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The Nature of Pain Offset Relief in Nonsuicidal Self-Injury: A Laboratory Study

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Abstract

The majority of people who engage in nonsuicidal self-injury (e.g., cutting) report that affect regulation is their primary motivation for these painful behaviors. Unfortunately, little is known about the mechanisms that regulate affect during nonsuicidal self-injury. In the present study, we examined the role of one potential mechanism known as pain offset relief. In 42 participants (21 self-cutters, 21 controls), we measured psychophysiological indices of positive affect (startle postauricular reactivity) and negative affect (startle eyeblink reactivity) after painful electric shocks. Results provided evidence that pain offset relief is a natural and unique mechanism of affect regulation that generates simultaneous—but independent—positive and negative reinforcement. However, associations between nonsuicidal self-injury frequency and pain offset relief variables were weak and nonsignificant. Contrary to contemporary theories, this suggests that the strong association between prior and future self-injury may not be driven by opponent processes that generate stronger relief (and, thus, stronger reinforcement of self-injury) across episodes.

Keywords

nonsuicidal self-injury, pain offset relief, affect regulation, psychophysiological indices

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Nonsuicidal self-injury (NSSI) is the intentional destruction of one's own body tissue in the absence of suicidal intent (e.g., cutting or burning; Nock, 2010). These behaviors are surprisingly prevalent, with studies reporting rates ranging from 6% in adult community samples (Klonsky, 2011) to 60% in adolescent clinical samples (DiClemente, Ponton, & Hartley, 1991; Nock & Prinstein, 2004). Although NSSI traditionally has been noted as a symptom of borderline personality disorder, recent empirical evidence suggests that it merits classification as its own disorder (Selby, Bender, Gordon, Nock, & Joiner, 2012).

Over the last decade, there have been large advances in the understanding of NSSI (see Nock, 2010). One particularly important advance is the knowledge that affect regulation is the primary reason that most people engage in NSSI (Klonsky, 2007; Nock & Prinstein, 2004). This insight has inspired models of NSSI based on potential affect regulation mechanisms, such as experiential avoidance and distraction from rumination-driven emotional cascades (Chapman, Gratz, & Brown, 2006; Selby & Joiner, 2009). Unfortunately, these models have not yet produced effective treatments for NSSI; indeed, despite attempts with a wide range of therapies, at present there are no empirically supported treatments for NSSI (see Nock, 2010). This suggests that other mechanisms may play a prominent role in driving affect regulation in NSSI, and it emphasizes the need for a greater knowledge about these mechanisms. A clearer understanding of how NSSI produces affect regulation would facilitate a clearer framework for establishing the first effective treatments for NSSI. One of the major goals of the present study was to investigate the role of a promising potential mechanism of affect regulation in NSSI known as *pain offset relief*.

A second recent advance concerns the link between NSSI and suicidal behavior. Not only does NSSI predict suicidal behavior (Wilkinson, Kelvin, Roberts, Dubicka, & Goodyer, 2011), but it appears to be a stronger predictor than prior suicidal behavior (Asarnow et al., 2011; Guan, Fox, & Prinstein, 2012). However, it remains unclear how NSSI increases risk for suicidal behavior. A second major goal of the present study

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was to test the hypothesis that this link is explained in part by opponent processes that heighten relief across repeated NSSI episodes (Heilbron, Franklin, Guerry, & Prinstein, in press; Franklin et al., 2010; Joiner, 2005; Joiner, Ribeiro, & Silva, 2012). Greater knowledge about this link may help to generate intervention targets that reduce suicidal behavior.

In the majority of cases, NSSI is directly preceded by acute emotional stress (Klonsky, 2007). This stress activates several brain areas, most notably the anterior cingulate cortex and the anterior insula (e.g., Eisenberger, 2012). These two areas primarily function to sense and mentally represent visceral activity (Craig, 2002, 2009). Because visceral activity is a core ingredient in the psychological construction of affect (Barrett & Bliss-Moreau, 2009), these two areas play a central role in many affective experiences (Lindquist, Wager, Kober, Bliss-Moreau, & Barrett, 2012), including both emotional and physical pain (Eisenberger, 2012). There are several ways to alter stress-induced activity in these two brain areas (e.g., social support, reappraisal, mindfulness), but many of these strategies require relatively high levels of effort, time, or skill. A comparatively simple method is to commandeer this emotional pain activity with physical pain activity. Because of the high degree of overlap between physical and emotional pain (cf. Eisenberger, 2012), the offset (i.e., removal) of one form of pain incidentally offsets the other form of pain. In other words, physical pain relief incidentally generates emotional pain relief. Emotional pain often is difficult to control and terminate (see Selby & Joiner, 2009), but the self-administration of pain (e.g., cutting) is relatively easy to control and terminate. Accordingly, many individuals may find that the physical pain involved in NSSI is worth the emotional relief that physical pain offset generates. This pain offset relief may be one of the primary mechanisms of affect regulation in NSSI.

Pain-induced affect regulation seems counterintuitive, leading many to assume that this is one of the most abnormal or pathological features of people who engage in NSSI. Surprisingly, however, research has shown that it is in fact normal for pain offset to have a beneficial effect on affect (Andreatta, Muhlberger, Yarali, Gerber, & Pauli, 2010; Bresin, Gordon, Bender, Gordon, & Joiner, 2010; Franklin et al., 2010; Franklin, Lee, Hanna, & Prinstein, in press; Leknes, Brooks, Wiech, & Tracey, 2008). Moreover, pain offset relief may be a fundamental characteristic of the nervous system, given that it has been observed in a wide range of paradigms and species (e.g., Leknes et al., 2008; Smith & Buchanan, 1954; Tanimoto, Heisenberg, & Gerber, 2004; Zanna, Kiesler, & Pilkonis, 1970). For example, Leknes et al. (2008) found that, compared to baseline, the removal of a painful heat stimulus diminished physiological arousal and increased self-reported relief in humans. Similarly, Tanimoto and colleagues (2004) found that fruitflies avoided odors that had been paired with shock onset but approached odors that had been paired with shock offset. These lines of evidence suggest that pain offset relief is a normal phenomenon that some people—either incidentally or intentionally—access with behaviors such as self-cutting.

