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Characterizing the course of non-suicidal self-injury: A cognitive neuroscience perspective

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ABSTRACT

Non-suicidal self-injury (NSSI) has received increasing recognition as a clinically significant phenomenon. Although in most individuals who engage in NSSI, this behavior is short-lived, for a significant proportion of these individuals, NSSI follows a chronic course. There is a need for research advancing our understanding of the mechanisms of risk for NSSI, and how these mechanisms may change over time to account for the persistence of this behavior. In the current paper, a conceptual framework is proposed for characterizing the processes underlying the transition from initial engagement in NSSI to a chronic trajectory of this behavior. In particular, a case is made for conceptualizing NSSI as a habitual behavior as defined within a cognitive neuroscience perspective, with support from the existing theoretical and empirical literature. Finally, potential mechanisms are articulated for the development of chronic NSSI within this conceptual framework and recommendations presented for empirically evaluating this conceptualization of NSSI in future research in this area.

1. Introduction

Non-suicidal self-injury (NSSI), defined as the direct and deliberate destruction of one's own bodily tissue in the absence of any suicidal intent (Nock, 2010), has only relatively recently received recognition as a clinically important phenomenon. That is, although NSSI has traditionally received less empirical attention than suicidal behaviors, it is increasingly recognized as a distinct and important clinical phenomenon in its own right (Muehlenkamp, 2005). In fact, NSSI as a distinct syndrome is included in DSM-5 as a disorder in need of further investigation (American Psychiatric Association, 2013). The neglect of NSSI in earlier research stemmed from the view that it exists on a continuum of severity with suicidal behaviors, with NSSI simply being a less severe form of self-injurious behavior (Brent, 2011; Liu et al., 2016). There is accumulating evidence, however, to challenge this assumption, with several recent studies suggesting that NSSI is a stronger predictor of future suicidal behavior than is its past history, particularly in adolescents (Asarnow et al., 2011; Wilkinson et al., 2011). Furthermore, a recent meta-analysis has found NSSI to be a significant predictor of prospective suicide attempts (Ribeiro et al., 2016). These findings highlight the clinical importance of this behavioral phenomenon.

NSSI is a highly prevalent behavior. This is especially true among adolescents, with lifetime prevalence rates of 13% to 24% in non-clinical samples (Heath et al., 2009; Jacobson and Gould, 2007; Muehlenkamp et al., 2012; Swannell et al., 2014), and 12-month prevalence rates ranging from 55% to 68% among psychiatric inpatients (Cha et al., 2016; Guerry and Prinstein, 2010). Although less research has focused on the course of NSSI than its prevalence, a review of longitudinal studies suggests that NSSI persists into adulthood for a substantial portion of individuals who initiate this behavior in adolescence (Selby et al., 2015). Indeed, although the majority of individuals who engage in NSSI more than once cease this behavior within a few years, it persists for more than five years for approximately 20% of these individuals (Whitlock et al., 2006). These findings regarding the persistence of NSSI are congruent with recent evidence that of all the risk factors studied to date, a past history of NSSI is the strongest predictor of its future recurrence, with a large pooled effect size observed for this relationship (Fox et al., 2015).

Although most studies to date have focused on identifying risk factors (i.e., what) for NSSI, there is a stated lack of research examining how risk develops and influences this behavior (Nock, 2012). Given that even a single incident of NSSI may be associated with significantly greater risk for negative psychiatric outcomes (Whitlock, 2010; Whitlock et al., 2006), there is a clear clinical need for studies delineating the mechanisms driving the transition from initial engagement in NSSI to a more persistent pattern of this behavior. Such work is important because, although many commonly studied risk factors for NSSI (e.g., female sex; Bresin and Schoenleber, 2015; Fox et al., 2015) help identify *who* is at risk, they are limited in their ability to advance

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our understanding of *how* to intervene with these individuals. In contrast, longitudinal studies designed specifically to elucidate the processes underlying risk for persistent NSSI can greatly inform the selection of meaningful targets for clinical intervention. Clarifying these underlying mechanisms is important for breaking the strong link that has been observed between past and future NSSI (Fox et al., 2015).

