

Cognitive and Affective Pathways to Nonsuicidal Self-Injury Among Youth in the Adolescent  
Brain Cognitive Development (ABCD) Study

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**ABSTRACT**

Nonsuicidal self-injury (NSSI) is the deliberate destruction of one's own body tissue (e.g., cutting, skin picking, biting, hitting) without conscious suicidal intent. Cognitive and affective difficulties may contribute to the development and maintenance of NSSI, such that emotion regulation may mediate the link between cognitive control difficulties and NSSI in youth. This study examined developmental links between cognitive control and emotion regulation on several facets of self-injurious thoughts and behaviors in a large sample of youth, collected via the ABCD Study (N=6447). Although a mediation of emotion regulation on cognitive control and self-injurious thoughts and behaviors was not supported, important direct effects were found between neural correlates of inhibition (at ages 9-10 years) on NSSI at 11-12 years, and behavioral measures of cognitive flexibility (at 10-11 years) and inhibition (at 9-10 years) on suicidality at 11-12 years. Further, links between poorer cognitive control and poorer emotion regulation were found. An exploratory aim of this study was examining the potential moderating role of autistic traits on significant associations. Although greater autistic traits significantly predicted presence of self-injurious thoughts and behaviors, this study did not find a moderation of autistic traits. These results provide developmental risk markers for NSSI and suicidality in youth.

**Cognitive and Affective Pathways to Nonsuicidal Self-Injury Among Youth in the  
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**GENERAL AUDIENCE ABSTRACT**

Nonsuicidal self-injury (NSSI) is the direct and intentional harm to one's own body (e.g., cutting, skin picking, biting, hitting) without suicidal intent. One's ability to regulate their cognitions and emotions may explain risk and continuation of NSSI and other suicidal thoughts and behaviors. In specific, one's ability to regulate their emotions may explain the relationship between cognitive control and NSSI in youth. This study examined the relationship between cognitive control and emotion regulation on NSSI and suicidality in a large sample of youth, collected from the ABCD Study (N=6447). Although emotion regulation did not explain the relationship between cognitive control and NSSI or suicidality, results showed that brain activation when trying to inhibit a response at ages 9-10 related to presence of NSSI at ages 11-12. Additionally, behavior related to one's ability to flexibly shift (at ages 10-11) and inhibit responses (at ages 9-10) related to suicidality at ages 11-12. Links between poorer cognitive control and poorer emotion regulation were also found. Recent work has also found that autistic youth have high rates of NSSI and suicidality, thus, the level of autistic traits on these relationships were evaluated. Although greater autistic traits significantly predicted presence of self-injurious thoughts and behaviors, this study did not find that level of autistic traits impacted links between cognitive control, emotion regulation, and NSSI or suicidality. These results provide developmental risk markers for NSSI and suicidality in youth.

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## Contents

Cognitive and Affective Pathways to Nonsuicidal Self-Injury Among Youth in the Adolescent Brain Cognitive Development (ABCD) Study.....		1
Existing Theoretical Frameworks of Self-Injurious Thoughts and Behaviors.....		1
Proposed Theoretical Model.....		4
Evidence for Cognitive and Affective Alterations in those with NSSI.....		5
Potential Moderating Role of Autistic Traits on Mechanisms of NSSI.....		9
Adolescent Brain Cognitive Development (ABCD) Study.....		9
Study Aims & Hypotheses.....		10
Method.....		11
Participants.....		11
Measures.....		12
Statistical Analysis Plan.....		17
Results.....		19
Correlations.....		19
Follow-up Regressions for Mediation Analyses.....		22
Moderation Analyses.....		28
Discussion.....		34
Limitations & Future Directions.....		39
Conclusion.....		42
References.....		44

## **Cognitive and Affective Pathways to Nonsuicidal Self-Injury in Youth**

Nonsuicidal self-injury (NSSI) is the deliberate destruction of one's own body tissue (e.g., cutting, skin picking, biting, hitting) without conscious suicidal intent (Favazza, 1989; Nock & Prinstein, 2005). Roughly 17% of adolescents in community samples (Swannell et al., 2014) and 60% of adolescents in psychiatric samples report history of NSSI (Kaess et al., 2013). Although some prior work has explored social and environmental correlates of NSSI in adolescence (i.e., bullying, childhood adversity; Hankin & Abela, 2011), considerably less is known about the cognitive and affective vulnerabilities that may potentiate and maintain NSSI in young ages. Consequently, cognitive and affective markers have not yet been used to develop mechanistically-informed prevention and intervention programs for NSSI in youth, despite the potential for such markers to predict and suppress self-injury. Accordingly, the purpose of this project is to test a novel model of NSSI in adolescents involving theoretically relevant cognitive mechanisms of NSSI such as inhibition, cognitive flexibility, and emotion regulation. More specifically, emotion regulation, or the ability to adaptively modulate emotional responses (Gross & Thompson, 2007; Mazefsky et al., 2013), may mediate the link between underlying cognitive control vulnerabilities (i.e., difficulties in impulse control and cognitive flexibility) and self-injury. Further, it is predicted that these links may be moderated by autistic traits, as emerging work has found that individuals with high autistic traits are at greater risk for self-injury and suicidality (Culpin et al., 2018). The over-arching working hypothesis of this work is that cognitive and affective vulnerabilities may contribute to the development and maintenance of self-injury, and specifically that emotion regulation may mediate the link between poor cognitive control and NSSI in adolescents, especially in adolescents with high autistic traits.

### **Existing Theoretical Frameworks of Self-Injurious Thoughts and Behaviors**

Prior evidence suggests that self-injurious thoughts and behaviors may serve to modulate current affect, similar in principle to the general function of broader emotion regulation capacities, such that engaging in self-injury typically allows for escape from a negative affective state (Brown et al., 2002; Chapman et al., 2006; Klonsky, 2007; McKenzie & Gross, 2014; Nock & Prinstein, 2004, 2005). In fact, some work has suggested that the self-regulatory aspect of NSSI may be a central function of NSSI in adolescents, as compared to other potential functional objectives such as generating a feeling, social attention, and social escape (Nock & Prinstein, 2004, 2005). Consistent with this perspective, young adults with self-injury have been found to endorse more difficulties related to accessing emotion regulation strategies, lack of emotional clarity, and difficulties with impulse control compared to typical peers (Gratz & Roemer, 2008; Heath et al., 2008; Whitlock et al., 2015). Moreover, this work has been extended by statistical models (i.e., path analysis, negative binomial hurdle model) which demonstrate that poorer emotion regulation capacities predict NSSI (Jenkins & Schmitz, 2012; Zelkowitz et al., 2016).

The idea that diminished emotion regulation capabilities are linked to NSSI is also consistent with the *Experiential Avoidance Model* of self-harm (Chapman et al., 2006), which posits that high intensity emotions in combination with poor emotion regulation capacities contribute to the development and maintenance of self-harm, such that individuals engage in self-harm behaviors when emotions are intensely experienced and adaptive emotion regulation strategies are unavailable or difficult to access. According to this model, individuals with this propensity may be prone to using self-harm behaviors to alleviate intense emotional experience. This model further posits that cognitive skills such as high impulsivity, and novelty-seeking may be key factors which contribute to experiential avoidance (Chapman et al., 2006). Although this theory has been developed primarily on the basis of data obtained from young adult samples, it



has recently been extended to adolescents (Brausch & Woods, 2018; Robinson et al., 2019). For example, one study found bidirectional relationships longitudinally between emotion regulation and self-harm in adolescents 12-15 years of age (Robinson et al., 2019). Moreover, various facets of impulsivity have been associated with self-harm in adolescents (Lockwood et al., 2017). Altogether, there is support for the *Experiential Avoidance Model*, such that difficulties with impulsivity as well as emotion regulation have been separately identified as precursors to self-harm in adolescents.

Another mechanism that has been highlighted as an important factor in self-harm is cognitive flexibility, or the ability to shift thoughts or responses and create new solutions to problems (Powell & Ragozzino, 2017). Cognitive flexibility was linked theoretically to NSSI by Selby and colleagues (2008), who proposed the *Emotional Cascade Model* of NSSI which identifies repetitive negative thinking, a form of cognitive inflexibility in which one gets perseverates on a negative feeling, thought, or state (Arditte et al., 2016; Burrows et al., 2017), as a key factor that intensifies negative emotions. Thus, this model posits that NSSI is used to alleviate the compounding cycle between repetitive negative thinking and negative emotion (Nicolai et al., 2016; Selby et al., 2010, 2013). This cycle is hypothesized to be further reinforced by the immediate relief that NSSI has on distressing thoughts and feelings, making it likely for individuals to continue using NSSI as a future coping method. This model has been supported by use of ecological momentary assessment in adolescents and young adults with NSSI, such that high levels of negative affect predicted NSSI, followed by increased positive affect and diminished negative affect after NSSI behavior (Kranzler et al., 2018). Moreover, in individuals with high impulsivity the compounding cycle between repetitive negative thinking and highly salient negative emotion is bidirectional, with exponential effects predicting self-harm (Selby et al., 2016). The role of

cognitive flexibility on NSSI has been supported with specific facets of repetitive negative thinking (i.e., brooding, reflection, rumination) as predicting NSSI, such that adolescents and adults with more repetitive negative thinking have more frequent NSSI (Gong et al., 2019; Polanco-Roman et al., 2015; Richmond et al., 2017). Despite work examining self-report of repetitive negative thinking, research examining performance on cognitive flexibility measures with NSSI has been limited. This is surprising considering that measures of diminished cognitive flexibility have been linked with related factors of depression and suicidality (Martin et al., 1991; McGirr et al., 2012).

### **Proposed Theoretical Model**

Although the *Emotional Cascade* and *Experiential Avoidance Models* highlight the importance of emotionally-valenced cognitive processes, there is little work examining the stable cognitive processes on NSSI (i.e., impulse control and cognitive flexibility), as they relate to and potentiate emotion regulation processes. Although these cognitive processes have been proposed as risk markers for self-injurious thoughts and behaviors (i.e., including suicidality) broadly, they have not been thoroughly examined in NSSI. Plus, prior work has established that several distinctive facets of impulsivity may mechanistically impact self-injurious thoughts and behaviors (Hamza et al., 2015; Lockwood et al., 2017). Moreover, although the link between cognitive inflexibility and NSSI has not been thoroughly examined, there is work supporting links between cognitive inflexibility and depression and suicidality (Martin et al., 1991; McGirr et al., 2012) two highly related outcomes to NSSI (Lin et al., 2018; Nock & Prinstein, 2005). Thus, given the current theoretical models of the *Experiential Avoidance* (Chapman et al., 2006) and *Emotional Cascade* (Selby et al., 2008) *Models* of NSSI, which highlight cognitive and affective processes, in addition to prior work in depression and self-injurious thoughts and behaviors (i.e., including suicidality) broadly, the current project proposes a theoretical model (see Figure 1), which emphasizes

cognitive and affective processes on NSSI. More specifically, emotion regulation was hypothesized to potentiate the link between difficulties with cognitive control (i.e., poor impulse control and cognitive flexibility) and NSSI. Further, given emerging work linking high rates of NSSI in autism spectrum disorder, a neurodevelopmental disorder characterized by restricted and repetitive behaviors and social communication difficulties (American Psychiatric Association, 2013; Cassidy et al., 2018; Culpin et al., 2018; Maddox et al., 2016; Moseley et al., 2019), we proposed that higher autistic traits may moderate the links between cognitive control, emotion regulation, and NSSI, such that the relationships between (1) poor cognitive control and poor emotion regulation, (2) poor cognitive control and NSSI, and (3) poor emotion regulation and NSSI, may be stronger among youth with higher autistic traits (Figure 1).

[Insert Figure 1]

## **Evidence for Cognitive and Affective Alterations in those with NSSI**

### ***The Role of Impulsivity on NSSI***

Consistent with the *Experiential Avoidance Model* of NSSI and the current proposed model, impulsivity may be both a risk and maintenance factor for NSSI. For example, individuals with self-harm describe the behavior as difficult to stop in the moment and “addicting” (Moseley et al., 2019). Impulsivity is broadly defined as encompassing behaviors that are characterized by an inclination toward quick, unplanned reactions to internal or external stimuli with little consideration for the consequences of these reactions on the self or others (Moeller et al., 2001). Under this definition, impulsivity encapsulates both inhibitory control and impulsive decision-making. Impulsivity has been measured through several self-report and task-based measures; more specifically, a variety of neurocognitive measures of inhibitory control have been used to examine the links between inhibition and self-injury, i.e., stroop, go/nogo, stop signal tasks (Allen &

Hooley, 2015; Giannetta et al., 2012). Tasks that measure impulsive decision-making, i.e., Iowa Gambling Task, have also been used to examine the links between impulsivity and self-injury (Oldershaw et al., 2009). A recent literature review collapsing these two types of tasks found a medium-to-large effect for the relationship between impulsivity (i.e., go/no go, stop signal task, continuous performance task, delay discounting) and both suicidality and self-harm (McHugh et al., 2019).

In terms of self-harm specifically, previous work has found that preadolescent youth with self-harm have demonstrated higher self-reports of impulsive behavior, but not with neurocognitive measures i.e., Stroop, when compared to healthy controls (Giannetta et al., 2012). Adolescents with current self-harm trended toward worse performance on the Iowa Gambling Task, such that they demonstrated more high-risk choices than both adolescents who self-harmed in the past and healthy controls (Oldershaw et al., 2009). One study examining executive function in adolescents with self-harm found that distinct facets of executive function characterize low-severity vs. high severity NSSI, such that adolescents with high-severity NSSI demonstrated poorer strategy use on a spatial working memory task, while those with low-severity NSSI demonstrated more inhibitory control deficits on a stop signal task (Fikke et al., 2011), suggesting that low impulse control may be an initial risk factor for NSSI, but broader executive functioning measures may characterize maintenance of NSSI long-term. Thus, although emerging work has demonstrated some differences with inhibitory control and impulsive decision-making with self-harm and suicidality more broadly, it is currently unknown how impulse control translates specifically to NSSI.