In the present study, we investigated the nature of pain offset relief in people with a history of NSSI. Four previous NSSI studies measured negative affect after pain offset (Bresin & Gordon, in press; Franklin et al., 2010; Russ et al., 1992; Weinberg & Klonsky, 2012); however, it was impossible to determine whether pain offset relief was the mechanism of negative affect reduction in these studies, because both distraction and pain offset relief should diminish negative affect. A unique feature of pain offset relief is that it simultaneously stimulates positive affect and diminishes negative affect (Franklin et al., in press). Accordingly, to experimentally test for a pain offset relief mechanism, we examined the effects of pain offset on psychophysiological measures of negative (startle eyeblink reactivity) and positive affective valence (startle postauricular reactivity). Importantly, these two measures are not strongly correlated (e.g., $r = -.07$: Franklin et al., in press; $r = -.11$: Sandt, Sloan, & Johnson, 2009), meaning that any increase in positive affect will not be an artifact of diminished negative affect. We hypothesized that pain offset would generate both diminished negative affect and increased positive affect in people with a history of NSSI, thereby supporting the existence of pain offset relief as a mechanism of affect regulation in NSSI.

This test also has the potential to clarify a controversial issue in NSSI research: the existence of a positive reinforcement mechanism in NSSI. Although many have proposed that NSSI primarily serves to reduce negative affect (e.g., Chapman et al., 2006; Klonsky, 2007; Victor, Glenn, & Klonsky, 2012), several self-report studies have found moderate evidence for a positive reinforcement mechanism (e.g., Muehlenkamp et al. 2009; Nock & Prinstein, 2004). Given certain theories of affect (e.g., Watson & Tellegen, 1985), however, some have proposed that these findings are artifacts of diminished negative affect (Klonsky, 2009). Unfortunately, previous laboratory NSSI studies have not been capable of testing for an independent positive affect mechanism; the present study has the potential to resolve this issue. Evidence for this mechanism would suggest the need to modify existing NSSI models and treatments, which are based on the assumption that NSSI primarily functions to reduce negative affect (e.g., Chapman et al., 2006; Selby & Joiner, 2009).

As noted, NSSI is a powerful longitudinal predictor of future suicidal behavior. Recent theoretical and empirical work (Franklin et al., 2010; Heilbron et al., in press; Joiner, 2005; Joiner et al., 2012) has drawn on the opponent process theory of acquired motivation (Solomon, 1980) to explain this link. Specifically, the opponent process theory posits that there are two reactions to every stimulus: a primary process and an opponent process. Across repeated stimulus presentations, this theory posits that the opponent process will become more powerful whereas the primary process will diminish. For NSSI, the primary processes are pain and fear of self-injury, and the opponent process is relief. Thus, across repeated NSSI episodes, self-injury (including suicidal self-injury) should become less painful, less aversive, and more emotionally

relieving. Partially supporting the habituation aspect of this theory, one laboratory study found that NSSI experience (i.e., number of years that an individual had engaged in NSSI) was positively correlated with diminished pain threshold (Hooley, Ho, Slater, & Lockshin, 2010). Similarly, preliminary evidence indicates that NSSI frequency (i.e., number of lifetime NSSI episodes) is positively correlated with diminished negative affective reactions to self-injury stimuli (Franklin, Puzia, et al., 2012; Glenn & Klonsky, 2010). These findings are consistent with the opponent process theory, but they also are consistent with the explanation that individuals simply habituate to the fear and pain involved in self-injury over time. To more definitively support the opponent process explanation, it should be shown that the opponent process (i.e., relief) intensifies across repeated NSSI episodes.

The present study provided a laboratory investigation of this hypothesis by examining the association between NSSI frequency and pain offset relief. Consistent with a role for opponent processes, we hypothesized that NSSI frequency would be positively correlated with heightened pain offset relief.

Methods

Participants

There were 42 total participants in the present study. There were 21 participants (11 males, 10 females) in the control group, with an average age of 19.29 (SD = 1.19) and a self-identified ethnic composition of 71.40% Caucasian Americans, 9.50% African Americans, 9.50% Asian Americans, 4.80% Hispanic Americans, and 4.80% others. These participants were recruited from introductory psychology classes and participated to partially fulfill a class research option. All participants in this group denied any lifetime history of self-injury. Although the present protocol is similar to that of Franklin et al. (in press), we note that participants in the two studies are completely independent.

There were 21 participants (7 males, 14 females) in the NSSI group, with an average age of 24.43 (SD = 7.95) and a self-identified ethnic composition of 76.20% Caucasian Americans, 4.80% African Americans, 14.30% Asian Americans, and 4.80% Hispanic Americans. These participants were recruited from campuswide e-mail and flyer advertisements and paid $35 for participation. Participants were included in this group if they had at least one lifetime episode of self-cutting that qualified as NSSI (as assessed by the Self-Injurious Thoughts and Behaviors Interview). The average number of lifetime self-cutting episodes was 231.76 (SD = 650.98), with a range of 1 to 3,000 and a median of 30. Participants reported an average of 2.24 NSSI methods (SD = 1.22). In addition to self-cutting, some participants scraped (42.86%), burned (38.09%), hit (33.33%), and inserted objects under their skin (19.05%). As assessed by the Mini International Neuropsychiatric Interview, some participants in this group qualified for major depressive disorder (38.09%), dysthymia (4.76%), panic disorder (28.57%), social phobia (9.52%), posttraumatic stress disorder (23.81%), alcohol dependence (19.05%), alcohol abuse (14.29%), substance abuse (9.52%), bulimia nervosa (4.76%), and generalized anxiety disorder (19.05%).

There were no significant associations between pain offset relief and gender, ethnicity, or psychiatric diagnoses ($F < 1.0$).

Self-report measures

Emotion Reactivity Scale. The Emotion Reactivity Scale (ERS; Nock, Wedig, Holmberg, & Hooley, 2008) measures emotion reactivity, a component of emotion regulation that specifically involves emotional sensitivity, intensity, and persistence. The ERS is a single-factor 21-item self-report questionnaire that displays strong internal consistency, convergent and divergent construct validity, and criterion-related validity (Nock et al., 2008). People with a history of NSSI (M = 43.30, SD = 16.00) have been shown to display higher ERS scores than control participants (M = 27.70, SD = 15.30), and ERS scores have been shown to mediate the association between psychopathology and self-injury (Nock et al., 2008).

Difficulties in Emotion Regulation Scale. The Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) is a 41-item self-report questionnaire about emotion regulation (and dysregulation), with higher scores indicating poorer regulation. Gratz and Roemer (2008) found that total DERS scores were significantly higher in people with a history of NSSI (M = 87.44, SD = 19.87) compared to people without a history of NSSI (M = 76.14, SD = 20.86). The DERS contains six subscales: Nonacceptance (e.g., "When I'm upset, I feel guilty for feeling that way"), Goals (e.g., "When I'm upset, I have difficulty concentrating"), Impulse (e.g., "When I'm upset, I lose control over my behaviors"), Awareness (e.g., "I am attentive to my feelings" [reverse scored]), Strategies (e.g., "When I'm upset, I believe I'll end up feeling very depressed"), and Clarity (e.g., "I have difficulty making sense of my feelings"). The DERS shows high internal consistency, good test-retest reliability, and adequate construct and predictive validity (Gratz & Roemer, 2004).

