suggest that emotion dysregulation is associated with diminished pain perception (e.g., [20]), to our knowledge this is the first direct demonstration of this association (Table 1). No specific facet of emotion dysregulation appeared to be primarily responsible for this effect; rather, it appeared that emotion dysregulation in general was associated with diminished pain perception. This effect existed across all pain perception variables, although it was strongest for pain tolerance and weakest for pain intensity ratings at the threshold point (Table 1). Within-group analyses showed that the association between emotion dysregulation and diminished pain perception existed in the NSSI group (Table 3). This provides further indirect support for the hypothesis that people with a history of NSSI may show diminished pain perception in part because of emotion dysregulation (with statistical mediation analyses providing more direct support, see discussion about this later). It should be noted that this result is also consistent with the alternative hypothesis that experience with NSSI increases emotion dysregulation and decreases pain perception. In this scenario, emotion dysregulation would be incidentally associated with diminished pain perception due to the third variable, NSSI. However, ruling out this alternative hypothesis as the complete explanation for our findings, emotion dysregulation was also significantly associated with diminished pain

perception (particularly pain tolerance) in the control group (Table 2). This demonstrates that this association can exist independently of NSSI and, as such, may represent a risk factor for NSSI (although it should be noted that this risk factor hypothesis could not be directly tested in the present cross-sectional design).

As expected, the NSSI group displayed significantly higher emotion dysregulation, pain threshold, and pain tolerance and significantly lower pain intensity ratings than the control group (Table 4). These findings are consistent with studies showing that NSSI is associated with increased pain threshold and endurance (e.g., [19]), decreased pain intensity ratings (e.g., [17]), and increased emotion dysregulation (e.g., [22,23]). We conducted mediation analyses to investigate whether emotion dysregulation partially accounts for group differences in pain perception. Other pain variables and subjective units of distress immediately before the cold pressor task were entered as competing mediators. These analyses showed that subjective units of distress did not account for significant variance within any analysis but that at least one pain variable contributed significant variance in each analysis (Table 6). Results specified that emotion dysregulation accounted for significant variance in the association between NSSI and pain tolerance but not pain threshold or pain intensity ratings (Table 6). In follow-up analyses, we found that no specific facet of emotion dysregulation accounted for significant variance in the association between NSSI and pain tolerance (Table 6). The combined effect of all of these facets, however, did account for significant variance in this association. Taken together, these analyses represent the first evidence that emotion dysregulation plays a direct role in the association between NSSI and diminished pain perception.

The finding that emotion dysregulation only accounts for significant variance in the association between NSSI and pain tolerance—but not pain threshold or pain intensity ratings—may help to clarify the role of emotion regulation in NSSI. This indicates that although NSSI is associated with increased pain threshold and decreased pain intensity ratings, emotion dysregulation appears to only contribute to pain tolerance. In other words, emotion dysregulation does not appear to play a prominent role in the altered sensory thresholds and subjective judgments about pain intensity that have been observed in NSSI. Instead, these results are consistent with the hypothesis that emotion dysregulation only makes people with a history of NSSI more able to tolerate pain. This is consistent with the preliminary findings and interpretations of Hooley and colleagues [19], leading to the hypothesis that emotion dysregulation may make people more accepting or desirous of pain. However, we emphasize that the present study was unable to directly test this intriguing motivational hypothesis. The present findings build on previous studies by examining this question with a measure of emotion dysregulation, using multiple mediation model analyses, providing evidence that distress levels after a stressful task do not explain this effect, and further specifying that this effect does not exist for pain threshold or pain intensity ratings.

These results should be interpreted in light of the limitations of the present study. First, the present study was cross-sectional, precluding any temporal conclusions about NSSI. The finding that emotion dysregulation was associated with decreased pain perception in the control group suggests that this effect may be a risk factor for NSSI, but longitudinal studies are required to draw such conclusions. Second, although the present study considered multiple contributors to variance within the NSSI pain association, many other viable potential contributors exist. For example, self-punishment beliefs [19], NSSI experience [19], a history of painful and provocative events [6], and many others may also play an important role in this association. In addition, although the measure of emotion dysregulation in the present study is multidimensional, future studies may benefit from exploring this association with other measures relevant to emotion dysregulation and NSSI, such as the Emotion Reactivity Scale [31].

Third, there are necessary limitations to laboratory replications of the stress and pain involved in NSSI. In addition, we only used one form of pain induction. Of note, the present results are similar to studies that have used other forms of pain induction (e.g., pressure pain, heat pain; [10,19,26]); nevertheless, it is unclear how well cold pain, heat pain, or pressure pain approximate cutting, burning, scraping, or other NSSI behaviors. Fourth, we used a college sample with relatively low levels of emotion dysregulation. Although the present results should generalize to other nonclinical populations (which have surprisingly high rates of NSSI; [2]), these results may not generalize to clinical populations. Interestingly, some of the associations between emotion dysregulation and pain perception held in the control group, which had very low levels of emotion dysregulation. This suggests that this association may be robust across populations.

Finally, stress was induced before the cold pressor task for all participants. This may have provided a closer approximation of the actual experience of NSSI because acute stress almost always precedes NSSI [7]. One previous study found that the association between NSSI pain tolerance was strongest after acute stress induction [26], though other studies have found such association under nonstress conditions (e.g., [19]). In any case, the present results may not generalize well to studies that do not induce acute stress before pain induction.

In sum, the present study demonstrated that emotion dysregulation is generally associated with diminished pain perception and that emotion dysregulation accounts for significant variance in the association between NSSI and pain tolerance but not other pain variables. In conjunction with Hooley and colleagues [19], this leads to the hypothesis that emotion dysregulation may increase the willingness to endure pain via the belief that pain or punishment is deserved. Future studies should test this hypothesis and

continue to investigate how and why people who engage in NSSI are able to overcome the instinct to avoid pain.

