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Associations Among Self-Regulation, Life Stress, and Suicidal Ideation in Adolescents: A Developmental Psychopathology Approach

A thesis submitted in partial fulfillment of the requirement
for the degree of Bachelor of Science in Psychological Sciences from
William & Mary

by

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Associations Among Self-Regulation, Life Stress, and Suicidal Ideation in Adolescents: A Developmental Psychopathology Approach

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Abstract

Background: Suicide is a major public health concern among adolescents. Although research has made progress in identifying risk factors for youth suicidality, there has been less focus on early developmental antecedents of youth suicidal thoughts and behaviors. Taking a developmental psychopathology perspective, we examined longitudinal associations among multiple aspects of self-regulation (i.e., emotion regulation, emotion reactivity, parasympathetic regulation, inhibitory control), life stress, and suicidal ideation. We hypothesized that deficits in self-regulation during middle childhood and early adolescence and greater life stress during early and middle childhood would predict higher lifetime suicidal ideation reported in adolescence.

Method: Participants were adolescents (N = 177) enrolled in a longitudinal follow-up of a randomized control trial evaluating the efficacy of a parenting intervention in infancy. Self-regulation was assessed using parent-reported emotion regulation, self-reported emotion reactivity, parasympathetic regulation (i.e., respiratory sinus arrhythmia at rest and in response to challenge), and inhibitory control during two behavioral tasks. Early life stress was scored based on parent report, and adolescents reported suicidal ideation. Bivariate and multivariate analyses were used to assess socio-demographics, risk group, self-regulation, and life stress as predictors of youth suicidality.

Results: Greater emotion reactivity significantly predicted greater suicidal ideation intensity in adolescence, and this association persisted after controlling for sex. Other candidate predictors were not significantly associated with youth suicidality, although life stress during early and middle childhood predicted worse emotion regulation and inhibitory control during middle childhood.

Conclusion: Results provide evidence for heightened emotion reactivity as a risk factor for

suicidal ideation in adolescents and have clinical implications for prevention and intervention targeting youth suicidality. More research is needed on the role of life stress in predicting self-regulation in middle childhood and beyond.

Keywords: Adolescents, suicide, suicidal ideation, self-regulation, early life stress

Associations Between Self-Regulation, Life Stress, and Suicidal Ideation in Adolescents: A Developmental Psychopathology Approach

Suicidal thoughts and behaviors (STBs) are major public health concerns in adolescents. Alarmingly, suicide is the second leading cause of death among adolescents aged 10-14 and the third leading cause of death among adolescents aged 15-19 in the United States (Centers for Disease Control and Prevention [CDC], 2020). In 2021, 22% of U.S. high school students reported seriously considering attempting suicide in the past year, and 10% reported having made one or more suicide attempts in the past year, with rates consistently increasing starting from 2017 (CDC, 2017; 2019; 2021). These statistics present an urgent need for a better understanding of the development of STBs among adolescents in order to inform timely, targeted prevention and intervention strategies.

Research over the past decades has made progress in identifying risk factors and correlates associated with youth STBs, including environmental (e.g., childhood maltreatment and bullying), psychological (e.g., affective, cognitive, and social processes), and biological factors (Cha et al., 2018). However, there remain significant gaps in the current literature on youth suicidality. First, most studies have focused on single, static risk factors using cross-sectional designs (Franklin et al., 2017), limiting their ability to compare effects across different candidate risk factors. Second, most of what we know about youth suicidality comes from data collected exclusively during adolescence. With limited research investigating the vulnerable periods before adolescence (Soole et al., 2015), we know little about the developmental antecedents of STBs from early or middle childhood into adolescence. These gaps limit our ability to effectively prevent and intervene youth STBs.

One promising way to bridge these gaps is to integrate a developmental psychopathology

perspective into youth suicide research. This perspective, which conceptualizes psychopathology as a deviation from normative development emerging dynamically from the interactions of internal systems and external contexts across the lifecourse, carries unique benefits (Cicchetti & Rogosch, 2002). First, it allows researchers to identify risk factors relevant to specific developmental periods and thus recognize potential mechanisms in the developmental progression of youth STBs (Oppenheimer et al., 2022). Specifically, studying the associations between deviations from typical developmental trajectories in early to middle childhood and suicide-related outcomes in adolescence enhances our understanding of the nature of youth STBs as well as possible causal pathways. Second, our knowledge of youth suicidality informed by a developmental psychopathology perspective can be utilized to guide the development of prevention and intervention strategies targeted to each developmental phase before the emergence of youth STBs. Waiting until suicidal behavior has already developed brings about significant healthcare costs (Babcock et al., 2021; Glenn et al., 2017) as well as considerable suffering on the individual, family, and social level. Thus, early preventive measures are needed to alleviate both the financial and social burden of youth STBs.

In view of the fact that our ability to predict suicidality is only slightly above chance levels (Franklin et al., 2017), the developmental psychopathology framework may improve the prediction of youth suicidality by examining the unique and combined effects of multiple risk factors from childhood to adolescence. Reflecting the commitment of developmental psychopathology to incorporating multiple levels of analysis (Cicchetti & Rogosch, 2002), researchers may conceptualize suicide as resulting from the complex interplay of many different environmental, psychological, and biological factors (Beurs et al., 2021). One candidate risk factor involves difficulty with self-regulation at both the physiological and behavioral levels.

Self-Regulation and Youth Suicidality

Self-regulation, an umbrella term that encompasses multiple regulatory factors, offers a promising framework for psychopathology research (Strauman, 2017). Self-regulation is defined as "the process by which people initiate, maintain, and control their own thoughts, behaviors, or emotions, with the intention of producing a desired outcome or avoiding an undesired outcome" (Strauman, 2017, p. 498). Dysfunction in self-regulation has been associated with a range of psychological disorders including eating disorders, substance use disorders, depression, and anxiety disorders (Ryan et al., 1997; Leary & Buttermore, 2003). Furthermore, research has demonstrated that difficulty self-regulating in early childhood predicts more internalizing and externalizing problems in middle childhood, and difficulty self-regulating in middle childhood predicts more depressive symptoms and externalizing problems in adolescence (Robson et al., 2020). Given the predictive power of self-regulation for general psychological functioning, researchers have examined the associations between youth suicidality and aspects of self-regulation, including emotion regulation, emotion reactivity, parasympathetic nervous system regulation, and inhibitory control (e.g., Brausch et al., 2022).