Self-Injurious Thoughts and Behaviors Interview. The Self-Injurious Thoughts and Behaviors Interview (Nock, Holmberg, Photos, & Michel, 2007) is a structured interview that measures the presence, frequency, and characteristics of various types of self-injurious thoughts and behaviors. It has modules for suicidal ideation, suicide plans, suicide gestures, suicide attempts, and NSSI. The interview has strong interrater reliability (average K = .99, r = 1.0) and test-retest reliability (average K = .70, intraclass correlation coefficient = .44) over a 6-month interval (Nock et al., 2007). It shows strong construct validity, converging with other measures of suicidal ideation (average K = .54), suicide attempts (average K = .65), and NSSI (average K = .87). In the present study, we used the

NSSI module of the interview to assess the presence and frequency of NSSI behaviors.

Mini International Neuropsychiatric Interview. The Mini International Neuropsychiatric Interview (Sheehan et al., 1998) is a short, structured diagnostic interview that assesses constructs from the text revision of the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition, text revision. Modules for each construct consist of 8 to 10 questions. The interview has shown high interrater reliability (K = 1.0) and test-retest reliability (K = .87) and strong convergent construct validity with longer structured clinical interviews of general psychopathology (Sheehan et al., 1998). In the present study, we employed the interview to assess the presence of Axis I disorders.

Psychophysiological measures

Startle stimuli and methods. Startle eyeblink and postauricular methods were in accordance with currently recommended guidelines (Benning, Patrick, & Lang, 2004; Blumenthal et al., 2005). Startle stimuli were 100-dB(A) broadband noises (20 Hz–20 kHz) with a near-instantaneous rise/fall time and a duration of 50 ms. These stimuli were delivered to participants through headphones. Startle was electromyographic activity measured from surface recording electrodes—two near the eye to measure eyeblink, two behind the ear to measure postauricular reactivity, and one on the temple to serve as a ground. This information was processed and sampled at 1000 Hz by a Biopac MP150 workstation, relayed through a bandpass filter at 28 to 500 Hz, and smoothed with a five-sample boxcar filter (cf. Blumenthal et al., 2005). Startle eyeblink and postauricular reactivity were measured simultaneously in response to the same stimuli (cf. Benning et al., 2004; Sandt et al., 2009). Although these reflexes are measured at the same time and display nearly inverse patterns with affective stimuli, startle eyeblink and postauricular reactivity show only a small inverse correlation (e.g., r = –.07: Franklin et al., in press; r = –.11: Sandt et al., 2009). These two measures had a similar near-zero correlation in the present study (average r = .08). This is consistent with the idea that these measures are independent and that positive affect and negative affect are two separate dimensions (e.g., Norman et al., 2011).

Startle eyeblink reactivity. Startle eyeblink is a defensive reflex that occurs in response to a sufficiently intense and sudden stimulus, such as a sudden and loud sound (Blumenthal et al., 2005). Because it is a defensive reflex, startle eyeblink reactivity is heightened by unpleasant stimuli and reduced by pleasant stimuli (see Lang, Bradley, & Cuthbert, 1990). Eyeblink reactivity was quantified as the electromyographic activity of the orbicularis oculi muscle, which surrounds the eye and contracts to produce an eyeblink (see Blumenthal et al., 2005). Startle eyeblink reactivity is one of the few psychophysiological measures specific to negative affective valence (Lang et al., 1990).

Startle postauricular reactivity. The postauricular muscle is located behind the ear and is used by most infant mammals to pull back the ear during nursing; however, it is largely vestigial in humans (Johnson, Valle-Inclán, Geary, & Hackley, 2012). Although it has no apparent functional use in humans, the postauricular muscle can still be primed by pleasant stimuli (especially those related to food, nursing, or reward; see Johnson et al., 2012; Sandt et al., 2009). When an intense stimulus is presented (e.g., a startling sound), it synchronizes the motor units of the postauricular muscle, generating a sudden spike in activity that is larger the more primed the muscle is (i.e., during pleasant affective states; see Johnson et al., 2012). Accordingly, startle postauricular reactivity shows a pattern of affective modulation that is opposite to that of startle eyeblink reactivity (Benning et al., 2004). Startle postauricular reactivity is one of the few psychophysiological measures specific to positive affective valence (Gable & Harmon-Jones, 2009).

Painful stimulus: NSSI proxy

Painful electrocutaneous stimulation (i.e., shocks) served as the laboratory NSSI proxy (cf. Andreatta et al., 2010; Franklin et al., in press). Previous laboratory NSSI studies that examined affect pre- and postpain employed cold pain (Franklin et al., 2010; Russ et al., 1992), heat pain (Bresin & Gordon, in press), and shock pain (Weinberg & Klonsky, 2012). Pain offset relief studies, which have methods that strongly resemble those from NSSI laboratory studies, have employed shock pain (Andreatta et al., 2010; Franklin et al., in press; Tanimoto et al., 2004; Yarali et al., 2008; Zanna et al., 1970), heat pain (Bresin et al., 2010; Leknes et al., 2008), and pressure pain (Bresin et al., 2010). Despite a wide range of painful stimuli and dependent measures, these studies have consistently found diminished negative affect or increased positive affect after pain offset. In the present study, we chose shocks because they allowed for high stimulus control in terms of intensity, timing, and repetition.

In terms of overall pain offset relief methodology, some studies have administered a painful stimulus and measured affect for the next several seconds (e.g., Franklin et al., in press; Leknes et al., 2008), and others have paired neutral stimuli with pain offset and measured affective reactions to the conditioned stimuli. For practical reasons, this latter paradigm is common in animal research (e.g., Tanimoto et al., 2004; Yarali et al., 2008), but this pain offset relief conditioning effect also has been demonstrated in humans (e.g., Andreatta et al., 2010; Zanna et al., 1970). In the present study, we chose to directly measure the effects of pain offset (cf. Leknes et al., 2008) because this paradigm most closely approximates the affective processes in NSSI.

Pain was induced with 200-ms 150-Hz shocks delivered via two electrodes attached to the left bicep. Similar to the methods of previous studies (Andreatta et al., 2010; Franklin et al., in press), stimulus intensity was adjusted for each participant until they rated the shocks as a 30 on a scale of 0 (no pain) to 100 (worst pain imaginable). This level was assessed twice to

ensure the validity of the ratings. During the experimental portion of the study, each participant received 20 shocks.

