TOWARDS A BIOPSYCHOSOCIAL MODEL OF ADOLESCENT SELF-INJURIOUS THOUGHTS AND BEHAVIORS

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A dissertation submitted to the faculty of the University of North Carolina at Chapel Hill in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the Department of Psychology (Clinical).

Chapel Hill
2011

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ABSTRACT

JOHN D. GUERRY: Towards a Biopsychosocial Model of Adolescent Self-Injurious Thoughts and Behaviors
(Under the direction of Mitchell Prinstein)

Adolescent self-injurious thoughts and behaviors (SITB) have been increasingly recognized as a major public health problem. Virtually absent from this literature are comprehensive, developmentally informed theoretical models which can account for the etiology and interrelationships among cognitive, social, and biological variables known to be associated with adolescent SITB. The present study preliminarily tested a biopsychosocial model of adolescent SITB which hypothesized that cognitive vulnerability and increased emotion reactivity in response to a laboratory social stress task would be related to greater engagement in SITB. Adolescent participants (n = 62; 73% female) completed measures of negative inferential style, past engagement in SITB, and participated in an in vivo speech task while samples of salivary cortisol were collected at regular intervals throughout the assessment. MANOVA and MANCOVA analyses and latent growth curve models provided inconclusive support for hypotheses and highlighted limitations related to the sample utilized in the present study. Several important directions for future research are discussed.

For Bama

And for June

ACKNOWLEDGEMENTS

Now that I've come to the end, I grapple with an old question that always seems new: how to begin to thank those who have made so much, including this dissertation, possible. First and foremost, I am indebted to Mitch Prinstein for his years of unwavering guidance, support, and encouragement. Mitch's singular combination of intellectual incisiveness, contagious enthusiasm and curiosity, and deep compassion for our social world—both at the (distal) empirical and (proximal) personal levels—truly embodies the best of mentorship. Mitch's influence will continue to inspire me as I begin my career as a clinical psychologist.

I am grateful to my loving wife and most admired colleague, Whitney. Whitney teaches me every day how to find more beauty and sense by stepping back a little, before listening and looking again. To my parents, DuPont and Sally, thanks for always centering me with love and a sense of home. You have instilled in me all of the things for which I am most proud. For support of this project, both directly and indirectly, I owe heartfelt thanks to my family—Sarah Guerry, Anne Guerry, Kennon Guerry, John Brady, Josh Lawler, Tim Dickinson, and, of course, Thelonius, Tristan, Finn, Cyrus, Emeline, and Everett. I'm also grateful for the love, support, inspiration, and humor of my second families, the Brechwalds and my lifelong friends Judah Dickstein, Dave Basner, and Bajir Cannon.

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INTRODUCTION

The Problem of Adolescent SITB

Self-injurious thoughts and behaviors (SITB) is an umbrella term referring to the broad class of cognitions and actions that produce direct and deliberate self-harm (Nock, Wedig, Janis, & Deliberto, 2008b). SITB are generally believed to fall along a continuum of severity (e.g., Claes et al., 2010; Walsh, 2006), from thoughts and behaviors performed by an individual with a perceptible intent to die (i.e., suicide ideation, plan, attempt, and completion) to the more recently recognized category of *non*suicidal self-injurious thoughts, gestures, and behaviors (see Nock et al., 2008b). While there are many important theoretical and empirical distinctions among different forms of SITB (e.g., Muehlenkamp, 2005), current research supports the view that these thoughts and behaviors are closely interrelated. For example, suicide ideation, a plan for how to carry out suicide, and engagement in nonsuicidal self-injury (NSSI) are all associated with an increased risk of suicide attempt (Kessler, Borges, & Walters, 1999; Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006; Reinherz, Tanner, Berger, Beardslee, & Fitzmaurice, 2006).

Across the spectrum of age, SITB is a major, worldwide public health problem, with nearly 1 million people dying annually from its direst expression, completed suicide (WHO, 2010). Recent epidemiological data, however, has raised particular concerns about the dramatic increase in SITB observed during the transition to adolescence. Suicide is currently

the third leading cause of adolescent death in the United States, following only accidents and homicides (CDC, 2010b), and its rate increases over twenty-fold from childhood into adolescence (from 0.46 to 9.76 per 100,000 for individuals aged 5-14 and 15-24, respectively; CDC, 2010a). This developmental period is also marked by corresponding increases in the occurrence of the common precursors to completed suicide, including suicide ideation, plans, and attempts, particularly among adolescent females. For example, while suicidal ideation remains relatively rare among children, nearly 20% of high school females and over 10% of males report that they have seriously contemplated suicide at some point in the past year (CDC, 2010b). It is also well known that adolescent females attempt suicide at approximately twice the rate of adolescent males (8.1% vs. 4.6%; CDC, 2010b). Alarmingly, extant longitudinal data suggests that far from representing normative adolescent angst, the experience of suicidal ideation during adolescence often portends severe distress and compromised functioning during later adulthood (Reinherz et al., 2006).

Prevalence data has likewise indicated that the rates of NSSI double from preadolescence (7%; Hilt, Nock, Lloyd-Richardson, & Prinstein, 2008) to adolescence (12-15%; Favazza, DeRosear, & Conterio, 1989; Ross & Heath, 2002). Moreover, adolescence is the period of development most associated with the initiation of chronic self-harming behaviors (e.g., Favazza & Conterio, 1988). As with suicidal thoughts and behaviors, some evidence suggests that adolescent females engage in NSSI more frequently than males (Ross & Heath, 2002; Bhugra, Thompson, Singh, & Fellow-Smith, 2003). These observations make it clear that the adolescent transition represents a developmental period associated with a critical vulnerability for the onset, maintenance, and possible long-term recurrence of

SITB. For these reasons, the study of adolescent SITB is a high public health priority (U.S. Department of Health and Human Services, 2001; WHO, 2010).

Despite this imperative, surprisingly little is known about many fundamental aspects of adolescent SITB. For instance, and in parallel to the adult literature, although decades of excellent research has revealed a constellation of both distal risk factors (e.g., Brent et al., 1993b; Lewinsohn, Rohde, & Seeley, 1994; McKeown et al., 1998) and more proximal warning signs of eventual suicide (see Rudd et al., 2006), the positive and negative predictive powers of these variables remain too low to have more than limited clinical utility. Perhaps as a result, the rates of SITB in the general population have remained virtually unchanged despite exponential increases in empirically informed treatment services, (Kessler, Berglund, Borges, Nock, & Wang, 2005).

Progress to date has been limited significantly by the paucity of longitudinal investigations that prospectively examine the development and recurrence of SITB among adolescent samples over multiple time points. This is a central failing: without establishing SITB's subtle temporal aspects, the causes, consequences, contributors, and correlates of these outcomes cannot be differentiated. What little longitudinal research is available has also been characterized by important methodological limitations, such as the utilization of single-item indices of SITB, the collection of data at only two time points, or the wide spacing of interval observations (e.g., between 1 and 15 years between assessments) (e.g., McKeown et al., 1998; Reinherz et al., 2006; see Prinstein et al., 2008, for a notable exception). This last issue is an especially important drawback of prior research, as basic clinical experience indicates that SITB often fluctuates with some rapidity over time.

Much of SITB research has also lacked theoretical sophistication. Few investigators, presumably daunted by the complex and heterogeneous nature of SITB, have articulated—much less rigorously tested—theoretical models that attempt to account for the etiology, causal development, and interrelationships among variables known to be associated with SITB. Many of the earlier models proposed, as Maris (1981) was quoted by Cornette, Abramson, and Bardone (2000), "tend not to be theories at all, but rather lists of factors believed somehow to be related to suicide" (p. 306). Although identifying candidate risk factors is a crucial early step in explanation and prediction, it is imperative early on to examine which factors and what relationships between them are in a causal pathway leading to SITB. This level of understanding will effectively identify more appropriate points for intervention (Smith, Alloy, & Abramson, 2006). Moreover, while certain prominent contemporary theories represent a considerable improvement over earlier models (see Cornette et al., 2000, for a review), these have considered SITB as unitary across the age spectrum; virtually absent from the theoretical literature has been attention to developmental considerations which might distinguish unique aspects of adolescent manifestations of SITB (e.g., Wagner, 2009).

One particularly salient and consistently documented risk factor for and presumed mediator of SITB that may have particular relevance for adolescents is stress. That SITB often occurs in response to a stressful precipitant (see Oquendo, Malone, & Mann, 1997, for a review) offers a potential explanation for both the exponential increase in SITB during adolescence and the observed gender differences. As compared to childhood, the transition to adolescence is marked by significant increases in stressful life events (Ge, Lorenz, Conger, Elder, & Simons, 1994; Larson & Ham, 1993). Related to the emerging prominence of the

peer group and an expanding social network, these stressors frequently occur within the interpersonal context (Rudolph & Hammen, 1999). Further, it has been found that adolescent females are exposed to both a higher number of interpersonal stressors and report greater distress in response to them, as compared to younger children and adolescent boys (Rudolph, 2002; Rudolph & Hammen, 1999; Hankin, Mermelstein, & Roesch, 2007). Unfortunately, little is known about how specific psychological and physiological stress responses may confer vulnerability—either individually or in their interaction—to adolescent SITB. Such an understanding would be extremely useful to understanding why some adolescents engage in SITB in the context of stress while others do not.

A research agenda in SITB should address the limitations inherent in prior work along four avenues. First, given that adolescence represents a period of particular vulnerability to the initiation and maintenance of SITB, research efforts need to be developmentally sensitive and aimed at understanding and predicting these outcomes among identifiable high risk samples of young people. Considering that SITB is most prevalent among adolescent females and most often co-occurs with psychopathology (Cavanagh, Carson, Sharpe, & Lawrie, 2003; Nock et al., 2006), utilizing a clinical sample of adolescent females is an important initial focus of this line of research. Second, the simultaneous consideration of psychological, biological, and social influences in the context of the adolescent stress response is a particularly promising way forward to generating a testable, developmental theory of SITB (e.g., Wagner, 2009). Based on prior work demonstrating that interpersonal stress represents a domain of particular vulnerability for adolescent females, it will be fruitful to begin by specifically examining stress responses to this class of stressors.

Third, there is a pressing need for such an evolving biopsychosocial model to drive longitudinal investigations in adolescent SITB. This would allow for examination of hypothesized associations among empirically chosen risk factors for adolescent SITB. Moreover, given the temporally fluctuating nature of SITB, such prospective work will benefit from the examination of these harmful outcomes across multiple time points. It will allow testing for the possible and more nuanced role of mediator and moderator influences in causal pathways to adolescent SITB. Finally, given that previous research is often limited by the use of single item (i.e., presence vs. absence) indices of SITB, future work must more comprehensively assess the severity, frequency, and duration of the full range of suicidal and nonsuicidal thoughts and behaviors. A better understanding of the range of shared and divergent causal pathways among the various features and forms of SITB will critically inform future prevention and intervention strategies.

The Hopelessness Theory of Suicidality: A Logical Place to Begin

Recently, cognitive models and risk factors have provided a useful framework to conceptualize the effects of stress on suicidal thoughts and behaviors (see Ellis, 2006; Ellis & Rutherford, 2008). One cogent and comprehensive theory is the hopelessness theory of suicidality (Abramson, Metalsky, & Alloy, 1989; Abramson et al., 2000). This theory, a corollary tenet of the broader hopelessness theory of depression, postulates that some individuals possess a certain cognitive vulnerability to the development of a subtype of depression (namely, hopelessness depression) that is particularly associated with suicidality. Consistent with a diathesis-stress framework, Abramson and colleagues (1989; 2000) argued that certain individuals manifest this cognitive vulnerability (i.e., a diathesis) when

confronted with negative life events (i.e., stress) through a generalized tendency to make negative attributions. This depressogenic attributional style leads individuals to make negative inferences regarding the causes and consequences of the event, as well as negative inferences about the self with respect to the event.

More specifically, Abramson and colleagues (1989; 2000) contend that individuals who demonstrate a consistent pattern of making stable (as opposed to transient) and global (as opposed to specific) causal attributions following negative life events—together with a tendency to infer negative consequences and/or self-characteristics regarding these events—are more likely to develop hopelessness and, in turn, suicidality (see Figure 1). The construct of hopelessness has been defined by these scholars as embodying two core elements: 1) negative expectations about the occurrence of highly valued outcomes (i.e., a negative outcome expectancy); and 2) expectations of the uncontrollably of the occurrence of these negative outcomes (i.e., a helplessness expectancy). In this way, suicidality, on a continuum from suicidal ideation to completed suicide, is believed to be a core symptom of hopelessness depression, mediated by the experience of hopelessness (Abramson et al., 1989).

Another essential component of hopelessness theory, the "specific vulnerability" hypothesis (see Abramson et al., 1989; Beck, 1967), maintains that an individual may possess one or more *specific* cognitive vulnerabilities that typically remain latent until activated or "triggered" by a relevant, domain-concurrent stressor. In other words, in order for core symptoms of hopelessness depression to emerge from the vulnerability-stress interaction (e.g., suicidal thoughts and behaviors), this hypothesis requires that there be a match between the content area(s) of an individual's negative attributional style (e.g., an achievement-related vulnerability vs. an interpersonal vulnerability) and the stressful life

events he or she experiences (e.g., “I failed a test” vs. “I broke up with my boyfriend”, respectively).

Over three decades of research conducted with adults has produced multiple lines of evidence in support of many facets of the hopelessness theory of suicidality. A great many studies have documented a powerful concurrent (e.g., Beck, Kovacs, & Weissman, 1975) and prospective link (e.g., Beck, Steer, Kovacs, & Garrison, 1985) between hopelessness and suicidal thoughts and behaviors among adults (see Abramson et al., 2000, for a review). In contrast, much less work has even begun to comprehensively test whether the more distal negative cognitive styles hypothesized to be relevant in hopelessness theory are prospectively associated with increased risk for suicidal thoughts and behaviors in conjunction with the occurrence of negative life events. Only four such studies have been published to date (Abramson et al., 1998; Joiner & Rudd, 1995; Priester & Clum, 1992; Smith et al., 2006), all of which were conducted with college-aged samples and two of which consisted of separate analyses conducted with the same dataset (Abramson et al., 1998; Smith et al., 2006).

Although each of these studies produced findings that were largely consistent with the hopelessness theory of suicidality, some important distinguishing features warrant a closer review. Priester and Clum (1992) conducted the earliest longitudinal examination of the cognitive vulnerability-stress hypothesis of suicidality using a naturalistic academic stressor. These investigators reported that college students who possessed a generalized tendency to attribute negative events to stable causes at baseline exhibited greater hopelessness and suicidal ideation following a low exam grade than did students with a more adaptive (i.e., unstable) attributional style. Importantly, these results were reportedly found after controlling for pre-exam levels of depression, hopelessness, and suicidal ideation.

Unfortunately, however, the data analyses presented by Priester and Clum (1992) did not allow for an examination of whether hopelessness mediated the association between the cognitive vulnerability-stress interaction and suicidality. Further, their measure of cognitive vulnerability, the Attributional Style Questionnaire (ASQ; Peterson et al., 1982), is an incomplete measure of the negative cognitive style consistent with hopelessness theory. Although the ASQ assessed attributions made to the *causes* of hypothetical negative events, it did not address the two other principal facets of hopelessness depression theory, attributions made as to the consequences of events and attributions made about the self with respect to events. Thus, it could be argued that because the ASQ lacks sufficient adherence to theoretically prescribed face validity Priester and Clum's (1992) study is an inadequate test of cognitive vulnerability as specified by hopelessness theory.

The study conducted by Joiner and Rudd (1995) is marked by a comparable lack of theoretical fidelity. These investigators, this time utilizing the Extended Attributional Style Questionnaire (EASQ; Metalsky, Halberstdt, & Abramson, 1987), also operationalized "cognitive vulnerability" as only a measure of negative *causal* attributional style. Nonetheless, Joiner and Rudd (1995) extended the findings of Priester and Clum (1992) in their prospective examination of the specific vulnerability hypothesis of suicidality. Controlling for baseline levels of depression and suicidality, they found that the combination of a stable, global attributional style specific to the domain of negative interpersonal life events and the self-reported occurrence of a greater number of such events were prospectively related to increases in suicidal ideation over a 10-week follow-up period. Consistent with the specific vulnerability hypothesis, these investigators reported that a negative attributional style for achievement-related stressors (e.g., exam failure) did not

predict suicidality in response to interpersonal stressors. Notably, and contrary to prediction, hopelessness was not found to mediate the relation between the attributional style-stress interaction and increases in suicidal ideation in this sample of young adults (Joiner & Rudd, 1995).

Perhaps the most powerful test to date of the hopelessness theory of suicidality was provided by a two-site collaborative project, the Temple-Wisconsin cognitive vulnerability to depression (CVD) project (see Alloy & Abramson, 1999). Utilizing a behavioral high-risk prospective design within a large sample of college students, Abramson et al. (1998) and Smith et al. (2006) distinguished among initially nondepressed participants who had been identified at the outset of the study as possessing either a high or low degree of cognitive risk (as determined by self-reported measures of negative attributional style and dysfunctional attitudes). Abramson et al. (1998) reported that, as compared to the low cognitive risk participants, the high cognitive risk participants were more likely to experience symptoms of suicidality, as measured by both self-report and structured diagnostic interview, over the 2 ½ year prospective follow-up period. As was the case with the studies conducted by Priester and Clum (1992) and Joiner and Rudd (1995), Abramson and colleagues' (1998) results were reportedly obtained after controlling for prior history of depression and suicidality. However, contrary to these previous studies and consistent with hopelessness theory, responses on a self-reported measure of hopelessness did appear to mediate the observed relationship between cognitive vulnerability and suicidality in this sample of university undergraduates (Abramson et al., 1998).