There is much evidence indicating that emotion regulation is associated with youth suicidality. Research with psychiatric inpatients found that limited access to emotion regulation strategies predicted past-year suicidal ideation (SI; Hatkevich et al., 2019) and suicide risk (Eaddy et al., 2019). Research with community-based youth has documented that different emotion regulation difficulties predict past-year presence of SI, recent SI severity, future SI severity, past-year suicide attempt (SA), and suicide risk (Brausch et al., 2022; Brausch & Woods, 2019; Haliczer et al., 2021; Pisani et al., 2013). Moreover, better emotion regulation is negatively associated with both SI and SA and may serve as a protective factor for youth suicidality (Barr et al., 2017; Ong

& Thompson, 2019). Interestingly, a study has shown that alexithymia, a factor associated with impaired emotion regulation (Preece et al., 2023) and characterized as the difficulty in identifying and describing feelings, is positively associated with SI in adolescents (Cerutti et al., 2018).

Emotion reactivity, a construct related to emotion regulation, refers to "the extent to which an individual experiences emotions (a) in response to a wide array of stimuli (i.e., emotion sensitivity), (b) strongly or intensely (i.e., emotion intensity), and (c) for a prolonged period of time before returning to baseline level of arousal (i.e., emotion persistence)" (Nock et al., 2008, p. 107). Findings regarding the relationship between emotion reactivity and youth suicidality are mixed. Studies have shown that emotion reactivity is positively correlated with SI in middle schoolers (Evans et al., 2016) and adolescents (Liu et al., 2020; Wu et al., 2021). However, one study found that emotion reactivity did not uniquely predict lifetime SI after including depressive symptoms in the model (Evans et al., 2016), and another study found no significant relationship between emotion reactivity and SI or SA in a sample of adolescents presenting to an emergency outpatient department (Kandsperger et al., 2021). Studies to date have yet to give a clear answer regarding the relationship between youth suicidality and emotion reactivity. Therefore, future research is needed to clarify conflicting findings.

Though multiple lines of evidence have highlighted the relationship between suicidality and emotional functioning in youth, most studies have relied on self-report measures of emotion regulation and emotion reactivity, which may be subject to social desirability and recall bias (Hunt et al., 2003). It is important to also utilize more objective measures of self-regulation. One example is using respiratory sinus arrhythmia (RSA) to assess the parasympathetic nervous system functioning as a physiological indicator of self-regulation. Specifically, numerous studies have pointed out the close connection between RSA and emotion regulation (Beauchaine & Thayer,

2015; Thayer & Lane, 2000; Thayer et al., 2012). There are two types of RSA data that are of particular interest to researchers: resting RSA and RSA in response to challenge. Higher levels of resting RSA are associated with relaxation and indicative of adaptive self-regulation (Beauchaine, 2001; Hastings et al., 2014). Conversely, lower levels of resting RSA are associated with an increased risk for internalizing and externalizing symptoms (Beauchaine, 2015; Beauchaine & Thayer, 2015). On the other hand, moderate RSA decreases in stressful environments are considered to be adaptive physiological functioning (Beauchaine, 2001; Hastings et al., 2014), whereas excessive decreases or increases in RSA reactivity to challenge are typically associated with psychopathology (Graziano & Derefinko, 2013).

The relationship between parasympathetic nervous system regulation and youth suicidality has received moderate empirical support. Current research has demonstrated that parasuicidal adolescents (i.e., suicidal and non-suicidal self-injurious adolescents) had lower resting RSA compared to their controls (Crowell et al., 2005). However, other studies found no relationship between resting RSA and youth suicidality. Specifically, one study found no difference in resting RSA among three groups: adolescents with lifetime depression and STBs, adolescents with lifetime depression but no STBs, and healthy controls (Yang et al., 2019). Another study found that resting RSA was not concurrently associated with or longitudinally predictive of SI in adolescent females (Giletta et al., 2017). Results regarding RSA reactivity to challenge in suicidal youth are also mixed. Studies have found that children and adolescents with a history of STBs did not display a normative RSA decrease in response to stressful or emotional contexts as exhibited by controls, instead showing stable RSA (James et al., 2017; Yang et al., 2019) or a greater RSA decrease than non-suicidal peers (Crowell et al., 2005; Kaufman et al., 2018). One study also found that adolescents who showed greater RSA decreases in response to challenge were at higher risk

for suicidal ideation in the subsequent nine months (Giletta et al., 2017). Further research is needed to clarify the link between suicidality and parasympathetic self-regulation, as measured by resting RSA and RSA reactivity to challenge.

Inhibitory control, another important aspect of self-regulation, has been defined as "an essential executive function" that includes the capacity to inhibit prepotent cognitive and motor responses (McHugh et al., 2019, p. 53). Inhibitory control is often measured using behavioral tasks that require individuals to suppress automatic responses: for example, Flanker tasks that require individuals to ignore distractor images while responding to a central prompt. Evidence regarding the relationship between inhibitory control and suicide-related outcomes in youth has been inconclusive. One study measuring inhibitory control with a two-choice oddball paradigm (i.e., a behavioral task that asks participants to respond to standard and deviant stimuli by pressing different keys as accurately and quickly as possible) found that college students with SI had worse inhibitory control than individuals without SI (Lin et al., 2020). However, other studies have found no significant relationship between youth STBs and inhibitory control as measured by the GoStop Impulsivity Paradigm (Mathias et al., 2011), Stop-Signal Reaction Time task (Millner, 2015), and Go/No Go task (Millner, 2015; Pan et al., 2011). Future research investigating inhibitory control measured by multiple behavioral tasks is needed to clarify mixed findings.

Taken together, results of prior research suggest that deficits in emotion regulation are associated with youth STBs, while findings are mixed regarding the relationship between youth suicidality and emotion reactivity, lower resting RSA, RSA reactivity to challenge, and inhibitory control. There are several limitations of the current literature. First, though previous studies have demonstrated the associations between youth suicidality and self-regulatory variables, few studies have conceptualized suicide as reflective of dysfunction in multiple aspects of self-regulation.