Procedure

Participants arrived at our laboratory, read and signed an informed consent form, completed a demographics questionnaire, were administered the NSSI module of the Self-Injurious Thoughts and Behaviors Interview and the Mini International Neuropsychiatric Interview, and completed the ERS and DERS. Next, electrodes were placed on participants to measure startle eyeblink and postauricular reactivity (see Benning et al., 2004; Blumenthal et al., 2005; Franklin et al., in press). Participants then underwent a startle habituation block designed to acclimate them to the procedure and to bring them to a startle reactivity asymptote. Evidence suggests that the majority of participants reach a startle reactivity asymptote at around 13 trials (Lane, Franklin, & Curran, in press); the present startle habituation block was 15 trials long. Next, participants underwent the pain calibration procedure described earlier. After this calibration, they underwent the experimental portion of the study, which consisted of 30 trials: (a) 10 startle-alone trials, (b) 10 trials for which startle reactivity was measured 3.5 s postshock, and (c) 10 trials for which startle reactivity was measured 6 s postshock. We chose these postshock intervals because this is the window within which affective startle modulation (cf. Benning et al., 2004; Lang et al., 1990) and pain offset relief are the strongest. Specifically, pain offset effects on affective valence (Franklin et al., in press), physiological arousal (Leknes et al., 2008), and conditioning (Andreatta et al., 2010) peak after 3 to 6 s and disappear around 14 s. Trials were presented in a random order and had an intertrial interval that varied randomly between 23 and 30 s. Finally, participants were then debriefed and compensated.

Data analytic plan

Startle scoring. Startle eyeblink reactivity was quantified as the difference between the peak and foot of the largest response that occurred between 20 and 100 ms after the onset of the startle stimulus (cf. Blumenthal et al., 2005). Startle postauricular reactivity was quantified as the difference between the peak within a window of 5 and 35 ms after the onset of the startle stimulus and the average voltage 50 ms before the startle stimulus (cf. Benning et al., 2004). Responses were averaged within each condition for each participant. Pain offset relief was operationalized as (a) the proportion of reduction in startle eyeblink reactivity in the postshock conditions compared to the control condition and (b) the proportion of increase in startle postauricular reactivity in the postshock conditions compared to the control condition. Mathematically, pain offset relief was defined as the percentage difference from control within each postshock condition: [(postshock condition mean – control condition mean)/control condition mean] × 100%. This resulted in a standardized variable for each of the four postshock conditions (3.5- and 6-s eyeblink and 3.5- and 6-s postauricular reactivity). These standardized variables reduced the skewing impact of particularly large or small levels of startle reactivity (see Blumenthal et al., 2005) and provided a direct index of pain offset relief magnitude.

Analyses. Our initial analyses were aimed at assessing the nature of pain offset relief. As such, these analyses tested hypotheses concerning pain offset relief as a mechanism of affect regulation in NSSI and the existence of an independent positive reinforcement mechanism. First, we assessed the consistency of pain offset relief to ascertain whether this was a rare or common phenomenon in the present study. To accomplish this, within each condition, we examined the percentage of participants who displayed pain offset relief. Second, we investigated the strength of pain offset relief. Specifically, for each of the four postshock conditions, we used one-sample *t* tests to determine whether the change in reactivity was significantly different from zero. We also calculated the effect size of the change in each condition with Cohen's *d*.

The next set of analyses tested the hypothesis that opponent processes heighten pain offset relief across repeated NSSI episodes. First, we investigated the association between pain offset relief and NSSI frequency within the NSSI group. As in many other studies, there was a large range of NSSI frequency (1 to 3,000 episodes) in the present study, which presented potential difficulties for analyses with this variable. To address this issue, we calculated NSSI frequency in three ways. The first method was to calculate raw frequency, for which values were the exact values that participants reported. To create a less skewed frequency distribution, the second method involved transforming raw frequency values into class intervals—specifically, (a) values between 1 and 10 were assigned a value of 1; (b) values between 11 and 50, a value of 2; (c) values between 51 and 100, a value of 3; (d) values between 101 and 500, a value of 4; and (e) values 501 and beyond, a value of 5. We then examined Pearson product-moment correlations between these frequency variables and the four pain offset relief variables. Because it could be argued that these class intervals were relatively arbitrary, we also examined these associations with Spearman's rho, which converted NSSI frequency data into ordinal data. Second, we examined the degree to which any history of NSSI heightened pain offset relief. Specifically, we employed one-way analyses of variance to examine the effect of group (NSSI vs. control) on pain offset relief in each of the four postshock conditions.

One prior study found that high emotion reactivity was associated with heightened pain offset relief (Bresin et al., 2010). Accordingly, any association between NSSI or NSSI frequency and heightened pain offset relief may be confounded by affective abnormalities. We accounted for this possibility in a final set of analyses. We first attempted to replicate previous findings of elevated scores on the ERS and DERS in people with a history of NSSI (Gratz & Roemer, 2008; Nock, et al., 2008). We then examined the associations among the

ERS, the DERS and its subscales, and the four pain offset relief variables. Finally, we controlled for ERS and DERS scores in any analyses that produced group differences in pain offset relief.

Results

Consistency of pain offset relief

Across all four conditions, analyses showed that the majority of participants displayed pain offset relief. That is, most participants evidenced reduced mean startle eyeblink reactivity and increased postauricular reactivity in the postshock conditions compared to the control condition. Specifically, 90.5% in the 3.5-s postshock condition and 92.9% in the 6-s postshock condition displayed reduced mean eyeblink reactivity. Likewise, 83.3% in the 3.5-s postshock condition and 71.4% in the 6-s postshock condition evidenced increased mean postauricular reactivity. These results suggest that pain offset relief is consistent phenomenon shown by most participants in most conditions.

Strength of pain offset relief

One-sample *t* tests revealed significant pain offset relief in all four postshock conditions (see Table 1). These effects were large in the postauricular conditions and very large in the eyeblink conditions (see Table 1). These findings demonstrate that pain offset has powerful effects on both positive and negative affect.

NSSI frequency and pain offset relief

Within the NSSI group, correlational analyses revealed no significant correlations between NSSI frequency and pain offset relief in any condition ($p > .05$; see Table 2). These null findings were consistent across all three NSSI frequency calculation methods. These findings suggest that repeated episodes of NSSI do not generate substantially heightened pain offset relief.