References

[1] Nock MK. Self-injury. Annu Rev Clin Psychol 2010;6:339-63.
[2] Jacobson CM, Gould M. The epidemiology and phenomenology of non-suicidal self-injurious behavior among adolescents: a critical review of the literature. Arch Suicide Res 2007;11:129-47.
[3] Darche MA. Psychological factors differentiating self-mutilating and non-self mutilating adolescent inpatient females. Psychiatric Hospital 1990;21:31-5.
[4] DiClemente RJ, Ponton LE, Hartley D. Prevalence and correlates of cutting behavior: risk for HIV transmission. J Am Acad Child Adolesc Psychiatry 1991;30:735-9.
[5] Andover MS, Gibb BE. Non-suicidal self-injury, attempted suicide, and suicidal intent among psychiatric inpatients. Psychiatry Res 2010; 178:101-5.
[6] Joiner T. Why people die by suicide. Cambridge (Mass): Harvard University Press; 2005.
[7] Klonsky ED. The functions of deliberate self-injury: a review of the evidence. Clin Psychol Rev 2007;27:226-39.
[8] Franklin JC, Hessel ET, Aaron RV, Arthur MS, Heilbron N, Prinstein MJ. The functions of nonsuicidal self-injury: support for cognitive–affective regulation and opponent processes from a novel psychophysiological paradigm. J Abnorm Psychol 2010;119:850-62.
[9] Niedtfeld I, Schulze L, Kirsch P, Herpertz SC, Bohus M, Schmahl C. Affect regulation and pain in borderline personality disorder: a possible link to the understanding of self-injury. Biol Psychiatry 2010;68: 383-91.
[10] Schmahl C, Bohus M, Esposito F, Treede R, Di Salle F, Greffrath W, et al. Neural correlates of antinociception in borderline personality disorder. Arch Gen Psychiatry 2006;63:659-67.
[11] Becerra L, Borsook D. Signal valence in the nucleus accumbens to pain onset and offset. Eur J Pain 2008;12:866-9.
[12] Leknes S, Brooks JW, Wiech K, Tracey I. Pain relief as an opponent process: a psychophysical investigation. Eur J Neurosci 2008;28: 794-801.
[13] Andreatta M, Muhlberger A, Yarali A, Gerber B, Pauli P. A rift between implicit and explicit conditioned valence in human pain relief learning. Proc Biol Sci 2010;277:2411-6.
[14] Bresin K, Gordon KH, Bender TW, Gordon LJ, Joiner T. No pain, no change: reductions in prior negative affect following physical pain. Motiv Emot 2010;34:280-7.
[15] Grill JD, Coghill RC. Transient analgesia evoked by noxious stimulus offset. J Neurophysiol 2002;87:2205-8.
[16] International Association for the Study of Pain Task Force on Taxonomy. Classification of chronic pain: description of chronic pain syndromes and definition of pain terms. 2nd ed. Seattle, WA, USA: IASP Press; 1994.
[17] Bohus M, Limberger M, Ebner U, Glocker FX, Schwarz B, Wernz M, et al. Pain perception during self-reported distress and calmness in patients with borderline personality disorder and self-mutilating behavior. Psychiatry Res 2000;95:251-60.
[18] Russ MJ, Roth SD, Lerman A, Kakuma T, Harrison K, Shindledecker RD, Hull J, et al. Pain perception in self-injurious patients with borderline personality disorder. Biol Psychiatry 1992;32:501-11.
[19] Hooley JM, Ho DT, Slater J, Lockshin A. Pain perception and nonsuicidal self-injury: a laboratory investigation. Personal Disord Theor Res Treat 2010;1:170-9.
[20] Klossika I, Flor H, Kamping S, Bleichhardt G, Trautmann N, Treede R, et al. Emotional modulation of pain: a clinical perspective. Pain 2006;124:264-8.
[21] Nock MK, Joiner TE, Gordon KH, Lloyd-Richardson E, Prinstein MJ. Non-suicidal self-injury among adolescents: diagnostic correlates and relation to suicide attempts. Psychiatry Res 2006;144:65-72.
[22] Adrian M, Zeman J, Erdley C, Lisa L, Sim L. Emotional dysregulation and interpersonal difficulties as risk factors for nonsuicidal self-injury in adolescent girls. J Abnorm Child Psychol 2011;39:389-400.
[23] Gratz KL, Roemer L. The relationship between emotion dysregulation and deliberate self-harm among female undergraduate students at an urban commuter university. Cogn Behav Ther 2008;37:14-25.
[24] Clarkin JF, Hull JW, Cantor J, Sanderson C. Borderline personality disorder and personality traits: a comparison of SCID-II BPD and NEO-PI. Psychol Assess 1993;5:472-6.
[25] Anestis MD, Bagge CL, Tull MT, Joiner T. Clarifying the role of emotion dysregulation in the interpersonal-psychological theory of suicidal behavior in an undergraduate sample. J Psychiatr Res 2011;45: 603-11.
[26] Gratz KL, Hepworth C, Tull MT, Paulson A, Clarke S, Remington B, et al. An experimental investigation of emotional willingness and physical pain tolerance in deliberate self-harm: the moderating role of interpersonal distress. Compr Psychiatry 2011;52:63-74.
[27] Preacher KJ, Hayes AF. Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. Behav Res Methods 2008;40:879-91.
[28] Gratz KL, Roemer L. Multidimensional assessment of emotion regulation and dysregulation: development, factor structure, and initial validation of the difficulties in emotion regulation scale. J Psychopathol and Behav Assess 2004;26:41-54.
[29] Lloyd EE, Kelley ML, Hope T. Self-mutilation in a community sample of adolescents: descriptive characteristics and provisional prevalence rates. Present at the meeting of the Society for Behavioral Medicine, New Orleans, LA; 1997.
[30] Klatzkin RR, Mechlin B, Girdler SS. Menstrual cycle phase does not influence gender differences in experimental pain sensitivity. Eur J Pain 2010;14:77-82.
[31] Nock MK, Wedig MM, Holmberg EB, Hooley JM. The emotion reactivity scale: development, evaluation, and relation to self-injurious thoughts and behaviors. Behav Therapy 2008;39:107-16.