Also representing a substantial methodological and theoretical improvement over prior work, Abramson et al. (1998) and Smith et al. (2006) were the first to report a study

which comprehensively assessed the construct of cognitive vulnerability as theoretically prescribed by hopelessness theory. These investigators collected baseline data regarding negative cognitive style pertaining to the causes, as well as to the consequences and self characteristics related to hypothetical negative events. Nevertheless, it is important to note that the study reported by Abramson et al. (1998) and Smith et al. (2006) is an incomplete test of the hopelessness theory in that the role of stressful life events was not explicitly evaluated.

In sum, all of the reviewed studies examining the hopelessness theory of suicidality have important limitations. First, no study to date has been able to comprehensively evaluate all the essential components hypothesized to be relevant to hopelessness theory. There is a need for future work to incorporate measures of specific cognitive vulnerabilities which completely assess individuals' inferential style for negative, domain-specific life events, including the causes, as well as inferred consequences and self characteristics. Additionally, when the role of stressful life events has been considered in past work, this variable was either evaluated using a simple checklist of items or an uncontrolled naturalistic stressor, such as a low exam grade. In neither case was it possible to assess participants' subjective, individualized responses to stressors. For example, in the Priester and Clum (1992) study it is possible that a low exam grade was not experienced as stressful to some participants. Therefore, to accurately test the hopelessness theory of suicidality, it is essential to simultaneously examine individuals' specific cognitive vulnerability together with their subjectively experienced response to the occurrence of a stressor that maps on to the corresponding domains of vulnerability. Arguably, the only method available to truly capture an individual's unbiased experience of stress is through the use of physiological

indices. Second, as noted previously, the power of this cognitive vulnerability-stress interaction to predict SITB needs to be prospectively evaluated over multiple time points at frequent intervals. Third, the extant data have all utilized convenience samples of college-aged students. No prior work has tested this theory among clinical samples of individuals known to be at the highest risk for SITB: females at the transition to adolescence.

Fourth, it is intriguing that the hypothesized role of hopelessness as the principal, proximal mediator of the longitudinal relationship between the cognitive vulnerability-stress interaction and SITB has received inconsistent support among samples of young adults (see Weishaar, 1996, for a review). This finding, albeit preliminary, is in contrast to the consistently observed and robust relationship found between hopelessness and suicidality in older adults and suggests a broader developmental pattern. The weakness of associations between hopelessness and suicidality among samples of children and adolescents and of young adults has prompted some to recommend that the explication of this developmental incongruity is a high priority for research (Abramson et al., 2000).

It is possible that the construct of hopelessness bears less significance to the proximal development and maintenance of SITB among populations of younger people. Adding some support to this notion, there is evidence from the literature on child and adolescent depression that depressive states among younger people results more directly from encountering negative life events, rather than from the proximal intermediary role of cognitive states (e.g., Cole & Turner, 1993). Extrapolating from these data, it may be similarly inferred that children and adolescents may be less capable of developing hopelessness (despite the enduring presence of alarming and increasing levels of suicidal thoughts and behaviors).

Indeed, at its essence, the relatively advanced, higher order cognitive state of hopelessness requires a (negative) future orientation. However, both psychological and biological research converge on the well-known conclusion that cognitive processing by adolescents is characterized by deficits in executive functioning, such as future planning, goal-directed activity, and the inhibition of maladaptive responses (e.g., Blakemore & Choudhury, 2006; Keating & Bobbitt, 1978). Indeed, it has been postulated that neurological immaturity in such areas as the prefrontal cortex may account for this lack of future orientation, for increases in impulsivity and risk-taking behavior, and for the undervaluation of aversive outcomes so often characteristic of adolescent thinking and behavior (e.g., Kelley, Schochet, & Landry, 2004). In sum, as Wagner (2009) succinctly paraphrased conclusions made by Steinberg et al. (2006), there is a "gap in early to mid-adolescence during which adolescents are prone to experiencing biologically driven, affect-laden motivations before they have the cognitive wherewithal to cope with them and so are prone to making poor, risky choices" (p. 73).

Distal Cognitive Vulnerability to Proximal Emotional/Physiological Risk

It follows that a developmentally sensitive cognitive vulnerability-stress model of adolescent suicidality (Abramson et al., 1989, 2000) would benefit from incorporation of proximal mechanisms that are additional or even alternative to hopelessness. As implied above, the negative, affectively-laden states experienced by adolescents (e.g., following stressful life events) may serve as immediate precipitants to SITB. Further, although no previous research has specifically examined how the negative inferential styles postulated by hopelessness theory are related to the physiological experience of distress in response to a

stressor, there is a strong theoretical and empirical rationale for doing so. Indeed, central to theories of psychological stress and emotion (Frijda, 1988; Lazarus, 1966, 1991; Lazarus & Folkman, 1984) is the notion that an individual's expectations and cognitive appraisals (and reappraisals) regarding potentially stressful situations shape his or her reactions to such situations. More specifically, according to Lazarus and Folkman's (1984) seminal theory cognitive appraisal processes intervene between the initial perception and the subsequent experience of stressful life events. In turn, these cognitive appraisals are essential for determining emotional, physiological, and behavioral responses to such events.

Empirical research examining Lazarus and Folkman's (1984) cognitive appraisal model of stress with children, adolescents, and adults has demonstrated that stressors perceived as uncontrollable, novel, challenging, or threatening (particularly to the social self) contribute to negative emotional and physiological stress response (Denson, Spanovic, & Miller, 2009; Dickerson & Kemeny, 2004; Gunnar, Talge, & Herra, 2009; Lazarus & Folkman, 1984). Considering this research in the context of hopelessness theory, the tendency to attribute negative life events to stable, global causes—as well as to infer negative consequences and self-characteristics with respect to the event—constitutes many of the same elements relevant to appraisal theory. Thus, it seems reasonable to expect that the cognitive vulnerabilities hypothesized to be relevant in the hopelessness theory of suicidality would—subsequent to the experience of a potentially stressful situation or negative life event—likewise lead to negative affective and physiological states. As argued above, this affectively charged, cognitively-mediated response to stress (i.e., emotion reactivity) may be particularly intense and/or overwhelming for adolescents and, thus, may set the stage for SITB.

Filling the Hole Left by Hopelessness: The Role of Emotion Reactivity

Substantial theoretical and empirical evidence is accumulating to support the association of emotion reactivity, in and of itself, to adolescent SITB. According to Nock, Wedig, Holmberg, and Hooley (2008a), emotion reactivity refers to the highly individualized extent to which emotions may be experienced across three dimensions. Individuals may differ in their emotional response to a wide array of stimuli (i.e., emotion sensitivity), the magnitude or strength of their emotional experience (i.e., emotion intensity), and/or the duration of an episode of emotional arousal before returning to baseline (i.e., emotion persistence). Nock and colleagues (2008a) postulated that the construct of emotion reactivity is of primary importance because it may serve as a proximal explanation for the functions underlying the onset and maintenance of many pathological (and ostensibly paradoxical) behaviors, including most centrally, SITB.

Indeed, descriptive studies have revealed that the primary reason given by self-injuring individuals for the engagement in both NSSI (e.g., Brown, Comtois, & Linehan, 2002; Chapman, Gratz, & Brown, 2006; Klonsky, 2007; Nock & Prinstein, 2004, 2005) and suicidal behaviors (Boergers, Spirito, & Donaldson, 1998; Hawton, Cole, O'Grady, & Osborn, 1982) is to escape from noxious and intolerable emotional experiences. In the case of NSSI more specifically, it has been hypothesized that escape in the form of the reduction of tension or more general negative affect serves as a primary motivation for these repetitive behaviors (i.e., automatic negative reinforcement; e.g., Nock & Prinstein, 2004, 2005; Suyemoto, 1998; Yip, 2005). In this way, it may be that the tendency of certain individuals to experience heightened and/or prolonged emotion reactivity in response to a range of stressors (or, alternatively, a domain of commonly experienced stressors) increases the

likelihood that these individuals will engage in SITB as an attempt to regulate or escape from these aversive internal states.

Of late, the association between emotion reactivity and adolescent SITB has begun to receive substantial empirical support. For instance, relative to non-self-injuring psychiatric controls, outpatient adolescent self-injurers have been found to report higher levels of subjectively experienced emotional distress in response to stressful events (Najmi, Wegner, & Nock, 2007; Nock et al., 2008a). Moreover, Nock and colleagues (2008a) in their cross-sectional validation of the self-reported Emotion Reactivity Scale (ERS) found that the construct of emotion reactivity mediated the concurrent association between overall level of psychopathology (represented by a composite score of mood, anxiety, or eating disorder symptoms as assessed by the K-SADS-PL) and the frequency of NSSI and suicidal ideation. The authors speculated that difficulties with emotion reactivity, a common feature to many types of psychopathology, may explain why the vast majority of individuals who engage in SITB also have a diagnosable psychological disorder (Nock et al., 2008a). Parallel evidence for the association between emotion reactivity and self-injury has been reported in a nonclinical sample (Klonsky, Oltmanns, Turkheimer, 2003). Klonsky and colleagues (2003) examined a sample of nearly 2000 military recruits and found that, as compared to their peers who had not reported a history of self-harm, self-harming individuals were viewed by themselves as well as peers as having more "strange and intense emotions" and a heightened sensitivity to interpersonal rejection.

More recently, psychophysiological research has been brought to bear on questions relating to the potential role of emotion reactivity in the development of adolescent SITB. Beyond the obvious appeal of objectivity, a crucial advantage of the physiological

measurement of emotion reactivity is that it allows for the individual or simultaneous quantification of emotion sensitivity, intensity, and persistence (e.g., Stern, Ray, & Quigley, 2001). For example, the study conducted by Nock and Mendes (2008), which incorporated measures of subjective distress and physiological arousal, demonstrated that adolescents who reported engaging in NSSI experienced both higher levels of negative affect during a distressing task and exhibited significantly lower levels of distress tolerance than those without histories of NSSI (Nock & Mendes, 2008). Similar results have recently been found among adolescent samples of suicidal and nonsuicidal self-injurers across many putative physiological indices of arousal, including skin conductance level (Nock & Mendes, 2008), serotonin (5-HT) concentration in peripheral blood (Crowell et al., 2008), and respiratory sinus arrhythmia (RSA; Crowell et al., 2005).

HPA Axis Reactivity as a Measure of Emotion Reactivity

The psychophysiological measurement of emotion reactivity from salivary cortisol has also received particular research attention (see Denson, Spanovic, & Miller, 2009). This is unsurprising given that cortisol is the end product of activation of the hypothalamic-pituitary-adrenal (HPA) axis, which—besides the sympathetic-adrenal-medullary (SAM) axis—is the major biological stress response system in humans. Since a pathway to activation of the HPA axis begins with affective information processed in the limbic system, the experience of emotions is considered an important trigger and modulator of this system (e.g., Adam, Sutton, Doane, & Mineka, 2008). Further, there is good evidence from naturalistic studies that collected multiple samples of cortisol throughout the day that

negative affect in response to stressors is associated with higher cortisol levels, whereas positive affect is associated with lower cortisol levels (see Smyth et al., 1998).

The dysfunction of HPA axis in adults (as a putative proxy for difficulties with emotion reactivity) has been commonly associated with completed suicide and, to a lesser extent, with attempted suicide (see Mann et al., 2009). In fact, in a recent review of twin, adoption, and family studies establishing the heritability of suicidality, Mann and colleagues (2009) concluded that cortisol response to social stress was one of the most promising endophenotypes associated with suicide attempts and suicide. Indeed, this possible trait-like pattern of hyper-responding to social stress might also help to explain increases in SITB during the adolescent transition. For example, it is intriguing that recent developmental studies of HPA axis reactivity during the adolescent transition have revealed increases in cortisol response to psychosocial stress from childhood to adolescence (Gunnar, Wewerka, Frenn, Long, & Griggs, 2009; Stroud et al., 2009).

The Current Study: Towards a Biopsychosocial Model of Adolescent SITB

The present study aimed to construct and preliminarily test a more developmentally-specific reformulation of Abramson and colleagues' (1989, 2000) hopelessness theory of suicidality. Given the particularly alarming increases in SITB during the transition to adolescence, as well as evidence that this developmental period is associated with the onset and maintenance of a chronic course of SITB, the explicit goal of this theoretical adaptation was to better characterize, explain, and predict SITB among young people. A sample predominantly composed of adolescent females was chosen because of the particularly high prevalence of SITB among females during this developmental period. Attempts were also

made to oversample adolescent participants from clinical referral sources, given the overall greater prevalence of SITB in the context of diagnosed psychopathology. Since the experience of SITB may fluctuate rapidly, a thorough assessment of these thoughts and behaviors was planned at frequent, temporally proximal longitudinal intervals.

The study addressed five central hypotheses related to the concurrent association and the prospective prediction of adolescent SITB. First, it was anticipated that a comprehensive baseline measure of self-reported cognitive vulnerability (in the form of a negative inferential style for causes, consequences, and self characteristics) would be associated with the self-reported occurrence of SITB both concurrently (i.e., at baseline) and over time. Second, it was predicted that the degree and duration of emotional reactivity to a laboratory-based, in vivo social stress task—as measured by subsequent and repeated salivary cortisol sampling—would similarly be associated with both concurrent and prospective levels of SITB. More specifically, it was hypothesized that those salivary cortisol levels which were higher in magnitude and maintained for longer periods of time after a stressor would be directly related to greater engagement in SITB.

Third, consistent with cognitive appraisal theory (e.g., Lazarus & Folkman, 1984), it was predicted that self-reported cognitive vulnerability at baseline would be concurrently associated with dysregulated emotion reactivity in response to an in vivo stressor designed to be experienced as uncontrollable, novel, challenging, and threatening to the social self. Fourth, it was predicted that dysregulated emotion reactivity is a mediator of the association between cognitive vulnerability and trajectories of SITB over time (see Figure 2).

A social evaluative speech task was selected as a stressor in the present study for two important reasons. It has been both theoretically argued and empirically demonstrated that

interpersonally-themed stress poses a particularly strong threat to adolescent samples generally and to adolescent girls specifically (Rudolph, 2002; Rudolph & Hammen, 1999; Hankin et al., 2007). In Gunnar and colleagues' (2009a) recent review of stressor paradigms in developmental studies, it was found that tasks which threaten the social self (i.e., public speaking tasks) produced the most reliable and pronounced increases in salivary cortisol. Fifth and finally, given the findings of prior work in this area related to the specific vulnerability hypothesis (Joiner & Rudd, 1995), it was predicted that a particular cognitive vulnerability for interpersonally-themed stressors as opposed to that for achievement-related stressors would confer heightened risk for subsequent emotional hyperreactivity following the speech task.

CHAPTER 2

METHODS

Participants

Adolescent participants were recruited through various clinical referral sources as part of a broader longitudinal investigation of adolescent self-injury funded by the American Foundation of Suicide Prevention. Potential participants were initially screened during recruitment for a number of predetermined exclusionary variables. First, adolescents were excluded from participation if they met criteria for any past or current diagnoses indicating psychosis, mental retardation, or pervasive developmental disorders. Second, detailed information on prescription medication usage was collected during the baseline assessment to assess for medications that would alter target variables. Given the significant and long-lasting effect of corticosteroid medication (potentially including inhaled agents such as flovent and ventolin) on circulating cortisol levels even on days when this class of medication is not taken, adolescents who had been prescribed these medications were excluded. Additionally, it was requested that adolescents refrain from taking any medication(s) on the day of testing until all procedures were completed.

Participants included 62 youths (50 community living and 12 inpatients) at the transition to adolescence, between the ages of 12 and 16 years (M = 14.70; SD = 1.33). Referral sources included local inpatient units (n = 12; 19%), outpatient clinics and community mental health agencies (n = 3; 5%), local high schools (n = 16; 26%), and mass-

email advertisements (n = 31; 50%). Forty-five participants were female (73%) and approximately 76% of all participants self-identified as White/Caucasian, 8% African-American, 8% Latino-American, 5% Asian-American, and 3% Mixed or Other Ethnicity. Approximately 65% of adolescents lived in a two-parent household, while 35% lived with their biological or adoptive mother only. Three percent of mothers reported that their highest level of education was a high school diploma or GED, 6% of mothers earned an associate's or trade degree, 29% of mothers reported that they had attended some undergraduate college, 13% earned a bachelor's degree, 6% reportedly attended some graduate school, 26% earned a master's degree, and 16% had earned a doctoral degree.

With respect to the recruitment of inpatients (and consistent with human subjects regulations), adolescent patients (n = 12; 19%) and their parents were approached for study participation only after hospital personnel had gained permission from adolescents' parents/guardians to be contacted about this investigation. For inpatients, data relevant to diagnostic status were collected by research staff during adolescents' admission, whereas the remaining laboratory-based aspects of the study were conducted four weeks post-discharge. This delay was chosen to allow for adequate time for the effects of the crises that may have precipitated hospitalization to subside. The psychiatric statuses of outpatient and community participants were determined at the initial laboratory baseline visit (see below). Diagnoses at baseline, as assessed by the Diagnostic Interview Schedule for Children (NIMH-DISC-IV – Adolescent Report; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000), included Major Depressive Disorder (15%), Oppositional Defiant Disorder (8%), Posttraumatic Stress Disorder (5%), and Generalized Anxiety Disorder (8%). Approximately 70% of participants

(n = 43) did not meet criteria for any psychiatric diagnosis (cumulative percentages exceeded 100% due to comorbidities).