Group differences in pain offset relief

Consistent with the weak association between NSSI frequency and pain offset relief in the NSSI group, one-way analyses of variance revealed no significant main effects of group on pain offset relief in the 3.5-s eyeblink ($d = .18$), 3.5-s postauricular ($d = .09$), or 6-s postauricular conditions ($d = .37$), $p > .05$ (see Fig. 1). However, there was a significant main effect of group on pain offset relief in the 6-s eyeblink condition, $F(1, 40) = 4.38$, $p = .04$, $d = .62$, such that the NSSI group displayed a greater reduction in eyeblink reactivity (see Fig. 1). A post hoc analysis of covariance showed that this effect did not remain significant after controlling for ERS scores, $F(1, 39) = 2.10$, $p = .16$, suggesting that this effect may be explained in part by emotion reactivity differences between the groups. Overall, these results provide evidence for an independent positive reinforcement mechanism in NSSI but indicate that experience with NSSI does not seem to substantially affect the degree of pain offset relief.

Table 1. Tests of Pain Offset Relief Across All Participants

Condition[a]	*M* (*SD*)[b]	*t*(40)	*d*
Eyeblink			
3.5	−38.17 (27.77)	−8.91	1.37
6	−31.60 (22.62)	−9.05	1.40
Postauricular			
3.5	28.25 (36.18)	5.06	0.78
6	23.36 (33.25)	4.55	0.70

Note: Each test, $p < .001$.
[a]3.5 = variable measured 3.5 s postshock; 6 = variable measured 6 s postshock.
[b]Means were calculated as the percentage difference from control trials.

Table 2. Correlations Between Pain Offset Relief and Nonsuicidal Self-Injury Frequency

Condition	1	2	3	4
1. Eyeblink, 3.5 s	—			
2. Eyeblink, 6 s	.35**			
3. Postauricular, 3.5 s	.08	.12		
4. Postauricular, 6 s	−.07	.22	.55***	
5. Frequency, raw[a]	.01	−.05	−.02	−.18
6. Frequency, interval[b]	−.24	−.01	−.11	.03
7. Frequency, ranked[c]	−.01	.25	.03	−.07

Note: Analyses were limited to the nonsuicidal self-injury group ($n = 21$).
[a]Frequency calculated as the exact number reported by participants.
[b]Frequency reports converted to class intervals.
[c]Spearman rank-order correlations.
**$p < .01$. ***$p < .001$.

Affective abnormalities and pain offset relief

Consistent with previous studies (Gratz & Roemer, 2008; Nock et al., 2008), one-way analyses of variance revealed that the NSSI group displayed significantly greater total ERS and DERS scores compared to the control group (see Table 3). Correlational analyses showed that ERS scores were significantly negatively associated with startle eyeblink reactivity in the 6-s postshock condition, $r(42) = –.26$, $p = .04$. No correlations between pain offset relief and the DERS total score or subscales trended toward significance ($p > .10$) or exceeded .2 or –.2. These results indicate that emotion reactivity may play a small role in heightening some components of pain offset relief, but they also suggest that affect dysregulation does not substantially influence pain offset relief intensity.

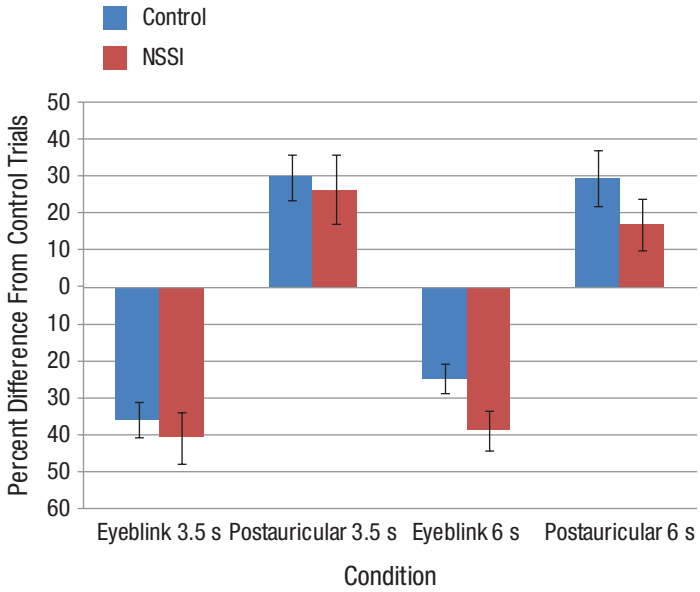

Fig. 1. Pain offset relief by group, measure, and time. 3.5 s = measurement 3.5 s postshock; 6 s = measurement 6 s postshock.

Discussion

It is critical to understand the mechanisms that drive affect regulation during self-injury. The present findings supported a unique role for pain offset relief, over and above other potential mechanisms, such as distraction. These findings also supported the existence of a positive reinforcement function that is independent of the well-known negative reinforcement function of NSSI. Surprisingly, however, the present results did not provide strong support for opponent processes as a major link between NSSI and suicidal behavior. We discuss each of these findings in greater detail.

Rumination-driven emotional cascades can generate intense emotional pain that is difficult to regulate (Selby, Anestis, Bender, & Joiner, 2009; Selby & Joiner, 2009). Self-administered physical pain may act as a distraction that serves to disengage these cascades, thereby diminishing negative affect (see Selby & Joiner, 2009). Taking advantage of the high degree of neural overlap between emotional and physical pain (see Eisenberger, 2012), self-administered physical pain may also serve to commandeer the emotional pain system. This neural overlap causes an incidental effect where physical pain offset (or even a slight reduction in physical pain intensity; cf. Grill & Coghill, 2002) simultaneously generates emotional pain offset (e.g., Leknes et al., 2008). The critical distinction between these two mechanisms is that whereas distraction would only diminish negative affect, pain offset relief would both diminish negative affect and stimulate positive affect (cf. Franklin et al., in press). Supporting the pain offset relief mechanism, the present results indicated that pain offset both diminished negative affect and stimulated positive affect in people with a history of NSSI (see Figure 1). Distraction may still play a role in affect regulation during NSSI, but the degree to which these two mechanisms overlap or interact remains unclear. Future studies should aim to disentangle these two mechanisms.