Second, since most findings come from cross-sectional studies, there is a relative lack of longitudinal data on the relationship between youth suicidality and self-regulation. Third, from the few longitudinal studies in the current literature (e.g., Brausch & Woods, 2019; Giletta et al., 2017), little is known about the prediction of youth suicidality from self-regulation in early and middle childhood.

Life Stress and Youth Suicidality

Beyond self-regulation, life stress is another broad construct that is commonly associated with psychopathology. Specifically, exposure to early life stress, commonly conceptualized as adverse childhood experiences (ACEs), is related to an increased risk of psychopathology in adolescence and adulthood. Early life stress includes a range of adverse experiences occurring in childhood, including exposure to violence, abuse, neglect, or family dysfunction (e.g., parental substance abuse, mental health problems, and/or incarceration). Such early life stressors have been linked to later psychopathology symptoms, including depression, bipolar disorder, posttraumatic stress disorder, substance use disorder, schizophrenia, antisocial personality disorder, and borderline personality disorder (Brietzke et al., 2012; DeLisi et al., 2019; Herzog & Schmahl, 2018; Lee et al., 2018; Leza et al., 2021; Merrick et al., 2017; Selous et al., 2020). Concerning suicide research, ACEs have been shown to be strongly related to greater odds of SI in adolescence (Ziker & Snopkowski, 2020) and young adulthood (Polanco-Roman et al., 2021) and risk for SA across the lifespan (Dube et al., 2001; Merrick et al., 2017; Stinson et al., 2021).

Moreover, research has shown that there is a strong relationship between early life stress (e.g., ACEs) and the development of self-regulation. Children and adolescents who have experienced early life stress tend to have difficulties regulating their behaviors and emotions, including deficits in inhibitory control (Evans & Kim, 2013; Lackner et al., 2018), emotion

regulation (Rollins et al., 2021; Tottenham et al., 2010), and increased use of maladaptive coping strategies (Hagan et al., 2017). Given the relationship between early life stress and deficits in self-regulation, impairment in self-regulation might be one possible pathway through which early life stress increases the risk of youth suicidality. Alternatively, early life stress and self-regulatory deficits may be correlated risk factors for youth suicidality. More research is needed to understand the unique effects of early life stress and difficulties in multiple aspects of self-regulation to identify which factors are independently predictive of youth suicidality.

There are limitations in the current literature on the relationship between life stress and youth suicidality. First, though evidence has clearly indicated that a relationship exists between life stress and suicidality, the roles of different timing and types of stress in the development of suicidality remain unexplored (Herzog & Schmahl, 2018; Juruena et al., 2021). This problem is relevant for research regarding the associations between life stress and the broader construct of psychopathology as well. Second, there is still much to know about the underlying mechanisms of the relationship between life stress and suicidality, as well as potential unique influences of life stress and self-regulation. Third, consistent with the largely cross-sectional literature on self-regulation, there is a relative lack of longitudinal data on the relationship between early life stress and youth suicidality. Longitudinal studies may both mitigate concerns about recall bias when participants retrospectively report their early experiences and afford the opportunity to investigate prediction from specific types and timing of life stressors on adolescent suicidality outcomes.

The Present Study

The present study used a longitudinal design to examine how multiple self-regulatory variables (i.e., emotion regulation, emotion reactivity, parasympathetic nervous system regulation, and inhibitory control) and life stress across different developmental stages confer risk for SI in

adolescents. There are several strengths of the present study. First, this study took a developmental psychopathology perspective, aiming to identify developmental antecedents of SI in adolescence, using data from early childhood, middle childhood, and adolescent research assessments. Second, this study aimed to examine multiple risk factors for SI in adolescents simultaneously, with the goal of increasing the predictive ability of youth suicidality using self-regulatory and life stress variables across early childhood to adolescence. Third, by studying life stress variables across different developmental periods, we hoped to examine the effect of the timing of life stress on adolescent SI and to compare prediction from different periods of life stress. Given the current state of knowledge, we hypothesized that deficits in self-regulation during middle childhood (i.e., worse emotion regulation, lower resting RSA, altered RSA reactivity to challenge, and worse inhibitory control) and early adolescence (i.e., greater emotion reactivity) as well as greater life stress during early and middle childhood would predict higher lifetime SI reported in adolescence.

Method

Participants and Procedures

Participants were adolescents (N = 177; 46.3% female) enrolled in a longitudinal study on the efficacy of an attachment-based parenting intervention in infancy. Participants in the risk group (N = 119) were originally recruited by referral from Child Protective Services (CPS) due to high risk for maltreatment when they were infants and were randomized to receive one of two parenting interventions. Participants in the comparison group (N = 58) were recruited from local community centers at the time of the middle childhood follow-up (age eight years). Comparison participants had no history of CPS involvement and did not participate in any parenting intervention.

Self-regulatory data were collected during laboratory assessments attended by parent-child

dyads during middle childhood (ages eight to 10) and early adolescence (ages 13 to 15). Life stress data were collected during middle childhood and supplemented with data from early childhood when available. Questionnaire and interview data were acquired by telephone or Zoom if in-person data collection was not feasible (e.g., because the family had moved out of state and/or due to a temporary suspension of in-person research during the COVID-19 pandemic). All procedures were approved by the Institutional Review Board of the University of Delaware and families received an honorarium for their participation.

Participant demographics were reported by their parents during early adolescent laboratory visits and are presented in Table 1. The sample was racially diverse, with most participants being African American (58.8%) or multiracial (16.9%).

Measures

Self-Regulation

Emotion Regulation Checklist (ERC; Shields & Cicchetti, 1997). The ERC is a 24-item parent-report measure that assesses children's ability to cope with their emotions. Parents rated their children's emotion regulation ability on a four-point Likert scale (1 = never, 2 = sometimes, 3 = often, 4 = almost always). This measure includes two subscales: Emotion Regulation (ER) and Lability/Negativity (L/N). The ER subscale assesses situational appropriateness of emotion expressions, empathy, and emotional self-awareness, with higher scores indicating better adaptive emotion regulation. This subscale contains eight items, such as, "Responds positively to neutral or friendly approaches by peers." The L/N subscale assesses emotional lability, lack of flexibility, and dysregulated negative emotions, with higher scores indicating greater emotion dysregulation. This subscale contains 15 items, such as, "Is prone to

angry outbursts/tantrums easily." The scores of both subscales are obtained by averaging all relevant scores, after reverse-coding negatively-worded items. Parents completed the ERC during laboratory visits when their children were ages eight, nine, and 10. The overall scores for middle childhood ERC ER and ERC L/N were obtained by averaging the relevant scores across three timepoints.

Emotion Reactivity Scale (ERS; Nock et al., 2008). At age 13, participants completed the ERS, a 21-item self-report measure that includes emotion sensitivity, intensity, and persistence subscales. Adolescents rated their experience of emotion reactivity on a five-point Likert scale (0 = not at all like me to 4 = completely like me). The sensitivity subscale contains 10 items, such as, "Even the littlest things make me emotional." The intensity subscale contains seven items, such as, "People tell me that my emotions are often too intense for the situation." The persistence subscale contains four items, such as, "When I feel emotional, it's hard for me to imagine feeling any other way." The scores for all the subscales are calculated by adding the scores for all the relevant items. A total emotion reactivity score is obtained by adding the scores of all the subscales, with higher scores indicating greater emotion reactivity.

Parasympathetic Nervous System Data Collection and Cleaning. During in-person laboratory assessments when children were age nine, children participated in a baseline task and an impossible task that was used to elicit feelings of frustration in the child. Children's heart rate (electrocardiogram) data were collected using disposable electrodes and wireless mobile devices throughout the task period. During the baseline task, the child viewed a nature image accompanied by nature sounds for two minutes. This task was designed to obtain children's heart rate data at rest and provide a baseline measure of their parasympathetic nervous system regulation. During the frustration task, the child was asked to complete perfect mazes. The

researcher first presented the child with a series of paper mazes one at a time and instructed them to complete each one perfectly. After the child finished each maze, the researcher provided neutral critiques to it, such as, “That one is too crooked. Try another one.” This process lasted for three and a half minutes. Then the researcher left the room, leaving the child in the room with a stack of mazes to complete while alone. After one minute, another researcher entered the room and conducted a one-minute interview with the child about their emotional responses to the task. The child was required to continue working towards completing the perfect maze throughout the task period and remained under task demands until the completion of the interview. The first researcher then returned to the room, asked the child to complete a final maze, and then praised the child. About one-third of the children completed perfect circles (Goldsmith et al., 1999) instead of perfect mazes. The change was made due to children’s recall of the perfect mazes task administered at a previous lab visit. The frustration task was designed to induce children’s negative emotions and used to collect data on children’s parasympathetic nervous system regulation in challenging situations.

Children’s heart rate data were recorded and cleaned using software and equipment provided by the James Long Company. The software algorithm detected R-waves (i.e., the electrical activity of the heart captured by an electrocardiogram), marked questionable interbeat intervals for visual inspection and correction, and computed RSA for each participant. All misidentified R-waves were manually corrected. Consistent with prior work measuring heart rate in children (Woody et al., 2016), segments of children’s heart rate data were excluded from analyses if at least 10% of the R-waves were manually corrected. Average RSA values for baseline and distress tasks were calculated using cleaned heart rate data.

Measures of Inhibitory Control. We used both the Flanker task and the Stop Signal

Reaction Time (SSRT) task as our measures of children's inhibitory control.

Flanker Task. At ages eight and 10, children completed a computerized Flanker task (Eriksen & Eriksen, 1974), which was programmed and administered using Presentation software (Neurobehavioral Systems, Inc.). Participants sat in front of a computer monitor on which trials of five arrows were serially presented. Half of the trials were congruent trials, in which all arrows pointed the same direction (e.g., > > > > >). The other half of the trials were incongruent trials, in which the middle arrow pointed the opposite direction of the other arrows (e.g., > > < > >). Children were asked to place their fingers on a two-button box and to respond by pressing the button corresponding to the direction of the middle arrow. The response deadline was 800 ms and an inter-trial interval of 900-1100 ms was used. The researchers instructed the children to emphasize accuracy over speed but encouraged them to respond more quickly if they repeatedly responded after the response deadline.

After researchers oriented children to the task using printed versions of arrow trials, children completed a practice block with corrective feedback. After a second practice block without feedback, children progressed to experimental trials or repeated additional practice blocks as needed until the researchers were confident that the children understood the task. Most children in our study proceeded to the experimental blocks without requiring additional practice. Children completed six sets of 50-trial blocks with short breaks between each set. The task took an average of 30 minutes to complete. The percentage of accurate responses and reaction time (RT) in milliseconds were recorded. Children who performed at chance levels (i.e., achieved less than 60% accuracy) were excluded from analyses. The Flanker RT interference score was calculated by subtracting the mean RT for congruent trials from the mean RT for incongruent trials. The Flanker accuracy interference score was calculated by subtracting the mean accuracy

for congruent trials from the mean accuracy for incongruent trials. Because accuracy and reaction time scores were positively correlated ($r_s = –.29$, $p < .001$), we calculated a composite Flanker score by averaging the standardized scores for Flanker RT and Flanker accuracy interference scores. The overall scores for all Flanker variables (i.e., Flanker RT interference score, Flanker accuracy interference score, and Flanker composite score) were averaged across scores at two timepoints, with lower scores indicating better performance.

SSRT Task. At ages eight and 10, children also completed the SSRT task (Logan & Cowan, 1984), which was again delivered using Presentation software (Neurobehavioral Systems, Inc.). They sat in front of a computer screen and a two-button box. Trials began with a blank screen. About two-thirds of the trials were "go trials," in which a green arrow (i.e., the go signal) pointing either right or left appeared on the screen. Children were asked to respond by pressing the button corresponding to the direction of the arrow. About one-third of the trials were "stop trials," in which a red circle superimposed on the green arrow (i.e., the stop signal) appeared after the go signal. Children were asked to stop responding upon seeing the stop signal. Children were encouraged to respond as quickly and accurately as possible when they saw the go signal and not to wait for the stop signal to appear. The stop signal delay (SSD) between the go and stop signal was initially 200 ms and increased or decreased by 50 ms after each successful or unsuccessful stop trial, respectively. The inter-trial interval was set at 2000 ms, and the trial order was completely randomized.

After the researchers explained the SSRT task to the children, the children completed two 50-trial practice blocks. The researchers offered feedback on children's performance during the first practice block and refrained from any feedback during the second practice block while observing children's behaviors to confirm that they were not deliberately waiting for stop signals

to appear. Additional practice was administered until the children gained understanding of the rules. Children completed four 60-trial experimental blocks after researchers left the room. The SSRT task lasted for an average of 30 minutes.

The integration method described by Verbruggen and colleagues (2019) was used to calculate an overall score for SSRT performance. The percentage of successful stop trials (SST) and the average SSD were calculated for each child across all four experimental blocks. The SSRT score for each child was calculated by subtracting the average SSD from the RT separating unsuccessful and successful stop trials (see Verbruggen et al. [2019] for detailed instructions). Children's data were excluded from analyses if their performance did not meet any of the following criteria: (1) at least 30% SST, (2) less than 25% go omissions (i.e., go trials without a response before the response deadline), (3) less than 25% incorrect go trials (i.e., go trials with incorrect button presses), and (4) length of the average unsuccessful stop trial shorter than that of the average successful stop trial. The overall SSRT score for each child was averaged across two timepoints, with lower scores indicating better inhibitory control.

Life Stress

During laboratory visits when children were ages eight, nine, and 10, their parents were interviewed with the Life Event Calendar that asked about children's stressful life events since birth (at age eight) or the last visit (at ages nine and 10). In line with the cumulative risk model in the current literature (Evans et al., 2013; Jensen et al., 2015), we developed a cumulative life stress score for the purpose of this study. Our life stress score included a primary caregiver risk index and a family instability risk index. The primary caregiver risk index contained four items: primary caregiver's mental health problem, criminal justice involvement, substance use, and single parenthood. The family instability risk index contained eight items: residential instability,

relationship instability, homelessness, separations from primary caregiver or changes in caregiver, CPS involvement, removal of other children from home, violence or abuse in the family, and deaths of close family members. The criteria indicating risk for each item are shown in Appendix A.

All the items were scored as zero (absence of risk) or one (presence of risk) so that they contributed the same weight to the overall cumulative risk. We scored cumulative risk index items for children's early childhood (birth to 48 months) and middle childhood (five to 10 years) and tallied all items for each developmental period to obtain two life stress scores: an early childhood life stress score and a middle childhood life stress score, with higher scores indicating more life stressors (i.e., greater levels of cumulative risk).

Suicidality Outcomes

Columbia-Suicide Severity Rating Scale (C-SSRS; Posner et al., 2011). At ages 13, 14, and 15, participants completed the C-SSRS, a validated, semi-structured interview designed to assess the severity of suicidal ideation and behavior. Adolescent SI intensity was scored using the maximum category endorsed for lifetime SI at ages 13-15 (0 = no SI, 1 = wish to be dead, 2 = non-specific active SI, 3 = active SI with any methods [no plan] without intent to act, 4 = active SI with some intent to act, without specific plan, 5 = active SI with specific plan and intent). A dichotomous SI score (i.e., rates of SI) was also constructed based on whether any lifetime SI was endorsed at ages 13-15. A dichotomous SA score was constructed based on endorsement of any lifetime SA.

Demographic Covariates

Parent-reported child sex and socioeconomic status (SES) from the early adolescent assessment were included as potentially relevant demographic covariates. We calculated a

composite SES score by averaging the standardized scores for family income and parental education, as reported by parents at the early adolescent assessment.

Data Analytic Plan

Descriptive analyses were conducted for all focal variables, including socio-demographics, self-regulation, life stress, and suicidality. A one-way ANOVA was conducted to examine whether SI intensity varied between different racial/ethnic groups. Independent samples t-tests were conducted to compare means of SI intensity between groups for binary demographic predictors including sex, risk group, and a dichotomous variable indicating whether the family received temporary assistance for needy families (TANF).

To test the primary study hypotheses, we conducted Spearman's correlation analyses among SI intensity and continuous predictor variables, including ERC (both Emotion Regulation and Lability/Negativity subscales), ERS, resting RSA, RSA reactivity to challenge, inhibitory control (as measured using a Flanker task and the SSRT), early childhood life stress, middle childhood life stress, and socioeconomic status. Significant findings using SI intensity were followed up with test(s) of dichotomous variable(s) to ensure that the results were not driven by outliers in maximum SI intensity.

Finally, we planned to conduct a multiple linear regression including all significant predictors of SI intensity at the bivariate level and controlling for sex as the most common demographic characteristic distinguishing suicidal ideators from non-ideators in adolescence (Cha et al., 2018). Exploratory follow-up analyses were planned to assess mediation of the effects of life stress through self-regulatory variables, if appropriate, and to test contributions of specific types of life stress (caregiver-level versus family instability) if overall stress indices were significant.

Results

Descriptive results for variables of self-regulation, life stress, and suicidality are displayed in Table 2. Concerning suicidality outcomes, 23.2% of participants (N = 41) reported experiencing any lifetime SI (M = 0.53, SD = 1.14), and 5.6% reported at least one lifetime SA (N = 10). There was no significant difference in SI intensity between different racial/ethnic groups as demonstrated by a non-significant one-way ANOVA, $F(3, 171) = .61$, $p = .613$. Independent samples t-tests revealed no significant differences in SI intensity based on participant sex ($p = .25$), risk group ($p = .37$), or receipt of TANF ($p = .29$).

Spearman's correlation analyses were conducted to examine the associations between SI intensity and predictor variables (i.e., socio-demographics, self-regulation, and life stress). Results of correlation analyses are shown in Table 3. Lifetime SI intensity measured in adolescence was positively correlated with age 13 emotion reactivity, $r_s = .34$, $p < .001$, but was not significantly related to other candidate predictors, including self-regulation during middle childhood or life stress.

Life stress during both early and middle childhood predicted worse parent-reported emotion regulation (early $r_s = –.26$, $p < .001$; middle $r_s = –.24$, $p = .002$) and more parent-reported lability/negativity (both r_ss = .34, $ps < .001$) on the ERC during middle childhood. Similarly, early and middle childhood life stress each predicted worse inhibitory control as measured during middle childhood using the SSRT (early $r_s = .22$, $p = .009$; middle $r_s = .17$, $p = .042$).

To ensure that significant associations between emotion reactivity and suicidality were not driven by outliers in maximum SI intensity, we conducted an independent samples t-test to

examine differences in age 13 ERS between groups based on presence or absence of lifetime SI in adolescence. Consistent with correlational findings, participants who endorsed any lifetime SI reported higher emotion reactivity (M = 42.95, SD = 20.24) than participants who denied any lifetime SI (M = 27.48, SD = 17.40), $t(152) = –4.60$, $p < .001$. An exploratory follow-up analysis showed that SI intensity was positively correlated with all three subscales of emotion reactivity, including emotion sensitivity, $r_s = .28$, $p < .001$, arousal/intensity, $r_s = .35$, $p < .001$, and persistence, $r_s = .32$, $p < .001$.

A multiple linear regression was used to test whether ERS uniquely predicted lifetime SI intensity controlling for participant sex (see Table 4). The results of the regression indicated the two predictors explained 15.3% of the variance, $F(2, 151) = 13.62$, $p < .001$, $R^2 = .153$. ERS significantly predicted SI intensity, $\beta = .38$, $t(151) = 4.99$, $p < .001$. Sex did not significantly predict SI intensity, $\beta = .03$, $t(151) = 0.40$, $p = .690$.

Because life stress variables were not significantly associated with emotion reactivity or SI intensity, exploratory analyses investigating possible mediation and/or specific contributions of different types of life stress were not conducted.

Discussion

This study took a developmental psychopathology approach to investigate the prediction of lifetime SI among adolescents from life stress and multiple aspects of self-regulation across early childhood to early adolescence, taking relevant socio-demographics into account. Specifically, we examined life stress during early and middle childhood, middle childhood self-regulation (i.e., emotion regulation, inhibitory control, parasympathetic nervous system regulation), and early adolescent self-regulation (i.e., emotion reactivity) as predictors of

adolescent SI.

Correlation analyses revealed a significant relationship between adolescent SI (ages 13-15) and concurrent emotion reactivity (age 13). This finding was further corroborated by a multiple linear regression controlling for participant sex, which indicated that emotion reactivity uniquely predicted lifetime SI in adolescence. Further, follow-up analyses showed that all three subscales of ERS were positively correlated with adolescent SI. Overall, our results suggested that adolescents who experience emotions to a broader range of stimuli, with greater intensity, and for a more extended period of time before returning to the usual level of arousal are more likely to have thoughts of suicide. Given the mixed findings of the relationship between emotion reactivity and youth suicidality in the current literature, our study helped clarify the role of emotion reactivity in conferring risk for suicidal ideation (Liu et al., 2020; Wu et al., 2021).

Inconsistent with previous findings suggesting that youth suicidality is associated with lower adaptive emotion regulation (Barr et al., 2017; Ong & Thompson, 2019) and/or more emotion dysregulation (Hatkevich et al., 2019, Brausch et al., 2022; Brausch & Woods, etc.), our study found no significant relationship between adolescent SI and adaptive emotion regulation (as measured by the ER subscale of ERC) or emotion dysregulation (as measured by the L/N subscale of ERC). There are two possible explanations for our finding. First, research examining emotion regulation and dysregulation relies primarily on self-reported measures (Wolff et al., 2019); this is true of the studies cited above identifying significant links with youth SI. In contrast, our study used a parent-report measure to evaluate children's emotion regulation. Parents may not always accurately report their children's emotion regulation abilities due to limited awareness or misinterpretation of their children's emotion expressions. Second, we measured children's parent-reported emotion regulation when they were aged eight to 10 and

their SI when they were aged 13-15. However, in studies documenting a significant relationship between emotion regulation and youth suicidality, both constructs were measured at the same time point. Because children's emotion regulation skills evolve over time, it is plausible that deficits in emotion regulation increase risk for youth suicidality concurrently but not prospectively.

Previous research has established a relationship between parasympathetic nervous system regulation and youth suicidality. However, our study found no relationship between parasympathetic nervous system regulation (either resting RSA or RSA reactivity to challenge) and SI intensity in adolescence. Regarding resting RSA, prior research has provided evidence for lower resting RSA among parasuicidal adolescents than non-parasuicidal controls (Crowell et al., 2005). This inconsistency between our findings and prior research may be due to several factors. First, Crowell et al. (2005) assessed adolescent parasuicidal behaviors, which included self-injurious behaviors with unknown suicide intent, whereas our study examined adolescent SI. This difference in the types of suicidality outcomes may be related to the inconsistency of results between the two studies. Second, we measured participants' parasympathetic nervous system regulation during middle childhood (age nine) as a distal predictor of adolescent SI. Considering that Crowell et al. (2005) assessed both predictor and outcome variables in adolescents, this inconsistency suggests that resting RSA might predict youth suicidality only in the short term.

Regarding RSA reactivity to challenge, prior findings are mixed, with studies showing either stable RSA or greater RSA decreases in response to challenge among suicidal versus non-suicidal youth. The discrepancy between our null findings and current literature may reflect differences in sample characteristics. Our study focused on a predominantly maltreatment-involved sample of adolescents. By contrast, other studies recruited participants based on

different criteria, such as a history of depression and STBs (Yang et al., 2019) and exhibiting parasuicidal behaviors (Crowell et al., 2005). Moreover, differences in study results may be attributed to the developmental period during which RSA data was collected. While our study collected RSA data from nine-year-olds, other studies have focused on RSA in adolescents. Nevertheless, it is important to note that limited research exists concerning the relationship between parasympathetic nervous system regulation and youth suicidality. Therefore, more work needs to be done to fully understand the extent of this relationship.

The non-significant finding regarding the relationship between inhibitory control and youth suicidality was unsurprising considering the inconclusive results in the current literature. One possible explanation is that the behavioral tasks used to measure inhibitory control in present study did not include negatively valenced affective materials that are typically associated with SI and suicidal decision-making (Cha et al., 2018; Millner, 2015). Moreover, in studies that find a significant relationship between inhibitory control and suicidality, researchers have focused more on suicide attempters than ideators (Wojnar et al., 2009; Swann et al., 2005; Dougherty et al., 2004). It may be that impulsive action (as measured by behavioral inhibitory control tasks) increases the likelihood of attempting suicide rather than having thoughts of suicide.

Early life stress (whether occurring in early or middle childhood) also did not significantly predict adolescent SI in our study. These results were surprising given the prior evidence indicating a graded association between cumulative life stressors and the severity of SI or SA in adolescence and adulthood (Enns et al., 2006; Johnson et al., 2002). Several factors may contribute to our findings. First, a majority of our sample (76.8%) did not report any lifetime SI, and among those who did, most had SI of low intensity. This low variability of SI data may have

contributed to the non-significant finding regarding the relationship between early life stress and adolescent SI. Second, data on early childhood life stress were collected at middle childhood laboratory assessments. Parents may be subject to recall bias and report fewer early childhood life stressors than there were, resulting in decreased variability of life stress data. Third, the significant relationship between early life stress and suicidality identified in prior studies may be partially explained by other factors associated with early life stress, such as higher prevalence of psychological disorders, that may not apply to the current sample (Enns et al., 2006). However, compared to abundant research in adults, few studies have examined early life stressors as predictors of suicidal outcomes in adolescents. More research is needed to clarify the relationship between early life stress and adolescent suicidality.

Although not related to youth SI, early life stress significantly predicted worse self-regulatory functioning in middle childhood including emotion regulation and inhibitory control. Impaired emotion regulation in childhood has been consistently linked with negative outcomes in social functioning and psychopathology during adolescence and adulthood (Robson et al., 2020; Zeman et al., 2006). Moreover, worse inhibitory control in childhood has been associated with higher externalizing and internalizing behaviors (Eisenberg et al., 2009; Rhoades et al., 2009), which increase the risk for psychopathology in adolescence and adulthood, including depression, anxiety disorders, substance use disorders, and suicide attempts (Campbell, 2006; Keiley et al., 2000; Taylor & Barch, 2022). Our findings provide preliminary evidence for the possible downstream effects of early life stress on other mental health-related outcomes, or on youth suicidality in samples with higher occurrences of STBs.

By integrating the developmental psychopathology perspective into research on youth suicidality (Oppenheimer et al., 2022), this study has important clinical implications for the

prevention and intervention of youth STBs. First, our study highlights the importance of recognizing elevated emotion reactivity as a marker of risk for adolescent suicidality, in order to initiate prevention and early intervention aimed at minimizing adolescent SI. Therapeutic interventions targeting emotion reactivity in early adolescence may be promising in reducing the severity of youth SI and the overall rates of youth STBs. Second, our study offers evidence for the effects of early life stress on self-regulatory functioning during middle childhood. Given that self-regulation deficits are associated with negative psychological and behavioral outcomes, our study suggests that one potential way to mitigate the effects of early life stress is by enhancing self-regulation in affected children.

This study displayed multiple strengths. First, our sample was racially/ethnically diverse with most identified as members of traditionally marginalized groups (58.8% African American; 16.9% multiracial). This enhances the generalizability of our findings to communities that are historically underrepresented in psychological research. Second, we utilized a multi-method assessment of self-regulation, incorporating parent-report measures, objective physiological and behavioral assessments, and self-report questionnaires. Third, this study implemented a longitudinal design to examine the prediction of self-regulation and early life stress from early childhood, middle childhood, and early adolescence to adolescent suicidality outcomes. This design enabled us to draw inferences about the relationship of predictor and outcome variables over a fairly long period of time.

There are several limitations of the current study. First, the sample was not representative of the general population of suicidal adolescents because most of our participants were initially recruited for indication of maltreatment-related risk in infancy. Additionally, our sample exhibited low levels of suicidality, which made it difficult to determine whether non-significant

results reflected an absence of associations between predictor and outcome variables or were due to insufficient variance in suicidality outcomes. Second, we developed the cumulative life stress score and the rubric for scoring each item for the purpose of this study. Although our approach was based on prior research (Sameroff et al., 1998; Obradović et al., 2012), this measure has not been previously validated against other assessments of early life stress. Third, since each aspect of self-regulation was assessed in a certain age range (e.g., parasympathetic regulation only at age nine and emotion reactivity only at age 13), we were unable to distinguish whether null associations with youth SI were driven by relevance of the construct, developmental timing of the assessment, or both.

The limitations of the current study highlight areas for future research. First, a sample with more variability of suicidality data may help clarify and better probe the associations between self-regulation, life stress, and youth suicidality. Second, it is crucial to validate our cumulative life stress score in future studies to enable more robust statistical analyses of the associations between early life stress and other psychological constructs. Third, future research should examine multiple aspects of self-regulation across the developmental periods of interest to examine more comprehensive associations between self-regulation and youth suicidality from a developmental perspective. Moreover, future studies are needed to clarify the relationship between early life stress and youth psychopathology, including STBs, and to identify potential mediators of any statistically significant associations.

Overall, findings indicate that heightened emotion reactivity in early adolescence predicts greater SI intensity among adolescents. Results also suggest that early life stress is associated with deficits in emotion regulation and inhibitory control in middle childhood, which has been associated with increased risk for psychopathology in adolescence and adulthood. Therefore,

targeting emotion reactivity may benefit the development of prevention and intervention strategies for reducing youth STBs.

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Tables

Table 1

Socio-demographic characteristics of participants

Socio-demographic characteristic	*n*	%
Sex		
Female	82	46.3
Male	95	53.7
Race/Ethnicity		
African American	104	58.8
Multiracial	30	16.9
Non-Hispanic White	17	9.6
Other (including Hispanic, Asian American, and Other)	24	13.6
Parental Education		
Less than High School	33	18.6
High School Degree or GED	74	41.8
Some College	45	25.4
Baccalaureate Degree	10	5.6
Post Baccalaureate Degree	7	4.0
Receiving TANF (Endorsing “Yes”)	42	23.7
Family Income		
< $10,000	10	5.6
$10,000 – $19,999	16	9.0
$20,000 – $29,999	23	13.0
$30,000 – $39,999	15	8.5
$40,000 – $59,999	23	13.0
$60,000 – $99,999	18	10.2
> $100,000	14	7.9

Note. $N = 177$. Percentages sum to less than 100% due to missing data. GED = general educational development test. TANF = temporary assistance for needy families.

Table 2

Descriptive statistics for self-regulatory, life stress, and suicidality variables

Predictor Variable	***M***	***SD***
ERS	31.40	19.31
ERC ER	3.32	0.37
ERC L/N	1.74	0.38
Resting RSA	0.13	0.06
RSA Reactivity	−0.02	0.06
Flanker Reaction Time	38.23	31.44
Flanker Accuracy	−0.12	0.12
Stop Signal Reaction Time	303.34	98.04
Early Childhood Life Stress	4.47	2.44
Middle Childhood Life Stress	3.87	2.22
Suicidality	***n***	**%**
SI Intensity (*n*, %)		
No SI	136	76.8
Wish to be dead	13	7.3
Non-specific active suicidal thoughts	17	9.6
Active SI with any methods (not plan) without intent to act	2	1.1
Active SI with some intent to act, without specific plan	5	2.8
Active SI with specific plan and intent	4	2.3
Lifetime Suicide Attempt (*n*, %)		
No	166	93.8
Yes	10	5.6

Note. N = 177. Percentages sum to less than 100% due to missing data. ERS = Emotion Reactivity Scale. ERC = Emotion Regulation Checklist. ER = emotion regulation (subscale). L/N = lability/negativity (subscale). RSA = respiratory sinus arrhythmia. SI = suicidal ideation.

Table 3

Bivariate correlations among main study variables

Variable	1	2	3	4	5	6	7	8	9	10
1. SI Intensity	—									
2. ERS	.34***	—								
3. ERC ER	−.05	−.28***	—							
4. ERC L/N	.02	.27***	−.57***	—						
5. Resting RSA	.03	.00	−.00	−.09	—					
6. RSA Reactivity	−.08	−.05	.04	−.00	−.59***	—				
7. Flanker	−.07	−.00	.19*	−.12	.10	−.05	—			
8. Stop Signal Reaction Time	.09	.09	−.07	.06	.03	.02	−.08	—		
9. Early Childhood Life Stress	.04	.09	−.26***	.34***	.07	−.02	−.11	.22**	—	
10. Middle Childhood Life Stress	.07	.04	−.24**	.34***	.01	−.07	−.04	.17*	.56***	—
11. SES	−.03	−.06	.38***	−.31***	.04	−.11	.16	−.35***	−.36***	−.39***

Note. SI = suicidal ideation. ERS = Emotion Reactivity Scale. ERC = Emotion Regulation Checklist. ER = emotion regulation (subscale). L/N = lability/negativity (subscale). RSA= respiratory sinus arrhythmia. SES = adolescent socioeconomic status.

*$p < .05$. **$p < .01$. ***$p < .001$.

Table 4

Multiple linear regression predicting SI intensity from ERS and sex

Variable	*B*	*SE B*	β	*t*	*p*
ERS	0.02	0.01	.38	4.99	< .001
Sex	0.07	0.19	.03	0.40	.690

Note. SI = suicidal ideation. ERS = Emotion Reactivity Scale.

Appendix A

Life Stress Scoring Rubric

Table A1

Primary caregiver risk index scoring rubric

Item	Scoring rubric	Notes
PCG mental health problem	1 = PCG reported mental health issue(s) 0 = PCG did not report any mental health issue	Seeking care (e.g., medication, therapy, talking to the doctor, going to the emergency room) was counted as a risk whether or not a diagnosis was specified or treatment plan was followed.
PCG criminal justice involvement	1 = PCG was involved with criminal justice system 0 = PCG was not involved with criminal justice system	Legal problems relating to civil suits and/or family court (visitation, child support, etc.) did not qualify for criminal justice system involvement.
PCG substance use	1 = PCG reported substance use (i.e., marijuana, alcohol, or illicit drug use) 0 = PCG did not report substance use	Marijuana use was counted as a risk if PCG had daily/problematic use or sought treatment. Alcohol use was counted as a risk if PCG had daily/almost daily/problematic use, engaged in binge drinking (≥ four drinks per day for women and ≥ five drinks per day for men), or sought treatment. Social drinking was not counted as a risk unless there was evidence indicating excessive drinking (e.g., binge drinking).
PCG single parenthood	1 = PCG did not identify any co-parent 0 = PCG identified co-parent(s)	

Note. PCG = primary caregiver.

Table A2

Family instability risk index scoring rubric

Item	Scoring rubric	Notes
Residential instability	1 = Family had one or more moves 0 = Family had no moves	
Relationship instability	1 = Relationship(s) ended or begun 0 = No relationships ended or begun	Temporary break-up and reunion with the same partner was counted as a risk.
Homelessness	1 = Homeless 0 = Not homeless	Living in the shelter/car or doubling up with family due to loss of housing qualified for homelessness, while receiving housing assistance did not.
Separations from PCG or changes in caregiver	1 = Child had separation(s) from PCG or change(s) in caregiver 0 = Child had no separations from PCG and no changes in caregiver	
CPS involvement	1 = Family had any involvement with CPS 0 = Family had no involvement with CPS	Allegations that did not require follow-up investigations qualified for CPS involvement. Purchase of care or childcare vouchers did not qualify for CPS involvement unless maltreatment allegations were mentioned.
Removal of other children from home	1 = Other children removed or placed with other caregivers 0 = No other children removed or placed with other caregivers	
Violence or abuse in the family	1 = Violence/abuse experienced by any family member or the child 0 = No violence/abuse experienced by any family member or the child	
Deaths of close family members	1 = Death(s) of close family members 0 = No deaths of close family members	Only deaths of close family members (e.g., parents, caregivers, siblings) were counted as a risk. Grandparents were only counted if identified as caregivers, lived with the child, or identified as having a close relationship with the child.

Note. PCG = primary caregiver. CPS = child protective services.