### ***The Role of Cognitive Flexibility on NSSI***

Cognitive flexibility is defined as the ability to switch thoughts or behaviors in order to generate new solutions in face of challenges (Powell & Ragozzino, 2017). As noted earlier, facets of cognitive flexibility have been highlighted in the *Emotional Cascade Model* of NSSI such as repetitive negative thinking and reinforcement from relief of distress as maintaining NSSI (Selby et al., 2008). Emerging work has identified the links between cognitive flexibility and habitual behaviors (i.e., obsessions, compulsions, behavioral addictions), such that those with poorer cognitive flexibility may be more prone to habitual behaviors and have greater difficulty breaking habits (Gillan et al., 2011; Robbins et al., 2012). Currently, there is little work examining cognitive flexibility in NSSI, though this mechanism has been proposed as a neurocognitive factor that may aid in understanding severe and chronic NSSI (Liu, 2017), with this perspective being supported by findings of altered frontal-striatal activation and circuitry in those with NSSI (Davis et al., 2014). Although there is sparse literature on cognitive flexibility in NSSI, a breadth of work has examined cognitive flexibility in related clinical outcomes, i.e., depression, suicidality.

Poor cognitive flexibility has specifically been associated with clinical outcomes such as depression and suicidality, though the link between cognitive flexibility and NSSI has been far less studied. One standardized measure of cognitive inflexibility is the Wisconsin Card Sorting Task (WCST). In the WCST participants are instructed to match cards that appear in a fixed random order to one of four cards. Cards can be matched by color, shape, or number, though participants are never told the sorting 'rule.' Feedback is given after each match attempt on whether the match was correct/incorrect. After 10 consecutive correct matches, the sorting rule changes. Cognitive inflexibility is typically measured by the amount of perseverative errors (i.e., the number of times the participant uses the old sorting rule after a rule change). Previous work has linked rumination in adults to more WCST perseverative errors and failure to maintain sets (Davis &

Nolen-Hoeksema, 2000). Further, participants with depression demonstrate more perseverative errors WCST as compared to non-psychiatric controls (Martin et al., 1991). Moreover, when examining WCST in an inpatient sample, Marzuk and colleagues (2005) found that depressed inpatient participants with suicidal ideation demonstrated more perseverative errors on the WCST than depressed inpatient participants without suicidal ideation, in addition to poorer performance on other cognitive measures, i.e., processing speed and attention. Further, WCST performance has been found to predict 6-month suicidal ideation in young adults with a history of suicide attempts (Miranda et al., 2012). Moreover, adults with high lethality suicide attempts have poorer WCST performance than those with low lethality attempts and nonsuicidal adults (McGirr et al., 2012). These differences in WCST performance are not always present when examining inpatient participants with suicidality vs. individuals without (Ellis et al., 2016) or in predicting self-harm (Polanco-Roman et al., 2015). Despite cognitive flexibility being extensively studied in the context of suicidality, little work has examined how NSSI and cognitive inflexibility specifically interact. This gap in work is surprising considering that NSSI is a precursor to suicide attempts and thus may share similar mechanistic pathways that reinforce and maintain self-harm. Moreover, identifying this mechanism in youth may be particularly beneficial in identifying those at highest risk for chronic and severe forms of NSSI (Liu, 2017).

### ***Mechanisms of Emotion Regulation in NSSI***

Several theories have implicated a strong relationship between emotion regulation and NSSI, due to the potential regulatory function NSSI plays on extreme negative affective states (Chapman et al., 2006; Nock & Prinstein, 2004; Selby et al., 2008). Although several studies have examined self-reported emotion regulation (e.g., Brausch & Woods, 2018; Davis et al., 2014; Robinson et al., 2019), few studies have examined emotion regulation in the context of behavioral

paradigms that can tap into task-based mechanisms. When using emotionally-valenced stimuli (i.e., positive/negative stimuli) within a stop signal task, adults with a history of NSSI demonstrated poorer inhibition toward negatively-valenced stimuli compared to controls (Allen & Hooley, 2015). Moreover, one study using a cognitive reappraisal paradigm in adults with and without self-harm found that those with self-harm had greater self-reported negative emotions following a sad film clip, and greater amygdala activity (Davis et al., 2014). Thus, in emotionally-valenced contexts, individuals with NSSI may demonstrate altered behavior reflecting poor emotion regulation.

### **Potential Moderating Role of Autistic Traits on Mechanisms of NSSI**

Recent work has demonstrated that individuals with autism spectrum disorder have strikingly high rates of NSSI (50-63.6%; Cassidy et al., 2018; Maddox et al., 2016), suicidal ideation (64%), and plans or attempts (32%; Cassidy et al., 2014). Moreover, autistic traits have been linked with suicidality and self-harm broadly (Culpin et al., 2018), though there is little work mechanistic work to understand why. Although NSSI in autism spectrum disorder has not been formerly studied in the context of cognitive control or emotion regulation, there is work to suggest links exist between autism symptoms (i.e., restricted and repetitive behaviors) linked to cognitive inflexibility and inhibition (e.g., Mosconi et al., 2009; Mostert-Kerckhoffs et al., 2015) and difficulties with emotion regulation (Gotham et al., 2014; Keenan et al., 2017; Mazefsky et al., 2012). Thus, it is important to understand the role that autistic traits may have on the proposed model, as there may be unique variance related to autistic traits which may not be captured by cognitive control and emotion regulation on NSSI.

### **Adolescent Brain Cognitive Development (ABCD) Study**

The Adolescent Brain Cognitive Development (ABCD) Study (Casey et al., 2018) is a prospective longitudinal study that is following 11,878 adolescents across ten years at 21 sites, starting at age 9-10 years. Data has been made available via the National Institutes of Health (NIH) Data Archive, with new data released yearly. Previous work from ABCD has found that at baseline, in a large sample of 9-10 year olds, 9.1% of participants reported presence of NSSI (DeVille et al., 2020). Moreover, this dataset is unique in that it has collected behavioral, cognitive, and biological measures in youth, making it the optimal dataset to examine cognitive and affective vulnerabilities that may underlie NSSI. Examining these links is particularly important during childhood and adolescence, as the brain undergoes several changes in the prefrontal cortex during time period (Giedd, 2004; Gogtay et al., 2004; Luna et al., 2010), which have been linked to a variety of cognitive control (Larsen & Luna, 2018) and emotion regulation (McRae et al., 2012) abilities, and thus may helpful in identifying youth at risk for NSSI.

### **Study Aims & Hypotheses**

**Primary Outcomes:** Based on mechanisms highlighted by the *Experiential Avoidance* and *Emotional Cascade Models* of NSSI, we aimed to examine emotion regulation as a mediator between measures of cognitive control and NSSI.

**Aim 1:** Determine whether emotion regulation difficulties explained the relationship between cognitive control (i.e., cognitive flexibility, inhibition) difficulties and presence of NSSI in youth.

**Hypothesis 1a:** Poorer emotion regulation (as evidenced by slower response time on the emotional stroop task) would mediate the link between poorer inhibition (as evidenced by poorer performance and diminished activation in lateral prefrontal cortex (LPFC), anterior cingulate cortex (ACC), and striatum on the stop signal task) and presence of NSSI. The specificity of these links would also be tested using outcome measures of suicidality.



**Hypothesis 1b:** Poorer emotion regulation (as evidenced by poor response time on the emotional stroop task) would mediate the link between poorer cognitive flexibility (as evidenced by poorer performance on the dimensional card sort task) and presence of NSSI. The specificity of these links would also be tested using outcome measures of suicidality.

**Exploratory Aim:** Based on emerging work of high rates of NSSI in autism, it was important to understand the role of autistic traits on links between cognitive control, emotion regulation, and NSSI.

**Aim 2:** Evaluate the moderating role of autistic traits on NSSI mechanisms in youth.

**Hypothesis 2a:** Those with higher autistic traits would have stronger links between cognitive control difficulties, emotion regulation difficulties, and presence of NSSI. The specificity of these links would also be tested using outcome measures of suicidality.

## Method

### Participants

Participant data that were used in analyses were collected by the Adolescent Brain Cognitive Development (ABCD) Study (<https://abcdstudy.org/>), which is a multisite (n=21) longitudinal study with aims of examining brain development and child health (Casey et al., 2018). Projected data collection of the ABCD cohort is yearly for 10 years, beginning with ages 9-10 years. Participants have completed measures of social, emotional, and cognitive development, in addition to health and environmental factors, including fMRI scanning every other year. Baseline data of the ABCD Study is available for 11,878 youths ages 9-10 years and their families. At the time of the present study, longitudinal follow-up was ongoing. The baseline sample is considered to be representative of the United States population in terms of sociodemographic factors (i.e.,

race, ethnicity, income and education levels, and living environments; Garavan et al., 2018). Measures from three timepoints of data were used in this study, the baseline data (at ages 9-10 years), second timepoint (at ages 10-11 years), and third timepoint (at ages 11-12 years). Data were included in this study for 6,447 youth that had either child-reported or parent-reported data for NSSI and suicidality at timepoint 3 (11-12 years). Tables 3 and 4 indicate participant characteristics and rates of NSSI and suicidality at 11-12 years in this sample.

[Insert Table 3]

[Insert Table 4]

## **Measures**

### ***Independent Variable: Stop Signal Task (SST)***

The SST (Logan, 1994) is a measure of inhibition and has been linked to neural mechanisms and measures of impulsivity and impulse control (Whelan et al., 2012). While undergoing fMRI participants completed the SST using e-prime. For this task, participants viewed a black arrow pointing left or right on a gray background, and were instructed to press the corresponding button on a button box to the direction of the arrow “as quickly and accurately as possible,” with their dominant hand. If a “Go” stimulus (i.e., right or left arrow) is followed unpredictably by a signal to stop (i.e., up-right arrow), participants must withhold their button response (Figure 2; Casey et al., 2018).

A total of 360 trials are completed across two runs. Each trial lasts 1000 ms; “Stop” trials include 300 ms of stop signal presentation at variable onset intervals (see description of tracking algorithm below) within the 1000 ms trial time. In order to establish a prepotent “Go” response, 83.33% (150/180 trials per run) were “Go” trials and 16.67% (30/180 trials per run) were “Stop” trials. Trials were evenly split between right and left facing arrows. Intertrial intervals range from

700 ms – 2000 ms, thus Stimulus Onset Asynchrony (SOA) ranges from 1700 ms to 3000 ms (mean SOA=1904 ms). Between 1 to 20 “Go” trials separated “Stop” trials (mean=4.91 trials). Twelve trial orders were developed; all orders had the first trial of the runs as a “Go” trial.

A tracking algorithm was used to guarantee that each participant had approximately 50% successful and 50% unsuccessful inhibition trials for Stop trials; this algorithm varied the interval between the onset of the right/left arrow and the onset of the stop signal. This interval was initially set to 50 ms; if a participant has unsuccessful inhibition, the task was made easier by reducing the interval by 50 ms on the next Stop trial, and if there was successful inhibition, the task is made more difficult by increasing the interval by 50 ms on the following Stop trial.

A key variable that has been previously identified in the literature are stop signal response time (SSRT). In order to more specifically capture inhibitory processes, regions of interest (ROIs) were probed from the “*correct stop trials vs. incorrect stop trials*” contrast. Preliminary results from the ABCD study have demonstrated activation of the LPFC, ACC, and striatum as involved in *correct* inhibition (Casey et al., 2018), thus these areas were used as ROIs. Independent variables in the analyses included SSRT mean, *correct stop vs. incorrect stop activation* of LPFC, ACC and striatum. Variables from this task were derived from baseline data (9-10 years).

[Insert Figure 2]

***Independent Variable: The Toolbox Dimensional Change Card Sort (TDCCS) Task<sup>®</sup>***

The TDCCS measures cognitive flexibility (Zelazo, 2006; Zelazo et al., 2013, 2014). Participants are presented with cards containing objects (i.e., white rabbit, green boat) at the bottom of the screen and are instructed to sort a third card that is presented in the middle of the screen by either color or shape to match cards at the bottom of the screen (Figure 3; Zelazo, 2006; Zelazo et al., 2013, 2014). Responses are recorded by touch input. Participants complete a practice

round, followed by a block of trials sorting on one of the dimensions (i.e., color), followed by a block of trials in which they are told to switch to the other dimension (i.e., shape). Lastly, they complete a block of trials where they alternate in pseudorandom order between sorting on shape versus color. TDCCS total score, which is age-normed and based on accuracy and RT, was used as an independent variable of interest. This variable was only collected at the second timepoint (10-11 years).

[Insert Figure 3]

### ***Mediator: Emotional Stroop Task***

An emotional word – emotional face Stroop task was developed for adolescents using age-appropriate face stimuli (Banich et al., 2019; Luciana et al., 2018). This task was administered through Inquisit ([www.millisecond.com](http://www.millisecond.com)). Participants were tasked with identifying the emotional valence of a word in the context of task-irrelevant faces that were either congruent or incongruent with the emotional valence of the word. Participants were instructed to determine whether each word describes a “good” feeling (i.e., happy, joyful, glad) or a “bad” feeling (i.e., angry, mad, upset) using a Right and Left key presses, respectively (Figure 4; (Banich et al., 2019)). Two blocks are administered and randomly ordered for each participant. Each block contains 48 trials. Each trial lasted for 2000 ms, followed by an inter-trial interval of 1500 ms consisting of a fixation cross. One block had an equal number of congruent vs. incongruent trials, while the other block had 75% congruent and 25% incongruent trials (36 congruent trials, 12 incongruent trials). This manipulation between blocks was included to capture reactive vs. proactive control, respectively (Braver, 2012; Kane & Engle, 2003), such that in the equally split congruent vs. incongruent condition, the higher proportion of incongruent trials serve as a stimulus-driven cue that reminds

the participant of task goals, and thus is reflective of reactive control, as compared to proactive control that is driven by internal reminders and is best captured by the 25/75 incongruent to congruent proportion (Braver, 2012). The key variable that was used in analyses is the incongruent RT total. This variable was only collected at the second timepoint (10-11 years).