One major implication of this finding is that it suggests that adaptive behaviors that activate pain offset relief may be particularly effective replacement behaviors for NSSI. For example, Wallenstein and Nock (2007) provided single case-study

Table 3. Group Differences in Emotion Reactivity and Emotion Dysregulation

Scale and subscales	Control *M* (*SD*)	Nonsuicidal self-injury *M* (*SD*)	*F*(1, 40)	*d*
Emotion Reactivity Scale	38.95 (14.67)	58.05 (22.20)	10.81**	.91
Difficulties in Emotion Regulation Scale				
Total	82.61 (13.59)	98.52 (22.21)	7.84**	.80
Nonacceptance	11.05 (4.39)	14.75 (5.49)	5.54*	.70
Goals	13.25 (3.42)	15.05 (3.46)	2.74	.51
Impulse	10.80 (2.95)	12.70 (4.65)	2.38	.48
Awareness	20.65 (3.77)	21.25 (6.14)	0.14	.12
Strategy	15.40 (3.62)	20.50 (6.45)	9.51**	.88
Clarity	12.60 (2.06)	12.20 (2.09)	0.37	−.19

*$p < .05$. **$p < .01$.

evidence that exercise significantly reduced NSSI behaviors. Future studies and treatments may benefit from similarly exploiting this pain offset relief mechanism. Another major implication of this finding is that it demonstrates the existence of an independent positive reinforcement function in NSSI. Previous self-report studies have detected this function, but it often has been either strongly correlated with a negative reinforcement function (e.g., r = .52: Nock & Prinstein, 2004) or interpreted as reflecting negative reinforcement (e.g., Klonsky, 2009). The present results provide experimental and psychophysiological evidence that most NSSI episodes may involve simultaneous—but independent—positive and negative reinforcement. Most models and treatments of NSSI focus solely on the negative reinforcement function (e.g., Chapman et al., 2006; Selby & Joiner, 2009); the present findings suggest that the conceptualization and treatment of NSSI would benefit from considering a prominent role for both functions.

Contrary to predictions, results indicated that NSSI frequency was not associated with heightened pain offset relief (see Table 2). Moreover, relative to the control group, the NSSI group displayed heightened pain offset relief in only one condition (i.e., greater reduction in startle eyeblink reactivity 3.5 s after pain offset; see Fig. 1), and this group difference did not hold after controlling for emotion reactivity. These findings provide little support for the hypothesis that heightened relief across self-injury episodes is one of the major mechanisms that increase the risk for future self-injury (Franklin et al., 2010; Heilbron et al., in press; Joiner, 2005; Joiner et al., 2012). Although the present investigation was not longitudinal, a lack of cross-sectional support for this association suggests that opponent processes may not be a major factor in the link between NSSI and suicidal behavior. This negates the need for longitudinal studies of the temporal association between NSSI frequency and pain offset relief. In sum, pain offset relief appears to be a robust and static phenomenon that is largely unaffected by experience with NSSI; similarly, results indicated that pain offset relief did not vary strongly with either affect dysregulation or emotion reactivity. These results stand in stark contrast to recent evidence that affect dysregulation, self-criticism, and NSSI experience are associated with diminished pain perception (Franklin, Aaron, Arthur, Shorkey, & Prinstein, 2012; Hooley et al., 2010) and preliminary evidence that NSSI frequency is associated with diminished negative affective reactions to self-injury stimuli (Franklin, Puzia, et al., 2012; Glenn & Klonsky, 2010).

Taken together, these findings suggest that although factors such as affect dysregulation and NSSI experience may substantially reduce the barriers to self-injury (e.g., pain, aversion to mutilation stimuli), these factors may not strongly amplify the benefits of self-injury (e.g., pain offset relief). This is inconsistent with the hypothesis that opponent processes drive the link between NSSI and suicidal behaviors (Heilbron et al., in press; Franklin et al., 2010; Joiner et al., 2012). Rather, we speculate that this link is driven primarily by habituation, self-criticism (Glassman, Weierich, Hooley, Deliberto, & Nock, 2007; Hooley et al., 2010; St. Germain & Hooley, 2012), pain offset relief conditioning (Franklin, Puzia, et al., 2012; cf. Andreatta et al., 2010), and other factors that serve to reduce the barriers to self-injury. We caution, however, that this model remains preliminary, and we emphasize the need for more experimental, psychophysiological, and longitudinal tests.

The present findings should be interpreted in accord with the limitations of the present study. First, ecological validity is a concern for all NSSI laboratory studies. However, across a wide range of paradigms, painful stimuli, and affect measures, these studies have found remarkably consistent results (present study; Bresin & Gordon, in press; Franklin et al., 2010; Russ et al., 1992; Weinberg & Klonsky, 2012). These findings are also highly consistent with preliminary findings from a naturalistic NSSI study (Nock, Mendes, Deliberto, & Dour, 2009). These lines of evidence suggest that the present findings may generalize beyond the laboratory; nonetheless, naturalistic studies are needed to ensure the ecological validity of the present study.

Second, startle eyeblink and postauricular reactivity are among the best-validated psychophysiological measures of affective valence available (Blumenthal et al., 2005; Johnson et al., 2012), but they are not perfect measures. In particular, these measures require the presentation of loud noises and can be vulnerable to habituation effects. Although these measures were well suited to investigate the present hypotheses, future studies would benefit from examining these hypotheses with other explicit, implicit, and biological measures. Third, a related limitation of the present study was the lack of self-reported affect after pain offset. Psychophysiological measures may be more effective at tapping into pain offset relief (see Andreatta et al., 2010; Franklin et al., 2010), and we reasoned that processes related to self-report (e.g., sensing, comparing, rating, and reporting affect) may interrupt the affective experience that we aimed to measure. However, multimethod information across several levels (e.g., explicit, implicit, physiological, genetic) would be ideal. Fourth, the NSSI group sample size was adequate to examine the present hypotheses, but future studies should utilize larger samples to investigate more fine-grained effects.

In summary, the present study found evidence that pain offset relief may be one of the primary mechanisms of affect regulation in NSSI and that NSSI may simultaneously evoke both positive and negative reinforcement. Additionally, the present findings were largely inconsistent with the hypothesis that opponent processes drive the link between prior and future self-injury. Specifically, the present results suggested that pain offset relief is normal and mostly unaffected by factors such as NSSI frequency and affect dysregulation. We speculate that this implies that the abnormal aspects of NSSI concern how some people overcome the instinctive barriers (e.g., pain, aversion to mutilation stimuli) that keep most people from engaging in these behaviors. Knowledge about how these

barriers are eroded may provide new insights into how NSSI develops and how to reduce NSSI. Future NSSI theories and treatments may benefit from targeting these processes.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

References

Andreatta, M., Muhlberger, A., Yarali, A., Gerber, B., & Pauli, P. (2010). A rift between implicit and explicit conditioned valence in human pain relief learning. *Proceedings of the Royal Society B*, *277*, 2411–2416. doi:10.1098/rspb.2010.0103

Asarnow, J. R., Porta, G., Spirito, A., Emslie, G., Clarke, G., Wagner, K. D., . . . Brent, D. A. (2011). Suicide attempts and nonsuicidal self-injury in the Treatment of Resistant Depression in Adolescents: Findings from the TORDIA study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *50*, 772–781.

Barrett, L. F., & Bliss-Moreau, E. (2009). Affect as a psychological primitive. *Advances in Experimental Social Psychology*, *41*, 167–218.

Benning, S. D., Patrick, C. J., & Lang, A. R. (2004). Emotional modulation of the post-auricular reflex. *Psychophysiology*, *41*, 426–432. doi:10.1111/j.1469-8986.00160.x

Blumenthal, T. D., Cuthbert, B. N., Filion, D. L., Hackley, S., Lipp, O. V., & van Boxtel, A. (2005). Committee report: Guidelines for human startle eyeblink electromyographic studies. *Psychophysiology*, *42*, 1–15.

Bresin, K., & Gordon, K. H. (in press). Changes in negative affect following pain (vs. nonpainful) stimulation in individuals with and without a history of nonsuicidal self-injury. *Personality Disorders: Theory, Research, and Treatment*.

Bresin, K., Gordon, K. H., Bender, T. W., Gordon, L. J., & Joiner, T. E. (2010). No pain, no change: Reductions in prior negative affect following physical pain. *Motivation and Emotion*, *34*, 280–287. doi:10.1007/s11031-010-9168-7

Chapman, A. L., Gratz, K. L., & Brown, M. Z. (2006). Solving the puzzle of deliberate self-harm: The experiential avoidance model. *Behaviour Research and Therapy*, *44*, 371–394. doi:10.1016/j.brat.2005.03.005

Craig, A. D. (2002). How do you feel? Interoception: The sense of the physiological condition of the body. *Nature Reviews Neuroscience*, *3*, 655–666.

Craig, A. D. (2009). How do you feel—Now? The anterior insula and human awareness. *Nature Reviews Neuroscience*, *10*, 59–70.

DiClemente, R. J., Ponton, L. E., & Hartley, D. (1991). Prevalence and correlates of cutting behavior: Risk for HIV transmission. *Journal of the American Academy of Child and Adolescent Psychiatry*, *30*, 735–739.

Eisenberger, N. I. (2012). Broken hearts and broken bones: A neural perspective on the similarities between social and physical pain. *Current Directions in Psychological Science*, *21*, 42–47.

Franklin, J. C., Aaron, R. V., Arthur, M. S., Shorkey, S. P., & Prinstein, M. J. (2012). Nonsuicidal self-injury and diminished pain perception: The role of emotion dysregulation. *Comprehensive Psychiatry*, *53*, 691–700.

Franklin, J. C., Hessel, E. T., Aaron, R. V., Arthur, M. S., Heilbron, N., & Prinstein, M. J. (2010). The functions of nonsuicidal self-injury: Support for cognitive-affective regulation and opponent processes from a novel psychophysiological paradigm. *Journal of Abnormal Psychology*, *119*, 850–862. doi:10.1037/a0020896

Franklin, J. C., Lee, K. M., Hanna, E. K., & Prinstein, M. J. (in press). Feeling worse to feel better: Pain offset relief simultaneously reduces negative affect and stimulates positive affect. *Psychological Science*.

Franklin, J. C., Puzia, M. E., Lee, K. M., Chung, G., Hanna, E. K., Spring, V. L., & Prinstein, M. J. (2012). *Pain offset relief and the development (and reversal) of positive associations with nonsuicidal self-injury*. Symposium presented at the Association for Behavioral and Cognitive Therapies, National Harbor, MD.

Gable, P. A., & Harmon-Jones, E. (2009). Postauricular reflex responses to pictures varying in valence and arousal. *Psychophysiology*, *46*, 487–490.

Glassman, L. H., Weierich, M. R., Hooley, J. M., Deliberto, T. L., & Nock, M. K. (2007). Child maltreatment, non-suicidal self-injury, and the mediating role of self-criticism. *Behaviour Research and Therapy*, *45*, 2483–2490.

Glenn, C. R., & Klonsky, E. D. (2010). The role of seeing blood in non-suicidal self-injury. *Journal of Clinical Psychology*, *66*, 466–473.

Gratz, K. L., & Roemer, L. (2004). Multidimensional assessment of emotion regulation and dysregulation: Development, factor structure, and initial validation of the difficulties in emotion regulation scale. *Journal of Psychopathology and Behavioral Assessment*, *26*, 41–54.

Gratz, K. L., & Roemer, L. (2008). The relationship between emotion dysregulation and deliberate self-harm among female undergraduate students at an urban commuter university. *Cognitive Behaviour Therapy*, *37*, 14–25.

Grill, J. D., & Coghill, R. C. (2002). Transient analgesia evoked by noxious stimulus offset. *Journal of Neurophysiology*, *87*, 2205–2208.

Guan, K., Fox, K. R., & Prinstein, M. J. (2012). Nonsuicidal self-injury longitudinally predicts adolescent suicide ideation and attempts in a diverse community sample. *Journal of Consulting and Clinical Psychology*, *80*, 842–849.

Heilbron, N., Franklin, J. C., Guerry, J. D., & Prinstein, M. J. (in press). Social and ecological approaches to suicidal behaviors. In M. Nock (Ed.), *The Oxford handbook of suicide and self-injury*. Oxford, England: Oxford University Press.

Hooley, J. M., Ho, D. T., Slater, J., & Lockshin, A. (2010). Pain perception and nonsuicidal self-injury: A laboratory investigation. *Personality Disorders: Theory, Research, and Treatment, 1*, 170–179.

Johnson, G. M., Valle-Inclán, F., Geary, D. C., & Hackley, S. A. (2012). The nursing hypothesis: An evolutionary account of emotional modulation of the postauricular reflex. *Psychophysiology*, *49*, 178–185. doi:10.1111/j.1469-8986.2011.01297.x

Joiner, T. (2005). *Why people die by suicide*. Cambridge, MA: Harvard University Press.

Joiner, T. E., Ribeiro, J. D., & Silva, C. (2012). Nonsuicidal self-injury, suicidal behavior, and their co-occurrence as viewed

through the lens of the interpersonal theory of suicide. *Current Directions in Psychological Science, 21*, 342–347.

Klonsky, E. D. (2007). The functions of deliberate self-injury: A review of the evidence. *Clinical Psychology Review*, *27*, 226–239.

Klonsky, E. D. (2009). The functions of self-injury in young adults who cut themselves: Clarifying the evidence for affect-regulation. *Psychiatry Research*, *166*, 260–268.

Klonsky, E. D. (2011). Non-suicidal self injury in United States adults: Prevalence, sociodemographics, topography and functions. *Psychological Medicine*, *41*, 1981–1986.

Lane, S. T., Franklin, J. C., & Curran, P. J. (in press). Clarifying the nature of startle habituation with latent curve modeling. *International Journal of Psychophysiology*.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1990). Emotion, attention, and the startle reflex. *Psychological Review*, *97*, 377–395. doi:10.1037/0033-295X.97.3.377

Leknes, S., Brooks, J. W., Wiech, K., & Tracey, I. (2008). Pain relief as an opponent process: A psychophysical investigation. *European Journal of Neuroscience*, *28*, 794–801. doi:10.1111/j.1460-9568.2008.06380.x

Lindquist, K. A., Wager, T. D., Kober, H., Bliss-Moreau, E., & Barrett, L. F. (2012). The brain basis of emotion: A meta-analytic review. *Behavioral and Brain Sciences*, *35*, 121–202.

Muehlenkamp, J. J., Engel, S. G., Wadeson, A., Crosby, R. D., Wonderlich, S. A., Simonich, H., & Mitchell, J. E. (2009). Emotional states preceding and following acts of non-suicidal self-injury in bulimia nervosa patients. *Behaviour Research and Therapy*, *47*, 83–87.

Nock, M. K. (2010). Self-injury. *Annual Review of Clinical Psychology*, *6*, 339–363. doi:10.1146/annurev.clinpsy.121208.131258

Nock, M. K., Holmberg, E. B., Photos, V. I., & Michel, B. D. (2007). Self-Injurious Thoughts and Behaviors Interview: Development, reliability, and validity in an adolescent sample. *Psychological Assessment*, *19*, 309–317.

Nock, M. K., Mendes, W. B., Deliberto, T. L., & Dour, H. J. (2009). *Evidence for the affect regulating properties of self-injury: Results from a real-time physiological monitoring study*. Symposium presented at the Association for Behavioral and Cognitive Therapies, New York, NY.

Nock, M. K., & Prinstein, M. J. (2004). A functional approach to the assessment of self-mutilative behavior. *Journal of Consulting and Clinical Psychology*, *75*, 885–890.

Nock, M. K., Wedig, M. M., Holmberg, E. B., & Hooley, J. M. (2008). The Emotion Reactivity Scale: Development, evaluation, and relation to self-injurious thoughts and behaviors. *Behavior Therapy*, *39*, 107–116.

Norman, G. J., Norris, C. J., Gollan, J., Ito, T. A., Hawkley, L. C., Larsen, J. T., . . . Bernston, G. G. (2011). Current emotion research in psychophysiology: The neurobiology of evaluative bivalence. *Emotion Review*, *3*, 349–359.

Russ, M. J., Roth, S. D., Lerman, A., Kakuma, T., Harrison, K., Shindledecker, R. D., . . . Mattis, S. (1992). Pain perception in self-injurious patients with borderline personality disorder. *Biological Psychiatry*, *32*, 501–511.

Sandt, A. R., Sloan, D. M., & Johnson, K. J. (2009). Measuring appetitive responding with the postauricular reflex. *Psychophysiology*, *46*, 491–497. doi:10.1111/j.1469-8986.2009.00797.x

Selby, E. A., Anestis, M. D., Bender, T. W., & Joiner, T. E. (2009). An exploration of the emotional cascade model in borderline personality disorder. *Journal of Abnormal Psychology, 118*, 375–387.

Selby, E. A., Bender, T. W., Gordon, K. H., Nock, M. K., & Joiner, T. E. (2012). Non-suicidal self-injury (NSSI) disorder: A preliminary study. *Personality Disorders: Theory, Research, and Practice*, 3, 167–175.

Selby, E. A., & Joiner, T. R. (2009). Cascades of emotion: The emergence of borderline personality disorder from emotional and behavioral dysregulation. *Review of General Psychology*, *13*, 219–229. doi:10.1037/a0015687

Sheehan, D. V., Lecrubier, Y., Sheehan, K. H., Amorim, P., Janavs, J., . . . Dunbar, G. C. (1998). The Mini-International Neuropsychiatric Interview (MINI): The development and validation of a structured diagnostic psychiatric interview for *DSM-IV* and ICD-10. *Journal of Clinical Psychiatry*, *59*, 22–33.

Smith, M. P., & Buchanan, G. (1954). Acquisition of secondary reward by cues associated with shock reduction. *Journal of Experimental Psychology*, *48*, 123–126.

Solomon, R. L. (1980). The opponent-process theory of acquired motivation: The costs of pleasure and the benefits of pain. *American Psychologist*, *35*, 691–712. doi:10.1037/0003-066X.35.8.691

St. Germain, S. A., & Hooley, J. M. (2012). Direct and indirect forms of non-suicidal self-injury: Evidence for a distinction. *Psychiatry Research*, *197*, 78–84.

Tanimoto, H., Heisenberg, M., & Gerber, B. (2004). Event timing turns punishment to reward. *Nature*, *430*, 983. doi:10.1038/430983a

Victor, S. E., Glenn, C. R., & Klonsky, E. D. (2012). Is non-suicidal self-injury an "addiction"? A comparison of craving in substance use and non-suicidal self-injury. *Psychiatry Research*, *197*, 73–77.

Wallenstein, M. B., & Nock, M. K. (2007). Physical exercise as a treatment for non-suicidal self-injury: Evidence from a single case-study. *American Journal of Psychiatry*, *164*, 350–351.

Watson, D., & Tellegen, A. (1985). Toward a consensual structure of mood. *Psychological Bulletin*, *98*, 219–235.

Weinberg, A., & Klonsky, E. D. (2012). The effects of self-injury on acute negative arousal: A laboratory simulation. *Motivation and Emotion*, *36*, 242–254.

Wilkinson, P., Kelvin, R., Roberts, C., Dubicka, B., & Goodyer, I. (2011). Clinical and psychosocial predictors of suicide attempts and nonsuicidal self-injury in the Adolescent Depression Antidepressants and Psychotherapy Trials (ADAPT). *American Journal of Psychiatry*, *168*, 495–501.

Yarali, A., Niewalda, T., Chen, Y., Tanimoto, H., Durrenagel, S., & Gerber, B. (2008). "Pain relief" learning in fruit flies. *Animal Behaviour*, *76*, 1173–1185. doi:10.1016/j.anbehav.2008.05.025

Zanna, M. P., Kiesler, C. A., & Pilkonis, P. A. (1970). Positive and negative attitudinal affect established by classical conditioning. *Journal of Personality and Social Psychology*, *14*, 321–328.