The current paper presents a conceptual framework for understanding and characterizing the processes underlying the transition from initial engagement in NSSI to a chronic trajectory of this behavior. Specifically, within a cognitive neuroscience perspective (Graybiel, 2008), habitual behaviors have been defined as: (i) not innate; (ii) evoked by specific contexts or stimuli; (iii) repetitive and becoming fixed over time; and (iv) occurring with little effort once fully acquired. With support from existing conceptualizations of NSSI models and empirical studies, a case is made for NSSI meeting this definition of a habitual behavior, focusing specifically on the latter two characteristics, and potential mechanisms are articulated for the development of chronic NSSI within this framework. Finally, included in this discussion are specific examples of how this conceptualization of NSSI may be empirically evaluated, with the view of guiding future research in this area. To guide this discussion, the main components of this cognitive neuroscience conceptualization of NSSI, along with specific hypotheses for testing each component, are presented in Fig. 1.

2. NSSI as a repetitive behavior that becomes fixed over time

In certain cases, behaviors become repetitive and fixed over time if they possess self-reinforcing properties. Based on DSM-5 criterion B (contingent response) for NSSI disorder (American Psychiatric Association, 2013), this may be the case for NSSI. This view is also consistent with the four-function model of NSSI (Bentley et al., 2014; Nock and Prinstein, 2004).[1] This model posits that NSSI is maintained by positive and negative self-reinforcing processes. These self-reinforcing processes include intrapersonal positive reinforcement (generating positive affective or cognitive states), intrapersonal negative reinforcement (reducing negative affective or cognitive states), interpersonal positive reinforcement (eliciting attention and help-seeking), and interpersonal negative reinforcement (facilitating removal from aversive social situations or decreasing interpersonal demands). In the case of intrapersonal negative reinforcement, for example, insofar as NSSI immediately reduces negative affect, the tendency to engage in this behavior should become stronger when confronted with negative affect in the future. This model has received empirical support, with intrapersonal negative reinforcement being the most commonly endorsed function (Bentley et al., 2014; Zetterqvist, 2015). Similarly consistent with this self-reinforcing conceptualization of NSSI, the experiential avoidance model of self-harm conceptualizes NSSI as a maladaptive emotion regulation strategy, specifically a form of emotional avoidance (Chapman et al., 2006). According to this model, when the individual experiences an aversive emotional response to a stimulus, avoidance behaviors such as NSSI are adopted. The short-term relief produced by NSSI serves to reinforce the adoption of this behavior as an avoidance strategy when confronted with future aversive stimuli. Furthermore, and of direct relevance to the conceptualization of NSSI as a repetitive and fixed behavior, its self-reinforcing nature is believed to lead over time to an automaticity in its adoption as an avoidance strategy.

Although these self-reinforcing characteristics of NSSI have been increasingly studied, they remain poorly characterized in the empirical literature. Specifically, the existing literature has been almost entirely reliant on self-report methodologies (Bentley et al., 2014). This is an important limitation for several reasons. First, recent studies have consistently revealed low congruency between self-report and behavioral or physiological measures of several constructs, including impulsivity (Cyders and Coskunpinar, 2012, 2011), self-control (Duckworth and Kern, 2011), emotion regulation in general (Vasilev et al., 2009), and distress tolerance in particular (Bernstein et al., 2011; McHugh et al., 2011). Therefore, self-report data on self-reinforcing properties of NSSI cannot be generalized to other measures of these properties. Second, self-report measures are limited inasmuch as individuals have imperfect insight into the processes underlying their behavior, and this is especially true for cognitive and affective processes that may exist, at least in part, outside of conscious awareness (Nisbett and Wilson, 1977). Thus, the validity and accuracy of self-report data regarding behavioral contingencies relating to NSSI must be viewed with a degree of caution (Nock et al., 2009), and laboratory tasks are required to clarify the mechanisms underlying NSSI (Bentley et al., 2014).

A few studies employing such tasks have been conducted, with one, for example, demonstrating a physically aversive stimulus (a frequently used experimental analogue for NSSI) to be associated with subsequent reduction in negative affect, as indexed by startle eye-blink reactivity (Franklin et al., 2010). Another study, albeit with a small college sample, has observed cessation of a physically aversive stimulus to be associated with a positive affective response and a reduced negative affective response, as indexed by the post-auricular reflex (PAR; Franklin et al., 2013a; Hebert et al., 2015; Quevedo et al., 2015) and startle eye-blink reflex, respectively (Franklin et al., 2013a). These findings were replicated in another study that included participants with NSSI (Franklin et al., 2013b). Although these studies are consistent with the view that NSSI has self-reinforcing properties, their cross-sectional nature cannot inform our understanding of the temporal dynamics of their relation to the development or maintenance of NSSI. Specifically, it is unclear to what degree these psychophysiological indices are prospectively predictive of NSSI re-engagement (i.e., a risk factor rather than concomitant or consequence; Kazdin et al., 1997; Kraemer et al., 1997). Moreover, it is unclear how the self-reinforcing properties of NSSI change over time in relation to the course of NSSI, particularly the trajectory of chronic NSSI.

Delineation of potential neural mechanisms underlying changes in self-reinforcing properties of NSSI during the course of this behavior is needed to identify promising targets for clinical intervention. That is, moving beyond a focus on self-reinforcement of NSSI solely at the physiological level to a study of changes in their associated neurocircuitry over the course of NSSI may yield specific, modifiable targets of intervention. The study of the neural processes underlying NSSI, however, is still in its infancy, and has predominantly involved assessing this behavior within the context of psychiatric diagnoses rather than as a transdiagnostic clinical phenomenon (Westlund Schreiner et al., 2015). One study of individuals who had engaged in NSSI (Osuch et al., 2014) observed a positive association between degree of relief after a physically aversive stimulus and blood oxygen level-dependent (BOLD) response in the dorsal striatum, a brain region involved in processing rewards (Everitt and Robbins, 2013; O'Doherty et al., 2004) and habitual behavior formation (Schiltz, 2006). This finding is notable because it mirrors prior research on habit formation in the broader literature, particularly in the context of other forms of psychopathology that similarly follow an often chronic course (e.g., substance use disorders and anorexia nervosa). Specifically, according to basic instrumental (operant) learning principles, if a behavior (e.g., NSSI) is followed immediately by a reward (e.g., reduced negative affect), the behavior is likely to be reinforced. Specific neural circuits, including the ventral striatal − posterior dorsomedial striatal network, have been linked with this effortful form of learning and involved in the acquisition of new behaviors (Everitt and Robbins, 2013; Walsh, 2013). With repeated engagement in the behavior (overtraining), followed

[1] In contrast to a syndromal approach focusing on categorizing behaviors based on topographical features (i.e., symptoms), functional approaches categorize behaviors based on functional processes underlying their occurrence and maintenance (i.e., their antecedents and consequences).

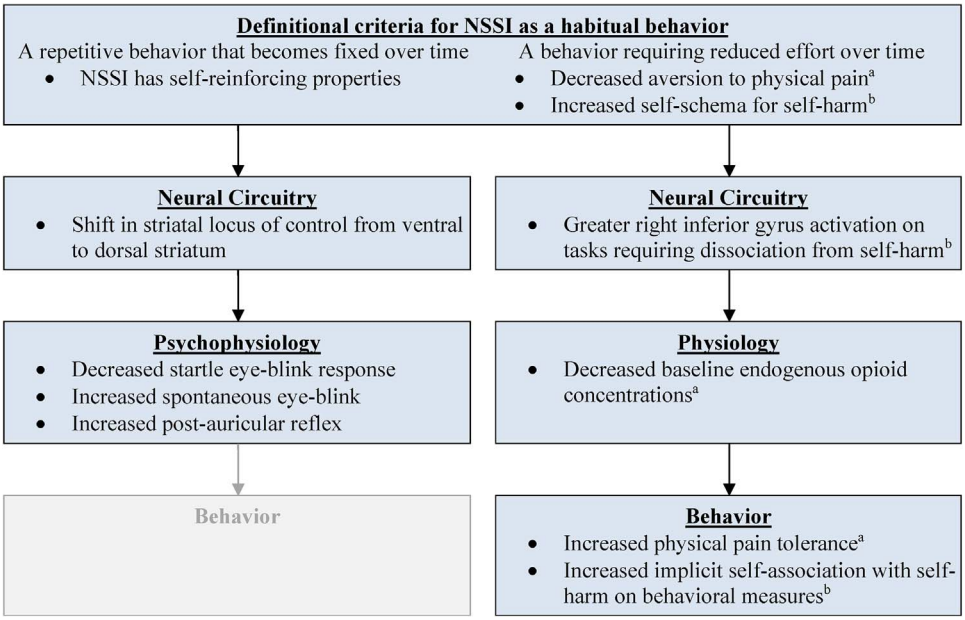

Fig. 1. A cognitive neuroscience model of NSSI as a habitual behavior and potential processes of change over time in response to self-harm stimuli across multiple units of analysis.
Note: For the second definitional criterion for habitual behaviors within a cognitive neuroscience framework, that NSSI in a behavior requiring reduced effort over time, corresponding processes are indicated by identical superscripts.

consistently by the reward, the behavior will become relatively insensitive to the reward, a process called stimulus-response (i.e., habit) learning. Here, the nucleus accumbens − anterior dorsolateral striatum circuit is involved (Everitt and Robbins, 2013). The transition from voluntary behavior (instrumental learning) to habitual behavior (stimulus-response learning) is reflected particularly by the shift in striatal locus of control from the ventral to dorsal striatum (Everitt and Robbins, 2013; Walsh, 2013). This shift in neural locus of control has been established in other areas with the progression from substance use to abuse and addiction (Everitt and Robbins, 2013; Schiltz, 2006). Support for this model has even been found for anorexia nervosa, with anorexic patients exhibiting greater BOLD activation in the dorsal striatum when making choices about food consumption than did healthy controls (Foerde et al., 2015). This finding suggests that self-starvation eventually becomes habitual and is not under effortful control. If this model holds true for NSSI, initial engagement in this behavior should be voluntary and goal-directed, the aim being to regulate affect. However, as NSSI is repeated over time, it becomes overtrained (i.e., a habit), being thereafter associated with greater activation of the dorsal striatum, as was found in the aforementioned NSSI neuroimaging study (Osuch et al., 2014). Given the cross-sectional nature of this study, however, the temporal changes in neural activation associated with the development of chronic NSSI put forth in this model remain to be evaluated.

Clarifying the neural pathways involved in the development of chronic NSSI is important inasmuch as it may yield novel targets for intervention. For example, deep brain stimulation, a reversible and non-destructive treatment that has been applied to other forms of habitual behaviors (e.g., obsessive-compulsive disorder), may offer some promise here, particularly when directed at specific brain regions implicated in these behaviors (e.g., the nucleus accumbens; Luigjes et al., 2012). In like manner, identifying the neural circuitry underlying persistent NSSI may yield potential targets for non-invasive neurostimulation methodologies (e.g., transcranial direct current stimulation [tDCS] and transcranial magnetic stimulation [TMS]) that have been found to produce a reduction in appetitive response for other self-reinforcing behaviors (e.g., disordered eating and substance dependence; Jansen et al., 2013).

Finally, an important limitation applicable to all studies to date interrogating the self-reinforcing aspects of NSSI on a physiological or neural regulatory level is the stimulus proxies used for NSSI. Prior studies in this area have used either electric shocks (Franklin et al., 2013a, 2013b) or thermal discomfort (Franklin et al., 2010; Osuch et al., 2014). Although such stimuli are well suited and frequently used in other studies assessing whether NSSI is associated with greater basic physical pain tolerance, they are limited in their ability to inform our understanding reinforcing properties of this specific behavior, due to the likely greater specificity of stimuli validly needed to conduct such assessments. Support for the importance of specificity of stimuli may be found in a recent study noting that individuals who engaged in cutting as a form of NSSI exhibited a greater implicit self-association with this behavior, when self-cutting stimuli were used, than did those with no history of NSSI (Cha et al., 2016). In contrast, those who only used other NSSI methods did not differ from those with no NSSI history.

Thus, although the aforementioned psychophysiological and neuroimaging studies are important to our basic understanding of psychophysiological experiences of physical pain, the possibility that their findings reflected a universal physiological response to painful stimuli in general, rather than to NSSI in particular, cannot be excluded from consideration. Consistent with this possibility, one of these studies featured an unselected undergraduate sample rather than individuals with NSSI (Franklin et al., 2013a), and another two found similar patterns of increased PAR and reduced startle eye-blink response to pain stimuli in both participants with NSSI and healthy controls, with essentially no between-group differences (Franklin et al., 2013b, 2010). Studies employing self-harm-related cues (e.g., self-harm concept words) are therefore needed to evaluate the specificity of the self-reinforcing properties of NSSI to individuals who engage in this behavior.

One potential approach for future research in this area may be to evaluate how the self-reinforcing properties of NSSI change over the trajectory of chronic NSSI by assessing whether this trajectory is associated with an increase in PAR (response to appetitive stimuli; Franklin et al., 2013a; Hebert et al., 2015; Quevedo et al., 2015) specifically to self-harm stimuli over time. Another promising possibility may be to examine how phasic shifts in eye-blink rate (EBR) may change across the course of NSSI. Spontaneous EBR is of particular relevance here for its value as an indicator of striatal dopamine, with tonic EBR reflecting tonic striatal dopamine levels and phasic changes in spontaneous EBR indicative of phasic release of striatal dopamine and thus reward responsivity (Peckham and Johnson, 2016). In support of the latter, phasic increases in EBR occurs as a result of pharmacological administration of a dopamine agonist (Blin et al., 1990) and in response to a positive affect induction and reward tasks (Akbari Chermahini and Hommel, 2012; Peckham and Johnson, 2016). Paralleling the finding

that drug-related cues lead to heightened phasic dopamine release with chronic drug use (Wanat et al., 2009), it may be that a chronic trajectory of NSSI is similarly associated with an increase in phasic EBR in response to self-harm stimuli over time. Finally, a complementary approach may be to assess whether a chronic trajectory of NSSI is associated with a shift over time in the striatal locus of control from the ventral striatum to the dorsal striatum in response to self-harm stimuli.

3. Repetitive NSSI as a behavior requiring reduced effort

This definitional criterion for habitual behavior is entirely consonant with DSM-5 criteria for NSSI disorder (criterion C: difficulty resisting the urge to engage in NSSI; American Psychiatric Association, 2013). Reduction in effort to engage in a behavior that is not innate, such as NSSI, may occur over time as a result of the erosion of natural impediments to the behavior (e.g., aversion to pain) following each occurrence. Indeed, theoretical support for this possibility is evident in the interpersonal theory of suicide (Joiner, 2005; Joiner et al., 2012). According to this theory, NSSI is a physically painful and initially fear-provoking behavior. Over time, however, with repeated engagement in NSSI, the individual habituates to the pain and fear associated with physical harm, thus reducing the effort required for future engagement in this behavior. Consistent with this view, a recent review found that in 10 out of 11 studies to date, individuals with NSSI exhibited greater pain tolerance than did healthy controls (Koenig et al., 2016). As noted in this review, however, none of these studies involved a prospective design. They leave unclear whether (i) differences in pain tolerance pre-exist and predict engagement in NSSI and/or (ii) pain tolerance increases subsequent to NSSI engagement and changes over the course of this behavior. More nuanced analyses employed within a longitudinal framework are therefore required to clarify the precise nature of the relation between NSSI and pain tolerance, particularly whether increased physical pain tolerance over time is associated with a chronic trajectory.

A potential mechanism underlying this greater pain tolerance, and resulting reduction in effort involved in initiating NSSI, is aberrant levels of endogenous opioids in individuals engaging in this behavior (Bresin and Gordon, 2013; Stanley et al., 2010). That endogenous opioids influence the perception of pain has been well documented (Fields, 2004; Yamada and Nabeshima, 1995). In support of the role of the endogenous opioid system in NSSI, individuals with a history of this behavior have been found to possess lower baseline cerebrospinal fluid β-endorphin and met-enkephalin relative to psychiatric controls (Stanley et al., 2010). One possible account of the lower baseline levels of β-endorphin and enkephalins evident in individuals with NSSI is that they result in hypersensitivity in corresponding opioid receptors, which in turn lead these individuals to experience the physiological effects of endogenous opioids (e.g., analgesia) all the more strongly when they are released following engagement in NSSI (Klonsky and Olino, 2008). In the absence of longitudinal studies in this area, however, it remains unclear whether these atypical baseline β-endorphin and enkephalins concentrations pre-exist NSSI onset or are an alteration consequent to engagement in this behavior. This issue is of particular relevance to the conceptualization of recurrent NSSI as a habitual behavior. Although the two possibilities are not mutually exclusive, evidence that aberrations in baseline endorphin levels are produced or accentuated by NSSI would be consistent with the view that decreased pain sensitivity may be a mechanism through which the effort required for this behavior reduces over time, facilitating the development of habitual NSSI. Future research employing multiple measures of endorphins across time is required adequately to evaluate this possibility.

An intriguing possible account of this increased pain tolerance, and resulting reduction of effort to engage in NSSI, may be found in studies of classical conditioning in the substance abuse literature. In particular, repeated pairing of a pre-drug conditional stimulus with a pharmacological unconditional stimulus has been noted to produce a conditional compensatory response (CCR) that counteracts the effect of the drug (Siegel, 2005). This phenomenon may in part account for physiological tolerance to drugs over time (Siegel et al., 2000). Such a process may be relevant to pain tolerance in recurrent NSSI. It may be, for example, that an individual who cuts as a form of NSSI experiences an anticipatory effect with repeated engagement in this behavior over time, such that picking up a sharp instrument with the intent of NSSI elicits a CCR. As a consequence of this increased tolerance, the individual may need to cut deeper to achieve the same effect. If this is indeed the case, empirical support for several hypotheses would need to be established in future studies. First, engagement in NSSI should generally worsen over the course of this behavior for a significant proportion of individuals (e.g., deeper cuts or increased number of cuts during subsequent occurrences of NSSI). Second, as mentioned above, increased pain tolerance over the course of this behavior would need to be demonstrated. Third, with this established, it would then be necessary to elucidate the physiological mechanisms through which tolerance develops, the aforementioned endogenous opioids hypothesis being one promising possibility.

Less effort may also be required over time to engage in NSSI if the individual increasingly identifies on some level with this behavior, as may occur through its repeated recurrence. One view that may provide an explanatory model for this possibility is the differential activation hypothesis (Teasdale, 1988). Although originally formulated as a cognitive reactivity model of depressive recurrence, it may be applicable to the development of chronic NSSI. Based upon semantic network theory, this cognitive model holds that early depressive episodes lead to the establishment of a depression-related cognitive processing network. In a manner not dissimilar to Hebbian learning at the neural level (i.e., the association between neurons that frequently fire together strengthen over time; Hebb, 1949), the association between depression and this negative cognitive network, particularly when activated by negative mood, may strengthen over time with their repeated temporal pairing. This model has received substantial empirical support, such that the cognitive reactivity it describes has been viewed as a candidate causal risk factor for depressive relapse and recurrence (Lau et al., 2004). If the differential activation hypothesis is applicable to NSSI, it may be that with repeated engagement in this behavior, NSSI may become increasingly entwined with the individual's self-schema. Relevant to this possibility, a few recent studies have assessed implicit self-associations with self-harm in individuals with a history of NSSI (Cha et al., 2016; Franklin et al., 2014; Glenn et al., 2016; Glenn and Klonsky, 2011; Nock and Banaji, 2007). Although the support is currently mixed for a longitudinal relation between implicit identification with self-harm and NSSI in the four studies to date (Cha et al., 2016; Franklin et al., 2014; Glenn et al., 2016; Glenn and Klonsky, 2011), there has been consistency across studies (Cha et al., 2016; Glenn et al., 2016; Glenn and Klonsky, 2011; Nock and Banaji, 2007; but also see Franklin et al., 2014, for an exception) in support of a significant cross-sectional relation. Furthermore, one recent study found implicit self-identification with self-harm to be positively associated with frequency of NSSI (Glenn et al., 2016). None of these studies assessed implicit self-identification with self-harm at multiple time-points, and thus it is unclear how it may change over time in relation to the course of NSSI. Within the current conceptual framework, a strengthening implicit self-association with self-harm over time should be associated with a chronic course of NSSI.

As with the study of the self-reinforcing properties of NSSI, research relating to reduced effort for chronic engagement in this behavior needs to extend beyond behavioral assessments toward an evaluation of potential neural underpinnings and how they may change over the development of chronic NSSI. As mentioned above, such work is important for its potential to lead to the identification of potential targets for intervention. Within this context, the right inferior frontal gyrus (rIFG) may be a region of particular interest, as it has been observed in several studies to be responsive to anodal tDCS (Cai et al., 2016;

Cunillera et al., 2014; Ditye et al., 2012; Stramaccia et al., 2015). The rIFG has been found to be involved in cognitive interference resolution (Berman et al., 2011). Lesions to this region result in impaired performance on neurobehavioral indices of interference resolution (Aron et al., 2003), and further support for IFG involvement in cognitive interference resolution comes from a meta-analysis of neuroimaging studies of such measures (Nee et al., 2007). If chronic NSSI indeed requires reduced effort relative to initial engagement in this behavior, it then follows that anything that is incongruent with this behavior would require increased effort. Moreover, if engagement in NSSI is positively associated with implicit self-identification with this behavior (Cha et al., 2016; Glenn et al., 2016; Glenn and Klonsky, 2011; Nock and Banaji, 2007), one would expect greater cognitive interference to be experienced and effort required (i.e., greater rIFG activation) on incongruent trials for a task measuring this implicit self-identification. The self-injury implicit association test offers a novel opportunity to assess this possibility. Specifically, in this paradigm, one would expect greater activation of the rIFG on trials requiring an individual with NSSI to associate the concept of "me" with the concept of "not harm" and reduced activation of this brain area on trials involving the pairing of the concepts "me" and "self-harm." Moreover, rIFG activation on incongruent trials should decrease over time with the development of chronic NSSI.

4. Conclusion

Although several psychological and behavioral models of NSSI have received increasing support in recent years, the current effort adds to these perspectives by advancing a cognitive neuroscience model of this behavior. Such advances are necessary to inform new areas of inquiry, particularly in uncovering the circuitry underlying the development of NSSI. There are relatively few longitudinal studies of NSSI risk factors, and the ones that do exist tend to assess these risk factors at a single time-point in relation to prospectively occurring NSSI. These static assessments of potential NSSI mechanisms cannot provide insight into how risk develops and changes over time. Therefore, research is particularly needed to examine how underlying processes of risk for NSSI change over its course.

Identifying processes of change over time in association with the development of a chronic trajectory of NSSI is important insofar as it may yield modifiable targets for intervention. That is, although identifying stable risk factors (e.g., sex, history of peer NSSI) has value for determining who is at risk, such risk factors cannot address clinically important questions of why these individuals are at risk, when they are most at risk, and thus when and how best to intervene. Contrastingly, processes of risk that change over time may hold value for safety monitoring of at-risk individuals. Targeting these modifiable processes may also avert the development of a chronic trajectory or benefit those who have already assumed this trajectory. In particular, identifying the mechanisms underlying the persistence of this behavior may inform our attempts effectively to replace it with more adaptive alternatives (e.g., adopting healthy behaviors with overlapping self-reinforcing properties; Wallenstein and Nock, 2007). Additionally, insofar as chronic NSSI is a habitual behavior, incorporating into treatment elements of habit reversal therapy, found to be efficacious with habit disorders such as trichotillomania (Bate et al., 2011), may offer promise for intractable cases.

This need for advances in our understanding of how to intervene with NSSI is pressing, given the current lack of empirically based treatment protocols for NSSI (Nock, 2012), with recent meta-analyses finding no evidence of an effect across 11 treatment studies for adolescents (Ougrin et al., 2015), and eight RCTs with adults (Calati and Courtet, 2016). Adding weight to this priority is the recent finding of a 24% increase in suicides in the U.S. over the last 15 years (Centers for Disease Control and Prevention, 2016). With NSSI being a stronger predictor of suicidal behavior than is its past occurrence (Asarnow et al., 2011; Ribeiro et al., 2016; Wilkinson et al., 2011), developing effective interventions for NSSI may indirectly address this public health concern. Finally, if supported, this cognitive neuroscience model of chronic NSSI as a habit may have some generalizability to other psychiatric phenomena. To provide just one example, depressive rumination shares certain properties that may warrant evaluation as a habitual behavior, particularly its fixed nature and the often reported feeling that it is productive, and thus self-reinforcing (Nolen-Hoeksema et al., 2008). Modeling the development of dynamic processes of risk for NSSI over its trajectory may therefore serve as a useful template for studying potentially comparable processes of risk underlying the development of these other psychiatric outcomes.

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