[Insert Figure 4]

### ***Dependent Variable: Self-Injurious Thoughts and Behaviors***

Self-injurious thoughts and behaviors were quantified using computerized self-administered versions of the Schedule for Affective Disorders and Schizophrenia for school-age children (K-SADS-5; Kaufman et al., 1997). Binary codes from the “diagnosis” of “self-injurious behavior without suicidal intent” were the main outcome of interest for NSSI. To test the specificity of NSSI (i.e., “self-injurious behavior without suicidal intent”) vs. other suicidal behaviors, K-SADS-5 diagnoses of (1) passive suicide ideation (i.e., “wish to be dead”); (2) nonspecific active suicidal ideation (i.e., “wanting to end one’s own life without consideration of a suicide method, intent, or plan”); (3) active suicidal ideation (i.e., “a desire to die by suicide and  $\geq 1$  of the following: a consideration of a specific suicide method, expression of the intent to act on suicidal thoughts, and/or formulation of a plan to commit suicide in the future”) or (4) suicide attempts (DeVille et al., 2020) were also probed. Child-report and parent-reported variables of NSSI and suicidality were used as the dependent variables of interest. Additionally, *current* (i.e., past two weeks), and *history of* (i.e., lifetime) these variables were examined. Variables from this measure were derived from third timepoint (11-12 years).

### ***Moderator: Autistic Traits***

Autistic traits were quantified using three measures: computerized self-administered versions of the 1) Child Behavior Checklist (CBCL) and 2) short Social Responsiveness Scale (sSRS), and 3) K-SADS-5.

**The Child Behavior Checklist (CBCL) Autism Spectrum Disorder (ASD) Subscales.**

The CBCL (Achenbach & Rescorla, 2001) is a caregiver report that measures problem behaviors in children. Items are rated on a 3-point scale from 0 (Not True) to 2 (Very True or Often True) with higher scores indicating higher autism spectrum symptoms. Several ASD screeners have been proposed with CBCL items, many of which use items primarily from Withdrawn/Depressed, Thought Problems, and Social Problems subscales (Biederman et al., 2010; Havdahl et al., 2016; Ooi et al., 2011; So et al., 2013). Based on previous work by Deckers, Muris, & Roelofs (2020) that tested five versions of CBCL ASD screening subscales against each other, the ASD screening subscales from So and colleagues (2013) and Ooi and colleagues (2011) were the most sensitive and specific in predicting DSM-5 ASD diagnosis and scores on the Autism Diagnostic Interview – Revised (Lord et al., 1994), and thus raw scores were used from these subscales. Internal consistency of the Ooi et al. (2011) CBCL ASD scale in this sample was questionable (Chronbach's  $\alpha=.66$ ) and internal consistency of the So et al. (2013) CBCL ASD scale in this sample was acceptable (Chronbach's  $\alpha=.73$ ). For more information on items see Table 1.

**The Short Social Responsiveness Scale (sSRS).** The Social Responsiveness Scale (SRS; Constantino, 2013; Constantino & Gruber, 2005) is a caregiver report that measures the presence and severity of autism spectrum symptoms (i.e., social communication difficulties, restricted and repetitive behaviors) in everyday settings. Caregivers rate items on a 4-point scale from 1 (Not True) to 4 (Almost Always True) with higher scores indicating greater severity in autism spectrum symptoms. An 11-item sSRS (Barch et al., 2017; Reiersen et al., 2008) was collected on 4,950

ABCD participants, though currently there is no work examining the psychometrics of this short form. Of note, the sSRS in this study did not overlap with items previously proposed as short SRS items (Kaat & Farmer, 2017; Sturm et al., 2017). A raw score was used. This variable was only collected at the second timepoint (10-11 years). Internal consistency of the sSRS in this sample was good (Cronbach's  $\alpha=.83$ ). For more information on items see Table 2.

**Neurodevelopmental Disorder Code from the K-SADS-5.** Using the K-SADS-5 (Kaufman et al., 1997), the binary code from “Diagnosis - Other Specified Neurodevelopmental Disorder Autism Spectrum Disorder full criteria not assessed (F88.0)” were used, which is based off of endorsement of symptoms of poor eye contact, unusual body movements, and strict routines.

### **Statistical Analysis Plan**

Data on K-SADS at ages 11-12 years of age were available for 6,351 parent-reported and 6,381 child-reported variables of NSSI and suicidality, as some participants only had parent- and some only had child-reported measures the total sample size 6,447 (47.4% [n=3056] female, 52.6% [n=3391] male). Independent, mediator, and moderator variables were tested for normality using skewness and kurtosis. SST brain activation measures were highly skewed and kurtotic, behavioral task data from the emotional stroop task, TDCCS task, and the SST were also skewed and kurtotic. Due to this, data were excluded for participants with Z-scores  $>3.5$  or  $<-3.5$  within each variable for the analyses. Data were excluded emotional stroop task (n=2), TDCCS (n=31), SST LPFC (n=27), SST ACC (n=44), SST striatum (n=26), SSRT mean (n=31). Of note, many participants had overlap in reasons for exclusion. Pairwise deletion was used for each regression model in order to maximize data used.

In order to test Aims 1 and 2, preliminary correlation and regression analyses using the Baron and Kenny (1986) methods were used to examine eligibility for mediation and moderation

testing. The steps for mediation testing included 1) correlations and regressions between predictor variables (X) and outcome variables (Y), 2) correlations and regressions between predictor variables (X) and mediator variable (M), 3) regressions between mediator (M) and outcome (Y) variables when controlling for predictor variable (X), and 4) evaluating whether the effect of the predictor variable (X) was diminished or not significant after adding the mediator (M). When appropriate these steps were modified for dichotomous outcome variables using a logistic rather than linear regression (Kenny, 2013). The steps for moderation included examining the effects of the predictor, moderator, and interaction term which multiplied the predictor and moderator. Planned analyses further involved conducting mediation and moderated mediation analyses (see below). All continuous variables were mean centered. All analyses were conducted using R and SPSS.

### ***Primary Model (Aim 1): Prediction of Self-Injurious Thoughts and Behaviors***

Planned analyses were to use Hayes' PROCESS model 4, to test two mediation models. Emotional stroop was planned to be evaluated as a potential mediator of the relationship between measures of 1) SST and NSSI (Figure 5a) and 2) TDCCS Task and NSSI (Figure 5b). Exploratory mediation models were run to examine the specificity of Emotional stroop as a potential mediator between 1) SST measures and other self-injurious thoughts and behaviors (as outlined above) and 2) TDCCS Task and other self-injurious thoughts and behaviors.

### ***Exploratory Models (Aim 2): Examining the Role of Autistic Traits as a Moderator***

In order to examine the moderating role of autistic traits on the primary model, Hayes' PROCESS model 59, which examines a moderated mediation, was planned to be used. This model allows testing of the individual moderated indirect effects on all paths, in addition to the moderated direct effects.



[Insert Figure 5a]

[Insert Figure 5b]

## Results

### Correlations

Consistent with preliminary steps to determine assumptions for a mediation analysis (Baron & Kenny, 1986), correlations and follow-up regressions were conducted amongst variables of interest to confirm the existence of relationships between (1) predictor variables (X) and outcome variables (Y), and (2) predictor variables (X) and the mediator (M). Correlations were bootstrapped 1000 times in order to calculate the 95% confidence interval (CI) of the correlations.

#### *Preliminary correlations for predictor (X) and outcome (Y) variables*

Point-biserial correlations, which capture relationships between dichotomous and continuous variables, were used rather than Spearman's rank and Pearson correlations. Consistent with the hypotheses from the proposed model (Figure 1), correlations between cognitive control [X] (i.e., inhibition, as measured by the ROIs from the *correct stop trials vs. incorrect stop trials* of the SST, and SSRT, and cognitive flexibility, as measured by TDCCS task score) and self-injurious thoughts and behaviors [Y] were evaluated.

**Child-Reported Variables.** Cognitive flexibility did not correlate with child-reported *current or history of* NSSI, passive suicidal ideation, nonspecific active suicidal ideation, active suicidal ideation, or suicide attempt ( $ps > .12$ ). SST LPFC activation did not correlate with child-reported *current or history of* NSSI, passive suicidal ideation, nonspecific active suicidal ideation, active suicidal ideation, or suicide attempt ( $ps > .11$ ). A weak but significant negative relationships emerged between diminished SST ACC activation and presence of child-reported *current* NSSI,  $r_{pb} (5091) = -.034$ ,  $p = .016$ , 95% CI [-0.071, .004]. Weak but significant negative relationships

emerged between diminished SST ACC activation and presence of child-reported *history of* nonspecific active suicidal ideation,  $r_{pb} (5091) = -.046$ ,  $p = .001$ , 95% CI [-0.077, -.015], and presence of child-reported *history of* active suicidal ideation,  $r_{pb} (5091) = -.036$ ,  $p = .009$ , 95% CI [-0.067, -.005]. SST ACC activation did not correlate with any other child-reported measures ( $ps > .07$ ). A weak but significant negative relationship emerged between diminished SST striatum activation and presence of child-reported *current* NSSI,  $r_{pb} (5109) = -.030$ ,  $p = .035$ , 95% CI [-0.061, .001]. SST striatum activation did not correlate with any other child-reported measures ( $ps > .11$ ). SSRT mean did not correlate with child-reported *current or history of* NSSI, passive suicidal ideation, nonspecific active suicidal ideation, active suicidal ideation, or suicide attempt ( $ps > .17$ ).

**Parent-Reported Variables.** A weak but significant negative relationship between poorer cognitive flexibility and presence of parent-reported *current* nonspecific active suicidal ideation,  $r_{pb} (6258) = -.035$ ,  $p = .005$ , 95% CI [-0.059, -.012], and presence of parent-reported *current* suicide attempt emerged,  $r_{pb} (6258) = -.026$ ,  $p = .04$ , 95% CI [-0.048, .005]. No other correlations were found for parent-reported measures ( $ps > .08$ ). Weak but significant negative relationships emerged between diminished SST LPFC activation and presence of parent-reported *current* NSSI,  $r_{pb} (5082) = -.036$ ,  $p = .011$ , 95% CI [-0.070, -.009], and presence of parent-reported *current* suicide attempts,  $r_{pb} (5082) = -.030$ ,  $p = .032$ , 95% CI [-0.111, .034]. Weak but significant negative relationships emerged between diminished SST LPFC activation and presence of parent-reported *history of* passive suicidal ideation,  $r_{pb} (5082) = -.034$ ,  $p = .017$ , 95% CI [-0.062, -.002], and presence of parent-reported *history of* nonspecific active suicidal ideation,  $r_{pb} (5082) = -.029$ ,  $p = .036$ , 95% CI [-0.060, .005]. No other significant correlations emerged between SST LPFC activation and parent-reported variables ( $ps > .27$ ). Weak but significant negative relationships emerged between diminished SST ACC activation and presence of parent-reported *current* NSSI,  $r_{pb} (5065) = -.029$ ,

$p=.040$ , 95% CI [-0.063, .002], and presence of parent-reported *current* active suicidal ideation,  $r_{pb}$  (5065)=-.042,  $p=.003$ , 95% CI [-0.087, .002]. A weak but significant negative relationship emerged between diminished SST ACC activation and presence of parent-reported *history of* passive suicidal ideation,  $r_{pb}$  (5065)=-.031,  $p=.030$ , 95% CI [-0.058, -.001]. SST ACC activation did not correlate with any other parent-reported variables ( $ps >.06$ ). A weak but significant negative relationship emerged between diminished SST striatum activation and presence of parent-reported *current* active suicidal ideation,  $r_{pb}$  (5083)=-.030,  $p=.034$ , 95% CI [-0.059, .001]. SST striatum activation did not correlate with any other parent-reported variables ( $ps >.05$ ). A weak but significant negative relationship emerged between faster SSRT mean and presence of parent-reported *current* suicide attempt,  $r_{pb}$  (5498)=-.029,  $p=.029$ , 95% CI [-0.078, .007]. No other correlations emerged between SSRT mean and parent-reported variables ( $ps >.29$ ).

***Preliminary correlations between predictor variables (X) and mediator (M)***

As all X and M variables were continuous, Pearson correlations were conducted. Consistent with the hypotheses from the proposed model (Figure 1), correlations between cognitive control [X] (i.e., inhibition, as measured by the ROIs from the *correct stop trials vs. incorrect stop trials* of the SST, and SSRT, and cognitive flexibility, as measured by scores from the TDCCS task) and emotion regulation [M] (as measured by emotional stroop incongruent RT) were evaluated.

Slower emotional stroop incongruent RT negatively correlated with poorer cognitive flexibility ( $r(6234)=-.317$ ,  $p<.001$ , 95% CI [-.340, -.296]), diminished SST LPFC activation ( $r(5078)=-.043$ ,  $p=.002$ , 95% CI [-.070, -.017]), diminished SST striatum activation ( $r(5079)=-.067$ ,  $p<.001$ , 95% CI [-.094, -.041]), and slower SSRT mean ( $r(5488)=.154$ ,  $p<.001$ , 95% CI [.127, .180]). Emotional stroop incongruent RT did not correlate with SST ACC ( $p >.39$ ).

### Follow-up Regressions for Mediation Analyses

Individual linear regressions were conducted on all significant correlated variables for  $X \rightarrow Y$  [c path] and  $X \rightarrow M$  [a path]. As  $M \rightarrow Y$  [b path], does not require a significant correlation to pursue within a mediation, these relationships were first tested at the regression level, while controlling for X (Baron & Kenny, 1986). Linear regressions were used to test  $X \rightarrow M$ , as both variables were continuous. Logistic regressions were used for  $X \rightarrow Y$  and  $M \rightarrow Y$ , as all Y variables were dichotomous. Statistics for regressions were bootstrapped at 1000 times with 95% CI and results are reported below.

#### ***Step 1: Follow-up logistic regressions examining the role of cognitive control (X) on self-injurious thoughts and behaviors (Y)***

Consistent with the hypotheses from the proposed model (Figure 1), logistic regressions between poorer cognitive control [X] (i.e., inhibition, as measured by the ROIs from the *correct stop trials vs. incorrect stop trials* of the SST, and SSRT, and cognitive flexibility, as measured by scores from the TDCCS task) and presence of self-injurious thoughts and behaviors [Y] were evaluated. Regression results for step 1 are presented in Table 5.

[Insert Table 5]

**Child-Reported Variables.** Diminished SST ACC activation significantly predicted presence of child-reported *current* NSSI,  $B=-1.399$ , 95% CI [-2.650, .045],  $Wald=6.422$ ,  $p=.045$ , and accounted for .9% of the variance in child-reported *current* NSSI, Nagelkerke  $R^2=.009$ ,  $\chi^2(1, N=5092)=6.108$ ,  $p=.013$ . Diminished SST ACC activation significantly predicted presence of child-reported *history of* active suicidal ideation,  $B=-1.176$ , 95% CI [-1.899, -.411],  $Wald=11.140$ ,  $p=.003$ , and accounted for .8% of the variance in child-reported *history of* active suicidal ideation, Nagelkerke  $R^2=.008$ ,  $\chi^2(1, N=5092)=10.977$ ,  $p=.001$ . Diminished SST striatum activation

significantly predicted presence of child-reported *current* NSSI,  $B=-1.564$ , 95% CI [-2.983, -0.80],  $Wald=5.404$ ,  $p=.036$ , and accounted for .8% of the variance in child-reported *current* NSSI, Nagelkerke  $R^2=.008$ ,  $\chi^2(1, N=5110)=5.252$ ,  $p=.022$ .

**Parent-Reported Variables.** Poorer cognitive flexibility significantly predicted presence of parent-reported *current* nonspecific active suicidal ideation,  $B=-.025$ , 95% CI [-.047, -.006],  $Wald=7.714$ ,  $p=.012$ , and accounted for 1.1% of the variance in parent-reported *current* nonspecific active suicidal ideation, Nagelkerke  $R^2=.011$ ,  $\chi^2(1, N=6257)=8.481$ ,  $p=.004$ . After bootstrapping, cognitive flexibility no longer significantly predicted parent-reported *current* suicide attempts,  $B=-.103$ , 95% CI [-.290, -.014],  $Wald=4.206$ ,  $p=.187$ , despite the original model being statistically significant, Nagelkerke  $R^2=.071$ ,  $\chi^2(1, N=6256)=5.767$ ,  $p=.016$ . Diminished SST LPFC activation significantly predicted presence of parent-reported *current* NSSI,  $B=-1.396$ , 95% CI [-2.386, -.328],  $Wald=6.718$ ,  $p=.009$ , and accounted for 0.8% of the variance in parent-reported *current* NSSI, Nagelkerke  $R^2=.008$ ,  $\chi^2(1, N=5081)=6.319$ ,  $p=.012$ . After bootstrapping, SST LPFC no longer significantly predicted parent-reported *current* suicide attempts,  $B=-3.998$ , 95% CI [-11.852, 5.730],  $Wald=5.201$ ,  $p=.181$ , and the overall model was not statistically significant, Nagelkerke  $R^2=.056$ ,  $\chi^2(1, N=5081)=3.629$ ,  $p=.057$ . Diminished SST LPFC activation significantly predicted presence of parent-reported *history of* passive suicidal ideation,  $B=-.723$ , 95% CI [-1.342, -.124],  $Wald=5.823$ ,  $p=.024$ , and accounted for 0.3% of the variance in parent-reported *history of* passive suicidal ideation, Nagelkerke  $R^2=.003$ ,  $\chi^2(1, N=5081)=5.740$ ,  $p=.017$ . After bootstrapping, SST LPFC no longer significantly predicted parent-reported *history of* nonspecific active suicidal ideation,  $B=-.729$ , 95% CI [-1.522, .026],  $Wald=4.408$ ,  $p=.061$ , despite the original model being statistically significant, Nagelkerke  $R^2=.003$ ,  $\chi^2(1, N=5081)=4.344$ ,  $p=.037$ . Diminished SST ACC activation significantly predicted presence of parent-reported

*current* NSSI,  $B=-1.009$ , 95% CI [-1.944, -.016],  $Wald=4.259$ ,  $p=.042$ , and accounted for .5% of the variance in parent-reported *current* NSSI, Nagelkerke  $R^2=.005$ ,  $\chi^2(1, N=5064)=4.149$ ,  $p=.042$ . Diminished SST ACC activation significantly predicted presence of parent-reported *current* active suicidal ideation,  $B=-3.464$ , 95% CI [-5.711, -.136],  $Wald=9.511$ ,  $p=.006$ , and accounted for 5% of the variance in parent-reported *current* active suicidal ideation, Nagelkerke  $R^2=.050$ ,  $\chi^2(1, N=5064)=7.730$ ,  $p=.005$ . Diminished SST ACC activation significantly predicted presence of parent-reported *history of* passive suicidal ideation,  $B=-.577$ , 95% CI [-1.121, -.086],  $Wald=4.789$ ,  $p=.035$ , and accounted for .2% of the variance in parent-reported *history of* passive suicidal ideation, Nagelkerke  $R^2=.002$ ,  $\chi^2(1, N=5064)=4.758$ ,  $p=.029$ . Diminished SST striatum activation significantly predicted presence of parent-reported *current* active suicidal ideation,  $B=-3.325$ , 95% CI [-5.634, .213],  $Wald=4.734$ ,  $p=.015$ , and accounted for 2.8% of the variance in parent-reported *current* active suicidal ideation, Nagelkerke  $R^2=.028$ ,  $\chi^2(1, N=5082)=4.253$ ,  $p=.039$ . Faster SSRT mean significantly predicted presence of parent-reported *current* suicide attempt,  $B=-0.13$ , 95% CI [-.034, .007],  $Wald=5.273$ ,  $p=.046$ , and accounted for 6.6% of the variance in parent-reported *current* active suicidal ideation, Nagelkerke  $R^2=.066$ ,  $\chi^2(1, N=5496)=4.312$ ,  $p=.038$ .

***Step 2: Follow-up linear regressions examining the role of cognitive control (X) on emotion regulation (M)***

Consistent with the hypotheses from the proposed model (Figure 1), linear regressions between poorer cognitive control [X] and poorer emotion regulation [M] (as measured by emotional stroop incongruent RT) were evaluated. Regression results for step 2 are presented in Table 6.

[Insert Table 6]

Poorer cognitive flexibility significantly predicted slower emotional stroop incongruent RT,  $B=-2.700$ , 95% CI [-2.897, -2.502],  $t=-26.395$ ,  $p<.001$ , which accounted for 10.1% of the variance in emotional stroop incongruent RT,  $R^2=0.101$ ,  $F(1, 6232)=696.710$ ,  $p<.001$ . Diminished SST LPFC activation significantly predicted slower emotional stroop incongruent RT,  $B=-28.904$ , 95% CI [-46.025 -9.500],  $t=-3.04$ ,  $p=.005$ , which accounted for 0.2% of the variance in emotional stroop incongruent RT,  $R^2=0.002$ ,  $F(1, 5076)=9.244$ ,  $p=.002$ . Diminished SST striatum activation significantly predicted slower emotional stroop incongruent RT,  $B=-47.91$ , 95% CI [-66.06, -29.57],  $t=-4.82$ ,  $p<.001$ , which accounted for 0.5% of the variance in emotional stroop incongruent RT,  $R^2=0.005$ ,  $F(1, 5077)=23.188$ ,  $p<.001$ . Slower SSRT Mean significantly predicted slower emotional stroop incongruent RT,  $B=.264$ , 95% CI [.214, .312],  $t=11.56$ ,  $p<.001$ , which accounted for 2.4% of the variance in emotional stroop incongruent RT,  $R^2=0.024$ ,  $F(1, 5486)=133.63$ ,  $p<.001$ .

***Step 3: Logistic regressions between examining the role of emotion regulation (M) on self-injurious thoughts and behaviors (Y) when controlling for cognitive control (X)***

As recommended by Baron & Kenny (1986), the predictor variable (X) must be included and controlled for when examining the effect of the mediator (M) on the outcome variable (Y). Thus, logistic regressions were run to examine the role of mediator (M) on the outcome variable (Y) while controlling for predictor variable (X). Only significant regression models from step 1 were probed. Additionally, no regression models including SST ACC activation and emotional stroop incongruent RT were not tested, as these variables were not correlated, and thus did not meet the assumptions to test the next step of a potential mediation.

Consistent with the hypotheses from the proposed model (Figure 1), logistic regressions between poorer emotion regulation [M] (as measured by emotional stroop incongruent RT) and

presence of self-injurious thoughts and behaviors [Y], while controlling for cognitive control [X] were evaluated. Regression results for step 3 are presented in Table 7.

[Insert Table 7]

**Child-Reported Variables.** The logistic regression model including SST striatum activation and emotional stroop incongruent RT was not significant in predicting child-reported *current* NSSI, Nagelkerke  $R^2=.006$ ,  $\chi^2(1, N=5035)=4.312$ ,  $p=.116$ . Within this model, SST striatum activation,  $B=-1.341$ , 95% CI [-2.737, .059],  $Wald=3.779$ ,  $p=.067$ , and emotional stroop incongruent RT were not significant in predicting child-reported *current* NSSI,  $B=.001$ , 95% CI [-.001, .002],  $Wald=.412$ ,  $p=.521$ .

**Parent-Reported Variables.** The logistic regression model including cognitive flexibility and emotional stroop incongruent RT was significant in predicting parent-reported *current* nonspecific active suicidal ideation, Nagelkerke  $R^2=.011$ ,  $\chi^2(1, N=6147)=8.577$ ,  $p=.014$ . Within this model, poorer cognitive flexibility significantly predicted presence of parent-reported *current* nonspecific active suicidal ideation,  $B=-.023$ , 95% CI [-.044, -.003],  $Wald=5.928$ ,  $p=.029$ , though emotional stroop incongruent RT was not significant in predicting parent-reported *current* nonspecific active suicidal ideation,  $B=.001$ , 95% CI [-.001, .003],  $Wald=.399$ ,  $p=.521$ . The logistic regression model including SST LPFC activation and emotional stroop incongruent RT was not significant in predicting parent-reported *current* NSSI, Nagelkerke  $R^2=.007$ ,  $\chi^2(1, N=5005)=5.701$ ,  $p=.058$ . Within this model, diminished SST LPFC activation significantly predicted presence of parent-reported *current* NSSI,  $B=-1.329$ , 95% CI [-2.361, -.319],  $Wald=5.828$ ,  $p=.012$ , though emotional stroop incongruent RT was not significant in predicting parent-reported *current* NSSI,  $B=.000$ , 95% CI [-.002, .002],  $Wald=.114$ ,  $p=.767$ . The logistic regression model including SST LPFC activation and emotional stroop incongruent RT was



significant in predicting parent-reported *history of* passive suicidal ideation, Nagelkerke  $R^2=.004$ ,  $\chi^2(1, N=5005)=7.205$ ,  $p=.027$ . Within this model, diminished SST LPFC activation significantly predicted presence of parent-reported *history of* passive suicidal ideation,  $B=-.787$ , 95% CI [-1.456, -.164],  $Wald=6.805$ ,  $p=.016$ , though emotional stroop incongruent RT was not significant in predicting parent-reported *history of* passive suicidal ideation,  $B=.000$ , 95% CI [-.001, .001],  $Wald=.360$ ,  $p=.535$ . The logistic regression model including SST striatum activation and emotional stroop incongruent RT was significant in predicting parent-reported *current* active suicidal ideation, Nagelkerke  $R^2=.042$ ,  $\chi^2(1, N=5006)=6.027$ ,  $p=.049$ . Within this model, diminished SST striatum activation significantly predicted presence of parent-reported *current* active suicidal ideation,  $B=-4.051$ , 95% CI [-6.274, -.996],  $Wald=6.827$ ,  $p=.001$ , though emotional stroop incongruent RT was not significant in predicting parent-reported *current* active suicidal ideation,  $B=-.001$ , 95% CI [-.006, .003],  $Wald=.244$ ,  $p=.514$ . The logistic regression model including SSRT mean and emotional stroop incongruent RT was not significant in predicting parent-reported *current* suicide attempt, Nagelkerke  $R^2=.090$ ,  $\chi^2(1, N=5409)=5.881$ ,  $p=.053$ . Within this model, faster SSRT mean was significant in predicting presence of parent-reported *current* suicide attempt,  $B=-.012$ , 95% CI [-.041, .005],  $Wald=5.687$ ,  $p=.017$ , though emotional stroop incongruent RT was not significant in predicting parent-reported *current* suicide attempt,  $B=.005$ , 95% CI [-.006, .010],  $Wald=1.562$ ,  $p=.087$ .

All Step 3 logistic regressions tested did not meet criteria for Step 4 of a mediation, such that including the mediator (M: emotional stroop incongruent RT) did not partially or completely diminish the relationship between the predictor (X) and outcome (Y) variables. Thus, emotional stroop incongruent RT did not mediate the relationship between variables of cognitive control and variables of NSSI and suicidality.

## Moderation Analyses

As a moderation of autistic traits was hypothesized to impact the relationships between all X->M, M->Y and X->Y, the potential moderations of autistic traits were tested on all significant regressions. Internal consistency analyses revealed that the sSRS had good reliability and the strongest internal consistency as compared to questionable reliability on the Ooi et al. (2011) CBCL ASD scale, and acceptable reliability on the So et al., (2011) CBCL ASD scale. Thus, sSRS was used as the moderator of interest. Additionally, the sSRS was used over the K-SADS-5 Neurodevelopmental Disorder code, as the sSRS captured more variability and was derived from a three-item screener on autistic traits, and the K-SADS-5 Neurodevelopmental Disorder code was a broader measure and not ASD-specific. Regression results evaluating moderation of autistic traits on the role of cognitive control on self-injurious thoughts and behaviors are presented in Table 8.

[Insert Table 8]

***Do autistic traits moderate the the role of cognitive control (X) on self-injurious thoughts and behaviors (Y)?***

**Child-Reported Variables.** The logistic regression model including SST ACC activation, sSRS, and the interaction term, was statistically significant,  $\chi^2(3, N=5083)=15.839, p=.001$ , and explained 2.4% (Nagelkerke  $R^2$ ) of the variance of child-reported *current* NSSI. Within this model, greater sSRS, ( $B=.077, 95\% \text{ CI } [.024, .113], \text{Wald}=11.467, p<.001$ ) was the only significant predictor. SST ACC activation ( $B=-1.332, 95\% \text{ CI } -2.612, .186], \text{Wald}=5.291, p=.071$ ) and the interaction term did not significantly contribute to the model ( $p>.87$ ), indicating no support for a moderation of autistic traits. The logistic regression model including SST ACC activation, sSRS, and the interaction term, was statistically significant,  $\chi^2(3, N=5083)=20.108, p<.001$ , and explained 3.1% (Nagelkerke  $R^2$ ) of the variance of child-reported *history of* active suicidal

ideation. Within this model, diminished SST ACC activation, ( $B=-1.279$ , 95% CI [-2.335, -.038],  $Wald=4.471$ ,  $p=.031$ ), and greater sSRS, ( $B=.087$ , 95% CI [.041, .120],  $Wald=15.234$ ,  $p<.001$ ) were significant predictors. The interaction term did not significantly contribute to the model ( $p>.82$ ), indicating no support for a moderation of autistic traits. The logistic regression model including SST striatum activation, sSRS, and the interaction term, was statistically significant,  $\chi^2(3, N=5102)=15.637$ ,  $p=.001$ , and explained 2.3% (Nagelkerke  $R^2$ ) of the variance of child-reported *current* NSSI. Within this model, greater sSRS, ( $B=.076$ , 95% CI [.032, .111],  $Wald=11.834$ ,  $p<.001$ ) was the only significant predictor. SST striatum activation ( $B=-1.470$ , 95% CI -2.835, .132],  $Wald=4.308$ ,  $p=.056$ ) and the interaction term did not significantly contribute to the model ( $p>.92$ ), indicating no support for a moderation of autistic traits.

**Parent-Reported Variables.** The logistic regression model including cognitive flexibility, sSRS, and the interaction term, was statistically significant,  $\chi^2(3, N=6242)=44.618$ ,  $p<.001$ , and explained 5.7% (Nagelkerke  $R^2$ ) of the variance of parent-reported *current* nonspecific active suicidal ideation. Within this model, poorer cognitive flexibility ( $B=-.026$ , 95% CI [-.049, -.006],  $Wald=6.892$ ,  $p=.021$ ), and greater sSRS, ( $B=.119$ , 95% CI [.084, .151],  $Wald=49.228$ ,  $p<.001$ ), were the only significant predictors. The interaction term did not significantly contribute to the model ( $p>.18$ ), indicating no support for a moderation of autistic traits. The logistic regression model including SST LPFC activation, sSRS, and the interaction term, was statistically significant,  $\chi^2(3, N=5072)=48.599$ ,  $p<.001$ , and explained 5.7% (Nagelkerke  $R^2$ ) of the variance of parent-reported *current* NSSI. Within this model, diminished SST LPFC activation ( $B=-1.344$ , 95% CI [-2.293, -.213],  $Wald=5.097$ ,  $p=.013$ ), and greater sSRS, ( $B=.121$ , 95% CI [.087, .150],  $Wald=55.494$ ,  $p<.001$ ), were the only significant predictors. The interaction term did not significantly contribute to the model ( $p>.58$ ), indicating no support for a moderation of autistic

traits. The logistic regression model including SST LPFC activation, sSRS, and the interaction term, was statistically significant,  $\chi^2(3, N=5072)=178.697, p<.001$ , and explained 8.8% (Nagelkerke  $R^2$ ) of the variance of parent-reported *history of* passive suicidal ideation. Within this model, greater sSRS, ( $B=.140, 95\% \text{ CI } [.121, .160], \text{Wald}=193.663, p<.001$ ) was the only significant predictor. SST LPFC activation ( $B=-.593, 95\% \text{ CI } [-1.236, .090], \text{Wald}=3.159, p=.086$ ) and the interaction term did not significantly contribute to the model ( $p>.70$ ), indicating no support for a moderation of autistic traits. The logistic regression model including SST ACC activation, sSRS, and the interaction term, was statistically significant,  $\chi^2(3, N=5054)=46.140, p<.001$ , and explained 5.5% (Nagelkerke  $R^2$ ) of the variance of parent-reported *current* NSSI. Within this model, greater sSRS, ( $B=.121, 95\% \text{ CI } [.088, .160], \text{Wald}=55.056, p<.001$ ) was the only significant predictor. SST ACC activation ( $B=-.941, 95\% \text{ CI } [-1.889, .162], \text{Wald}=3.009, p=.071$ ) and the interaction term did not significantly contribute to the model ( $p>.57$ ), indicating no support for a moderation of autistic traits. The logistic regression model including SST ACC activation, sSRS, and the interaction term, was statistically significant,  $\chi^2(3, N=5054)=12.133, p=.007$ , and explained 7.8% (Nagelkerke  $R^2$ ) of the variance of parent-reported *current* active suicidal ideation. Within this model, diminished SST ACC activation ( $B=-2.779, 95\% \text{ CI } [-5.368, .563], \text{Wald}=4.284, p=.049$ ) was the only significant predictor. sSRS ( $B=.073, 95\% \text{ CI } [-.121, .145], \text{Wald}=1.343, p=.126$ ) and the interaction term did not significantly contribute to the model ( $p>.408$ ), indicating no support for a moderation of autistic traits. The logistic regression model including SST ACC activation, sSRS, and the interaction term, was statistically significant,  $\chi^2(3, N=5054)=176.143, p<.001$ , and explained 8.7% (Nagelkerke  $R^2$ ) of the variance of parent-reported *history of* passive suicidal ideation. Within this model, greater sSRS, ( $B=.141, 95\% \text{ CI } [.120, .161], \text{Wald}=189.220, p<.001$ ) was the only significant predictor. SST ACC activation ( $B=-.499, 95\% \text{ CI } [-.998, .000], \text{Wald}=3.159, p=.086$ ) and the interaction term did not significantly contribute to the model ( $p>.70$ ), indicating no support for a moderation of autistic traits.

[-1.064, .083],  $Wald=2.906$ ,  $p=.079$ ) and the interaction term did not significantly contribute to the model ( $p>.44$ ), indicating no support for a moderation of autistic traits. The logistic regression model including SST striatum activation, sSRS, and the interaction term, was statistically significant,  $\chi^2(3, N=5062)=8.297$ ,  $p=.040$ , and explained 5.4% (Nagelkerke  $R^2$ ) of the variance of parent-reported *current* active suicidal ideation. Within this model, greater sSRS, ( $B=.103$ , 95% CI [-.024, .151],  $Wald=3.924$ ,  $p<.001$ ) was the only significant predictor. SST striatum activation ( $B=-3.075$ , 95% CI [-5.720, .650],  $Wald=3.223$ ,  $p=.051$ ) and the interaction term did not significantly contribute to the model ( $p>.92$ ), indicating no support for a moderation of autistic traits. The logistic regression model including SSRT mean, sSRS, and the interaction term, was statistically significant,  $\chi^2(3, N=5484)=10.327$ ,  $p=.016$ , and explained 15.8% (Nagelkerke  $R^2$ ) of the variance of parent-reported *current* suicide attempts. Faster SSRT mean ( $B=-.015$ , 95% CI [-.041, .007],  $Wald=5.090$ ,  $p=.013$ ) and greater sSRS, ( $B=.196$ , 95% CI [-.111, .250],  $Wald=8.136$ ,  $p=.001$ ) were significant predictors in the model. The interaction term did not significantly contribute to the model ( $p>.23$ ), indicating no support for a moderation of autistic traits.

***Do autistic traits moderate the role of cognitive control (X) on emotion regulation (M)?***

The linear regression model including cognitive flexibility, sSRS, and the interaction term, was statistically significant,  $F(3, 6224)=234.497$ ,  $p<.001$ , and explained 10.2% ( $R^2$ ) of the variance of emotional stroop incongruent RT. Within this model, poorer cognitive flexibility  $B=-2.691$ , 95% CI [-2.881, -2.469],  $t=-26.238$ ,  $p<.001$ , was the only significant predictor. sSRS,  $B=.775$ , 95% CI [.027, 1.589],  $t=2.072$ ,  $p=.051$ , and the interaction term did not significantly contribute to the model ( $p>.31$ ), indicating no support for a moderation of autistic traits. The linear regression model including SST LPFC activation, sSRS, and the interaction term, was statistically significant,  $F(3, 5070)=5.651$ ,  $p<.001$ , and explained 0.3% ( $R^2$ ) of the variance of emotional stroop

incongruent RT. Within this model, diminished SST LPFC activation,  $B=-27.650$ , 95% CI [-46.764,-9.433],  $t=-2.899$ ,  $p<.001$ , and greater sSRS,  $B=1.235$ , 95% CI [.425, 2.161],  $t=2.786$ ,  $p=.006$ , were the only significant predictors. The interaction term did not significantly contribute to the model ( $p>.60$ ), indicating no support for a moderation of autistic traits. The linear regression model including SST striatum activation, sSRS, and the interaction term, was statistically significant,  $F(3, 5071)=10.404$ ,  $p<.001$ , and explained 0.6% ( $R^2$ ) of the variance of emotional stroop incongruent RT. Within this model, diminished SST striatum activation,  $B=-47.300$ , 95% CI [-67.628, -27.811],  $t=-4.746$ ,  $p<.001$ , and greater sSRS,  $B=1.245$ , 95% CI [.313, 3.414],  $t=2.821$ ,  $p=.007$ , were the only significant predictors in the model. The interaction term did not significantly contribute to the model ( $p>.67$ ), indicating no support for a moderation of autistic traits. The linear regression model including SSRT mean, sSRS, and the interaction term, was statistically significant,  $F(3, 5478)=47.408$ ,  $p<.001$ , and explained 2.5% ( $R^2$ ) of the variance of emotional stroop incongruent RT. Within this model, slower SSRT mean,  $B=.262$ , 95% CI [.218, .311],  $t=11.474$ ,  $p<.001$ , and greater sSRS,  $B=1.261$ , 95% CI [.363, 2.092],  $t=2.969$ ,  $p=.004$ , were the only significant predictors. The interaction term did not significantly contribute to the model ( $p>.57$ ), indicating no support for a moderation of autistic traits. Regression results evaluating moderation of autistic traits on the role of cognitive control on emotion regulation are presented in Table 9.

[Insert Table 9]

*Do autistic traits moderate the role of emotion regulation (M) on self-injurious thoughts and behaviors (Y)?*

**Logistic regressions between mediator (M) and outcome (Y) variables.** As relationships between M->Y were not evaluated in the regressions for mediation above, all M->Y

relationships were tested at this point, in order to examine which relationships should be further probed for moderation effects of autistic traits.

**Child-Reported Variables.** Faster emotional stroop incongruent RT significantly predicted presence of child-reported *history of NSSI*,  $B=-.001$ , 95% CI [-.002, .000],  $Wald=4.087$ ,  $p=.048$ , and accounted for .2% of the variance in child-reported *history of NSSI*, Nagelkerke  $R^2=.002$ ,  $\chi^2(1, N=6266)=4.111$ ,  $p=.043$ . Emotional stroop incongruent RT did not predict any other child-reported variables ( $ps>.06$ ).

**Parent-Reported Variables.** Faster emotional stroop incongruent RT significantly predicted presence of parent-reported *history of NSSI*,  $B=-.001$ , 95% CI [-.002, .000],  $Wald=5.118$ ,  $p=.014$ , and accounted for .3% of the variance in parent-reported *history of NSSI*, Nagelkerke  $R^2=.003$ ,  $\chi^2(1, N=6237)=4.312$ ,  $p=.023$ . Emotional stroop incongruent RT did not predict any other parent-reported variables ( $ps>.09$ ).

#### **Moderation effects of autistic traits on M->Y.**

**Child-Reported Variables.** The logistic regression model including emotional stroop incongruent RT, sSRS, and the interaction term, was statistically significant,  $\chi^2(3, N=6260)=47.015$ ,  $p<.001$ , and explained 2.8% (Nagelkerke  $R^2$ ) of the variance of child-reported *history of NSSI*. Within this model, faster emotional stroop incongruent RT ( $B=-.001$ , 95% CI [-.002, .000],  $Wald=4.286$ ,  $p=.026$ ), and greater sSRS, ( $B=.087$ , 95% CI [.061, .108],  $Wald=50.875$ ,  $p<.001$ ) were the only significant predictors. The interaction term did not significantly contribute to the model ( $p>.74$ ), indicating no support for a moderation of autistic traits.

**Parent-Reported Variables.** The logistic regression model including emotional stroop incongruent RT, sSRS, and the interaction term, was statistically significant,  $\chi^2(3, N=6231)=110.395$ ,  $p<.001$ , and explained 6.3% (Nagelkerke  $R^2$ ) of the variance of parent-reported

*history of* NSSI. Within this model, faster emotional stroop incongruent RT ( $B=-.001$ , 95% CI  $[-.002, .000]$ ,  $Wald=4.462$ ,  $p=.038$ ), and greater sSRS, ( $B=.122$ , 95% CI  $[.102, .142]$ ,  $Wald=122.957$ ,  $p<.001$ ), were the only significant predictors. The interaction term did not significantly contribute to the model ( $p>.36$ ), indicating no support for a moderation of autistic traits. Regression results evaluating moderation of autistic traits on the role of emotion regulation on self-injurious thoughts and behaviors are presented in Table 10.

[Insert Table 10]

### **Discussion**

This study aimed to examine the roles of cognitive control at ages 9-10 and 10-11, and emotion regulation at ages 10-11, on NSSI and measures of suicidality at ages 11-12. Additionally, this study aimed to examine the potential moderating role of autistic traits on these links. Although the overarching model of a mediating effect of emotion regulation on the links between cognitive control and NSSI was not supported, results indicated support for direct effects of diminished inhibition on presence of NSSI. This effect was found only for neural correlates of inhibition and not behavioral inhibition (i.e., SSRT mean) or cognitive flexibility. Moreover, these links were supported with both child- and parent-reported NSSI. As for the specificity of these links on NSSI, neural correlates of inhibition also predicted active suicidal ideation and passive suicidal ideation, though this was less consistent across parent and child variables as compared to NSSI. Behavioral markers of cognitive flexibility were linked with parent-reported *current* nonspecific suicidal ideation and behavioral markers of inhibition were related to *current* suicide attempts. Additionally, direct effects of cognitive control on emotion regulation were supported with the exception of SST ACC activation. Surprisingly, when controlling for cognitive control, no associations between emotion regulation and measures of NSSI or suicidality were found. An



exploratory aim of this study was examining the potential moderating role of autistic traits on significant associations. Although greater autistic traits frequently significantly predicted presence of NSSI and suicidality, a moderation of autistic traits was not found.

Although limited, previous work has examined links between cognitive control and NSSI and suicidality (Hamza et al., 2015; Liu, 2017; McHugh et al., 2019). Despite some preliminary work linking neurocognitive measures to NSSI and suicidality, very few studies have used neural markers of cognitive control (Allen & Hooley, 2017; Dahlgren et al., 2018). Moreover, few studies have specifically examined these links in youth, with the majority of this work in adolescents and adults (Fikke et al., 2011; Giannetta et al., 2012; Oldershaw et al., 2009). This gap in knowledge is surprising considering the potential for development of prevention and intervention programs. One study to date, has examined predictive ability of diminished childhood response inhibition on presence of adolescent NSSI and suicidality in a sample of females with ADHD vs. typical controls, and found that difficulties in inhibition predict NSSI in adolescence (Meza et al., 2016). The present study further expands upon these results with a larger transdiagnostic sample of pre-adolescents, which examines the predictive role of these links more proximally in time and with both neural and behavioral correlates of cognitive control.

Consistent with previous work, difficulties in inhibition and not cognitive flexibility were predictive of NSSI (Allen & Hooley, 2015; Fikke et al., 2011; Giannetta et al., 2012; Hamza et al., 2015; McHugh et al., 2019; Oldershaw et al., 2009). The *Experiential Avoidance Model* of NSSI (Chapman et al., 2006) has proposed impulsivity as one specific risk marker for NSSI, while theoretical work of the *Emotional Cascades Model* (Selby et al., 2008) has proposed perseverative thinking (i.e., cognitive inflexibility) as a risk marker for broad behavioral dysregulation, with applications to NSSI (Selby et al., 2010). The results from the present study support a role of

difficulties with inhibition on NSSI. Interestingly, only neural markers of inhibition (i.e., diminished LPFC, ACC, and striatum activation) were linked with NSSI, while behavioral markers of inhibition (i.e., SSRT mean) did not predict NSSI. These results are consistent with one study which found that although women with NSSI vs. controls demonstrated similar behavioral performance on a cognitive interference processing task, they demonstrated diminished activation of the dorsolateral prefrontal cortex, as compared to controls (Dahlgren et al., 2018). Similarly, in adults with NSSI vs. controls, behavioral markers of inhibition as measured by SSRT from a valenced SST did not differ between groups. Together, this work points toward the potential for neural markers of inhibition to capture the inhibitory control differences in youth who self-injure. Diminished LPFC and ACC activation predicted parent-reported NSSI, while diminished ACC and striatum activation predicted child-reported NSSI. The altered roles of diminished LPFC activation on parent-reported NSSI vs. diminished striatum activation on child-reported NSSI may be related to mechanisms which were not captured in this study (i.e., alexithymia, parent involvement) and future work may want to disentangle these associations.

Neural correlates of inhibition were also related to other areas of suicidality, though of note most of these associations were related to *history* of suicidality rather than *current* suicidality. It is known that there is a strong relationship between suicidality and NSSI (Nock & Prinstein, 2004, 2005), and *history* of suicidality may further potentiate NSSI, as NSSI many times serves as a coping mechanism for intense emotions (Brown et al., 2002; Chapman et al., 2006; Nock & Prinstein, 2004, 2005; Selby et al., 2008). The exception to this pattern, were findings of diminished SST ACC and striatum activation impacting parent-reported *current* active suicidal ideation, though of note these relationships did not survive bootstrapping. The overlap between NSSI and suicidal ideation may greatly overlap for 11–12-year-olds; In this sample about 56% of

those with parent-reported *current* active suicidal ideation also reported NSSI, this contrasts an overlap of 35% in those with child-reported *current* active suicidal ideation that also reported NSSI. The variability in how these constructs are perceived by parents vs. youth may also relate to how these measures perform in their predictive ability and parallel findings. More work is needed in understanding the risk for NSSI vs. suicidality in youth (Oppenheimer et al., 2022) and the development and application of more detailed measures of self-injurious thoughts and behaviors in youth (i.e., Self-Injurious Thoughts and Behaviors Interview; SITBI, or Columbia Suicide Severity Rating Scale; CSSR-S; Nock & Prinstein, 2004; Posner et al., 2011). Altogether, this work is a first step in identifying early cognitive and affective risk factors in pre-adolescence that relate to NSSI and suicidality, it will be important to examine whether these predictors continue to predict longitudinal outcomes in adolescence and adulthood. Moreover, it is imperative to also understand how changes over time in cognitive control and emotion regulation may impact NSSI and suicidality, as this can help inform targeted interventions. Findings of neural, and not behavioral, markers of cognitive control predicting NSSI underscores a need to examine neural mechanisms of cognitive control as risk markers, as they may capture individual differences that cannot be captured via task performance.

Cognitive flexibility is a broad construct, and as defined in this study relied on age-normed accuracy and RT in a card sorting task. Our findings suggested that poorer cognitive flexibility had an effect on parent-reported *current* nonspecific active suicidal ideation, and *current* suicide attempts. This measure may not have captured the learning and habit related mechanisms that are related to cognitive flexibility and thought to underlie persistent and chronic NSSI (Gillan et al., 2011; Liu, 2017; Robbins et al., 2012) and more work is needed in understanding how these mechanisms may relate to one another and at varying degrees of NSSI. Moreover, as the dependent

variables of NSSI and suicidality were dichotomous, much of the heterogeneity of this behavior was lost. Although a relationship between cognitive flexibility and NSSI was not found, this study replicated findings of cognitive inflexibility, as indicated via perseverative errors on the WCST, and suicidality and suicide attempts (Davis & Nolen-Hoeksema, 2000; Martin et al., 1991; Marzuk et al., 2005; McGirr et al., 2012; Miranda et al., 2012). Future work should further specifically examine the role of perseverative errors on NSSI. This specific association could not be probed in this study, as that data was not publicly available. The present study extends findings of links between cognitive flexibility and suicidality into a large transdiagnostic longitudinal sample of youth.

Consistent with a large body of literature, mechanisms of cognitive control predicted emotion regulation in this study. The overlap between cognitive control and emotion regulation has been well documented (Hendricks & Buchanan, 2016; Joormann & Tanovic, 2015; Ochsner & Gross, 2005; Pruessner et al., 2020). For the purposes of this study, difficulties in emotion regulation, as measured by the emotional stroop, was conceptualized as slower incongruent RT. This hypothesis was based off of previous work which has demonstrated that individuals with emotional and behavioral difficulties have slower incongruent RT (Algom et al., 2004; Williams et al., 1996) and activation of LPFC, ACC and striatum differentiating successful inhibition (Casey et al., 2018). Within this sample, we found support for diminished cognitive flexibility impacting slower emotional stroop incongruent RT. Results from this study are similar to reported difficulties in cognitive flexibility in those with internalizing difficulties (Grant et al., 2001; Martin et al., 1991; McGirr et al., 2012; Snyder, 2013). Further, diminished LPFC and striatum activation predicted slower emotional stroop incongruent RT. Broadly, this work converges with overlapping neural mechanisms of cognitive control and emotion regulation (Ochsner & Gross, 2005). Slower

SSRT also was associated with slower emotional stroop incongruent RT. This is consistent with work finding slower SSRT in disorders related to impulsive decision making and emotional reactivity (i.e., youth with ADHD, women with borderline personality disorder, and substance use disorders; Alderson et al., 2007; LeGris et al., 2012; Li et al., 2006). Altogether, poorer cognitive control at ages 9-11 were linked to poorer emotion regulation at ages 11-12. Surprisingly, emotion regulation did not predict NSSI or suicidality above and beyond measures of cognitive control. This may in part be related to the emotional stroop task requiring an inhibitory response and thus relies on cognitive control mechanisms. Additionally, similar to neural, and not behavioral, correlates of inhibition predicting NSSI, emotion regulation captured by behavioral measures of the emotional stroop task may not fully capture individual differences in emotion regulation. Thus, it will be important for future work to incorporate biomarkers and other measures that may index emotion regulation, as well as emotion reactivity.

Autistic traits were predictive of most NSSI and suicidality measures above and beyond cognitive control measures, though autistic traits did not play a moderating role on any of the proposed paths. It will be important for future work to examine the potential mediating role of autistic traits. Further, core autistic features (i.e., restricted and repetitive behaviors vs. reciprocal social interaction) may play different roles on these relationships. For example, restricted and repetitive behaviors have been strongly linked with both emotion regulation and cognitive control (Greenlee et al., 2021; Kenworthy et al., 2008; Lopez et al., 2005; Miller et al., 2015; Samson et al., 2014), and thus high restricted and repetitive behaviors may more specifically impact these links.

### **Limitations & Future Directions**

Although there are strengths to the present work, i.e., a robust sample size, longitudinal variables to examine the predictive role of cognitive control and emotion regulation on NSSI and suicidality, and neural correlates of cognitive control, there are also several limitations. First, as ABCD is a multisite study, there are several variables that may add to the heterogeneity in these findings. More specifically, although the baseline data was recruited to be representative of sociodemographic factors in the United States, self-selection biases by families in their enrollment of the study was possible (Garavan et al., 2018), and may be particularly evident at follow-up timepoints. For example, at timepoint three (ages 11-12), only about half of individuals had measures of NSSI and suicidality available, and of note, the rates for NSSI and suicidality were significantly less than ABCD's rates of NSSI and suicidality at baseline (e.g., presence of parent-reported *history of* NSSI at 11-12 years was 3.89% vs. prevalence rate of 9.14% at baseline of 9-10 years of age; DeVille et al., 2020). There is a dearth of work noting that rates of NSSI increase from childhood to adolescence, and that the prevalence rates in general adolescent samples are about 17-18% (Swannell et al., 2014; Zetterqvist et al., 2013). Moreover, considering the large amount of missing data at follow-up timepoints, it may be possible that participants at risk for highest stress (who may also be at risk for NSSI and suicidality) may not return for longitudinal follow-up. More work is needed understanding factors that impact the retention rate in the ABCD Study. In addition to missing data for timepoint three, only about half of the participants had all variables and the majority of missing data was from the second timepoint, in which measures of emotion regulation, cognitive flexibility, and autistic traits were collected at. Relatedly, these measures being collected at the same time may partially explain the increased strength in connectivity between emotion regulation and cognitive flexibility vs. weak but significant relationships with measures of inhibition, which were collected at the first time point. Moreover,

although significant, the strength of the connections between inhibition and NSSI and suicidality were very weak and explained very little variability, thus, it will be important for future work to examine other relevant mediators of inhibition on NSSI and suicidality. Finally, although the model proposed included mediations and moderated mediations, these final models were ultimately not run, as the preliminary steps to test a mediation were not met. A significant limitation of this work was the dichotomous nature of NSSI and suicidality measures. Statistically, classic mediation and moderation is completed with continuous dependent variables. Recent work has tested methods for mediation with dichotomous dependent variables (Rijnhart et al., 2019; Samawi et al., 2018), and these methods have been derived from mediation steps proposed by Baron & Kenny (1986) within a logistic regression framework (Kenny, 2013), as used in the present study. There is evidence to suggest that structural equation modelling may reduce bias with dichotomous dependent variables, though development of statistical analyses that support dichotomous outcomes is just emerging (Rijnhart et al., 2019).

Several limitations related to the measures should also be noted. The K-SADS-5 constructs may be both too broad and too narrow. For example, “nonspecific active suicidal ideation” is specific, though functionally may be subsumed under active suicidal ideation. Additionally, although the K-SADS-5 provides codes for both *current*, and *history of* NSSI/suicidality, there is no index for how severe these thoughts and behaviors are when they are present. For example, a child who has frequently self-harmed and a child who has self-harmed once are weighted equally. Similarly, a child who has self-harmed and left deep scars and a child who has superficially self-harmed are also weighted equally. The dimensionality and nature of such complex behaviors were not fully captured by the K-SADS-5. Additionally, although there is a strength of including both child- and parent-reported NSSI/suicidality in this study, there may be mediating or moderating

variables (i.e., alexithymia, parent's involvement with child, emotion socialization) which may explain why these relationships exist between only parent- vs. child-reported measures.

Although the SRS has been well-validated in autistic and general samples as a screening measure and a measure of autistic traits (Constantino et al., 2003; Constantino & Todd, 2005), the sSRS has not yet been validated as a measure of autistic traits. This is particularly difficult to do within the ABCD data, as no reported measures of diagnosis of autism spectrum disorder were not collected and thus, the validity of the sSRS in this sample cannot be evaluated. Moreover, as the sSRS was limited in items, the restricted and repetitive behavior index vs. the social communication index could not be parsed apart to further examine the role of these core autistic features on the relationships of interest.

It will be important for cognitive and affective risk markers of NSSI and suicidality to continue to be evaluated across development, as it will allow understanding of risk and maintenance factors (Oppenheimer et al., 2022). These defined developmental risk markers of cognitive control on NSSI may point to potentially modifiable targets that may influence the relationship between pre-existing risk factors and eventual self-injurious thoughts and behaviors among youth. Ultimately, this may open up new horizons in research and clinical care for at-risk youth, by discovering targets that may reduce risk for self-injurious thoughts and behavior when engaged directly.

## **Conclusion**

This study examined developmental links between cognitive control and emotion regulation on several facets of self-injurious thoughts and behaviors in a large sample of youth. Although a mediation of emotion regulation on cognitive control and self-injurious thoughts and behaviors was not supported, important direct effects were found between neural correlates of



inhibition on NSSI, and behavioral measures of cognitive flexibility and inhibition on suicidality. Further, links between poorer cognitive control and poorer emotion regulation were found. An exploratory aim of this study was examining the potential moderating role of autistic traits on significant associations. Although greater autistic traits significantly predicted presence of self-injurious thoughts and behaviors, this study did not find a moderation of autistic traits. This work is an important step in identifying neural and behavioral risk markers for self-injurious thoughts and behaviors in youth and developing novel targeted prevention and intervention programs.

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Figure 1. Proposed latent model.

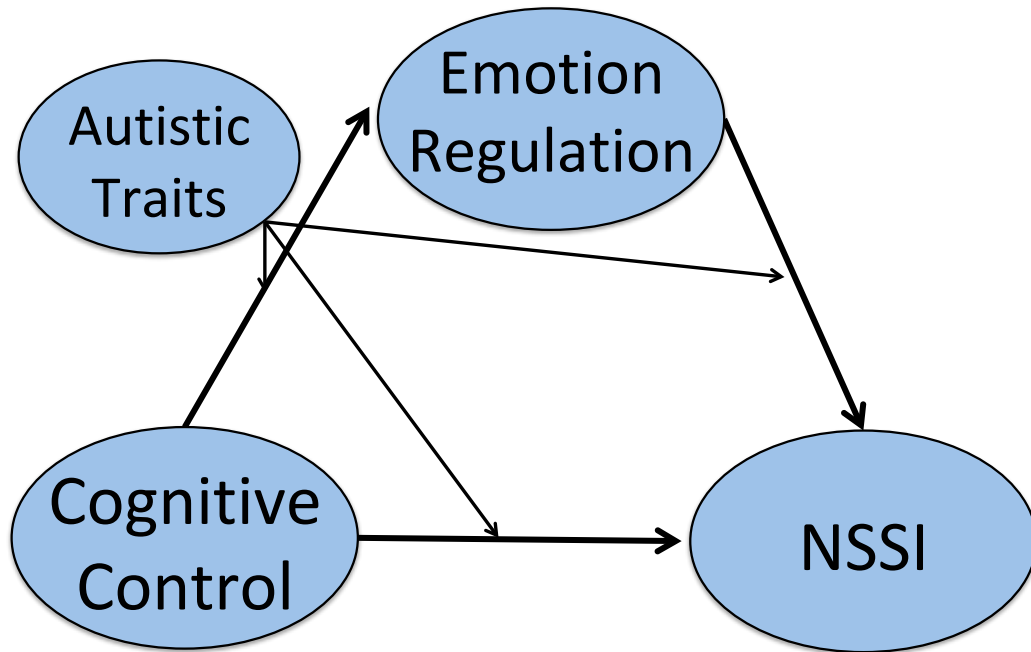
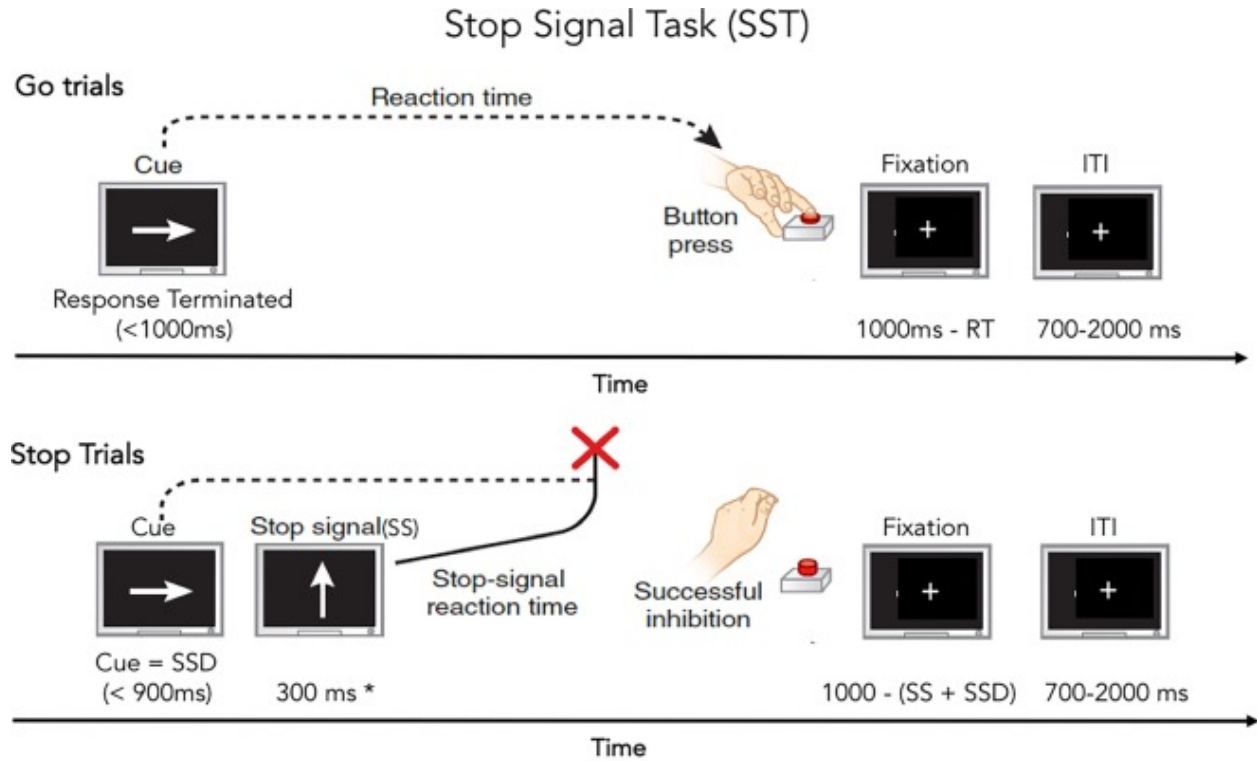


Figure 2. Trial sequence for Stop Signal Task (SST). Of note, although images depict black background with white stimuli, publicly available ABCD SST uses a gray background and black stimuli. Figure image from Casey et al., 2018.



\* If the SSD > 700 ms then the SS duration = 1000-SSD.

Figure 3. Trial sequence for the NIH Toolbox Dimensional Change Card Sort (TDCCS) Test using practice stimuli. Figure image from Zelazo et al., 2013; Zelazo et al., 2014. © 2006–2012 National Institutes of Health and Northwestern University.

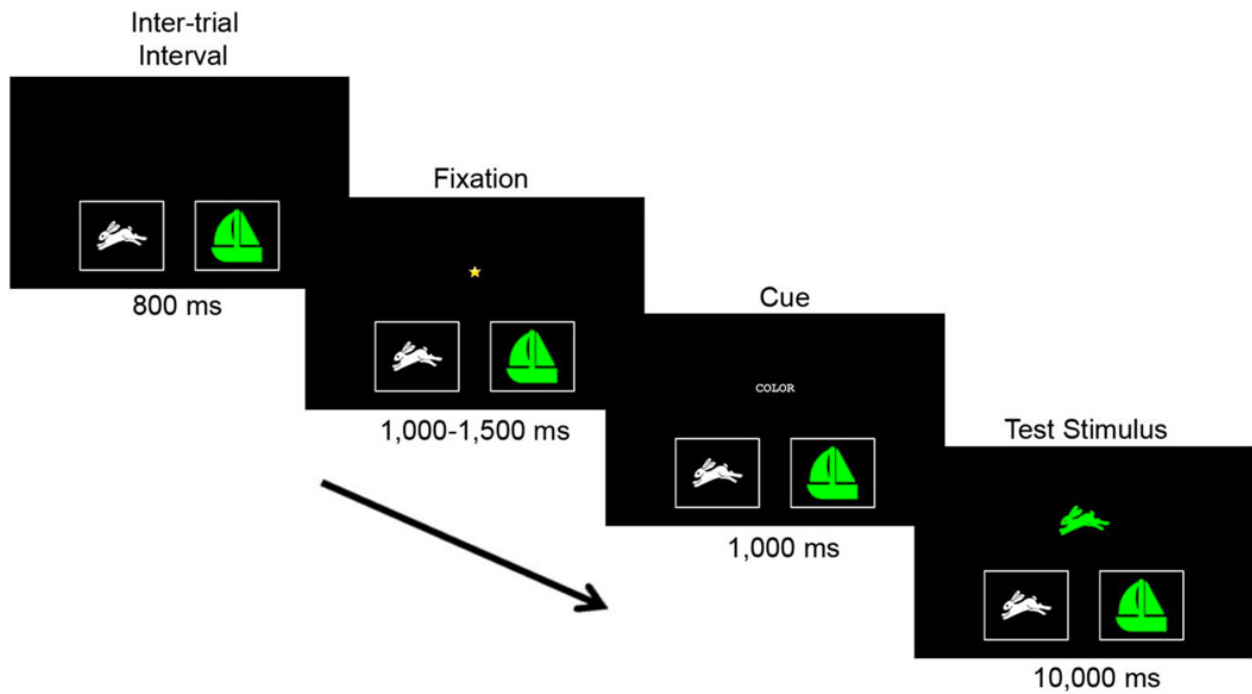
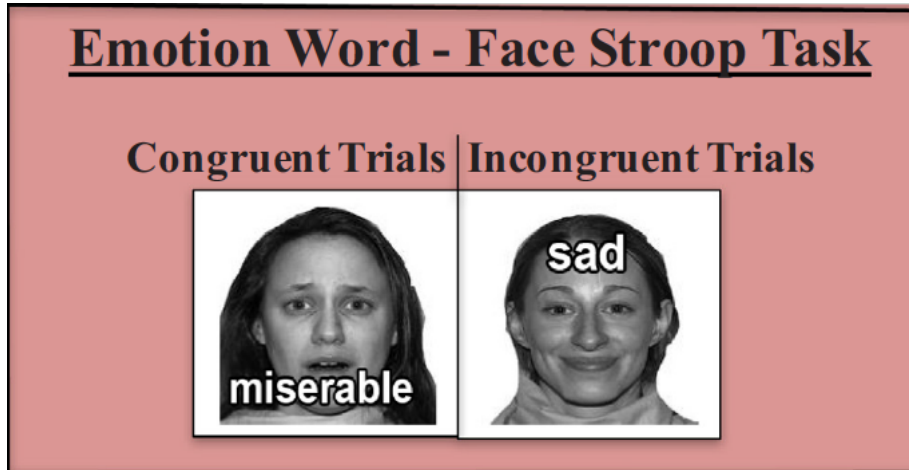
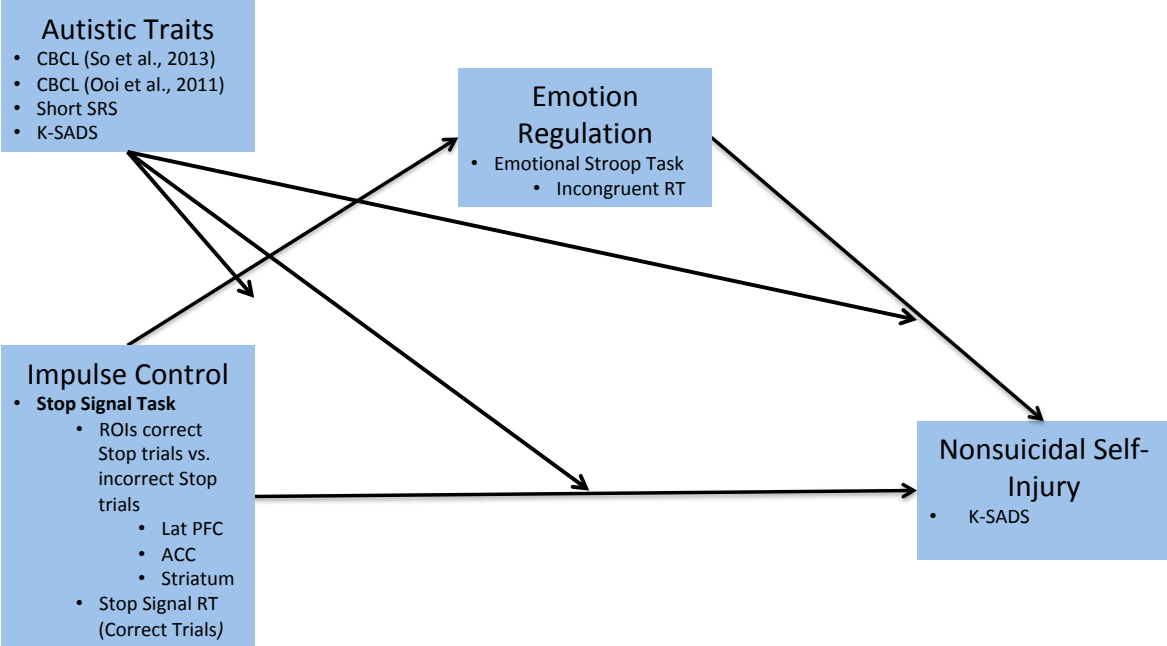




Figure 4. Example of stimuli used for the Emotional Stroop Task. Image adapted from as Banich and colleagues (2019).



Figures 5a. Planned moderated mediation analyses with manifest variables. Figure 5a depicts emotion regulation mediating the path between impulse control and NSSI, with autistic traits moderating all paths.



Figures 5b. Planned moderated mediation analyses with manifest variables. Figure 5b depicts emotion regulation mediating the path between cognitive flexibility and NSSI, with autistic traits moderating all paths.

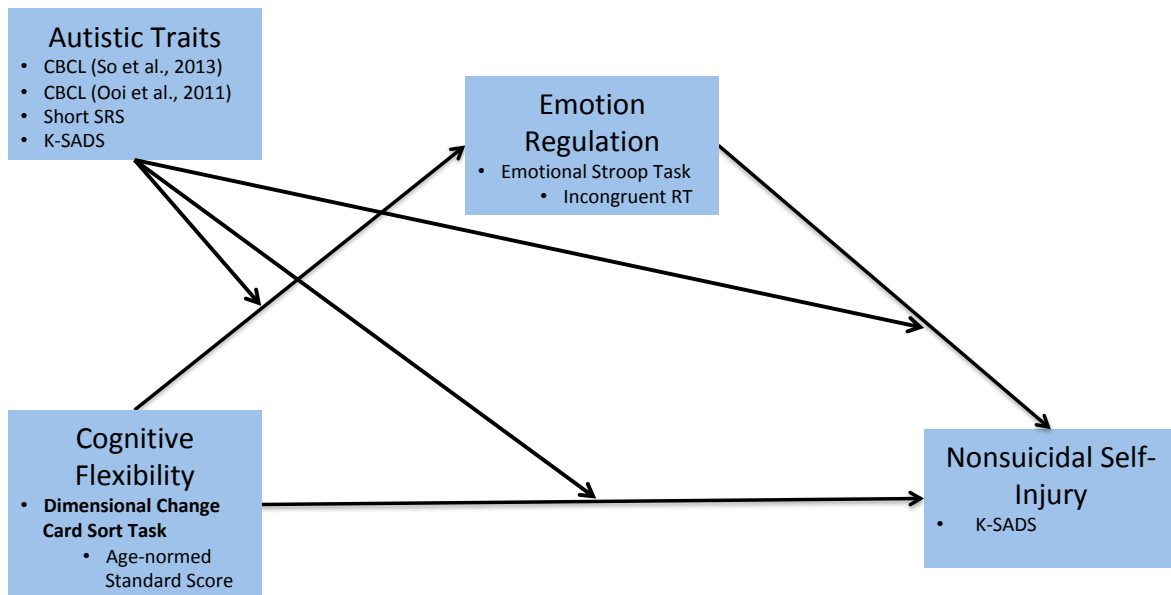


Table 1. Items from CBCL ASD measures that were identified as being the most sensitive and specific with DSM-5 ASD diagnosis.

<b>Ooi et al., 2011</b>	<b>So et al., 2013</b>
1. Acts too young for his/her age (Attention Problems)	1. Acts too young for his/her age (Attention Problems)
25. Doesn't get along with other kids (Social Problems)	9. Cannot get his/her mind off certain thoughts/obsessions (Thought Problems)
29. Fears certain animals, situations (Anxious/Depressed)	17. Daydream or gets lost in his/her thoughts (Attention Problems)
42. Would rather be alone than with others (Withdrawn/Depressed)	42. Would rather be alone than with others (Withdrawn/Depressed)
46. Nervous movements or twitching (Thought Problems)	62. Poorly coordinated or clumsy (Social Problems)
66. Repeats certain acts over and over (Thought Problems)	66. Repeats certain acts over and over (Thought Problems)
79. Speech problem (Social Problems)	79. Speech problem (Social Problems)
84. Strange behavior (Thought Problems)	80. Stares blankly (Attention Problems)
111. Withdrawn, doesn't get involved with others (Withdrawn/Depressed)	84. Strange behavior (Thought Problems)
	111. Withdrawn, doesn't get involved with others (Withdrawn/Depressed)

Table 2. Items from the Short Social Responsiveness Scale (SRS).

<b>Short Social Responsiveness Scale Items</b>
6. Would rather be alone than with others.
15. Is able to understand the meaning of other people's tone of voice and facial expressions.
16. Avoids eye contact or has unusual eye contact.
18. Has difficulty making friends, even when trying his or her best.
24. Has more difficulty than other children with changes in his or her routine.
29. Is regarded by other children as odd or weird.
35. Has trouble keeping up with the flow of normal conversation.
27. Has difficulty relating to peers.
39. Has an unusually narrow range of interests.
42. Seems overly sensitive to sounds, textures or smells.
58. Concentrates too much on parts of things rather than seeing the whole picture. For example, if asked to describe what happened in a story, he or she may talk only about the kind of clothes the characters were wearing.

Table 3. Sample characteristics (N=6447)

	<b>n</b>	<b>Mean</b>	<b>SD</b>	<b>Minimum</b>	<b>Maximum</b>
Age at K-SADS (months)	6447	143.480	7.750	121.000	163.000
SST LPFC	5157	0.000	0.183	-1.050	1.030
SST ACC	5140	0.005	0.210	-1.010	1.020
SST striatum	5158	0.032	0.174	-0.730	0.770
SSRT mean	5580	300.683	72.699	3.946	592.623
sSRS raw score	6431	3.390	4.036	0.000	31.000
Emotional stroop incongruent RT	6328	1084.128	124.628	673.424	1512.317
TDCCS score	6351	97.280	14.654	68.000	148.000

Table 4. Rates of self-injurious thoughts and behaviors.

	<b>Present</b>	<b>Not Present</b>	<b>Rate</b>
<b>Parent-reported variables (n=6,351)</b>			
Current NSSI	124	6227	1.95%
History of NSSI	247	6104	3.89%
Current Passive Suicidal Ideation	42	6309	0.66%
History of Passive Suicidal Ideation	449	5902	7.07%
Current Nonspecific Active Suicidal Ideation	79	6272	1.24%
History of Nonspecific Active Suicidal Ideation	331	6020	5.21%
Current Active Suicidal Ideation	16	6335	0.25%
History of Active Suicidal Ideation	98	6253	1.54%
Current Suicide Attempt	5	6346	0.08%
History of Suicide Attempt	21	6330	0.33%
<b>Child-reported variables (n=6,381)</b>			
Current NSSI	89	6292	1.39%
History of NSSI	229	6152	3.59%
Current Passive Suicidal Ideation	54	6327	0.85%
History of Passive Suicidal Ideation	386	5995	6.05%
Current Nonspecific Active Suicidal Ideation	74	6307	1.16%
History of Nonspecific Active Suicidal Ideation	230	6151	3.60%
Current Active Suicidal Ideation	36	6345	0.56%
History of Active Suicidal Ideation	82	6299	1.29%
Current Suicide Attempt	7	6374	0.11%
History of Suicide Attempt	72	6309	1.13%

Table 5. Logistic regression results for step 1: the role of cognitive control on self-injurious thoughts and behaviors (X->Y).

	Parent-Reported		Child-reported	
	<i>Estimate</i>	<i>Nagelkerke R<sup>2</sup></i>	<i>Estimate</i>	<i>Nagelkerke R<sup>2</sup></i>
<b>Current NSSI</b>				
TDCCS score	--	--	--	--
SST LPFC	-1.396**	.008*	--	--
SST ACC	-1.009*	.005*	-1.399*	.009*
SST striatum	--	--	-1.564*	.008*
SSRT mean	--	--	--	--
<b>History of NSSI</b>				
TDCCS score	--	--	--	--
SST LPFC	--	--	--	--
SST ACC	--	--	--	--
SST striatum	--	--	--	--
SSRT mean	--	--	--	--
<b>Current Passive Suicidal Ideation</b>				
TDCCS score	--	--	--	--
SST LPFC	--	--	--	--
SST ACC	--	--	--	--
SST striatum	--	--	--	--
SSRT mean	--	--	--	--
<b>History of Passive Suicidal Ideation</b>				
TDCCS score	--	--	--	--
SST LPFC	-0.723*	.003*	--	--
SST ACC	-0.577*	.002*	--	--
SST striatum	--	--	--	--
SSRT mean	--	--	--	--
<b>Current Nonspecific Active Suicidal Ideation</b>				
TDCCS score	-.025*	.011**	--	--
SST LPFC	--	--	--	--
SST ACC	--	--	--	--
SST striatum	--	--	--	--
SSRT mean	--	--	--	--
<b>History of Nonspecific Active Suicidal Ideation</b>				
TDCCS score	--	--	--	--



SST LPFC	-0.729 <sup>^</sup>	.003*	--	--
SST ACC	--	--	--	--
SST striatum	--	--	--	--
SSRT mean	--	--	--	--
<b>Current Active Suicidal Ideation</b>				
TDCCS score	--	--	--	--
SST LPFC	--	--	--	--
SST ACC	-3.464**	.050**	--	--
SST striatum	-3.325*	.028*	--	--
SSRT mean	--	--	--	--
<b>History of Active Suicidal Ideation</b>				
TDCCS score	--	--	--	--
SST LPFC	--	--	--	--
SST ACC	--	--	-1.176**	.008**
SST striatum	--	--	--	--
SSRT mean	--	--	--	--
<b>Current Suicide Attempt</b>				
TDCCS score	-.103	.071*	--	--
SST LPFC	-3.998	.056 <sup>^</sup>	--	--
SST ACC	--	--	--	--
SST striatum	--	--	--	--
SSRT mean	-0.13*	.066*	--	--
<b>History of Suicide Attempt</b>				
TDCCS score	--	--	--	--
SST LPFC	--	--	--	--
SST ACC	--	--	--	--
SST striatum	--	--	--	--
SSRT mean	--	--	--	--

Note: <sup>^</sup> =  $p < .10$ , \* =  $p < .05$ , \*\* =  $p < .01$ , \*\*\* =  $p < .001$ . Logistic regressions were only conducted on significant correlations. P-values for bootstrapping at 1000 times with a CI of 95% are represented here.

Table 6. Linear regression results for step 2: the role of cognitive control of emotion regulation (X->M).

	<i>Estimate</i>	<i>R</i> <sup>2</sup>
<b>Emotional Stroop Incongruent RT</b>		
TDCCS score	-2.700***	.101***
SST LPFC	-28.904**	.002**
SST ACC	--	--
SST striatum	-47.91***	.005***
SSRT mean	.264***	.024***

Note:  $\wedge = p < .10$ , \* =  $p < .05$ , \*\* =  $p < .01$ , \*\*\* =  $p < .001$ . Linear regressions were only conducted on significant correlations. P-values for bootstrapping at 1000 times with a CI of 95% are represented here.

Table 7. Logistic regression results for step 3: the role of emotion regulation on self-injurious thoughts and behaviors, when controlling for cognitive control (M->Y when controlling for X).

	Parent-Reported <i>Nagelkerke</i>		Child-reported <i>Nagelkerke</i>	
	<i>Estimate</i>	<i>R<sup>2</sup></i>	<i>Estimate</i>	<i>R<sup>2</sup></i>
<b>Current NSSI</b>				
SST LPFC	-1.329*	.007 <sup>^</sup>	--	--
Emotional stroop incongruent RT	.000		--	
SST striatum	--	--	-1.341 <sup>^</sup>	.006
Emotional stroop incongruent RT	--		.001	
<b>History of Passive Suicidal Ideation</b>				
SST LPFC	-0.787*	.004*	--	--
Emotional stroop incongruent RT	.000		--	
<b>Current Nonspecific Active Suicidal Ideation</b>				
TDCCS score	-.023*	.011*	--	--
Emotional stroop incongruent RT	.001		--	
<b>Current Active Suicidal Ideation</b>				
SST striatum	-4.051**	.042*	--	--
Emotional stroop incongruent RT	-.001		--	
<b>Current Active Attempt</b>				
SSRT	-0.012*	.090 <sup>^</sup>	--	--
Emotional stroop incongruent RT	.005		--	

Note: <sup>^</sup> =  $p < .10$ , \* =  $p < .05$ , \*\* =  $p < .01$ , \*\*\* =  $p < .001$ . Logistic regressions for this step were only conducted if there were significant step 1 and step 2 regressions. P-values for bootstrapping at 1000 times with a CI of 95% are represented here.

Table 8. Regression results examining whether autistic traits moderated the role of cognitive control on self-injurious thoughts and behaviors (X->Y).

	Parent-Reported		Child-reported	
	<i>Nagelkerke</i>		<i>Nagelkerke</i>	
	<i>Estimate</i>	<i>R<sup>2</sup></i>	<i>Estimate</i>	<i>R<sup>2</sup></i>
<b>Current NSSI</b>				
SST LPFC	-1.344*	.057***	--	--
sSRS	.121***		--	
SST LPFC x sSRS interaction	.029		--	
SST ACC	-.941 <sup>^</sup>	.055***	-1.332 <sup>^</sup>	.024**
sSRS	.121***		.077***	
SST ACC x sSRS interaction	.032		.016	
SST striatum	--	--	-1.470 <sup>^</sup>	.023**
sSRS	--		.076***	
SST striatum x sSRS interaction	--		-.011	
<b>History of Passive Suicidal Ideation</b>				
SST LPFC	-0.593 <sup>^</sup>	.088***	--	--
sSRS	.140***		--	
SST LPFC x sSRS interaction	.017		--	
SST ACC	-0.499 <sup>^</sup>	.087***	--	--
sSRS	.141***		--	
SST ACC x sSRS interaction	.038		--	
<b>Current Nonspecific Active Suicidal Ideation</b>				
TDCCS score	-.026*	.057***	--	--
sSRS	.119***		--	
TDCCS score x sSRS interaction	.001		--	
<b>Current Active Suicidal Ideation</b>				
SST ACC	-2.779*	.078**	--	--
sSRS	.073		--	
SST ACC x sSRS interaction	-.123		--	
SST striatum	-3.075 <sup>^</sup>	.054*	--	--
sSRS	.103***		--	
SST striatum x sSRS interaction	-.017		--	
<b>History of Active Suicidal Ideation</b>				
SST ACC	--	--	-1.279*	.031***
sSRS	--		.087***	
SST ACC x sSRS interaction	--		-.018	
<b>Current Suicide Attempt</b>				

SSRT mean	-0.015*	.158**	--	--
sSRS	.196**		--	
SSRT mean x sSRS interaction	.000		--	

Note:  $\wedge = p < .10$ ,  $* = p < .05$ ,  $** = p < .01$ ,  $*** = p < .001$ . Logistic regressions were only conducted for significant regressions from step 1 of mediation. P-values for bootstrapping at 1000 times with a CI of 95% are represented here.

Table 9. Regression results examining whether autistic traits moderated the role of cognitive control on emotion regulation (X->M).

	<i>Estimate</i>	<i>R<sup>2</sup></i>
<b>Emotional Stroop Incongruent RT</b>		
TDCCS score	-2.691***	.102***
sSRS	.775 <sup>^</sup>	
TDCCS score x sSRS interaction	-.025	
SST LPFC	-27.650***	.003***
sSRS	1.235**	
SST LPFC x sSRS interaction	-.964	
SST striatum	-47.300***	.006***
sSRS	1.245**	
SST striatum x sSRS interaction	-.943	
SSRT	.262***	.025***
sSRS	1.261**	
SSRT x sSRS interaction	.003	

Note: <sup>^</sup> =  $p < .10$ , \* =  $p < .05$ , \*\* =  $p < .01$ , \*\*\* =  $p < .001$ . Linear regressions were only conducted for significant regressions from step 2 of mediation. P-values for bootstrapping at 1000 times with a CI of 95% are represented here.

Table 10. Regression results examining whether autistic traits moderated the role of emotion regulation on self-injurious thoughts and behaviors (M->Y).

	Parent-Reported		Child-reported	
	<i>Estimate</i>	<i>Nagelkerke R<sup>2</sup></i>	<i>Estimate</i>	<i>Nagelkerke R<sup>2</sup></i>
<b>History of NSSI</b>				
Emotional stroop incongruent RT	-.001*	.063***	-.001*	.028***
sSRS	.122***		.087***	
Emotional stroop x sSRS interaction	.000		.000	

Note:  $\wedge = p < .10$ , \* =  $p < .05$ , \*\* =  $p < .01$ , \*\*\* =  $p < .001$ . Logistic regressions were conducted for any significant regressions of M->Y. P-values for bootstrapping at 1000 times with a CI of 95% are represented here.