Procedures

Recruited adolescents completed an initial baseline assessment in a laboratory setting. During this visit, participants completed all questionnaire data and were administered structured interviews, including those aiming to provide a comprehensive assessment of past and current engagement in SITB (see below). In addition, adolescents participated in an in vivo, social stress-induction paradigm while salivary cortisol samples were collected at regular intervals (described in detail below). Collateral data related to pubertal development, depressive symptoms, and time of awakening on the day of testing were also collected, given the known influence of these variables on diurnal cortisol secretion (e.g., Gunnar and Quevedo, 2007). Subsequent to this baseline assessment, telephone follow-up interviews were conducted at 3-, 6-, and 9-months post-baseline to reassess the presence of SITB in the time period since the preceding assessment. Three-month intervals were chosen on the basis of data suggesting that adolescent inpatients are at the greatest risk for making a suicide attempt within 6 months following discharge (e.g., Brent et al., 1993b). Adolescents received incrementally increasing monetary compensation for their participation at various stages of the study (up to $80 for the completion of all lab and telephone-based data collection).

Of the 62 adolescents who completed baseline assessments, 55 (89%) participated in the 3-month follow-up assessment, 44 (71%) participated in the 6-month follow-up, and 41 (66%) completed the 9-month follow-up. Although many retention strategies were utilized

(e.g., frequent phone, mail, and email contact with participants and their families, the provision of monetary incentives to encourage continuing participation, etc.), attrition over longitudinal intervals reflected reasons common to research of this type, including family relocation, study drop-out, and hospital readmission.

Primary Measures

Self-injurious Thoughts and Behaviors. Adolescents' suicidal and nonsuicidal self-injurious thoughts and behaviors were assessed using the Self-Injurious Thoughts and Behaviors Interview (SITBI, Nock, Holmberg, Photos, & Michel, 2007). The SITBI is a structured, clinician-administered interview (3-15 minutes) which uses 169 items across five modules to assess the presence, frequency, severity, duration, age-of-onset, and other characteristics of a broad range of SITB. Importantly, the modules included on the SITBI correspond to the full range of constructs specified by O'Carroll and colleagues' (1996) authoritative taxonomy of self-injury. These include suicide ideation, suicide plans, suicide gestures (i.e., instrumental suicide-related behaviors performed without intent to die), suicide attempts, and NSSI.

The adolescent-specific version of this interview was administered at the baseline laboratory visit and during each of the three follow-up time points. Nock and colleagues (2007) provided evidence for the strong psychometric characteristics of the SITBI in an adolescent sample. The SITBI has strong inter-rater reliability (average κ = .99, r = 1.0) and test-retest reliability over a 6-month period (average κ = .70, ICC = .52). Further, the construct validity of the SITBI has been demonstrated by its strong correspondence with

other measures of suicide ideation (average κ = .54), suicide attempt (κ = .65), and NSSI (average κ = .87).

Cognitive Vulnerability. Participants' negative inferential style was assessed during the baseline laboratory visit using the Adolescent Cognitive Style Questionnaire (ACSQ; Hankin & Abramson, 2002). The ACSQ is a 12-item, self-reported measure of cognitive vulnerability to depression designed for use with high school-age adolescents. Consistent with hopelessness theory (Abramson et al., 1989; Abramson et al., 2000), the ACSQ assesses adolescents' tendencies to make negative inferences regarding the causes, consequences, and the self in response to stressful events. The questionnaire consists of 12 hypothetical scenarios (6 each related to interpersonal or achievement domains) relevant to adolescents. Each scenario presents the participant with a hypothetical negative event (e.g., "Your girlfriend/boyfriend breaks up with you, but you still want to stay together") and allows the participant to record one cause for the event in the unstructured space provided. Respondents then rate the degree to which the cause of the hypothetical negative event is internal, stable, and global (i.e., negative inferences for causal attributions). In addition, they rate the likelihood that further negative consequences will result from the occurrence of the negative event (i.e., negative inferences for consequences), as well as the degree to which the occurrence of the event signifies that the person's self is flawed (i.e., negative inferences for self).

Average item scores on the ACSQ range from 1 to 7, with higher scores indicating more negative cognitive styles. The ACSQ has demonstrated sound psychometric properties, including excellent internal consistency, good test-retest reliability, and factor structure consistent with hopelessness theory (Hankin & Abramson, 2002). Validity for the ACSQ

also is shown by research in which the ACSQ, either alone or in interaction with negative events, predicts depressive symptoms and episodes (e.g., Hankin, 2008). Given the previously noted theoretical and empirical importance of assessing a specific domain of cognitive vulnerability in the context of a specific class of stressors (see Abramson et al., 1989; Beck, 1967) and that the present research design incorporated an in vivo social stressor paradigm, composite averages of "interpersonal cognitive vulnerability" and "achievement cognitive vulnerability" were computed across each of the two sets of six interpersonally- and achievement-themed hypothetical events on the ACSQ. Internal consistency for the interpersonal and achievement subscales were both excellent ($\alpha = .92$ for both).

In Vivo Social Stressor Paradigm. Similar to paradigms commonly used in psychophysiological research (e.g., Hastings, Zahn-Waxler, & Usher, 2007; Klimes-Dougan, Hastings, Granger, Usher, & Zahn-Waxler, 2001), adolescents participated in a social stressor speech task during the baseline laboratory assessment. Participants who had been acclimated to an observational setting were oriented towards a camera connected to a closed-circuit "feedback screen" displaying their own live image. Adolescents were instructed to face this camera and feedback screen while preparing (for one minute) and subsequently delivering a three-minute speech. The explicit goal of the speech, as explained to participants, was to convince an audience of their peers (presumably watching the live video feed in a nearby room) that they should be selected to star in a fictional television show about teens' ability to form and maintain friendships.

Immediately prior to the adolescents' delivery of the speech, a male undergraduate research assistant (who had been previously selected on the basis of youthful appearance)

entered the room, ostensibly to evaluate participants' performance. Although this "observer" remained in the room at close proximity to the participant for the duration of the speech task, he was given instructions to fix his gaze on the feedback screen and withhold direct eye contact with the participant at all times. At approximate intervals of 20 seconds, the observer was instructed to make a small mark on a clipboard in order to give the appearance of continuous evaluation. Immediately following the speech task, the observer asked a series of brief, structured questions designed to elicit adolescents' self-evaluations of their speech performance (e.g., "How do you think you did on the speech?", "Do you think that you would be selected for the TV show, if this were an actual audition?"). As with the "evaluative" component of the speech task, the observer was instructed to withhold during this "debriefing" session any verbal or nonverbal feedback while in the presence of the participant.

A speech task of this kind has been shown to elicit meaningful variability in adolescents' physiological responses, including those specifically pertaining to adolescents' neuroendocrine responses (e.g., Hastings et al., 2007; Klimes-Dougan et al., 2001). In fact, in Dickerson and Kemeny's (2004) meta-analysis of laboratory studies of acute psychological stressors conducted with adults, it was found that speech tasks characterized by both uncontrollable and social-evaluative elements in which others could judge performance negatively are associated with greater cortisol responses than other types of stressors. A similar conclusion was reached in a recent review of stressor paradigm studies conducted with children and adolescents (Gunnar et al., 2009a).

Measurement of HPA Axis Reactivity. Adolescents were asked to provide salivary cortisol samples using a passive drool procedure (see Klimes-Dougan et al., 2001). This

procedure, developed by Klimes-Dougan and colleagues (2001), involves chewing a piece of sugar-free gum for one minute, swallowing the accumulated saliva (to avoid potential contamination of the sample by the "flavor burst"), chewing for an additional minute without swallowing any saliva, and then expectorating 5 milliliters into a vial. Participants gave salivary samples on four occasions during the baseline laboratory assessment described above: 1) immediately prior to the speech task (following a 10-minute break from experimental procedures and questionnaires during which adolescents were asked to sit quietly in the observational room); 2) 20 minutes post-speech; 3) 30 minutes post-speech; and 4) 40 minutes post-speech. The timing of these samples was determined by the reliable empirical finding that cortisol will reach peak levels in human saliva approximately 20-30 minutes after the onset or peak of a stressor (e.g., Adam et al., 2008; Gunnar et al., 2009a). Additionally, it is important to note that in the time period immediately preceding the 20-minute, 30-minute, and 40-minute collections of saliva, participants were given a break from all experimental activities and were instructed to wait quietly.

Salivary samples were frozen for storage at -25°C and then shipped on dry ice to Pennsylvania State University's Behavioral Endocrinology Laboratory for assay (Salimetrics, PA). Samples were assayed for cortisol using a 510-k cleared high-sensitive enzyme immunoassay designed to assess adrenal function. This test, which uses 25 µl of saliva (for singlet determinations), has a lower limit sensitivity of .007 µg/dl and a range of sensitivity from .007 to 1.2 µg/dl. Average intra- and inter-assay coefficients of variation were computed. Sample pH were screened for levels less than 4 and greater than 9 prior to assay in accordance with guidelines set by Schwartz, Granger, Susman, Gunnar, and Laird (1998).

Covariate Measures

Cortisol Timing. Cortisol production is known to be influenced by a combination of physiological, psychological, and environmental factors. One salient variable is the time of day. Over the circadian cycle of day and night, or waking and sleeping, normative cortisol production follows a predictable pattern (Lovallo & Thomas, 2000). The normative pattern is for cortisol levels to be fairly high by the end of the sleeping period and to continue increasing until it peaks 30 to 40 minutes after awakening. This is the "cortisol awakening response" (CAR) (Adam et al., 2008; Chida & Steptoe, 2009). Circulating cortisol levels then drop rapidly over the morning, drop more slowly through the afternoon, and reach their nadir in the evening (Lovallo & Thomas, 2000). Thus, over the waking (daytime or diurnal) period, the change in cortisol levels is characterized by a negative slope. Cortisol levels then increase again during sleep, until the waking level is reached in the morning hours. The diurnal rhythm constitutes "baseline" or "basal" HPA activity, representing the predictable, circadian cycle-dependent, physiologic fluxes of blood cortisol that are expected at various times throughout the day, all other things being equal.

Given that the present study is concerned with individuals' cortisol response to a discrete stressor, it is important to note that cortisol levels observed immediately following a stressor represents the sum of the acute cortisol response together with the basal cortisol level for that particular time of day (e.g., Gunnar & Quevedo, 2007). In other words, in the current study it was essential to control for the time of day relative to the time of awakening, when interpreting cortisol response to the in vivo laboratory stress task. Thus, adolescent participants were asked to report their time of awakening and the times of cortisol collections were recorded by laboratory personnel. For each individual, a "cortisol timing" variable was

computed representing the duration of time elapsed between the time of awakening and the time at which the first cortisol sample was collected.

Pubertal stage. Pubertal development was assessed using adolescent' self-report on the Udry questionnaire. This questionnaire presents two sets of five serial line drawings representing the development of two secondary sexual characteristics and corresponding to the five Tanner stages, from prepubertal (stage = 1) to postpubertal (stage = 5) (Morris & Udry, 1980). Female and male participants were presented, respectively, with drawings depicting breast development/pubic hair growth and genital development/pubic hair growth. For each of the two sets, all participants were instructed to circle the picture that is "closest to your stage of growth." Adolescent self-ratings of pubertal stage on the Udry questionnaire are highly correlated with physician assessment and are considered sufficient for a general estimation of pubertal stage (Dorn, Susman, Nottelmann, Inoff-Germain, & Chrousos, 1990; Morris & Udry, 1980).

For the purpose of the present study and in accordance with other investigations (e.g., Negriff, Fung, & Trickett, 2008), pubertal stage was defined as the score on the breast pictures for females and the genital pictures for males. Data from the question relating to pubic hair stage was not included for two reasons. First, as compared to estimations of pubic hair growth, breast/genital development are more revelatory secondary sexual characteristics and have been found to be more reliably measured (Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987). Second, pubic hair growth and breast/genital development are linked to differently timed hormone systems and correspond to disparate age norms (Grumbach, 2002). In the current sample, breast/genital stage and pubic hair stage scores were strongly correlated ($r = .61$, $p < .001$).

Depressive symptoms. Depressive symptoms were assessed using the Mood and Feelings Questionnaire (MFQ; Costello & Angold, 1988). The MFQ was designed for use as a self-reported screening instrument for major depression among children and adolescents aged 8-18 years. The questionnaire, which consists of 33 items rated on a three point scale (0 = Not True; 1 = Sometime True; 2 = Mostly True), includes content conforming to DSM criteria for Major Depressive Disorder. Evidence from psychometric studies of the MFQ indicate that the questionnaire has strong internal consistency, acceptable test-retest reliability, and high convergent validity with semi-structured diagnostic measures of MDD such as the Schedule for Affective Disorders and Schizophrenia—Child Version (Angold, 1989; Wood, Kroll, Moore, & Harrington, 1995). In the present sample, internal consistency was excellent ($\alpha = .97$). A mean score across all 33 items was computed at baseline.

Data Analytic Plan

Three sets of analyses were conducted to examine study hypotheses. First, descriptive statistics were conducted to examine the means and standard deviations on all study variables over the 9-month longitudinal period. Correlational analyses also were performed between all study variables. Given the hypothesized concurrent associations between baseline measures of cognitive vulnerability and SITB and between emotion (i.e., cortisol) reactivity and SITB, correlational data among these measures were of particular interest. Consistent with appraisal theory (e.g., Lazarus & Folkman, 1984), the hypothesis that cognitive vulnerability would be concurrently associated with observed dysregulated emotion reactivity in response to a performance-based laboratory stressor was tested by examining the bivariate relationship between the measure of cognitive vulnerability and salivary cortisol data.

Second, an unconditional growth curve model using latent curve analysis was examined to better understand the pattern of emotion reactivity and recovery as reflected in the salivary cortisol samples. The use of latent curves allowed for estimation of the slope and pattern of growth within the entire sample, as well as predictors of individual temporal growth trajectories (Bollen & Curran, 2006). All latent curve analyses were conducted using AMOS 16.0. Cortisol samples measured pre-speech task (Time 1), 20 minutes post-speech (Time 2), 30 minutes post-speech (Time 3), and 40 minutes post-speech (Time 4) were included as observed indicators, with latent intercept and slope factors estimated. Adapting the analytic procedures recommended by Willoughby, Vandergrift, Blair, and Granger (2007) for use with cortisol data using "pre-post-post designs", this model examined a three-slope, or piecewise linear model, where each piece consists of only two time points. The first slope function modeled the curve between Time 1 and Time 2 measures of cortisol (i.e., a "reactivity" curve), the second slope function modeled the curve between Time 1 and Time 3 (i.e., an initial "regulation" curve), and the third slope modeled the curve between Time 1 and Time 4 (i.e., a "recovery" curve). This parameterization is equivalent to a simple difference score approach wherein reactivity refers to the simple differences between cortisol values obtained at Times 2 and 1, initial regulation refers to the differences between Times 3 and 1, and recovery refers to the differences between Times 4 and 1 (Willoughby et al., 2007). A latent intercept factor with paths to each observed indicators set to 1 was modeled. Path weights between the reactivity, regulation, and recovery latent slope factors and Time 2, Time 3, and Time 4 cortisol observed indicators, respectively, were all set to 1 (see Figure 3). Third, to examine central study hypotheses related to the prospective prediction of SITB, the unconditional model specified above was built upon. Hypotheses tested a conditional growth

curve model. The proposed model tested whether inter-individual differences in baseline cortisol values, cortisol reactivity, cortisol regulation, and cortisol recovery were associated with engagement in SITB. Further, it was intended that the measure of cognitive vulnerability would be entered into the model and tested as a predictor of SITB. Lastly, assuming cognitive vulnerability would itself be associated with measures of SITB, cortisol reactivity would then be tested as a mediator of the prospective association between cognitive vulnerability and SITB. All paths would be estimated between these additional indicators and the latent intercept and slope factors for cortisol (see Figure 4).

CHAPTER 3

RESULTS

Descriptive Statistics

Measures of SITB

Descriptive statistics for SITB outcome measures are provided in Table 1 and Table 2. To allow for a more complete exploration of the prevalence and course of the various forms of SITB across the 9-month interval, separate data are reported for suicide ideation, suicide attempt, and nonsuicidal self-injury (NSSI). Overall, the data indicated that in regard to the month preceding baseline assessment 12 individuals (approximately 19% of the sample) reported the experience of suicide ideation, 5 individuals (approximately 8%) reported attempting suicide, and 5 individuals (approximately 8%) reported engaging in NSSI.

Given the relatively rare occurrence of individual forms of SITB, two composite indices were also computed, revealing that 13 individuals (21%) reported engaging in any of the above forms of SITB in the month prior to baseline assessment (i.e., "SITB composite") and 9 individuals (14.5%) reported engaging in any suicidal or nonsuicidal self-injurious behavior in this timeframe (i.e., suicide attempts and/or NSSI; "SIB composite"). Similarly and as would be expected, a substantially greater proportion of adolescents reported lifetime (as opposed to past-month) engagement in SITB at the baseline assessment. For example, approximately 44% of participants (27 individuals) reported the lifetime experience of any

form of SITB and over a quarter of the sample reported the lifetime performance of suicidal or nonsuicidal self-injurious behaviors.

As can be seen from Table 1, the already low baseline rates of SITB decline precipitously over the 9-month longitudinal interval, with between 0 and 5 individuals (9.1%) reporting engaging in suicide ideation, suicide attempts, or NSSI in the month preceding either the 3-month, 6-month, or 9-month follow-up assessments. There was also evidence for biased attrition: as compared to individuals who denied engaging in any form of SITB in the month prior to baseline assessment, adolescents who reported past-month engagement in SITB at baseline were more likely to drop-out of study participation over the 9-month follow-up interval, $\chi^2(1) = 3.94$, $p < .05$. Further, there was a notably low incidence of individuals in the present sample who reported the first onset/initiation of SITB during the follow-up time period. Between the interval of time between baseline assessment and the 9-month post-baseline assessment, no participants reported the first experience of suicide ideation, only 2 individuals reported first suicide attempts, and 5 individuals reported the onset of NSSI.

Thus, given the particularly low rates of prevalence, onset, and maintenance of SITB over time in the present sample, it was determined that power was insufficient to examine central study hypotheses related to the prospective prediction of SITB. As a result, subsequent analyses involving past-month and lifetime measures of SITB were limited to the examination of study hypotheses using concurrent baseline data.

Additionally, although it was initially important to provide separate data regarding individual forms of SITB for descriptive purposes, it is both intuitively apparent and empirically demonstrated that suicide ideation, suicide attempts, and NSSI often co-occur

(e.g., Kessler, Borges, & Walters, 1999; Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006; Reinherz, Tanner, Berger, Beardslee, & Fitzmaurice, 2006). Indeed, when present, forms of SITB were often highly comorbid in the present sample. For example, of the 14 individuals who reported at baseline lifetime engagement in NSSI, 10 of these also reported lifetime experience of suicide ideation and 7 acknowledged at least one previous suicide attempt. Thus, to better represent the occurrence of SITB in the present sample and increase both ecological validity and statistical power, adolescents were divided into four conceptually-based categories in preparation for subsequent analyses conducted separately for past-month versus lifetime measures of SITB. The descriptive data regarding the four categories, which are presented in Table 2, were as follows: individuals who reported the absence of any SITB (past-month, $n = 47$; lifetime, $n = 35$), individuals who reported the experience of suicide ideation only but engaged in no self-injurious behaviors (past-month, $n = 4$; lifetime, $n = 11$), individuals who reported ideation combined with a history of either suicide attempt(s), NSSI, or both behaviors (past-month, $n = 8$; lifetime, $n = 12$), and individuals who reported engaging only in NSSI, in the absence of ideation or attempts (past-month, $n = 1$; lifetime, $n = 4$). Finally, to fully capitalize on power, the analyses were re-run for lifetime versus past-month SITB with the groups described above collapsed into the following dichotomized categories: the previous experience of any form of SITB (past-month, $n = 13$; lifetime, $n = 27$) versus the absence of prior history of any SITB (past-month, $n = 47$; lifetime, $n = 35$).

Independent Variables

Table 3 presents descriptive data for independent variables and covariates. Consistent with previous studies examining stress reactivity of the HPA axis using measures of salivary cortisol, raw cortisol values were highly positively skewed (e.g., Klimes-Dougan et al., 2001). Therefore, log transformations of the 4 cortisol samples were conducted to establish normal distributions prior to analyses. All subsequent analyses of cortisol data utilized the log-transformed values. Paired samples *t*-tests were conducted to preliminarily characterize (unadjusted) mean differences among the cortisol samples taken immediately prior to the speech task (i.e., Time 1), 20 minutes post-speech task (i.e., Time 2), 30 minutes post-speech task (i.e., Time 3), and 40 minutes post-speech tasks (i.e., Time 4). These analyses indicated first that the mean Time 1 cortisol value was significantly lower than that for Time 2, t (61) = -3.65, $p < .01$, and Time 3, t (60) = -2.06, $p < .05$, but were not significantly different than that for Time 4, t (60) = -1.16, $p = .25$. This indicates that, relative to baseline values, while participants tended to experience significant increases in cortisol production at Time 2 and Time 3 in response to the in vivo stressor, their cortisol had recovered to a level comparable to that of baseline samples by Time 4. Second, the mean Time 2 cortisol value was significantly greater than the means obtained at both the Time 3, t (60) = 3.98, $p < .001$, and Time 4 collections, t (60) = 4.19, $p < .001$, indicating that, as expected, the Time 2 cortisol values represent the peak of HPA axis responsiveness to the stressor. Third, the mean Time 3 cortisol value was significantly greater than the mean Time 4 cortisol value, t (60) = 2.24, $p < .05$.

Inspection of descriptive data derived from the Adolescent Cognitive Style Questionnaire (ACSQ) revealed that the interpersonal and achievement cognitive

vulnerability subscales were both fairly normally distributed. Interestingly, and contrary to what might be expected given that the sample was composed predominantly of adolescent females, participants' mean levels of achievement vulnerability was significantly higher (i.e., more negative) than those of their interpersonal vulnerability, t (60) = -4.88, $p < .001$. This difference remained significant when only females were considered in this analysis, t (43) = -4.00, $p < .001$.

Correlational Analyses

Intercorrelations between all continuous study variables are presented in Table 4. Note that, where indicated, partial correlations are displayed between cortisol samples and other study variables, controlling for age, pubertal status, and the duration of time between awakening and the collection of the first cortisol sample. For the most part, results from these correlational analyses were as expected. For example, there was a significantly negative and increasing correlation between cortisol samples 1 through 4 and the duration of time between awakening and the time of Sample 1. As expected from a normative diurnal cycle in which basal cortisol production steadily decreases throughout the day (e.g., Lovallo & Thomas, 2000), later cortisol samples and correspondingly longer times elapsed since awakening are associated with smaller concentrations of cortisol in saliva.

Also broadly consistent with previous evidence demonstrating a link between depression and cortisol hypersecretion (Hankin, Badanes, Abela, & Watamura, 2010; Luby et al., 2003; Luby, Mrakotsky, Heffelfinger, Brown, & Spitznagel, 2004; Rao, Hammen, Ortiz, Chen, & Poland, 2008), in the present sample there were positive correlations at the level of a trend between cortisol levels and depressive symptoms. However, these correlations were

small and failed to reach statistical significance (all *p*s > .05). Finally, data presented in Table 4 indicate that interpersonal but not achievement-related cognitive vulnerability was associated with depressive symptoms. These data converge with prior work which has demonstrated that cognitive vulnerability—and particularly interpersonally-oriented as opposed to achievement-related attributional style (e.g., Robins, Block, & Peselow, 1985; see Hankin & Abramson, 2000; Coyne & Whiffen, 1995, for reviews)—is positively associated with depressive symptoms.

Given study hypotheses related to the concurrent positive association between cognitive vulnerability and cortisol reactivity, these bivariate correlations were of particular interest. Although these correlations were weak and failed to reach statistical significance (all *p*s > .05), all were in the expected direction. It is likely that power was insufficient to detect statistically significant findings related to the bivariate association between cognitive vulnerability and cortisol response. Contrary to expectations, however, there was no evidence within the full sample that mean interpersonally-oriented cognitive vulnerability scores were more associated with cortisol response to a social stressor than were mean achievement-related vulnerability scores (all *p*s > .05). But when correlational analyses were conducted separately by gender, adolescent females' mean interpersonal vulnerability scores were significantly and moderately correlated with all four cortisol values (*r* s ranging from .36 to .42, *p*s < .05). On the other hand, correlations between girls' achievement vulnerability scores and cortisol values were all relatively lower in magnitude and, with the exception of the association with Time 2 ($r = .34$, $p < .05$), failed to reach statistical significance. For adolescent boys, no significant associations were found between the

cortisol values and either mean interpersonal or mean achievement-oriented vulnerability scores.

SITB Group Differences among Study Variables

Overview of MANOVA/MANCOVA Analyses

As previously described, to assist with statistical comparisons adolescents were divided into four conceptually-based categories according to SITB status: individuals who reported the absence of any SITB; individuals who reported the experience of suicide ideation without any past engagement in self-injurious behaviors; individuals who endorsed ideation combined with a history of either suicide attempt(s), NSSI, or both behaviors; and individuals who reported engaging only in NSSI, in the absence of suicide ideation or attempts. In accordance with study hypotheses, a series of multiple analyses of variance (MANOVAs) and multiple analyses of covariances (MANCOVAs) were performed to establish and explore mean SITB group differences with respect to the cognitive vulnerability and cortisol variables. The results of these analyses for lifetime and past-month SITB are presented in Table 5 and Table 6, respectively.

Of note, all of these analyses were initially run as factorial with gender as an additional independent variable (i.e., simultaneously with life-time or past-month SITB groups, respectively). However, since no significant effect for gender was ever found, this independent variable was dropped from all analyses reported below. Additionally, given the previously cited relevance of the construct of interpersonal cognitive vulnerability and this specific domain of stressors (and thus, stress response) to adolescent females in particular, each MANOVA and MANCOVA analysis described below was first conducted for the entire

sample and then separately for adolescent females. For the most part, separate analyses for adolescent females did not alter the pattern of results reported below. The few differences that were found are reported in the relevant section of results.

Group Differences in Cognitive Vulnerability by SITB Status – Lifetime and Past-month

It was hypothesized that, as compared to adolescents without such histories, adolescents engaging in SITB would possess a more negative attributional style (i.e., cognitive vulnerability), particularly with respect to interpersonally-oriented attributional style. To test this hypothesis, one-way MANOVAs were conducted with mean interpersonally- and achievement-related vulnerability entered as dependent variables and categories of SITB engagement entered as the independent variable. These analyses were conducted separately for lifetime SITB (Table 5) and past-month SITB (Table 6). Of note, given that cognitive vulnerability and cortisol data were only available for a single participant who had reported engaging in NSSI in the past month, this category was removed from all past-month analyses.

A significant overall effect for lifetime SITB group was found, F (6, 112) = 2.97, p = .01. As can be seen in Table 5, follow-up analyses of between-subject effects provided some support for the hypotheses; marginally significant mean differences were found with respect to lifetime SITB status for interpersonal cognitive vulnerability, F (3, 57) = 2.31, $p < .10$, whereas no such differences in achievement vulnerability were found ($p > .10$). Post hoc comparisons using Tukey's HSD revealed that individuals with a lifetime history of suicide ideation combined with either attempts, NSSI, or both had significantly more negative interpersonally-oriented attributional styles than did individuals without any life history of

SITB (p = .05). No other mean group differences reached statistical significance. To determine whether the significant mean difference in interpersonal vulnerability between the two groups would be found over and above the effect of depressive symptoms, a follow-up MANCOVA was conducted controlling for the effect of mean MFQ scores at baseline. The significant effect for SITB group was no longer found, F (6, 110) = 1.66, p = .14.

A broadly similar pattern of results were found for the cognitive vulnerability variables using MANOVA and MANCOVA analyses considering past-month SITB (see Table 6). A significant effect for past-month SITB group was found, F (4, 108) = 4.44, p = .002. As with analyses conducted with lifetime SITB, follow-up analyses of between-subject effects revealed significant means differences among past-month SITB groups with respect to interpersonal cognitive vulnerability, F (2, 55) = 5.70, p < .01. Post hoc Tukey's HSD analyses indicated that individuals who reported suicide ideation combined with either attempts, NSSI, or both in the past month had significantly more negative interpersonally-oriented attributional styles than did individuals without any history of SITB (p < .05). As well, individuals reporting a history of suicide ideation (in the absence of self-injurious behaviors) in the past month had marginally significantly more negative interpersonally-oriented attributions styles than did individuals reporting no SITB in the past month (p < .10). Contrary to the result found with lifetime SITB, however, significant means differences were also found for past-month SITB groups with respect to achievement-related cognitive vulnerability, F (2, 55) = 3.84, p < .05. Tukey's HSD analyses revealed that individuals who reported suicide ideation combined with either attempts, NSSI, or both in the past month had significantly more negative achievement-oriented attributional styles than did individuals who reported no SITB in the past month (p < .05). It is important to note, however, that

when these analyses were conducted with adolescent females only, there were no longer significant differences in achievement related vulnerability between past-month SITB groups, F (2, 39) = 2.72, $p > .05$.

Contrary to the corresponding MANCOVA conducted with lifetime SITB groups, certain mean group differences with respect to past-month SITB remained significant after controlling for depressive symptoms within the full sample, F (4, 108) = 2.68, $p = .04$. Unexpectedly, follow-up analyses of between-subject effects after controlling for depressive symptoms indicated a marginally significant effect for achievement-related vulnerability, F (2, 54) = 2.79, $p = .07$, but not for interpersonally-oriented cognitive vulnerability, F (2, 54) = .99, $p = .38$. Pairwise (LSD) comparison analyses after partialling out depressive symptoms indicated that individuals who reported suicide ideation in combination with either suicide attempts, NSSI, or both in the past month had significantly more negative achievement-oriented attributional styles than did individuals reporting no SITB in the past month ($p < .05$), as well as marginally significantly more negative achievement-oriented attributional styles than did individuals who reported ideation only ($p < .10$). Again, however, when this MANCOVA was re-run considering only adolescent females, the significant effect for achievement-related vulnerability was no longer found, F (2, 38) = 1.75, $p > .10$.

Group Differences in Cognitive Vulnerability by Dichotomized SITB Status – Lifetime and Past-month

As mentioned above, to further explore SITB group differences and increase power to detect these differences, the four SITB groups were collapsed into dichotomous groups

representing the absence versus the presence of any form of SITB. Subsequently, the MANOVAs and MANCOVAs described above were repeated—again separately for lifetime and past-month history of SITB—to examine whether mean differences among the cognitive vulnerability measures existed among the dichotomized groups. As expected, the results of these analyses were largely consistent with those above. Significant overall effects on cognitive vulnerability were found for the dichotomized lifetime SITB groups, F (2, 58) = 5.87, p = .005, and past-month occurrence of SITB groups, F (2, 56) = 5.94, p = .005. Follow-up multivariate comparisons revealed that individuals who reported lifetime engagement in SITB had significantly more negative interpersonally-oriented cognitive vulnerability than did those without such histories, F (1, 59) = 4.27, $p < .05$, whereas no such mean differences in achievement vulnerability were found (p = .85). As with the 4 subgroup analyses considering lifetime SITB above, however, the adjusted mean difference in interpersonal vulnerability between the SITB group and controls were no longer significant after controlling for depressive symptoms (see Table 5).

The results of follow-up multivariate comparisons for dichotomized past-month SITB were identical to those conducted with the 4 subgroups of past-month SITB (see Table 6). Individuals who reported past-month engagement in SITB had significantly more negative interpersonally-oriented cognitive vulnerability, F (1, 57) = 12.02, p = .001, and achievement-related vulnerability, F (1, 57) = 4.99, $p < .05$, than did those without such histories. However, when a separate analysis was conducted with only females, the effect for interpersonal cognitive vulnerability remained significant, F (1, 40) = 7.55, $p < .001$, whereas no significant effect was found for achievement-related vulnerability, F (1, 40) = 1.98, $p >$ .10.

The results of MANCOVA analyses controlling for depressive symptoms within the full sample revealed that although there was no longer a significant overall effect for past-month SITB group on the cognitive vulnerability variables, F (2, 55) = 1.57, p = .22, a marginally significant between-subjects effect was found for achievement-related vulnerability, F (1, 56) = 2.97, p = .09, but not for interpersonally-oriented vulnerability, F (1, 56) = 2.46, p = .12. On the other hand, an examination of this MANCOVA analysis re-run with only adolescent females revealed no significant between-subject effects for either interpersonally- or achievement-oriented vulnerability for dichotomized past-month SITB group (ps > .10).

Group Differences in Cortisol by SITB Status – Lifetime and Past-Month

It was also hypothesized that, relative to adolescents without a history of SITB, adolescents engaging in SITB would demonstrate higher magnitudes of cortisol response to the laboratory stressor. To test this hypothesis, four one-way MANCOVAs were conducted—two considering lifetime SITB divided either into four groups or dichotomized (see Table 5) and two considering past-month SITB divided either into three groups or dichotomized (see Table 6). For all MANCOVAS, the mean values of the four logarithmically transformed cortisol samples were entered as dependent variables and categories of SITB engagement were entered as the independent variable. Age, pubertal status, and the duration of time between awakening and collection of the first cortisol sample were entered as covariates.

The results of MANCOVAs conducted with respect to lifetime SITB will be discussed first. Although no significant overall effect for SITB group was found for cortisol,

F (12, 111.41) = .81, *p* = .64, a marginally significant between-subject effect was found among lifetime SITB groups for cortisol Sample 1, *F* (3, 45) = 2.30, *p* < .10. Post-hoc Tukey's HSD analyses revealed that individuals with a lifetime history of NSSI (and no other SITB) had significantly higher (i.e., less extremely negative) levels of cortisol at Time 1 than did individuals with no prior history of SITB and individuals with a history of suicide ideation combined with either attempts, NSSI, or both (*p*s < .05). Individuals with a lifetime history of NSSI (and no other SITB) also had marginally significantly higher levels of cortisol at Time 1 than did individuals with a history of ideation only (*p* = .08). A parallel MANCOVA conducted using the dichotomized sample of presence versus absence of SITB yielded no significant results. The results of MANCOVAs conducted with respect to past-month SITB indicated no significant overall effect for the SITB group, considered as 3 categories, *F* (12, 111.41) = .65, *p* = .80, or dichotomized, *F* (4, 44) = .50, *p* = .50.

Unconditional Model: Characterizing Cortisol Changes in Response to the Stressor Task

The model depicted in Figure 3 was fit to the logarithmically transformed cortisol data. Given that the unconditional model is just identified, there are no formal indices of model fit. To recapitulate, the primary goals of the unconditional model were to establish the following: 1) the average values for baseline cortisol level, reactivity, initial regulation, and recovery (i.e., latent means); 2) to determine whether there is significant variation around these average values (i.e., latent variances); and 3) to determine the interrelations between baseline cortisol level, reactivity, regulation, and recovery scores (i.e., latent covariances).

The means of the latent variables that correspond to baseline cortisol level (i.e., Time 1), reactivity (i.e., Time 2), initial regulation (i.e., Time 3), and subsequent recovery (i.e.,

Time 4), respectively, were -1.114 ($p < .001$), 0.096 ($p < .001$), 0.051 ($p < .05$), and 0.027 ($p = .26$). Thus, the mean cortisol value at Time 1 of -1.114 was significantly different than 0. The mean difference between cortisol values at Times 1 and 2, defined here as reactivity, was 0.096. This value was positive and significantly different than 0, indicating an overall increase in cortisol between the initial baseline cortisol level and that of the sample taken 20 minutes following the in vivo stressor task. The mean difference between cortisol values taken at Times 1 and 3, defined here as initial regulation, was 0.051. This value was also positive and significantly different than 0; although the mean level of cortisol for the overall sample at Time 3 (i.e., 30 minutes following the stressor task) was a decline from the peak reactivity sample (taken at Time 2), this value was still significantly different from the mean initial baseline cortisol level. Finally, the mean difference between cortisol values taken at Times 1 and 4, defined as recovery, was 0.027. Although positive, this difference was not significantly different than 0, indicating that, by 40 minutes post-stressor task, mean cortisol levels had returned to a level comparable to the mean baseline value. Notably, these patterns of differences found between latent cortisol means using unconditional growth curve analyses were identical to those described previously using paired samples *t*-tests.

The variances of the latent variables corresponding to baseline cortisol level, reactivity, initial regulation, and recovery were 0.063 ($p < .001$), 0.042 ($p < .001$), 0.038 ($p < .001$), and 0.034 ($p < .001$), respectively. Thus, there was significant variability in the baseline level of cortisol, as well as in the magnitude of change in cortisol between Times 2 and 1 (i.e., reactivity), Times 3 and 1 (i.e., initial regulation), and Times 4 and 1 (i.e., recovery).

Finally, the latent correlations between baseline cortisol level with reactivity, initial regulation, and recovery was .01 (p = .93), -.09 (p = .50), -.17 (p = .20), respectively. Thus, baseline cortisol levels were not significantly related to the magnitude of reactivity, regulation, or recovery. The latent correlations between reactivity with initial regulation and recovery were .91 (p <.001) and .79 (p <.001), respectively. Thus, the magnitude of reactivity scores were significantly and positively related to the magnitude of initial regulation and recovery scores. In other words, individuals who showed greater amounts of change between Times 1 and 2 also showed greater amounts of change between Times 1 and 3, as well as between Times 1 and 4. Last, the latent correlation between regulation and recovery was .90 (p <.001), meaning adolescents who demonstrated greater amounts of change in cortisol levels between Times 1 and 3 also demonstrated great amounts of change between Times 1 and 4.

Conditional Models: Are Cortisol Changes in Response to the Stressor Task Concurrently Associated with SITB?

Due to the previously noted low prevalence rates of SITB over time in the present sample, recall that power was far from sufficient to examine central study hypotheses related to the prospective prediction of SITB (see Figure 4). However, it was possible to test conditional models using concurrent baseline data. It was also initially proposed that the cognitive vulnerability variables would be entered into conditional models to test whether these variables would be directly associated with SITB and, if so, whether cortisol reactivity would mediate the association between cognitive vulnerability and SITB. However, cognitive vulnerability variables were not considered in any of the forthcoming conditional

models for several reasons. First, the MANOVA and MANCOVA analyses considered above revealed little evidence of significant mean differences between measures of cognitive vulnerability according to SITB status, particularly after controlling for depressive symptoms (see Tables 5 and 6). Second, no evidence was found for any concurrent association between measures of cognitive vulnerability and cortisol (see Table 4). Third, it would not have been appropriate to conduct the proposed meditational analyses given the exclusive examination of concurrent data and in light of the fact that significant cognitive vulnerability-SITB and cognitive vulnerability-cortisol bivariate associations are necessary preconditions for conducting such analyses (see Holmbeck, 1997). Finally, reducing the number of variables entered into conditional models would increase the power to detect significant associations among variables of primary interest (i.e., changes in cortisol over time in response to the stressor task and SITB).

The results of the unconditional model outlined above demonstrated that there was significant variability in baseline levels of cortisol, as well as in cortisol reactivity, initial regulation, and recovery. The primary goal of the conditional models were to test whether this interindividual variability among baseline cortisol level, reactivity, regulation, and recovery would be concurrently associated with engagement in SITB (see Figure 5). Given the low prevalence of various individual forms of SITB reported in the present sample at baseline and the need to fully capitalize on power, the SITB variables described below were again dichotomized to represent previous engagement in any form of SITB versus the absence of prior history of any SITB. Similar to the MANCOVA analyses described above, age, pubertal status, and the duration of time between awakening and collection of the first cortisol sample were simultaneously entered in all models as covariates. Since these

covariates are known to influence overall cortisol levels, these three factors were also considered as independent predictors of baseline cortisol level to provide rigorous control. Paths were estimated between all exogenous variables and baseline cortisol level (i.e., latent intercept) and reactivity, regulation, and recovery slope factors. A total of 4 variants of this model were fit to the observed data. A summary of model description and fit indices is provided in Table 7.

The primary goal of the first conditional model was to test whether interindividual differences in baseline cortisol level, reactivity, regulation, and recovery were associated with adolescents' lifetime engagement in any form of SITB. The first conditional model fit the observed data well, $\chi^2(9) = 11.277$, NS; $\chi^2/df = 1.253$. The duration of time between awakening and the collection of the first cortisol sample (i.e., cortisol timing) emerged as the only variable significantly associated with baseline cortisol levels (i.e., latent intercept). Neither the age nor the pubertal status of participants was significantly associated with baseline cortisol levels. While none of the paths between lifetime SITB and the cortisol latent variables were significant (all *p*s > .10), potentially important trends were noted. As compared to adolescents who had never engaged in any form of SITB, adolescents who reported engaging in SITB tended to have higher baseline levels of cortisol (i.e., latent intercept) and lower levels cortisol reactivity, regulation, and recovery.

The primary goal of the second conditional model was to replicate the first while at the same time testing whether the latent cortisol variables (i.e., the four cortisol periods) were additionally associated with adolescents' depressive symptoms (see Figure 5). The inclusion of a measure of depressive symptoms into this model was considered likely to allow for the simultaneous comparison of the relative associations between depression vs. the variable

representing lifetime SITB and the latent cortisol variables. The fit of the second conditional model was good, $\chi^2(9) = 10.444$, NS; $\chi^2/df = 1.160$. In the full model, the cortisol timing variable again emerged as the only variable significantly associated with baseline cortisol levels (i.e., latent intercept). No other significant paths were observed between the latent cortisol variables and other outcome variables. However, with respect to substantive paths of interest, there was a trend whereby increased depressive symptoms were associated with elevated cortisol levels at baseline and lower cortisol reactivity, regulation and recovery. A parallel pattern emerged for the nonsignificant paths between the latent cortisol variables and lifetime SITB. However, as compared to those found in the first conditional model, the magnitude of the regression weights between lifetime SITB and the latent cortisol variables were reduced in the model including depressive symptoms.

The final two conditional models replicated the first two models described above but substituted a dichotomous measure representing the presence vs. absence of engagement in any form of SITB in the past month for the life-time measure of SITB. The third model fit the observed data well, $\chi^2(9) = 11.564$, NS; $\chi^2/df = 1.285$. The fourth model, which simultaneously tested whether depressive symptoms and past-month SITB would be associated with the latent cortisol variables, also fit the data well, $\chi^2(9) = 10.031$, NS; $\chi^2/df = 1.115$. As can be seen from Table 8, the third and fourth models yielded a pattern of result that were comparable to those found from testing the first and second models.

CHAPTER 4

DISCUSSION

SITB represent a major, worldwide public health problem (WHO, 2010). Recent epidemiological data has raised particular concern about the dramatic increase in SITB and corresponding increase in deaths by suicide observed among youths during the transition from childhood to adolescence (e.g., CDC, 2010b). Despite national and global recognition that adolescent SITB represents a critical priority for research (U.S. Department of Health and Human Services, 2001; WHO, 2010), surprisingly little is known to date about many fundamental aspects of these phenomena.

A principal limitation of the contemporary study of SITB across the age spectrum—and of adolescent SITB in particular (e.g., Wagner, 2009)—has stemmed from a primarily pragmatic approach to research. With the obvious goal of facilitating clinical recognition of individuals who may engage in SITB to better inform prevention and intervention strategies, decades of excellent research has been devoted to the empirical identification of risk factors associated with completed suicide and nonfatal SITB (e.g., Brent et al., 1993b; Lewinsohn, et al., 1994; McKeown et al., 1998; Roberts, Roberts, & Xing, 2010; Rudd et al., 2006). Although identifying risk factors is a crucial early step in explanation and prediction, there is little evidence that this reductionistic, pragmatic approach has enabled the mental health field to effectively impact the rates of SITB or even attain a better understanding of SITB in general (Kessler et al., 2005; Rogers, 2001a, 2001b, 2003). There is a pressing need for

comprehensive and developmentally sensitive theoretical models to drive research examining which specific factors and what relationships between them are in a causal pathway leading to adolescent SITB. More specifically, as Wagner (2009) surmised, "the biopsychosocial framework probably offers the most fertile ground for generating a developmental theory of suicidal behaviors" (p. 76).

The present study aimed to propose and preliminarily test one such biopsychosocial model of adolescent SITB, namely, a developmentally-specific reformulation of Abramson and colleagues' (1989, 2000) hopelessness theory of suicidality. This theoretical adaptation, which is the first of its kind, was articulated with the goal of examining whether certain cognitively mediated vulnerabilities and psychophysiological stress response profiles—either alone or in conjunction—may confer increased risk for the onset and recurrence of SITB.

Unfortunately, several characteristics of the sample utilized in the present study limited the ability to adequately examine many central study hypotheses. First, the small sample size available (i.e., overall 62 and fewer participants across baseline analyses) substantially reduced power to detect significant effects and limited the ability to conduct multivariate analyses integrating all constructs of interest. Second, although attempts were made to oversample participants from clinical referral sources given the greater prevalence of SITB in the context of diagnosed psychopathology, the majority of participants (i.e., over 75%) were ultimately recruited from normative samples, such as local high schools and mass email advertisements. As a result, the prevalence of various forms of SITB found at baseline was lower than expected and more closely resembled the rates found among community samples of high-school-aged adolescents (CDC, 2010b) than among clinical samples of adolescents at high-risk for SITB. Third, the compound problems of small sample size and

low prevalence rates of SITB led to even more precipitous declines in the number of individuals reporting SITB at each of the 3-month, 6-month, and 9-month follow-up time points. Due to the low rates of onset and maintenance of SITB over time in the present sample, it was determined that power was far from sufficient to examine central study hypotheses related to the prospective prediction of SITB. Thus, only concurrent baseline data could be utilized to examine study hypotheses.

A fourth limitation of the sample utilized in the current study may have stemmed from the unbalanced gender composition. A sample predominantly composed of adolescent females (i.e., approximately 73%) was utilized for reasons related both to practicality and theoretical fidelity. As a logical beginning to this novel line of research and in anticipation of analytic restrictions related to low power, adolescent females were oversampled due to the generally higher prevalence rates of SITB among females as compared to males during this developmental period. More importantly, females were selectively recruited given the theoretically-based decision to incorporate an in vivo social evaluative speech task to induce stress (i.e., to test the specific vulnerability hypothesis; see Abramson et al., 1989; Beck, 1967). It has been empirically demonstrated that interpersonally-themed stress poses a particular area of vulnerability for adolescent girls (Hankin et al., 2007; Rudolph & Hammen, 1999; see Rudolph, 2002 for a review).

Nonetheless, adolescent males were included in the present sample to reflect the likely reality that adolescence in general is associated with greater overall interpersonal stress exposure, irrespective of gender (e.g., Rudolph & Asher, 2000; Rudolph & Hammen, 1999). However, it is possible that inclusion of males in the full sample may have been problematic. On the one hand, a sample which included 17 male participants for whom interpersonally-

themed cognitive vulnerability and stress may not have been as theoretically relevant may have diluted the present study's ability to detect possible gender biased (i.e., female) effects. On the other hand, removing all male participants from the present analyses might unnecessarily have reduced the already low power available. Accordingly, all analyses of interest were conducted first with all available participants and subsequently with females only. (Insufficient numbers of males were available to examine any post-hoc hypotheses with respect to males.) Where any evidence of gender-specific effects for females were found, these will be discussed below.

Despite the limitations imposed by study sample size and makeup, the present investigation yielded several notable findings. The available concurrent data allowed for important—albeit preliminary—examinations of several hypotheses. First, it was anticipated that higher scores on a baseline measure of self-reported, interpersonally-relevant cognitive vulnerability (in the form of a negative inferential style for causes, consequences, and self characteristics) would be concurrently associated with the past occurrence of SITB. The results of several multiple analyses of variance (MANOVAs) appeared to provide qualified support for this hypothesis. As compared to adolescents who denied any past engagement in SITB, individuals who reported the lifetime experience of suicidal ideation combined with either suicide attempts, nonsuicidal self-injury (NSSI), or both of these behaviors had marginally significantly more negative interpersonal cognitive vulnerability. This trend became statistically significant when considering SITB dichotomously; adolescents with past histories of any SITB had more negative interpersonal inferential styles than did adolescents with no prior history of SITB. However, it is important to note that these mean group differences were no longer significant in multiple analyses of covariance (MANCOVAs)

which controlled for the effect of depressive symptoms. It appears as though adolescents engaging in past SITB tended to have more negative interpersonal inferential styles but only in concurrence with greater symptoms of depression. This pattern of results remained the same when these analyses were re-run with only adolescent females.

A generally consistent pattern of results emerged when considering group differences in cognitive vulnerability according to the past-month experience of SITB. Groups of adolescents who reported suicide ideation only in the past month, as well as those who engaged in suicidal ideation with either suicide attempts, NSSI, or both of these self-harming behaviors each had significantly more negative interpersonal vulnerability than did individuals who reported no SITB in the past month. Similar to the lifetime analyses above, these mean SITB group differences in interpersonal vulnerability were no longer significant after controlling for depressive symptoms.

Contrary to the lifetime analyses, however, past-month SITB group differences were also found with respect to achievement-related vulnerability. Specifically, individuals who experienced past-month suicidal ideation in conjunction with either suicide attempts, NSSI, or both behaviors reported having more negative achievement-related inferential styles at baseline than did adolescents denying any past-month SITB. Interestingly, these SITB group differences in achievement-related vulnerability were still marginally significant after controlling for depressive symptoms. These findings suggest that whereas interpersonal cognitive vulnerability may be more related to recent engagement in SITB in the context of acute depressive symptoms, the association between achievement-related vulnerability and recent SITB appears to be less contingent upon comorbid depressive symptoms. Additionally, considering the mean SITB group differences found for interpersonal

vulnerability but not for achievement vulnerability in the lifetime analyses, it is possible that cognitive vulnerability in general—rather than specific vulnerabilities related to interpersonal versus achievement domains—becomes more relevant as a risk factor for SITB as the experience of SITB become more recent or acute (i.e., in the past month).

Nevertheless, it is important to consider the shifting pattern of results when these past-month analyses were conducted only with adolescent females. While identical, statistically significant mean SITB group differences were found with respect to interpersonal cognitive vulnerability, the mean group differences in achievement-related vulnerability were no longer found among a sample of girls only. Thus, synthesizing across lifetime and past-month SITB analyses, findings were generally consistent with the hypothesis that adolescents who experience SITB have greater interpersonal cognitive vulnerability. As expected, it appeared that this effect particularly pertains to adolescent females. Moreover, it is unsurprising that comorbid depressive symptoms statistically account for the higher levels of interpersonal vulnerability among adolescents who engage in SITB as compared to individuals who do not. In fact, recall that Abramson and colleagues' (1989, 2000) theory of suicidality specifies that negative inferential styles render individuals more likely to first develop symptoms of (hopelessness) depression and, in turn, SITB. Thus, the results of the present study conducted with adolescents offers indirect support for the hypothesis that cognitive vulnerability may be associated with SITB, and this association may be mediated by the experience of depressive symptoms. The finding also represents important continuities with prior work with adults in this area (Abramson et al., 1998).

A second major study hypothesis predicted that those salivary cortisol levels that were higher in magnitude and maintained for longer periods of time in response to the

laboratory-based speech task would be related to greater prior engagement in SITB. No support was found for this dysregulated stress-reactivity/stress recovery hypothesis. This was despite conditional growth curve models revealing a robust, statistically significant overall cortisol response to the in vivo stressor for all individuals (regardless of SITB status). The MANCOVA analyses examining lifetime and past-month SITB, considered as both SITB subgroups and dichotomized, revealed no significant group differences in mean cortisol levels measured post-speech task (i.e., at Times 2, 3, and 4) between individuals who engaged in any prior SITB and those who had not. Similarly, the results of latent growth curve analyses using baseline data found no evidence for an association between any prior engagement in SITB and changes in baseline cortisol levels, cortisol reactivity in response to the laboratory stressor, initial regulation, or recovery.

In fact, very little support was found in the present study for the hypothesis that individuals engaging in any form of SITB exhibit aberrant cortisol profiles on any index. A potential exception lies in the results of a MANCOVA analysis (controlling for age, pubertal status, and duration of time between awakening and the collection of the first cortisol sample) that revealed a marginally significant effect in the overall lifetime SITB group. Specifically, it was found that individuals who reported engaging in lifetime NSSI (only) exhibited significantly higher mean levels of salivary cortisol as measured at Time 1 (i.e., prior to the speech task) than did individuals from the other three SITB categories (i.e., those without any previous history of SITB, individuals who reported the lifetime experience of suicide ideation, and individuals who reported the lifetime experience of ideation in conjunction with suicide attempts, NSSI, or both behaviors). This statistically significant result, which was limited to the lifetime NSSI only group, was unexpected. Given the

absence of previous research in this area, no specific, a priori hypotheses were made about the possible differential stress responses of individuals who engage in separate forms of SITB. There is an important caveat to pursuing this finding: the NSSI only group consisted of only two individuals, and no claims can be made their representativeness.

Nonetheless, it will be important for future research to address the question of whether and how individuals engaging in specific forms of SITB may differ in their stress response profiles. Further, the idea that there may be baseline or basal differences in cortisol levels between individuals who engage in SITB and those who do not is intriguing. The significant mean group differences between the NSSI only group and the other groups with respect to the first cortisol sample raises the possibility that some individuals at risk for SITB may subjectively experience coming to the laboratory as more inherently stressful in and of itself. Alternatively, perhaps some at-risk adolescents are less characterized by dysregulated responses to acute stressors than by the subjective experience of more chronic stress throughout the day. These adolescents would thus appear to have higher overall cortisol profiles. As anecdotal support for this contention, a visual inspection of the graph depicting cortisol levels over time for groups of adolescents who reported engaging in lifetime SITB versus those who did not (see Figure 6) suggests a trend whereby the SITB individuals appear to have more chronically aroused HPA axes than do non-SITB individuals. Further, as the graph suggests, it is possible that these chronically aroused individuals, already nearing the peak of HPA stress response may even appear hypo-responsive to discrete stressors.

Obviously, these explanations are purely speculative. It is unfortunate, perhaps, that the present study was explicitly designed to measure whether post-baseline *changes* in

cortisol levels in response to an acute social stressor may be associated with SITB. The possibility remains that the null findings in the present study with respect to the association between SITB and cortisol reactivity, regulation, and recovery were more an artifact of the limitations of sample number and heterogeneity. Alternatively, as suggested above, it is possible that individuals at risk for engaging in SITB demonstrate differences in HPA axis functioning that are important but difficult to detect. The existence of subtly but chronically increased cortisol levels among SITB adolescents would not be detected by the present, low-powered study.

There is some empirical support for the notion that higher basal cortisol rhythms may be associated with increased suicidality. For example, Mathew and colleagues (2003) used continuous blood sampling to study 24-hour cortisol cycles in 42 adolescents with Major Depressive Disorder (MDD) and 35 without, and examined these youths again 10 years later with the goal of predicting suicide attempts. Although cortisol levels had not distinguished those with and without depression in concurrent analyses, cortisol rhythms predicted trajectories toward suicidality. Compared to all other adolescents, the 13 youths with lifetime diagnoses of MDD who attempted suicide in the subsequent 10 years all had elevated cortisol levels in the 6 hours before sleep onset, from late afternoon through evening. They also had lower cortisol levels 2-4 hours after sleep onset when, normatively, the HPA axis would be expected to increase cortisol production. Thus, a systemic dysregulation of diurnal HPA axis activity predicted, at least in this study, subsequent suicidal behavior in young people with depression. Other studies have presented similar findings with respect to elevated basal cortisol functioning exhibited by suicidal adolescents, particularly around the sleep onset period (e.g., Dahl et al., 1991). Future research into the

HPA axis characteristics of adolescents at-risk for engaging in SITB will benefit from examination of indices of basal cortisol functioning, as well as those that measure stress reactivity.

The positive finding of the Mathew and colleagues' (2003) study also underscores other critical avenues for future research. First, and perhaps most importantly, there remains a pressing need for investigations to test biopsychosocial models of adolescent SITB using prospective, longitudinal designs. An important limitation of the past and current research is the general inability to establish the temporal precedence of study variables. Until sophisticated and large-scale longitudinal designs can be conducted, SITB's correlates, contributors, causes, and consequences cannot be distinguished from one another. Of particular importance will be study designs that establish whether potential HPA axis dyregulation precedes the development of SITB and, if so, which factors may mediate or moderate this association. Moreover, since the experience of SITB among adolescents may fluctuate rapidly over time (see Prinstein et al., 2008), such prospective work will also likely benefit from the examination of these outcomes across multiple, temporally proximal longitudinal intervals.

A second future research direction highlighted by the Mathew and colleagues' (2003) study pertains to the study sample recruited. The extant research which has found positive evidence for dysregulated HPA activity among adolescents engaging in SITB has tended to utilize more diagnostically homogenous samples. For example, both Dahl and colleagues (1991) and Mathew and colleagues (2003) utilized rigorous diagnostic procedures such as repeated, multi-informant, semistructured clinical interviews to recruit samples of clinically depressed and non-depressed adolescents. It will be important for future research to

determine whether the possible (cognitively precipitated) abnormal stress system functioning found among adolescents engaging in SITB is limited to clinically depressed individuals. Further, it is interesting that the empirical studies mentioned above focused on one particular form of SITB as their outcome measure, attempted suicide. In short, it is possible that the sample utilized in the present study—though more ecologically representative—was too heterogeneous in terms of diagnostic comorbidities and diversity of SITB forms to allow for the detection of effects that may be specific to certain types of psychopathology or forms of SITB.

A third major study hypothesis predicted that, consistent with cognitive appraisal theory (e.g., Lazarus & Folkman, 1984), more negative inferential style would be concurrently associated with increased cortisol levels in response to the stressor task. More specifically, it was predicted that adolescents with greater tendencies to make negative inferences about the causes, consequences, and self characteristics in response to hypothetical social- and achievement-related scenarios would also demonstrate increased cortisol response to an actual, in vivo stressor. (Although not directly tested, this increased stress response would have presumably emanated from the tendency of these "cognitively vulnerable" adolescents to make the same negative, and thus stress-inducing, inferences in response to the speech task.) However, the results of bivariate correlation analyses between interpersonally- and achievement-related inferential style and the four measures of salivary cortisol provided no statistical support for this hypothesis.

However, it is intriguing that the magnitudes of correlations between the cognitive vulnerability and cortisol variables are generally comparable to (and in most cases exceeds) those of the correlations between depressive symptoms and the cortisol samples. These latter

set of correlations also failed to reach statistical significance in the present study, despite generally consistent empirical evidence which supports the tendency for depressed adolescents to exhibited higher baseline cortisol values and overactive responses to psychological stressors (for reviews, see Guerry & Hastings, 2011; Lopez-Duran, Kovacs, & George, 2009). Again, to the extent that power was insufficient in the current study to detect weak correlations between cortisol levels and depression, it is possible that the same Type II error occurred in failing to detect a correlation between cognitive vulnerability and cortisol stress response.

Null findings notwithstanding, there was also no evidence of a great concurrent association between negative interpersonally-oriented (as opposed to achievement-related) inferential style and the four cortisol measures. This finding was also contrary to hypotheses. It was predicted that a particular cognitive vulnerability for interpersonally-themed stressors would confer heightened risk for subsequent HPA axis dysregulation following the social stress (i.e., speech) task. This null finding was unexpected given prior work in this area supporting the specific vulnerability hypothesis (e.g., Joiner & Rudd, 1995). To review, this important corollary of hopelessness theory specifies that in order for core symptoms of hopelessness depression to emerge from a cognitive vulnerability-stress interaction (e.g., the experience of distress, suicidal thoughts and behaviors, etc.), there needs to be congruence between the content area(s) of an individual's negative inferential style (e.g., an achievement-related vulnerability vs. an interpersonal vulnerability) and the stressor he or she experiences (e.g., a failing exam grade vs. a break-up of a romantic relationship, respectively).

It is possible that while the speech task used in the present study was experienced as stressful overall, it was not subjectively interpreted by adolescent participants as falling within the social domain. Recall that the explicit goal of the speech task, as explained to participants, was to convince a hypothetical audience of their peers that they should be selected to star in a fictional television show about teens' ability to form and maintain friendships. Some have argued (e.g., Stroud, Tanofsky-Kraff, Wilfley, & Salovey, 2000) that speech tasks of this kind which incorporate either silent or implied audiences elicit performance-related distress, which are more accurately characterized as related to goal-directed or achievement-related domains. Stroud and colleagues (2000) content that in order for laboratory paradigms to truly induce "interpersonal stress" these must involve direct social interactions as the primary means of inducing distress. Accordingly, it is possible that the speech task utilized in the present study, as well as many others (e.g., Hastings et al., 2007; Klimes-Dougan et al., 2001), was more generally stressful than interpersonally so. Future research in this area may benefit from the incorporation of more explicitly *social* stressors such as live rejection paradigms (e.g., the Yale Interpersonal Stressor; Stroud et al., 2000).

A final, untested hypothesis in the current study related to whether dysregulated stress responses might mediate the association between cognitive vulnerability and trajectories of SITB over time (see Figure 2). As discussed previously, this hypothesis could not be examined for several reasons. First, the results of several MANOVA and MANCOVA analyses revealed little evidence of significant mean differences either in measures of cognitive vulnerability or cortisol according to SITB status. Second, no evidence was found for any concurrent association between measures of cognitive vulnerability and cortisol.

Third, it would have been inappropriate to conduct meditational analyses given the exclusive examination of concurrent data and in light of the fact that significant cognitive vulnerability-SITB and cognitive vulnerability-cortisol bivariate associations are necessary preconditions for conducting such analyses (see Holmbeck, 1997). Finally, the low power available to examine conditional growth curve models in the present study precluded the simultaneous examination of the cognitive vulnerability variables with constructs of primary interest (i.e., changes in cortisol over time in response to the stressor task and SITB). Thus, this important tenet of the present biopsychosocial theory of SITB remains to be examined by future longitudinal investigations with adequate sample sizes.

In summary, limitations with respect to the sample size and characteristics of the present study precluded adequate examination of the proposed biopsychosocial model of adolescent SITB. Future work in this area will benefit from addressing these and other major limitations. First, potential investigations should utilize a large, clinical sample of adolescents at high-risk for engaging in SITB. Second, preliminary examinations of this model should either exclusively sample adolescent females or, if resources allow, recruit an adequate number of males to more fully examine differential hypotheses with respect to gender. The possibility remains that boys' and girls' respective tendencies to have greater achievement- and interpersonally-oriented cognitive vulnerabilities may potentiate different physiological stress response profiles to corresponding domains of stressors. These theoretically gendered pathways to the same overwhelming experience of negative affect could, in turn, contribute to the onset or recurrence of SITB. Third, and relatedly, particular care should be taken in the selection of appropriate laboratory stressor task to ensure that "interpersonal" and "achievement" stressors explicitly (and exclusively) tap into these

domains of vulnerability. Fourth, future work will benefit from the inclusion of alternative or additional measures of HPA axis system functioning to more completely capture the acute stress responsiveness and basal cortisol profiles which might confer proximal vulnerability to the engagement in SITB. Lastly, a central failing of this and other extant research into adolescent SITB is the paucity of longitudinal designs. The establishment of temporal precedence among theoretically-determined risk factors and the testing of potential mediator and moderator influences are essential for the development of any cogent biopsychosocial model of adolescent SITB.

Table 1

Descriptive Statistics for SITB Variables

	Past Month	Lifetime
	N (%) reporting SITB	N (%) reporting SITB
Suicide Ideation		
Baseline (*n* = 62)	12 (19.4)	23 (37.1)
3 Months (*n* = 55)	5 (9.1)	
6 Months (*n* = 41)	1 (2.4)	
9 Months (*n* = 38)	3 (7.9)	
Attempts		
Baseline (*n* = 62)	5 (8.1)	9 (14.5)
3 Months (*n* = 54)	0 (0)	
6 Months (*n* = 43)	1 (2.3)	
9 Months (*n* = 38)	1 (2.6)	
NSSI		
Baseline (*n* = 62)	5 (8.1)	14 (22.6)
3 Months (*n* = 54)	3 (5.6)	
6 Months (*n* = 43)	2 (4.7)	
9 Months (*n* = 38)	1 (2.6)	
SITB composite [a]		
Baseline (*n* = 62)	13 (21.0)	27 (43.5)
SIB composite [b]		
Baseline (*n* = 62)	9 (14.5)	16 (25.8)

Note. SITB = Self-injurious thoughts and behaviors; NSSI = Nonsuicidal self-injury; SIB = Self-injurious behaviors.

[a] Those endorsing suicidal ideation, suicide attempts, or NSSI. [b] Those endorsing self-injurious behaviors only (i.e., suicide attempts and NSSI).

Table 2

Descriptive Statistics for Conceptually-based SITB Categorical Variables

	Past Month	Lifetime
	N (%) reporting SITB	N (%) reporting SITB
	(*n* = 60)	(*n* = 62)
No SITB	47 (78.3%)	35 (56.5%)
Ideation Only	4 (6.7%)	11 (17.7%)
Ideation with either Attempts, NSSI, or Both	8 (13.3%)	12 (19.4%)
NSSI Only	1 (1.7%)	4 (6.5%)
No SITB	47 (78.3%)	35 (56.5%)
Any SITB	13 (21.7%)	27 (43.5%)

Note. SITB = Self-injurious thoughts and behaviors; NSSI = Nonsuicidal self-injury.

Table 3

Descriptive Statistics for Independent Variables and Covariates

	N	*M*	*SD*	*Skew*	*Kurtosis*
Cortisol					
Time 1	62	.09	.05	.64	-.31
Time 2	62	.12	.09	1.43	2.29
Time 3	61	.11	.08	1.80	5.23
Time 4	61	.10	.06	1.54	4.31
Ln-Time 1	62	-1.11	.25	-.45	-.30
Ln-Time 2	62	-1.02	.33	-.35	-.02
Ln-Time 3	61	-1.07	.31	-.36	-.10
Ln-Time 4	61	-1.09	.29	-.52	.03
Cognitive Vulnerability					
Interpersonal	61	3.02	.94	.85	2.46
Achievement	61	3.46	1.01	.06	.85
Covariates					
Depression	62	.47	.47	1.46	1.28
Pubertal Status	56	4.04	.81	-.71	.37
Age	62	14.70	1.33	-.38	-1.12
Cortisol timing	59	7:29	3:05	.42	-.73

Note. Time 1 = measurement of salivary cortisol pre-speech task; Time 2 = measurement of salivary cortisol 20 minutes post-speech task; Time 3 = measurement of salivary cortisol 30 minutes post-speech task; Time 4 = measurement of salivary cortisol 40 minutes post-speech task; Ln-Time 1, Ln-Time 2, Ln-Time 3, Ln-Time 4 = Log-transformed values of salivary cortisol measures; Interpersonal = composite average of interpersonal vulnerability on the Adolescent Cognitive Style Questionnaire; Achievement = composite average of achievement vulnerability on the Adolescent Cognitive Style Questionnaire; Depression = Depressive

symptoms as assessed by average scores on the Mood and Feelings Questionnaire; Pubertal Status = Self-reported pubertal stage as measured by the Udry questionnaire; Age = Age of participant; Cortisol Timing = Duration of time elapsed between self-reported time of awakening and the collection time of the first cortisol sample.

Table 4

Pearson Correlations among Continuous Study Variables

Variable	N	Achievement	Cortisol Sample 1	Cortisol Sample 2	Cortisol Sample 3	Cortisol Sample 4	Depression	Pubertal Status	Awake Time	Age
Inferential Style										
Interpersonal	61	.74***	.21	.13	.14	.17	.41**	.11	-.03	.07
Achievement	61	--	.18	.14	.17	.17	.17	.21	.02	.07
Cortisol – Time 1	62		--	.75***	.73***	.74***	.15	.06	-.38**	.07
Time 2	62			--	.95***	.89***	.06	.07	-.46***	.15
Time 3	61				--	.95***	.10	.09	-.47***	.15
Time 4	61					--	.18	.11	-.51***	.20
Depression	62						--	.03	.13	-.16
Pubertal Status	56							--	-.01	.65***
Cortisol Timing	59								--	-.06
Age	62									--

Note. Shaded cells indicate partial correlations reported among cortisol sample values and other study variables after controlling for age, pubertal status, and duration of time between awakening and collection of cortisol sample 1 (i.e., "cortisol timing"). Interpersonal = composite average of interpersonal vulnerability on the Adolescent Cognitive Style Questionnaire; Achievement = composite average of achievement vulnerability on the Adolescent Cognitive Style Questionnaire; Time 1 = measurement of salivary cortisol pre-speech task; Time 2 = measurement of salivary cortisol 20 minutes post-speech task; Time 3 = measurement of salivary cortisol 30 minutes post-speech task; Time 4 = measurement of salivary cortisol 40 minutes post-speech task; Depression = Depressive symptoms as assessed by average scores on the Mood and Feelings Questionnaire; Pubertal Status = Self-reported pubertal stage as measured by the Udry questionnaire; Cortisol Timing = Duration of time elapsed between self-reported time of awakening and the collection time of the first cortisol sample; Age = Age of participant.
**p < .01; ***p < .001.

Table 5

Means (Standard Errors) from MANOVA and MANCOVA Analyses – Lifetime SITB

	SITB Categories					Dichotomized Sample		
Variable	No SITB	Ideation Only	Ideation with either Attempts, NSSI, or Both	NSSI Only	Statistic	No SITB	SITB	Statistic
ACSQ	(*n* = 34)	(*n* = 11)	(*n* = 12)	(*n* = 4)		(*n* = 34)	(*n* = 27)	
Interpersonal	2.80 (.16) [a]	3.01 (.28)	3.61 (.26) [a]	3.12 (.46)	F(3,57) = 2.31[†]	2.80 (.16)	3.29 (.18)	F(1, 59) = 4.27*
Achievement	3.49 (.17)	2.92 (.30)	3.94 (.28)	3.34 (.49)	F(3, 57) = 2.10	3.49 (.18)	3.44 (.20)	F(1,59) = 0.40
ACSQ - Adjusted Means [d]								
Interpersonal	2.95 (.17)	2.94 (.27)	3.25 (.30)	3.14 (.44)	F(3, 56) = .29	2.96 (.16)	3.09 (.19)	F(1, 58) = .23
Achievement	3.55 (.19)	2.89 (.30)	3.14 (.44)	3.35 (.49)	F(3, 56) = 1.76	3.60 (.19)	3.29 (.21)	F(1, 58) = 1.06
Log - Cortisol [e]	(*n* = 32)	(*n* = 10)	(*n* = 8)	(*n* = 2)		(*n* = 32)	(*n* = 20)	
Sample 1	-1.17 (.04) [a]	-1.10 (.08) [c]	-1.22 (.09) [b]	-.76 (.17) [a b c]	F(3, 45) = 2.30 [†]	-1.17 (.04)	-1.11 (.05)	F(1, 47) = .76
Sample 2	-1.05 (.05)	-.98 (.10)	-1.15 (.11)	-.83 (.21)	F(3, 45) = .76	-1.05 (.05)	-1.03 (.07)	F(1, 47) =.05
Sample 3	-1.09 (.05)	-1.04 (.09)	-1.19 (.11)	-.93 (.20)	F(3, 45) = .58	-1.09 (.05)	-1.08 (.06)	F(1, 47) = .01
Sample 4	-1.12 (.04)	-1.08 (.08)	-1.17 (.09)	-.90 (.18)	F(3, 45) = .72	-1.12 (.04)	-1.10 (.06)	F(1, 47) = .13

[a, b] Denote significant pairwise mean difference ($p < .05$). [c] Denotes marginally significant pairwise mean difference ($p < .10$). [d] Controlling for depressive symptoms (i.e., mean MFQ scores). [e] Statistics reported after entering age, pubertal status, and duration of time between awakening and collection of cortisol sample #1 as covariates.

[†] $p < .10$ * $p < .05$

Table 6

Means (Standard Errors) from MANOVA and MANCOVA Analyses – Past Month SITB

	SITB Categories				Dichotomized Sample		
Variable	No SITB	Ideation Only	Ideation with either Attempts, NSSI, or both	Statistic	No SITB	SITB	Statistic
ACSQ	(*n* = 46)	(*n* = 4)	(*n* = 8)		(*n* = 46)	(*n* = 13)	
Interpersonal	2.78 (.13) [a b]	3.73 (.44) [b]	3.73 (.31) [a]	F(2, 55) = 5.70**	2.78 (.13)	3.71 (.24)	F(1, 57) =12.02**
Achievement	3.30 (.14) [a]	3.26 (.49)	4.32 (.34) [a]	F(2, 55) = 3.84*	3.30 (.15)	3.99 (.27)	F(1, 57) = 4.99*
ACSQ – Adjusted Means [c]							
Interpersonal	2.87 (.14)	3.48 (.46)	3.37 (.39)	F(2, 54) = .99	2.86 (.14)	3.43 (.30)	F(1, 56) = 2.46
Achievement	3.27 (.16) [a]	3.32 (.53) [b]	4.41 (.45) [a b]	F(2, 54) = 2.79†	3.29 (.16)	4.02 (.35)	F(1, 56) = 2.97†
Log - Cortisol [d]	(*n* = 43)	(*n* = 3)	(*n* = 5)		(*n* = 43)	(*n* = 9)	
Sample 1	-1.16 (.04)	-1.04 (.14)	-1.18 (.12)	F(2, 45) = .35	-1.16 (.04)	-1.09 (.09)	F(1, 47) = .46
Sample 2	-1.04 (.05)	-.88 (.17)	-1.18 (.15)	F(2, 45) = .94	-1.04 (.05)	-1.06 (.11)	F(1, 47) = .03
Sample 3	-1.09 (.04)	-.95 (.16)	-1.22 (.14)	F(2, 45) = .91	-1.09 (.04)	-1.10 (.10)	F(1, 47) = .02
Sample 4	-1.12 (.04)	-.97 (.14)	-1.20 (.12)	F(2, 45) = .80	-1.12 (.04)	-1.08 (.09)	F(1, 47) = .18

[a] Denotes significant pairwise mean difference ($p < 05$). [b] Denotes marginally significant pairwise mean difference ($p < .10$). [c] Controlling for depressive symptoms (i.e., mean MFQ scores). [d] Statistics reported after entering age, pubertal status, and duration of time between awakening and collection of cortisol sample #1 as covariates.

† $p < .10$ * $p < .05$ ** $p < .01$

Table 7

General SEM Model Fit and Description

Model Descriptions	χ^2	*df*	χ^2/df	*p*	CFI	NFI	RMSEA	AIC
1. Lifetime SITB	11.277	9	1.253	.257	.994	.974	.064	81.277
2. Lifetime SITB, MFQ	10.444	9	1.160	.316	.997	.977	.051	100.444
3. Past month SITB	11.564	9	1.285	.239	.994	.974	.068	81.564
4. Past month SITB, MFQ	10.031	9	1.115	.348	.998	.979	.043	100.031

Note. $N = 62$ for all models. SITB = Self-injurious thoughts and behaviors; MFQ = Depressive symptoms as assessed by average scores on the Mood and Feelings Questionnaire.

Table 8

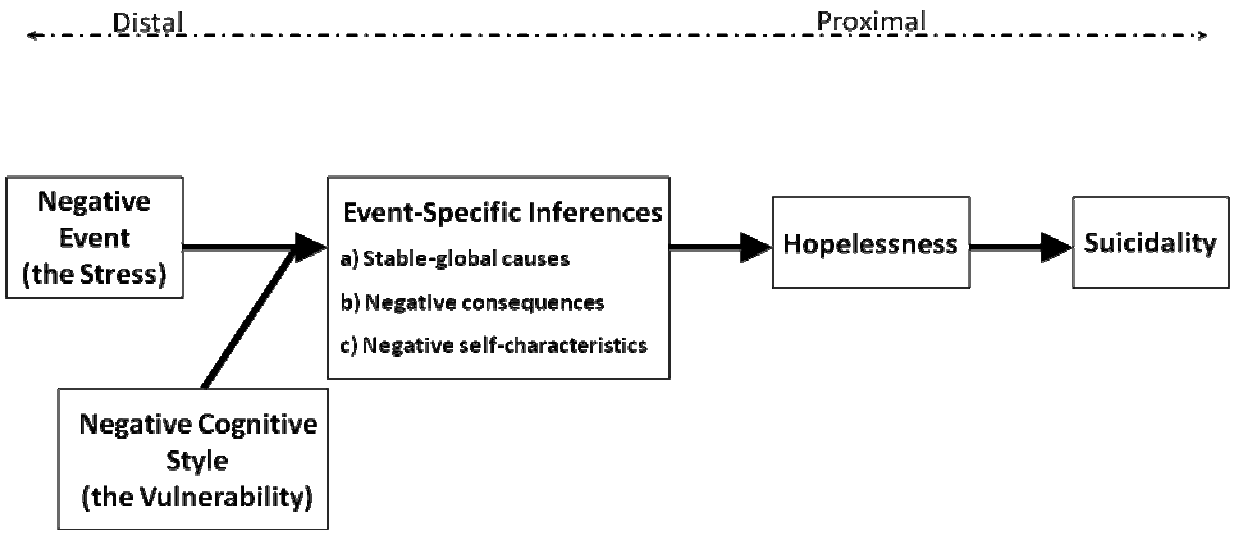
Association between cortisol and exogenous variables; ***Standardized Regression Weights****, Unstandardized Regression Weights (and Standard Errors)*

	Cortisol			
	Intercept	Reactivity Slope	Regulation Slope	Recovery Slope
Model #1				
Lifetime SITB	***.17***, .09 (.06)	***-.13***, -.05 (.05)	***-.15***, -.06 (.05)	***-.16***, -.06 (.05)
Cortisol Timing	***-.42***, .00 (.00)*			
Age	***.04***, .01 (.03)			
Pubertal Status	***.06***, .02 (.05)			
Model #2				
Lifetime SITB	***.09***, .05 (.07)	***-.03***, -.01 (.06)	***-.07***, -.03 (.06)	***-.08***, -.03 (.06)
Depressive Symptoms	***.17***, .09 (.08)	***-.19***, -.08 (.07)	***-.15***, -.06 (.06)	***-.15***, -.06 (.06)
Cortisol Timing	***-.43***, .00 (.00)*			
Age	***.09***, .02 (.03)			
Pubertal Status	***.02***, .01 (.05)			
Model #3				
Past-month SITB	***.15***, .09 (.08)	***-.08***, -.04 (.07)	***-.07***, -.04 (.06)	***-.06***, -.03 (.06)
Cortisol Timing	***-.42***, .00 (.00)*			
Age	***.11***, .02 (.03)			
Pubertal Status	***-.02***, -.01 (.05)			
Model #4				
Past-month SITB	***.07***, .04 (.11)	***.07***, .03 (.09)	***.02***, .01 (.08)	***.07***, .03 (.08)
Depressive Symptoms	***.17***, .09 (.09)	***-.25***, -.11 (.08)	***-.21***, -.09 (.08)	***-.25***, -.10 (.07)
Cortisol Timing	***-.43***, .00 (.00)*			
Age	***.11***, .02 (.03)			
Pubertal Status	***-.01***, .00 (.03)			

Note. SITB = Self-injurious thoughts and behaviors; Depression = Depressive symptoms as assessed by average scores on the Mood and Feelings Questionnaire; Cortisol Timing = Duration of time elapsed between self-reported time of awakening and the collection time of the first cortisol sample; Age = Age of participant; Pubertal Status = Self-reported pubertal stage as measured by the Udry questionnaire.
*$p < .001$.

Figure 1

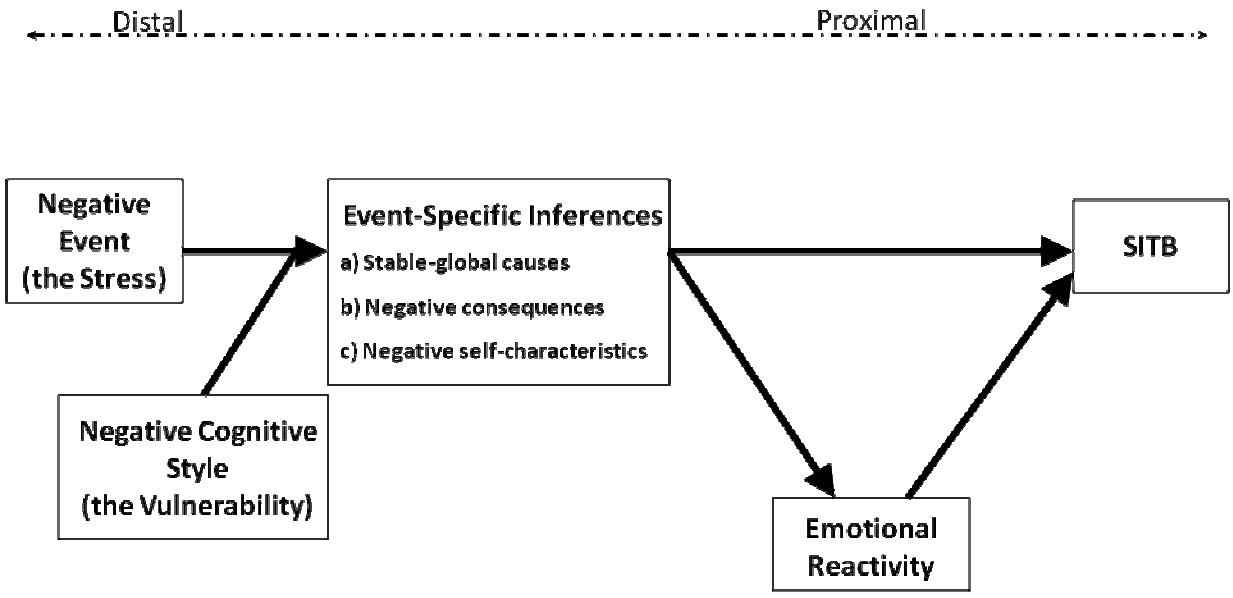
The Hopelessness Theory of Suicidality



Note. Adapted from Abramson and colleagues (2000).

Figure 2

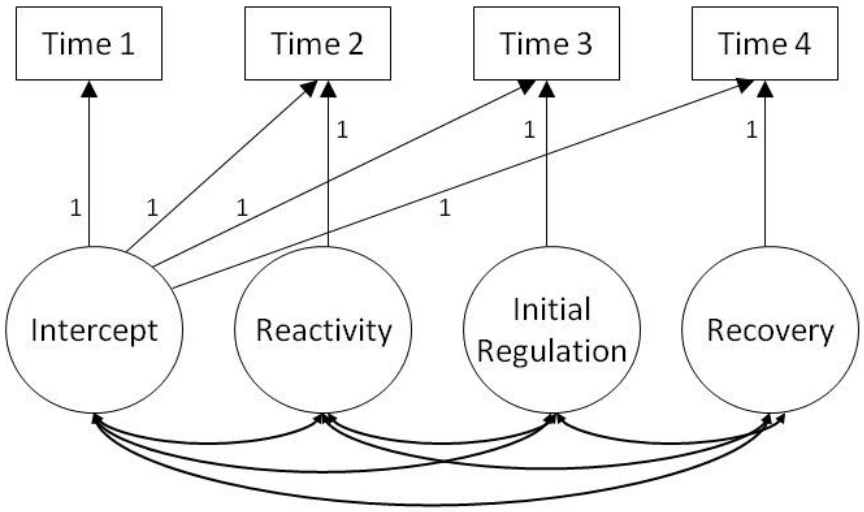
The Proposed Biopsychosocial Model of Adolescent SITB



Note. SITB = Self-injurious thoughts and behaviors.

Figure 3

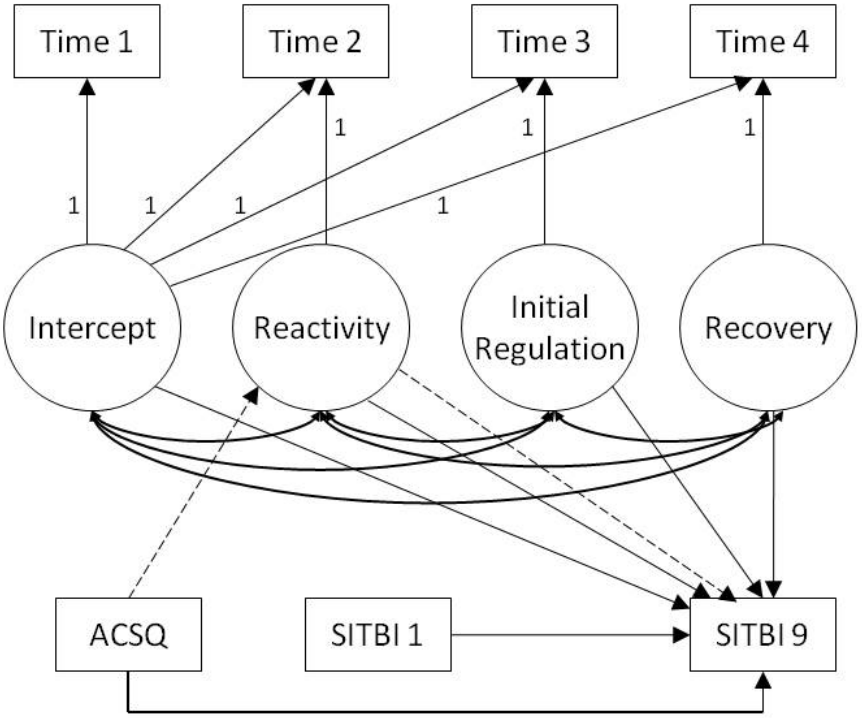
Unconditional Model



Note. Time 1 = measurement of salivary cortisol pre-speech task; Time 2 = measurement of salivary cortisol 20 minutes post-speech task; Time 3 = measurement of salivary cortisol 30 minutes post-speech task; Time 4 = measurement of salivary cortisol 40 minutes post-speech task.

Figure 4

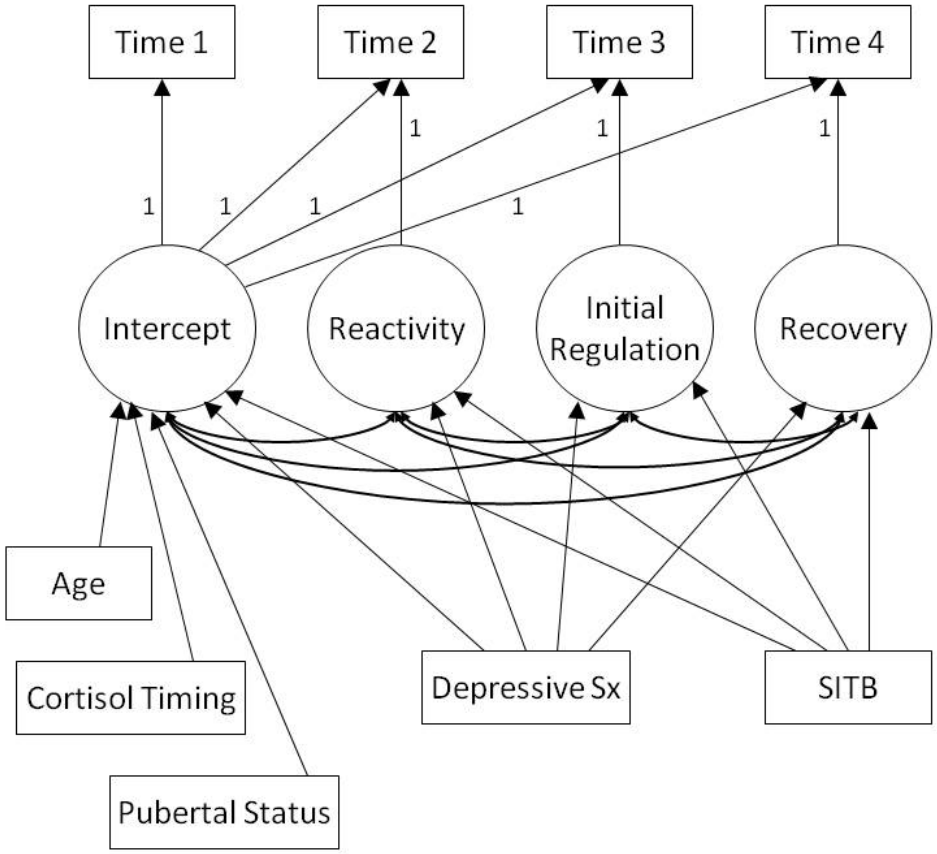
Proposed Conditional Model



Note. ACSQ = Attributional Style Questionnaire; SITBI 1 = Self-Injurious Thoughts and Behaviors Inventory – baseline measure; SITBI 9 = Self-Injurious Thoughts and Behaviors Inventory – 9-month follow-up measure; Time 1 = measurement of salivary cortisol pre-speech task; Time 2 = measurement of salivary cortisol 20 minutes post-speech task; Time 3 = measurement of salivary cortisol 30 minutes post-speech task. Time 4 = measurement of salivary cortisol 40 minutes post-speech task.

Figure 5

Tested Conditional Model

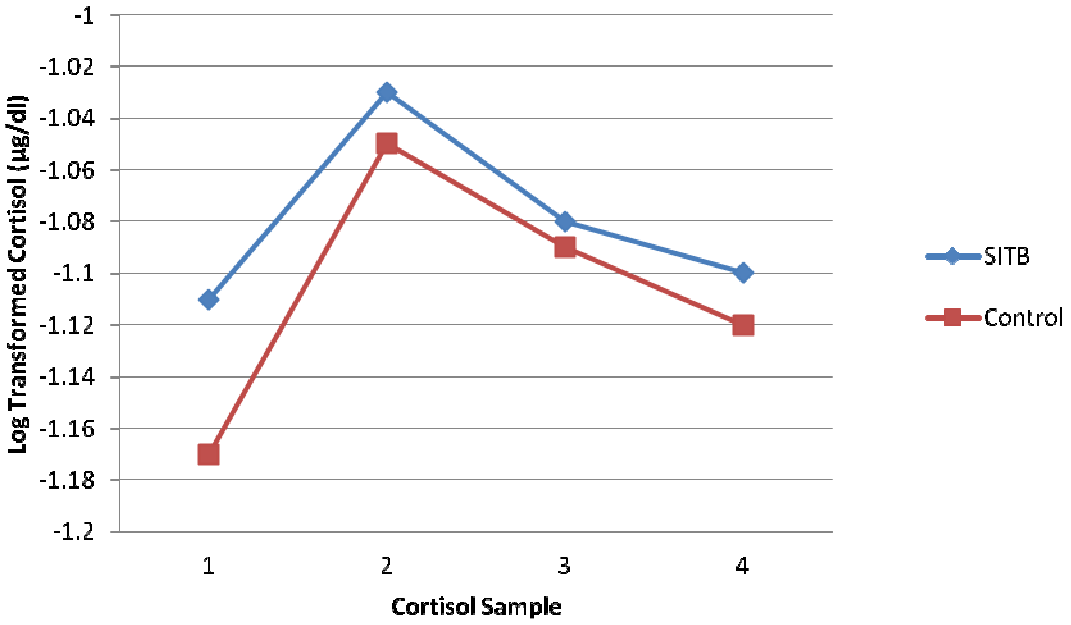


All predictors allowed to covary

Note. Time 1 = measurement of salivary cortisol pre-speech task; Time 2 = measurement of salivary cortisol 20 minutes post-speech task; Time 3 = measurement of salivary cortisol 40 minutes post-speech task; Time 4 = measurement of salivary cortisol 40 minutes post-speech task; Age = Age of participant; Cortisol Timing = Duration of time elapsed between self-reported time of awakening and the collection time of the first cortisol sample; Pubertal Status = Self-reported pubertal stage as measured by the Udry questionnaire; Depressive Sx = Depressive symptoms as assessed by average scores on the Mood and Feelings Questionnaire; SITB = The presence versus absence of self-injurious thoughts and behaviors (past-month or lifetime) as measured by the Self-Injurious Thoughts and Behaviors Inventory.

Figure 6

Adjusted Mean Cortisol Values for Dichotomized Study Groups - Lifetime SITB, controlling for cortisol timing, age, and pubertal status



Note. Adjusted means controlling for age, pubertal status, and duration of time between awakening and collection of the first cortisol sample. SITB = group of individuals reporting lifetime engagement in self-injurious thoughts and behaviors; Control = group of individuals reporting no prior history of self-injurious thoughts and behaviors; 1 = measurement of salivary cortisol pre-speech task; 2 = measurement of salivary cortisol 20 minutes post-speech task; 3 = measurement of salivary cortisol 30 minutes post-speech task. 4 = measurement of salivary cortisol 40 minutes post-speech task.

REFERENCES

Abramson, L. Y., Alloy, L. B., Hogan, M. E., Whitehouse, W. G., Cornette, M., Akhavan, S., & Chiara, A. (1998). Suicidality and cognitive vulnerability to depression among college students: A prospective study. *Journal of Adolescence, 21*, 473-487.

Abramson, L. Y., Alloy, L. B., Hogan, M. E., Whitehouse, W. G., Gibb, B. E., Hankin, B. L., & Cornette, M. M. (2000). The hopelessness theory of suicidality. In T. Joiner & M. D. Rudd (Eds.), *Suicide Science: Expanding the boundaries* (pp. 17-32). Norwell, Massachusetts: Kluwer Academic Publishers.

Abramson, L. Y., Metalsky, G. I., & Alloy, L. B. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological Review, 96*, 358-372.

Adam, E. K., Sutton, J. M., Doane, L. D., & Mineka, S. (2008). Incorporating hypothalimic-pituitary-adrenal axis measures into prentative interventions for adolescent depression: Are we there yet? *Development and Psychopathology, 20*, 975-1001.

Alloy, L. B., & Abramson, L. Y. (1999). The Temple-Wisconsin Cognitive Vulnerability to Depression Project (CVD): Conceptual background, design, and methods. *Journal of Cognitive Psychotherapy: An International Quarterly, 13*, 227-262.

Angold, A. (1989). Structured assessments of psychopathology in children and adolescents. In C. Thompson (Ed.), *The intstruments of psychiatric research* (pp. 271-304). New York: Wiley.

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, stategic, and statistical considerations. *Journal of Personality and Social Psychology, 51*, 1173-1183.

Beck, A. T. (1967). *Depression: Clinical, experimental, and theoretical aspects*. New York: Harper & Row.

Beck, A. T., Kovacs, M., & Weissman, A. (1975). Hopelessness and suicidal behavior: An overview. *Journal of the American Medical Association, 234*, 1146-1149.

Beck, A. T., Steer, R. A., Kovacs, M., & Garrison, B. (1985). Hopelessness and eventual suicide: A 10-year prospective study of patients hospitalized with suicdal ideation. *American Journal of Psychiatry, 142*, 559-563.

Bhugra, D., Thompson N., Singh, J., & Fellow-Smith, E. (2003). Inception rates of deliberate self-harm among adolescents in West London. *International Journal of Social Psychiatry, 49*, 247-250.

Blakemore, S., & Choudhury, S. (2006). Development of the adolescent brain: Implications for the executive function and social cognition. *Journal of Child Psychology and Psychiatry, 47*, 296-312.

Boergers, J., Spirito, A., & Donaldson, D. (1998). Reasons for adolescent suicide attempts: Associations with psychological functioning. *Journal of the American Academy of Child and Adolescent Psychiatry, 37*, 1287-1293.

Bollen, K. A., & Curran, P. J. (2006). *Latent curve models. A structural equation perspective*. Hoboken, NJ: Wiley.

Brent, D. A., Kolko, D. J., Wartella, M. E., Boylan, M. B., Moritz, G., Baugher, M., & Zelenak, J. P. (1993a). Adolescent psychiatric inpatients' risk of suicide attempt at 6-month follow-up. *Journal of the American Academy of Child and Adolescent Psychiatry, 32*, 95-105.

Brent, D. A., Perper, J. A., Moritz, G., Allman, C., Friend, A., Roth, C., Schweers, J., Balach, L., & Baugher, M. (1993b). Psychiatric risk factors for adolescent suicide: A case control study. *Journal of the American Academy of Child and Adolescent Psychiatry, 32*, 521-529.

Brooks-Gunn, J., Warren, M. P., Rosso, J., & Gargiulo, J. (1987). Validity of self-report measures of girls' pubertal status. *Child Development, 58*, 829-841.

Brown, M. Z., Comtois, K. A., & Linehan, M. M. (2002). Reasons for suicide attempts and nonsuicidal self-injury in women with borderline personality disorder. *Journal of Abnormal Psychology, 111*, 198-202.

Cavanagh, J. T., Carson, A. J., Sharpe, M., & Lawrie, S. M. (2003). Psychological autopsy studies of suicide: A systematic review. *Psychological Medicine, 33*, 395-405.

Center for Disease Control and Prevention (2010a). Web-based Injury Statistics Query and Reporting System (WISQRS) - United States 2007. Retrieved 2010 July 14, from http://www.cdc.gov/violenceprevention/suicide/statistics/aag.html

Center for Disease Control and Prevention (2010b). Youth Risk Behavior Surveillance System. *Morbidity and Mortality Weekly Report*. http://www.cdc.gov/mmwr/pdf/ss/ss5905.pdf

Chapman, A. L., Gratz, K. L., & Brown, M. Z. (2006). Solving the puzzle of deliberate self-harm: The experiential avoidance model. *Behavior Research and Therapy, 44*, 371-394.

Chida, Y., & Steptoe, A. (2009). Cortisol awakening response and psychosocial factors: A systematic review and meta-analysis. *Biological Psychology, 80*, 265-278.

Claes, L., Muehlenkamp, J., Vandereycken, Hamelinck, L., Martens, H., & Claes, S. (2010). Comparison of non-suicidal self-injurious behavior and suicide attempts in patients admitted to a psychiatric crisis unit. *Personality and Individual Differences, 48,* 83-87.

Cole, D. A., & Turner, J. E. Jr (1993). Models of cognitive mediation and moderation in child depression. *Journal of Abnormal Psychology, 102,* 271-281.

Cornette, M. M., Abramson, L. Y., & Bardone, A. M. (2000). Toward an integrated theory of suicidal behaviors: Merging the hopelessness, self-discrepancy, and escape theories. In T. Joiner & M. D. Rudd (Eds.), *Suicide Science: Expanding the boundaries* (pp. 43-66). Norwell, Massachusetts: Kluwer Academic Publishers.

Costello, E., & Angold, A. (1988). Scales to assess child and adolescent depression. *Journal of the American Academy of Child and Adolescent Psychiatry, 27,* 726-737.

Coyne, J. C., & Whiffen, V. E. (1995). Issues in personality as diathesis for depression: The case of sociotropy-dependency and autonomy-self-criticism. *Psychological Bulletin, 108,* 358-378.

Crowell, S. E., Beauchaine, T. P., McCauley, E., Smith, C. J., Stevens, A. L., & Sylvers, P. (2005). Psychological, autonomic, and serotonergic correlates of parasuicide among adolescent girls. *Development and Psychopathology, 17,* 1105-1127.

Crowell, S. E., Beauchaine, T. P., McCauley, E., Smith, C. J., Vasilev, C. A., & Stevens, A.L. (2008). Parent-child interactions, peripheral serotonin, and self-inflicted injury in adolescents. *Journal of Consulting and Clinical Psychology, 76,* 15-21.

Dahl, R. E., Ryan, N. D., Puig-Antich, J., Nguyen, N. A., Al-Shabbout, M., Meyer, V. A., & Perel, J. (1991). 24-hour cortisol measures in adolescents with major depression: A controlled study. *Biological Psychiatry, 30,* 25-36.

Denson, T. F., Spanovic, M., & Miller, N. (2009). Cognitive appraisals and emotions predict cortisol and immune responses: A meta-analysis of acute laboratory social stressors and emotion inductions. *Psychological Bulletin, 135,* 823-853.

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol response: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130,* 355-391.

Dorn, L. D., Susman, E. J., Nottelmann, E. D., Inoff-Germain, G., & Chrousos, G. P. (1990). Perceptions of puberty: Adolescent, parent, and health care personnel. *Developmental Psychology, 26,* 322-329.

Ellis, T. E. (2006). *Cognition and suicide: Theory, research, and therapy.* Washington, D.C.: American Psychological Association.

Ellis, T. E., & Rutherford, B. (2008). Cognition and suicide: Two decades or progress. *International Journal of Cognitive Therapy, 1*, 47-68.

Favazza, A. R., Conterio, K. (1988). The plight of chronic self-mutilators. *Community Mental Health Journal, 24*, 22-30.

Favazza, A. R., DeRosear, L., & Conterio, K. (1989). Self-mutilation and eating disorders. *Suicide & Life-Threatening Behavior, 19*, 352-261.

Frijda, N. H. (1988). The laws of emotion. *American Psychologist, 43*, 349-358.

Ge, X., Lorenz, F. O., Conger, R. D., Elder, G. H., & Simons, R. L. (1994). Trajectories of stressful life events and depressive symptoms during adolescence. *Developmental Psychology, 30*, 467-483.

Guerry, J. D., & Hastings, P. D. (2011). In search of HPA axis dysregulation in child and adolescent depression. *Clinical Child and Family Psychology Review, 14*, 135-160.

Gunnar M. & Quevedo K. (2007). The neurobiology of stress and development. *Annual Review of Psychology, 58*, 145–173.

Gunnar, M. R., Talge, N. M., & Herrera, A. (2009a). Stressor paradigms in developmental studies: What does and does not work to produce mean increases in salivary cortisol. *Psychoneuroendocrinology, 34*, 953-967.

Gunnar, M. G., Wewerka, S., Frenn, K., Long, J. D., & Griggs, C. (2009b). Developmental changes in hypothalamus-pituitary-adrenal activity over the transition to adolescence: Normative changes and associations with puberty. *Development and Psychopathology, 21*, 69-85.

Grumbach, M. M. (2002). The neuroendocrinology of human puberty revisited. *Hormone Research, 57*, 2-14.

Hankin, B. L. (2008). Cognitive vulnerability-stress model of depression during adolescence: Investigating depressive symptom specificity in a multi-wave prospective study. *Journal of Abnormal Child Psychology, 36*, 999-1014.

Hankin, B. L., & Abramson, L. Y. (2001). Development of gender differences in depression: An elaborated cognitive vulnerability-transactional stress theory. *Psychological Bulletin, 127*, 773-796.

Hankin, B. L., Badanes, L. S., Abela, J. R. Z., & Watamura, S. E. (2010). