

Negative Emotional Action Termination (NEAT): Support for a Cognitive Mechanism Underlying Negative Urgency in Nonsuicidal Self-Injury

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Negative urgency, the self-reported tendency to act impulsively when distressed, increases risk for nonsuicidal self-injury (NSSI). NSSI is also associated with impaired negative emotional response inhibition (NERI), specifically negative emotional action *termination* (NEAT), a cognitive process theoretically related to negative urgency. We previously found that adults with NSSI history had difficulty inhibiting behavioral responses to affective images depicting negative content (but not positive or neutral images) in an Emotional Stop-Signal Task. We sought to replicate this finding, determine whether this deficit extends to negative emotional action *suppression* (NEAS; an earlier stage of NERI), and explore whether impairment in these two stages of emotional response inhibition helps explain the relationship between negative urgency and NSSI. Eighty-eight adults with NSSI history ($n = 45$) and healthy control participants ($n = 43$) without NSSI history or psychopathology completed a clinical interview, symptom inventories, an impulsivity questionnaire, and behavioral assays of early and late NERI (NEAS and

NEAT, respectively). The NSSI group had worse NEAT than the control group in the Emotional Stop-Signal Task, but no group differences in NEAS were observed in an Emotional Go/No-go task. However, both early and late stages of NERI accounted for independent variance in negative urgency. We additionally found that NEAT explained variance in the association between negative urgency and NSSI. These results suggest that impulsive behavior in NSSI may involve specifically impaired inhibitory control over *initiated* negative emotional impulses. This deficit in late response inhibition to negative emotional stimuli might reflect a cognitive mechanism or pathway to elevated negative urgency among people who self-injure.

Keywords: self-injurious behavior; inhibitory control; impulsiveness; emotionality; emotion dysregulation

NONSUICIDAL SELF-INJURY (NSSI), direct and deliberate self-harm without suicidal intent, is a common behavior with potentially serious consequences. Lifetime community prevalence rates include 17.2% of adolescents, 13.4% of young adults, and 5.5% of adults (Swannell, Martin, Page, Hasking, & St. John, 2014). Although NSSI acts are “nonsuicidal” by definition, they are also strongly associated with suicide (e.g., Klonsky, May, & Glenn, 2013); NSSI is statistically equivalent to prior suicide attempts in predicting future suicidal behavior (Ribeiro et al., 2016).

Dispositional impulsivity is one risk factor for NSSI (Fox et al., 2015). However, although people with

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NSSI history report impulsive traits on various measures, they generally do not behave impulsively in laboratory tasks (Hamza, Willoughby, & Heffer, 2015). This is likely because “impulsivity” is a multidimensional construct, encompassing subcomponents with theoretically distinct contributions to psychopathology and behavior. The model of impulsive personality developed by Whiteside and Lynam (2001) and extended by Cyders and Smith (2008) supports the idea that certain traits are especially involved in NSSI. Individuals who engage in NSSI report low levels of perseverance and premeditation, but meta-analysis indicates that NSSI is most strongly associated with high levels of *negative urgency* ($g = 0.59$; Hamza et al., 2015). Longitudinal research suggests that negative urgency may specifically increase risk for NSSI onset, whereas lack of perseverance might promote NSSI maintenance (Riley, Combs, Jordan, & Smith, 2015). Evidence that NSSI is particularly related to impulsivity in response to negative emotion accords with theories conceptualizing emotion dysregulation as its core feature.

Despite its consistent link with self-reported impulsivity, few studies have observed impulsive behavior in NSSI. Empirical work by MacKillop and colleagues (2016) suggests that impulsivity’s latent structure comprises three categories: impulsive personality, impulsive action, and impulsive choice. This study thus sought to reconcile the discrepancy between elevated negative urgency (i.e., impulsive personality) and mixed findings for impulsive action in NSSI.

INHIBITORY CONTROL IN NSSI

Impulsive action results from impaired *inhibitory control*: a set of neurocognitive processes responsible for suppressing undesirable or inappropriate information and behavioral responses (Bari & Robbins, 2013). Inhibitory control includes three stages (Sebastian et al., 2013): (a) interference inhibition; (b) action restraint or *suppression* (early response inhibition); and (c) action cancellation or *termination* (late response inhibition). Interference inhibition refers to attentional/behavioral control in the face of distracting or competing stimulus characteristics (Nigg, 2000). Response inhibition involves restraining prepotent responses to infrequently presented stimuli (suppression) or “cancelling” already-initiated responses (termination), which demands the most cognitive resources (Sebastian et al., 2013). Failure in any of these processes may result in impulsive behavior, and studies have examined functioning at each stage in NSSI.

Two recent studies indicate comparable interference inhibition among participants with and without NSSI histories (Allen & Hooley, 2017;

Dahlgren et al., 2018). Research examining action suppression suggests no differences between NSSI and matched healthy control (Janis & Nock, 2009) or clinical control groups (McCloskey, Look, Chen, Pajoumand, & Berman, 2012). Past-month NSSI was also not associated with action suppression deficits among adolescents in residential treatment (Auerbach et al., 2014). Finally, four studies report equivalent overall action termination in NSSI and control groups (Allen & Hooley, 2015; Fikke, Melinder, & Landrø, 2011; Glenn & Klonsky, 2010; Lengel, DeShong, & Mullins-Sweatt, 2016).

What accounts for this inconsistency between self-reported and behavioral impulsivity? A large literature confirms that the relationship between self-report and performance-based impulsivity indices is weak, perhaps because they tap distinct aspects of this construct (MacKillop et al., 2016; Sharma, Markon, & Clark, 2014; Stahl et al., 2014). Indeed, Hamza et al. (2015) proposed that negative mood may be necessary to elicit impulsive behavior in laboratory tasks, given the role of negative urgency in NSSI etiology (Riley et al., 2015).

EMOTIONAL INHIBITORY CONTROL IN NSSI

Three recent studies have attempted to address this possibility. Allen and Hooley (2017) observed no difference between participants with and without NSSI histories on an interference inhibition task at baseline mood. Following a negative mood induction designed to activate self-critical schemas (participants wrote about a time they felt worthless, shameful, or failed to meet the expectations of someone important), the NSSI group reported increased negative mood but did not show worse interference inhibition than the control group. There was no relationship between changes in negative mood and interference inhibition, indicating that NSSI may not involve impaired inhibitory control over interference from *either* cognitive or emotional sources.

Lengel et al. (2016) similarly assessed impulsive action in NSSI before and after a negative mood induction. The authors asked participants to write a detailed account of their most stressful experience within the past year, which they listened to as an auditory recording in a subsequent session to induce negative mood. In line with other work, this study found no baseline response inhibition differences between NSSI and control groups, and consistent with Allen and Hooley (2017), increasing negative mood did not impair response inhibition in the NSSI group. These findings suggest that negative mood does not promote impulsive action in NSSI. In sum, NSSI is not apparently associated with inhibitory deficits at baseline mood or with difficulty inhibiting emotional interference during negative mood.

If negative mood does not impair inhibitory control in NSSI, elevated negative urgency may instead reflect failure to inhibit impulses directly motivated by negative emotion, or deficits in negative emotional response inhibition (NERI). The above studies indicate that the ability to redirect one's attention from distracting negative emotional interference (e.g., inhibiting negative mood states to focus on a task) is unaffected in NSSI. Difficulty controlling *urges* that naturally ensue from negative emotions (e.g., yelling when angry or crying when sad) may alternatively contribute to negative urgency in this population. Allen and Hooley (2015) directly assessed this possibility using a novel task requiring participants to quickly discriminate the valence (positive or negative) of affective images by keypress, while occasionally terminating initiated motor responses (and associated emotional reactions). Adults reporting NSSI demonstrated worse response inhibition *specifically* to negative affective stimuli, i.e., when attempting to terminate negative emotional reactions. To our knowledge, NERI dysfunction observed in that study—impaired *negative emotional action termination* (NEAT)—remains the only evidence of behavioral impulsivity in this population. This process fits theoretically with negative urgency in NSSI, which may reflect specifically impaired inhibitory control over negative emotional impulses, rather than generally impaired inhibitory control during negative emotional states.

This study's primary aim was to evaluate NERI dysfunction as a potential cognitive mechanism supporting negative urgency in NSSI. We thus sought to: (a) replicate the finding that NSSI involves impairment in late NERI, or NEAT; (b) explore if this deficit extends to an earlier stage of NERI, *negative emotional action suppression* (NEAS); and (c) examine associations between early and late NERI, negative urgency, and NSSI history. Deficits in either inhibitory process may contribute to negative urgency and help explain its relationship to NSSI.

Late emotional response inhibition was measured using the Emotional Stop-Signal Task (Allen & Hooley, 2015). We hypothesized that participants with a history of NSSI would commit more errors during stop trials with negative affective images, but not those with positive or neutral images, based on this prior work. We also predicted that the NSSI group would fail to terminate a greater proportion of negative emotional impulses, regardless of image valence (i.e., commission errors during stop trials in which participants classify stimuli as "negative"), compared to the control group. That is, accounting for the tendency to categorize images as negative in the absence of stop signals, we expected NSSI to be associated with impaired NEAT: difficulty interrupt-

ing negative emotional reactions *after* their initiation. We used the Emotional Go/No-go task to assess early emotional response inhibition (Hare et al., 2008). We defined NEAS as commission errors to negative no-go stimuli, which the task developers interpret as inhibitory deficits in negative affective contexts (cf. negative urgency; Tottenham, Hare, & Casey, 2011). We predicted that the NSSI group would demonstrate worse NEAS than the control group. Finally, we hypothesized associations between negative urgency and NSSI, as well as between negative urgency and impaired NERI on both tasks. We predicted that early and late NERI would each explain unique variance in negative urgency itself, as well as in its relationship with NSSI. In other words, we expected to observe indirect effects of negative urgency on the presence of NSSI history through NERI deficits. Although the cross-sectional design of this study precludes true mediation testing, observing indirect effects (indicating that NERI accounts for shared variance between negative urgency and the presence of NSSI history) provide preliminary evidence that these deficits might represent mechanisms underlying this trait in NSSI. Given this conceptualization of NERI as a set of cognitive processes contributing to negative urgency, we also expected associations between poor NERI and symptoms of psychopathology involving negative urgency (e.g., borderline personality traits, cyclothymia, and disordered eating; see Berg, Latzman, Bliwise, & Lilienfeld, 2015 for a meta-analytic review).

Method

PARTICIPANTS

Participants aged 18+ were recruited online via the university study pool and through advertisements posted in the community. The sample comprised a mix of local students and nonstudent adults seeking paid research opportunities. Interested individuals completed a web screening that collected demographic and psychiatric information. Eligibility criteria included English proficiency, no concussion history, and no impairments in motor ability, hearing, or vision. Participants provided informed consent before completing a 90-minute IRB-approved protocol that included counterbalanced self-report measures, a clinical interview, and cognitive tasks. Two groups of participants completed this session: those reporting a history of at least one NSSI episode using any method (confirmed via clinical interview; $n = 45$; 35 female) and control adults ($n = 43$; 34 female) reporting no history of NSSI, suicidality, psychiatric treatment, or psychological problems (including scores beneath thresholds on the symptom measures below).

Most participants in the NSSI group endorsed a history of at least one psychiatric diagnosis ($n = 31$, 69%), and many had multiple comorbidities ($n = 21$, 47% of the NSSI group). The most common reported diagnosis was depression ($n = 25$, 56%), followed by anxiety disorders ($n = 19$, 42%: generalized anxiety disorder $n = 8$; social phobia $n = 6$; posttraumatic stress disorder $n = 4$; panic disorder $n = 1$). Six participants reported eating disorders (13%), five reported developmental disorders/learning disabilities (11%), three reported bipolar disorder (7%), and two each (4%) reported alcohol or other substance use disorders, obsessive-compulsive disorder, and personality disorders. One participant (2%) reported a history of psychotic disorder. Eighteen participants endorsing NSSI history (40%) had received psychological or psychiatric treatment in the past month; 13 (29%) reported current psychiatric medication use.

PSYCHOPATHOLOGY MEASURES AND SELF-REPORTED IMPULSIVE TRAITS

We used the Self-Injurious Thoughts and Behaviors Interview (Nock, Holmberg, Photos, & Michel, 2007) to evaluate NSSI history. This measure has strong inter-rater reliability (mean $\kappa = 0.99$, $r = 1.0$), 6-month test-retest reliability (mean $\kappa = 0.70$, ICC = 0.44), and construct validity via correspondence with other measures of NSSI (Nock et al., 2007).

We assessed recent symptoms of emotional disorders using the Beck Anxiety Inventory (BAI; Beck & Steer, 1993) and the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996). Each contains 21 Likert-type items rated on a severity scale of 0–3. Both have established psychometric properties, including 1-week test-retest reliability (BAI: $r = 0.75$, Beck, Epstein, Brown, & Steer, 1988; BDI-II: $r = 0.93$, Beck et al., 1996), internal consistency (BAI: $\alpha = 0.92$ and BDI-II: $\alpha = 0.94$ in this study), as well as concurrent, discriminant, and criterion validity (BAI: Beck & Steer, 1993; BDI-II: Arnau, Meagher, Norris, & Bramson, 2001; Beck et al., 1996). We used cutoff scores of 26 (BAI) and 29 (BDI-II), corresponding to “severe” symptom levels (Beck & Steer, 1993; Beck et al., 1996) for healthy control (HC) group inclusion.

The Eating Disorder Examination-Questionnaire (EDE-Q; Fairburn & Beglin, 1994) is a 22-item measure rated on 7-point symptom frequency and severity scales. It has high internal consistency ($\alpha = 0.96$ in this sample), as well as concurrent and criterion validity in community samples of adults (Mond, Hay, Rodgers, Owen, & Beumont, 2004). We used a cutoff score of 2.3 on the global EDE-Q scale for inclusion in the HC group, which yields the best compromise between sensitivity and specificity in identifying individuals meeting DSM-IV

(American Psychiatric Association, 1994) eating disorder criteria (Mond et al., 2004).

We assessed borderline traits using the McLean Screening Instrument for borderline personality disorder (MSI-BPD; Zanarini et al., 2003), a 10-item yes/no self-report checklist. We used the recommended cutoff score (7 affirmative items), which is both highly sensitive and specific (both above 0.90 in young adults). This measure’s criterion validity is established in community samples (Patel, Sharp, & Fonagy, 2011). Zanarini et al. (2003) also report adequate 1-week test-retest reliability ($r = 0.72$) and internal consistency ($\alpha = 0.82$ in this sample).

Participants also completed the 17-item cyclothymia scale of the TEMPS-A (Akiskal, Akiskal, Haykal, Manning, & Connor, 2005), a yes/no self-report measure of temperamental mood lability. It has been validated in healthy and clinical samples (Akiskal, Mendlowicz, et al., 2005), has adequate test-retest reliability (6- to 12-month interval: $r = 0.68$; Akiskal, Akiskal, et al., 2005), and demonstrated acceptable internal consistency in the current sample ($\alpha = 0.87$). We used Akiskal and Akiskal’s (2005) suggested cutoff of 11 to identify HC group participants.

We used the UPPS-P (Lynam et al. 2006) to evaluate impulsivity, a 59-item self-report inventory with items rated on a 4-point Likert scale. It measures five traits: (a) negative and (b) positive urgency, rash action in response to negative or positive emotion, respectively; (c) (lack of) premeditation, action without forethought; (d) (lack of) perseverance, low tolerance for difficult or boring tasks; and (e) sensation seeking, pursuit of exciting or novel experiences. These subscales demonstrate convergent and discriminant validity (Cyders et al., 2007; Smith et al., 2007), as well as internal consistency in this sample (negative urgency $\alpha = 0.92$; positive urgency $\alpha = 0.95$; premeditation $\alpha = 0.81$; perseverance $\alpha = 0.87$; sensation seeking $\alpha = 0.88$).

EMOTIONAL RESPONSE INHIBITION TASKS

The Emotional Go/No-go task (Hare et al., 2008) measures early emotional response inhibition, using images of 10 adult faces (five males and five females) in five different expressions (neutral, happy, fearful, angry, and sad). Research shows convergent (Schulz et al., 2007) divergent validity (Casey et al., 2011) between nonemotional and emotional versions of this task. The Emotional Go/No-go requires participants to rapidly respond *only* to a given facial target that varies by task block. These “go” trials occur frequently in order to generate a prepotent response tendency. Participants must withhold responses to nontarget (“no-go”) facial stimuli that appear in 33% of trials. Emotional expressions are paired with

neutral expressions, and depending on the block, either the emotional face serves as the go stimulus (with neutral as no-go) or the no-go stimulus (with neutral as go). Participants completed eight randomized blocks of 30 trials in each condition. We defined early NERI/NEAS as the commission error rate to negative emotional nontarget faces. Prior studies have found associations between worse NEAS and childhood adversity (Johnson & Tottenham, 2015), depression-related constructs (Hjordt et al., 2017; Vanderhasselt, Kühn, & De Raedt, 2011), as well as aggression (Denny & Siemer, 2012), violence (Brugman et al., 2016), and psychopathy (Iria, Barbosa, & Paixão, 2012). Negative urgency is also related to recruitment of inhibitory regions during NEAS in functional imaging (Chester et al., 2016). We used E-Prime to present stimuli and collect data, excluding participants with less than 75% accuracy (see Johnson & Tottenham, 2015). Two NSSI participants had missing data, yielding a sample of $n = 83$ (NSSI: $n = 41$; HC: $n = 42$).

The Emotional Stop-Signal Task (Allen & Hooley, 2015) is a modification of the traditional version, which evaluates late response inhibition (Verbruggen & Logan, 2008). In the original, participants discriminate between serially displayed left and right arrows by pressing the key corresponding to the direction of the arrow (“go” trials). However, unpredictable auditory stop signals appear almost immediately after arrows on 25% of trials, indicating that participants should terminate their initiated response (“stop” trials). These stop signals occur with a variable delay that is adjusted to ensure that roughly half of stop trials result in errors, to calculate the time necessary for successful inhibition, known as stop-signal reaction time.

In lieu of arrow stimuli, the emotional version uses 36 images from the International Affective Picture System (Lang et al., 2008) comprised of neutral, positive, and negative categories (12 of each type).¹ Images were selected based on equivalent standardized ratings of valence intensity in the positive and negative categories, with neutral images being rated as neither positive nor negative. All three categories were matched in terms of arousal, with normative ratings within the “not arousing and not unarousing” range. Participants quickly and accurately distinguish the valence of each image (positive or “pleasant” vs. negative or “unpleasant”) by key-press. As in the standard task, 25% of trials include a stop signal indicating that participants should inhibit behavioral responses and associated emotional

reactions. Participants completed four blocks of randomized images from all three categories, for 192 total trials. Given that this task’s overall commission error rate approximates 50% by design, deviation from this rate indicates the relative difficulty of inhibiting reactions to different stimuli. We thus examined commission error rate separately in each image category to replicate the previous association between NSSI and worse response inhibition to negative stimuli. The primary outcome was late NERI/NEAT: failure to terminate initiated motor responses reflecting negative emotional impulses across all stimulus categories. We used Inquisit software to present stimuli and collect data. We restricted task analyses to participants with hit rates above 75% and omission error rates under 25%, consistent with the Emotional Go/No-go; omission errors occur when participants wait for stop signals, rather than quickly responding as instructed. This resulted in a sample of $n = 79$ for analyses involving this task (NSSI: $n = 41$; HC: $n = 38$).

STATISTICAL PROCEDURES

We first derived descriptive statistics from interview responses in the NSSI group and then conducted χ^2 and t -tests comparing groups on demographic, clinical, and self-reported impulsivity variables, after examining normality and applying transformations as appropriate; we report skewness (R_{sk}) and kurtosis (R_{ku}) for transformed variables with absolute values above 1 (descriptive statistics reflect original values to aid interpretation). We also adjusted outliers in past-year and lifetime NSSI frequency to account for potential recall biases and improve correlation estimates, replacing values above three standard deviations from the mean with the next value higher than the largest number of NSSI episodes beneath this threshold. This resulted in two adjustments in past-year and one in lifetime frequency. We used t -tests to examine group differences in overall task performance; repeated-measures ANOVAs to examine the effects of group membership, stimulus valence, and trial type (to assess our central hypotheses regarding NSSI history and NERI) and follow-up t -tests to probe significant main effects and interactions. We evaluated linear associations among task performance and self-report measures with bootstrapped bias-corrected and accelerated (BCa) 95% confidence intervals (CIs) to reduce the likelihood of error, given that most variables violated normality assumptions even after applying transformations and the number of tests performed.

We conducted linear regression analysis with group membership and symptom measures as independent variables predicting task performance to determine the specificity of associations between NSSI history

¹ Neutral images: 2102, 2215, 2280, 2305, 2385, 2396, 2411, 2440, 2480, 2516, 2840, 8312; positive images: 1340, 2045, 2075, 2091, 2209, 2550, 4614, 5470, 5831, 8190, 8200, 8470; negative images: 2053, 2205, 2456, 2800, 2900, 3350, 6370, 6821, 9040, 9417, 9800, 9810.

and NERI, and also conducted linear regression to evaluate our hypothesis that early and late NERI account for separate variance in negative urgency. We used the PROCESS macro (Hayes, 2012) to test our hypothesis that NERI explains variance in the association between negative urgency and NSSI history (i.e., that negative urgency exerts an indirect effect on NSSI through NERI), and to examine alternative models. We also performed a series of exploratory, post-hoc zero-inflated negative binomial (ZINB) regression analyses to determine the presence of an indirect effect of negative urgency on past-year and lifetime NSSI frequency through NERI (see online supplementary materials). Note that NERI variables are “reverse coded” akin to (lack of) premeditation on the UPPS-P, as positive values indicate more errors and worse response inhibition; we use “NERI” rather than “worse NERI” throughout the Results section for simplicity. Participants with missing data were excluded from the appropriate analyses. We conducted analyses in SPSS 25.0 using two-tailed tests with $p < .05$ as the significance threshold and bootstrapping with 5000 samples to estimate 95% CIs for coefficients.

Results

SELF-REPORT MEASURES

The average age of NSSI onset was 14.45 years ($SD = 3.00$). The most commonly endorsed method of NSSI was cutting/carving skin (57.78%), followed by scraping skin (35.56%), self-battery (26.67%), burning (24.44%), and picking skin (22.22%). Forty percent endorsed some other method (e.g., self-flagellation). Twenty-five participants (55.56%) endorsed multiple methods (two: $n = 8$, 17.78%; three: $n = 9$; 20%; four to six: $n = 8$; 17.78%). Four participants (8.89%) reported past-week NSSI episodes ($M = 0.62$, $SD = 3.06$, $Mdn = 0$; $R_{sk} = 4.30$, $R_{ku} = 19.60$; within-group: $M = 7.00$, $SD = 8.83$, $Mdn = 4$), 12 (26.67%) reported past-month NSSI episodes ($M = 2.79$, $SD = 12.26$, $Mdn = 0$; $R_{sk} = 3.03$, $R_{ku} = 9.73$; within-group: $M = 10.46$, $SD = 22.66$, $Mdn = 1$), and 27 (60%) reported past-year NSSI episodes ($M = 22.24$, $SD = 61.31$, $Mdn = 2$; within-group: $M = 37.07$, $SD = 76.09$, $Mdn = 5$). Mean lifetime NSSI frequency was 187.40 episodes ($SD = 519.08$, $Mdn = 20$; $R_{sk} = 1.34$; $R_{ku} = 1.11$).

There were no group differences in gender, ethnicity, age ($R_{ku} = 2.87$), or education; the NSSI group had more participants who identified as nonheterosexual, and reported more psychopathology symptoms (Table 1). Five NSSI participants (11.11%) each endorsed severe symptoms of anxiety and depression on the BAI and BDI-II, respectively; 18 surpassed the EDE-Q threshold for eating disorders (40%); nine met MSI-BPD criteria (20%),

and 11 exceeded the TEMPS-A cyclothymia cutoff (24.44%). The NSSI group also reported higher negative urgency [$M = 29.20$, $SD = 8.64$; $t(75.39) = 5.57$, $p < .001$, Cohen's $d = 1.18$], lack of perseverance [$M = 22.07$, $SD = 6.37$; $t(86) = 3.77$, $p < .001$, Cohen's $d = 0.81$], and positive urgency [$M = 23.80$, $SD = 9.28$; $t(75.93) = 3.17$, $p = .002$, Cohen's $d = 0.71$], compared to the control group (negative urgency: $M = 20.63$, $SD = 5.54$; perseverance: $M = 17.63$, $SD = 3.88$; positive urgency: $M = 18.67$, $SD = 5.00$). There were no group differences in premeditation or sensation seeking.

TASK PERFORMANCE

Emotional Go/No-Go

There were no group differences in reaction time, omission errors, or commission errors. We conducted a 2 (valence: positive vs. negative) \times 2 (trial: go vs. no-go) \times 2 (group: control vs. NSSI) repeated-measures ANOVA for commission errors, observing main effects of valence [$F(1, 81) = 18.76$, $p < .001$, partial $\eta^2 = .19$] and trial [$F(1, 81) = 157.85$, $p < .001$, partial $\eta^2 = .66$], as well as a significant interaction between the two [$F(1, 81) = 43.26$, $p < .001$, partial $\eta^2 = .38$]. Follow-up tests revealed that participants had a higher commission error rate during blocks containing negative emotional stimuli ($M = 11.14\%$, $SD = 7.30\%$) compared to positive stimuli ($M = 9.16\%$, $SD = 8.62\%$), $t(82) = 2.17$, $p = .03$, Cohen's $d = 0.24$. Participants also made more errors when trying to suppress emotional responses, i.e., during emotional no-go/neutral go blocks ($M = 15.74\%$, $SD = 10.50\%$) than during neutral no-go/emotional go blocks ($M = 4.56\%$, $SD = 6.08\%$, kurtosis = 2.16), $t(82) = 10.33$, $p < .001$, Cohen's $d = 1.36$. These main effects were qualified by a Valence \times Trial interaction: participants made more errors to neutral no-go faces when go faces were positive ($M = 6.60\%$, $SD = 10.39\%$, kurtosis = 2.36) rather than negative ($M = 2.49\%$, $SD = 3.64\%$, kurtosis = 3.00), $t(82) = 3.87$, $p < .001$, Cohen's $d = 0.53$. Commission error rate was highest during NEAS blocks (negative no-go/neutral go faces; $M = 19.80\%$, $SD = 12.89\%$), which involved more errors than positive no-go/neutral go blocks [$M = 11.70\%$, $SD = 11.88\%$; $t(82) = 5.62$, $p < .001$, Cohen's $d = 0.62$] and negative go/neutral no-go blocks [$t(82) = 13.74$, $p < .001$, Cohen's $d = 2.05$]. We did not observe any effects of group membership on early emotional response inhibition in this task. Given these null findings, we ran exploratory ANOVAs restricting the NSSI group to participants who surpassed clinical thresholds on symptom measures, and also using three comparison groups (control vs. past-year NSSI vs. prior NSSI), which both yielded comparable results.

Table 1
Demographic and Clinical Characteristics

	NSSI group (<i>n</i> = 45)	HC group (<i>n</i> = 43)
Sex: $\chi^2(1, 88) = 0.02, p = .88, \phi = .02$		<i>n</i> (%)
Female	35 (77.78)	34 (79.07)
Male	10 (28.57)	9 (20.93)
Orientation: $\chi^2(1, 88) = 10.61, p = .001, \phi = .02$		<i>n</i> (%)
Heterosexual	29 (64.44)	40 (93.02)
LGBT/Q	16 (35.56)	3 (6.98)
Ethnicity: $\chi^2(4, 88) = 6.01, p = .20, \phi = .26$		<i>n</i> (%)
White/Caucasian	28 (62.22)	18 (41.86)
Black/African-American	6 (13.33)	4 (9.30)
Asian	8 (17.78)	13 (30.23)
Hispanic/Latinx	1 (2.22)	3 (6.98)
Mixed/Other	2 (4.44)	5 (11.63)
Age: $t(86) = 0.63, p = .53, \text{Cohen's } d = 0.10$	24.22 (6.53)	23.58 (7.18)
Years education: $t(86) = 0.98, p = .33, \text{Cohen's } d = 0.21$	14.58 (1.84)	14.18 (1.92)
BAI: $t(86) = 5.50, p < .001, \text{Cohen's } d = 1.16$	12.91 (9.03)	4.09 (4.36)
BDI-II: $t(79.59) = 7.69, p < .001, \text{Cohen's } d = 1.63$	15.91 (10.78)	3.67 (4.28)
EDE-Q Global: $t(74.12) = 3.85, p < .001, \text{Cohen's } d = 0.82$	1.96 (1.59)	0.82 (0.70)
MSI-BPD ¹ : $t(75.44) = 3.96, p < .001, \text{Cohen's } d = 2.18$	4.84 (2.13)	0.71 (1.13)
TEMPS-A Cyclothymia: $t(86) = 7.42, p < .001, \text{Cohen's } d = 1.58$	7.93 (3.99)	2.74 (2.67)

Note. NSSI = nonsuicidal self-injury; HC = healthy control; BAI = Beck Anxiety Inventory; BDI-II = Beck Depression Inventory-II; EDE-Q = Eating Disorder Examination-Questionnaire; MSI-BPD = McLean Screening Instrument for Borderline Personality Disorder. Values are *M* (*SD*) unless otherwise indicated. ¹One NSSI participant had incomplete MSI-BPD data.

Emotional Stop-Signal Task

We did not observe group effects in overall reaction time, stop-signal reaction time ($R_{ku} = 5.38$), omission errors, or commission errors ($R_{sk} = 2.59, R_{ku} = 11.00$). A 3 (valence: neutral vs. positive vs. negative) \times 2 (group: control vs. NSSI) repeated-measures ANOVA revealed a main effect of valence [$F(2, 154) = 217.80, p < .001, \text{partial } \eta^2 = .74$] as well as an interaction between valence and group [$F(2, 154) = 4.24, p = .02, \text{partial } \eta^2 = .05$] on commission errors. Post-hoc *t*-tests indicated worse inhibition to emotional stimuli (positive: $M = 51.97\%, SD = 14.89\%$; negative: $M = 49.63\%, SD = 17.04\%$) than to neutral stimuli [$M = 37.80\%, SD = 14.15\%, \text{kurtosis} = 2.55; t(78) = 22.25, p < .001, \text{Cohen's } d = 3.46$ and $t(78) = 18.08, p < .001, \text{Cohen's } d = 2.92$ for positive and negative images, respectively]. There was no difference in commission errors to positive and negative stimuli in both groups combined; however, follow-up *t*-tests confirmed that the NSSI group made more commission errors to negative stimuli [Table 2; $t(77) = 2.05, p = .04, \text{Cohen's } d = 0.46$], in the absence of group differences to positive or neutral stimuli.

We ran a 2 (trial: go vs. stop) \times 2 (group: control vs. NSSI) repeated measures ANOVA on negative emotional classification responses to assess the association between NSSI history and NEAT (Figure 1). This analysis evaluated the tendency to

judge images as negative in the absence of inhibitory demands (i.e., the percentage of go trials in which participants made negative emotional responses, regardless of image valence) relative to the percentage of stop trials in which participants failed to inhibit negative emotional judgments across all image types (i.e., late NERI). There was a main effect of group, indicating that NSSI participants made more negative judgments overall [$F(1, 77) = 11.78, p = .001, \text{partial } \eta^2 = .13$], and a Group \times Trial interaction [$F(1, 77) = 10.84, p = .002, \text{partial } \eta^2 = .12$]. As predicted, *t*-tests confirmed that the NSSI group had worse NEAT than the control group [$t(77) = 3.73, p < .001, \text{Cohen's } d = 0.84$; see Figure 1 and Table 2], with no group differences in negative emotional judgments during go trials. The NSSI group also made more negative emotional classification responses during stop trials relative to go trials, $t(40) = 3.15, p = .003, \text{Cohen's } d = 0.49$. The control group, in contrast, had equivalent rates of negative emotional responses across trial type.

ASSOCIATIONS BETWEEN NERI TASK PERFORMANCE AND SELF-REPORT

Reaction time and omission errors on the tasks were correlated, $r(74) = .31, \text{BCa } 95\% \text{ CI: } .10 - .48$ and $r(74) = .29, \text{CI: } .01 - .53$, respectively. There were no associations between total commission errors, or

Table 2
Emotional Response Inhibition Task Performance

Commission Error %	NSSI group ¹	HC group ²
Emotional Go/No-go task		
Neutral stimuli no-go	4.76 (6.50)	4.37 (5.71)
Positive stimuli no-go	11.20 (11.44)	12.10 (12.40)
Negative stimuli no-go (NEAS)	21.14 (9.96)	18.49 (15.23)
Emotional Stop-Signal Task		
Neutral stimuli stop	37.24 (13.19)	38.41 (15.27)
Positive stimuli stop	50.06 (15.13)	54.03 (14.55)
Negative stimuli stop	53.34 (16.78)*	45.62 (16.62)
Negative stop responses across stimuli (NEAT)	46.22 (14.05)***	35.28 (11.85)

Note. NEAS = negative emotional action suppression; NEIT = negative emotional action termination. Values are given as *M* (*SD*) in percent. **p* < .05; ****p* ≤ .001. ¹NSSI group *n* = 41 for both tasks; ²Control group *n* = 42 for Emotional Go/No-go and *n* = 38 for Emotional Stop-Signal Task analyses.

between commission errors to specific types of stimuli. As Table 3 shows, there was an association between early and late NERI (CI: .003 – .39), and both correlated with negative urgency (early CI: .05 – .45; late CI: .11 – .46). NEAS was further associated with cyclothymic temperament scores on the TEMPS-A (CI: .06 – .36) and positive urgency (CI: .05 – .42). Partial correlations suggested that negative and positive urgency accounted for overlapping variance in NEAS, i.e., neither correlation was significant when controlling for the other variable. NEAT was correlated with scores on all clinical measures. We conducted a linear regression predicting NEAT from group membership and scores on significantly associ-

ated clinical measures to confirm that the association between NSSI history and NEAT was not accounted for by psychopathology symptoms. This analysis determined that NSSI history was the only variable to explain significant variance in NEAT (Table 4). Our hypothesis that early and late NERI account for distinct variance in negative urgency was supported by another linear regression shown in Table 4. NSSI frequency was not associated with NERI on either task (see online supplementary materials). We also conducted *t*-tests comparing individuals who engaged in past-year NSSI vs. those who did not and observed no group differences in NERI. We observed no other correlations with NSSI frequency aside from

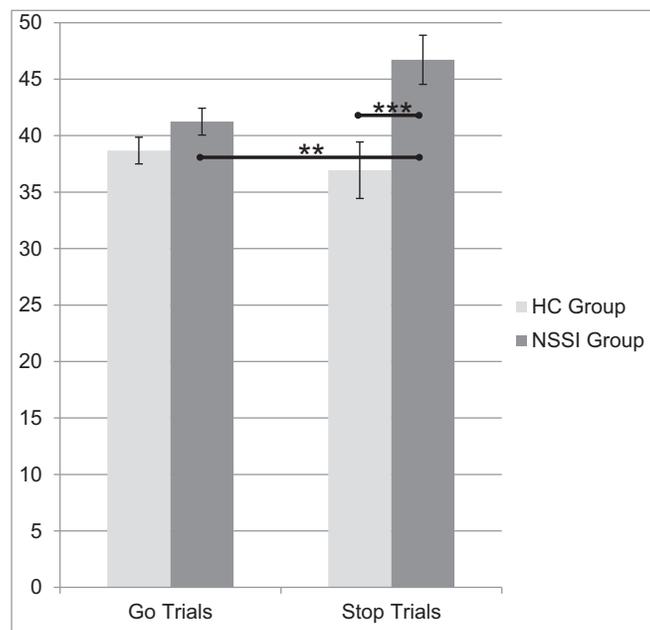


FIGURE 1 Percentage of negative responses by trial type on the Emotional Stop-Signal Task. Note. ***p* ≤ .01; ****p* ≤ .001. Note. HC = healthy control; NSSI = nonsuicidal self-injury.

Table 3
Pearson Correlations Among NERI, Psychopathology Symptoms, and Impulsive Traits (N = 88)

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. NEAS (early NERI) ¹											
2. NEAT (late NERI) ²	.21*										
3. BAI	.17	.25*									
4. BDI-II	.17	.34*	.66*								
5. EDE-Q Global score	.08	.31*	.31*	.46*							
6. MSI-BPD ³	.14	.25*	.53*	.69*	.44*						
7. TEMPS-A Cyclothymia	.22*	.34*	.53*	.71*	.39*	.72*					
8. Negative urgency	.26*	.27*	.44*	.62*	.40*	.64*	.63*				
9. Lack of premeditation	.05	-.07	.09	.13	-.01	.22*	.14	.41*			
10. Lack of perseverance	-.01	.22	.23*	.42*	.08	.37*	.37*	.47*	.40*		
11. Sensation seeking	.09	.11	-.13	-.10	-.15	-.03	.11	.05	.14	.04	
12. Positive Urgency	.24*	.26*	.29*	.38*	.27*	.47*	.50*	.73*	.34*	.35*	.34*

Note. NERI = negative emotional response inhibition; NERI variables are reverse-coded, such that higher values reflect worse inhibitory control over negative emotional impulses. *Indicates significant linear correlation coefficients, based on bootstrapped bias corrected and accelerated confidence intervals (CIs) with 5000 samples, i.e., 95% CIs that do not include zero. ¹n = 83; ²n = 79; ³n = 87.

BDI-II scores [past-month $r(45) = .24$, CI: .03 – .50; past-year $r(45) = .26$, CI: .02 – .48; lifetime $r(45) = .31$, CI: .05 – .52].

We next examined a model employing logistic regression to predict the presence or absence of NSSI history (i.e., group membership) from negative urgency, with NEAT entered as a “mediator” to estimate an indirect effect. This model significantly predicted NSSI history overall, $\chi^2(2, N = 79) = 30.99$, $p < .001$, Nagelkerke’s $R^2 = .43$, with significant direct effects of negative urgency on NEAT, NEAT on NSSI, and negative urgency on NSSI (see Figure 2). As hypothesized, negative urgency had a significant indirect effect on NSSI history through NEAT ($b = 0.02$, $SE = 0.02$, CI: 0.004 – 0.06). The results of this analysis were supported by comparing correlations between group membership and negative urgency before [$r(79) = .51$, CI: .34 – .67] and after controlling

for NEAT [$r(76) = .46$, CI: .28 – .61], which suggested that this cognitive process accounted for approximately 5-8% of the variance in the association between negative urgency and NSSI history. We evaluated two alternative models to elucidate the specificity of these relationships. First, we evaluated whether perseverance also exerted an indirect effect on NSSI group membership through NEAT, which produced a significant overall model [$\chi^2(2, N = 79) = 23.81$, $p < .001$, Nagelkerke’s $R^2 = .35$], with direct effects observed for perseverance ($b = 0.16$, $SE = 0.06$, CI: 0.06 – 0.31) and NEAT ($b = 6.13$, $SE = 2.09$, CI: 2.76 – 11.08) on NSSI group, but no effect of perseverance on NEAT (and no indirect effect on NSSI). We constructed a final alternative model using BDI-II scores to predict NSSI group, again including NEAT as a “mediator” to determine the presence of an indirect effect. This model was also

Table 4
Modeling Relations Among Early and Late NERI, Psychopathology, and Negative Urgency

DV: NEAT	B (SE)	Bootstrapped p	BCa 95% CI	β
Constant	0.30 (0.04)	$p < .001$	0.21 – 0.37	
Group (HC = 0, NSSI = 1)	0.10 (0.05)	$p = .04$	0.01 – 0.19	.34
BAI	-0.01 (0.05)	$p = .90$	-0.11 – 0.12	-.02
BDI-II	0.01 (0.02)	$p = .71$	-0.03 – 0.03	.07
EDE-Q Global score	0.04 (0.04)	$p = .20$	-0.02 – 0.12	.18
MSI-BPD	-0.01 (0.01)	$p = .21$	-0.03 – 0.01	-.24
TEMPS-A Cyclothymia scale	0.02 (0.02)	$p = .36$	-0.02 – 0.06	.17
Model summary: $F(6, 71) = 3.09$, $p = .01$, $R^2 = .21$				
DV: Negative Urgency	B (SE)	Bootstrapped p	BCa 95% CI	β
Constant	16.21 (2.38)	$p < .001$	11.33 – 21.01	
NEAS (early NERI)	39.28 (18.40)	$p = .03$	3.10 – 76.01	.22
NEAT (late NERI)	14.25 (5.60)	$p = .01$	3.70 – 24.55	.24
Model summary: $F(2, 71) = 5.26$, $p = .007$, $R^2 = .13$				

Note. Estimates derived from bootstrapping with 5000 samples.

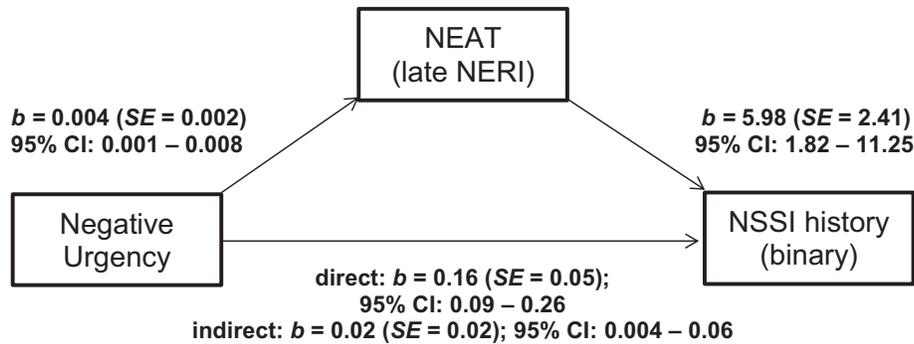


FIGURE 2 Proposed conceptual model of negative emotional response inhibition and statistical results. Note. Direct and indirect effects are expressed on a log-odds metric. Unstandardized coefficients and 95% confidence intervals (CI) derived from bootstrapping with 5000 samples.

significant [$\chi^2(2, N = 79) = 47.47, p < .001$, Nagelkerke's $R^2 = .60$], including direct effects of BDI-II on NEAT ($b = 0.03, SE = 0.009, CI: 0.01 - 0.05$) and on NSSI ($b = 1.22, SE = 0.29, CI: 0.66 - 1.77$). However, NEAT did not have a direct effect on NSSI in this model, nor was there evidence for an indirect effect. Post-hoc ZINB regression models suggested no effects of negative urgency or NEAT on past-year or lifetime NSSI frequency (see online materials), consistent with correlation estimates but in contrast to logistic regression models treating NSSI history as a binary outcome.

Discussion

This study sought to extend previous work by clarifying the nature of emotional response inhibition in NSSI and how it relates to self-reported negative urgency. We replicated the previous association between NSSI and poor late response inhibition to negative affective images in the Emotional Stop-Signal Task, without group differences to neutral or positive images (Allen & Hooley, 2015). On each trial in this task, participants rapidly judge the valence of the presented image as positive or negative. Although the NSSI and control groups were equally likely to categorize images as negative in the absence of a stop signal, the NSSI group had more difficulty inhibiting negative categorization responses (theoretically reflecting negative emotional reactions) when a stop signal occurred—a process we define as *late NERI* or *NEAT* (Figure 1). In other words, participants from the NSSI and control groups demonstrated a similar amount of negative behavioral/emotional responses in contexts without inhibitory demand, but those with NSSI history were less able to terminate those responses after they were initiated.

Our primary aim was to evaluate whether impaired NERI represents a cognitive process underlying the association between negative urgency and NSSI. Our

hypotheses were partially supported: early and late NERI explained independent variance in negative urgency, but only NEAT accounted for a small but significant portion of the variance in the relationship between negative urgency and NSSI history. Alternative models evaluating lack of perseverance and depressive symptoms did not produce indirect effects through NEAT, providing preliminary evidence for the specificity of this proposed mechanism. Although worse NEAT correlated with various psychiatric symptoms, NSSI history accounted for these associations in regression. Our findings suggest that NEAT dysfunction may reflect one pathway to negative urgency in NSSI.

People who engage in NSSI might report that they behave impulsively when distressed due to difficulty controlling urges accompanying their negative affect—only *after* those urges surpass a certain temporal or intensity threshold. This implies that a causal event could actually precede NSSI by hours, through a slow increase in negative affect that goes unnoticed until it reaches sufficient intensity to generate a behavioral impulse that cannot be suppressed. This creates the impression of acting “impulsively” under distress, because the emotional state and urge were not attended to until after the behavior occurred. From a clinical perspective, promoting self-awareness (of urges/emotional states) and implementing alternative behavioral routines *preventatively* could thus be a helpful strategy; for example, learning to respond quickly and habitually to mild frustration or a fleeting desire to self-injure with a distracting activity.

Another goal of the study was to assess emotional impulse suppression in NSSI using the Emotional Go/No-go task. As in prior research, participants made more commission errors to emotional faces than to neutral faces and during blocks including negative faces (Johnson & Tottenham, 2015; Tottenham et

al., 2011). We found an exception for more commission errors during neutral no-go blocks with positive faces compared to negative faces, possibly reflecting a positive interpretive bias. This could also explain the relatively low percentage (below 50%) of negative classification responses on the Emotional Stop-Signal Task (Figure 1).

Although results supported the hypothesized association between NEAS and negative urgency, we found no evidence of impaired NEAS in the NSSI group. This follows work suggesting that NSSI does not involve deficient negative emotional interference inhibition (Allen & Hooley, 2017; Lengel et al., 2016), the earliest stage of inhibitory processing (Sebastian et al., 2013). As predicted, NEAS and NEAT were correlated, but accounted for separate variance in negative urgency and showed distinct (if overlapping) patterns of association with clinical measures. These results are consistent with the notion that action restraint and action cancellation represent neurocognitively dissociable processes, and support the validity of these tasks to probe different stages of emotional response inhibition. Results also accord with meta-analysis indicating that response inhibition is the facet of impulsivity most related to negative urgency (Cyders & Coskunpinar, 2011). However, the magnitude of negative urgency's association with NERI is larger than with traditional measures of (nonemotional) response inhibition, and conceptually, NERI fits closely within urgency theory articulated by Cyders and Smith (2008).

Prior studies have established that NSSI is not associated with impulsive action in nonemotional contexts. This study adds to the line of research examining inhibitory control over negative emotion at each stage of inhibition in NSSI, as negative urgency might reflect deficits in any of these processes. The present study extends prior research demonstrating normative interference inhibition in the context of negative mood (stage 1), by also finding no evidence for NEAS deficits (stage 2). This stage involves restraining an impulsive act before it is initiated, as opposed to action termination (stage 3), which involves terminating an ongoing response *after* initiation. The present findings, in combination with Allen and Hooley (2015), provide evidence that impulsive behavior in NSSI might be restricted to terminating ongoing negative emotional action impulses. Although NEAT accounted for relatively small statistical variance, the observed indirect effect of negative urgency on NSSI history through this cognitive deficit provides initial support for the idea that it might comprise one mechanism underlying impulsive personality associated with NSSI. These results suggest that the tendency to behave impulsively during negative mood associated with NSSI might be

specifically related to difficulty inhibiting already-initiated impulses motivated by negative emotion. Lack of deficits in the initial stages of emotional inhibitory control could be interpreted as normative abilities disengaging from negative mood (interference inhibition) and suppressing negative emotional impulses (e.g., to harm oneself or others) before acting (NEAS). However, once these impulses surpass a certain threshold (NEAT), individuals with NSSI history may find them more difficult to control.

Study groups were demographically similar. More NSSI participants identified as LGBT/Q, consistent with evidence for higher NSSI rates among sexual minorities (Fox et al., 2017). By design, control participants also reported fewer psychopathology symptoms. NEAT correlated with all symptom measures; although NSSI history explained the most variance in regression (i.e., symptoms did not fully account for group differences), more research is needed to draw firm conclusions regarding NERI deficits across psychopathology. Negative urgency is transdiagnostic, and as a proposed mechanism for this trait, we expect that NERI might operate similarly. This study adds to evidence that NSSI may be distinct from other conditions, however, in that NEAT is thus far the *only* inhibitory process implicated in these behaviors.

The specificity of this deficit also has therapeutic implications. Treatments promoting awareness of one's internal experiences (e.g., mindfulness-based interventions; see Heath, Carsley, De Riggi, Mills, & Mettler, 2016) may therefore prove clinically useful, by empowering clients to address mild distress and/or self-harm urges before they reach a "tipping point." Concrete strategies to delay or interrupt this process may also be important. For example, treatment could focus on increasing the latency between urges and action by limiting access to preferred self-injury tools and/or environments, in line with lethal means restriction, an empirically supported intervention for suicide (Yip et al., 2012). Consistent with Dialectical Behavior Therapy skills training, which reduces NSSI in patients with BPD (Linehan et al., 2015), clients might attach written reminders of goals or self-encouraging statements to NSSI tools or place pleasant visual cues in the environments in which they typically self-injure.

This study had several limitations. We cannot determine whether images were similarly perceived across study groups, as we did not directly assess valence intensity or arousal associated with task stimuli in this sample. The negative affective images may have elicited stronger aversive reactions in the NSSI group, possibly contributing to their relative difficulty inhibiting responses to those images. However, NSSI and control participants were equally likely

to categorize images as positive or negative in the Emotional Stop-Signal task, suggesting that negativity bias does not fully explain the observed NEAT deficit among NSSI participants.

The present findings may not be generalizable to severe psychiatric populations, given the relatively low NSSI frequency observed in this sample. Likely due to this limited variance and insufficient statistical power, we did not detect an association between NSSI frequency and NERI. In fact, BDI-II scores were the only correlate of NSSI frequency, which might also reflect low levels of overall psychopathology in this sample compared to other studies. Future work investigating whether these neurocognitive deficits are associated with NSSI in psychiatric patients is therefore needed. Post-hoc power analysis indicated that to detect a modest effect of $r = 0.30$, we achieved a power level of 0.55 for these correlations. We were thus adequately powered (at ~ 0.80) to detect only a relatively large minimum effect size of $r = 0.40$. Results from ZINB regression analyses likewise contrasted with models treating NSSI history as a binary outcome. In light of power analyses and substantial literature indicating associations between NSSI frequency and many of the variables we tested in the current study (e.g., negative urgency, BPD symptoms), we believe that limited sample size was the primary reason for failing to reject the null hypothesis in linear models (vs. logistic regression). However, individuals with a lifetime NSSI history comprise a heterogeneous population, warranting examination of these constructs and their underlying mechanisms in larger, more diverse samples.

We similarly found no NEAT differences among participants in the NSSI group with and without past-year episodes. This implies that NEAT dysfunction may not maintain NSSI, as individuals seem to “recover” from these behaviors while this underlying deficit remains. It is therefore possible that impairment in this process might be a risk factor for NSSI or a “scar” (i.e., a consequence that persists after NSSI cessation). As negative urgency precedes NSSI onset (Fox et al., 2015; Riley et al., 2015), we expect that NEAT deficits may correspondingly increase NSSI risk. These data are cross-sectional, however, and cannot disambiguate such temporal relationships. More, the indirect effect indicating overlapping variance in these constructs was small, suggesting that negative urgency in NSSI is likely supported by multiple mechanisms that have yet to be determined. The present results suggest that NEAT may be less sensitive to changes in frequency of NSSI engagement than other constructs involving impulsive behavioral reactivity to negative affective states (e.g., risky decision-making; see Allen, Fox, Schatten, & Hooley, 2019).

This study provides further evidence for a specific inhibitory deficit in NSSI: difficulty terminating initiated impulses driven by negative emotion. NSSI may be the manifestation of such impulses, providing quick relief from unpleasant affective states, before or despite the consideration of potentially enduring adverse consequences. We found preliminary support for our assertion that NERI is a cognitive mechanism underlying the tendency to act impulsively in the context of negative emotions. These data help reconcile discrepant findings between heightened negative urgency and inconsistent evidence for impulsive behavior in NSSI. This work further encourages future research elucidating the neural circuit underpinnings of NERI and negative urgency in NSSI, which might reveal objective biomarkers that could serve as novel intervention targets. Prospective studies with multimodal assessment (e.g., functional neuroimaging) are needed to identify the biological substrates of NEAT impairment and determine whether this process represents a modifiable neurocognitive risk factor for NSSI.

Conflict of Interest Statement

The authors declare that there are no conflicts of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.beth.2019.02.001>.

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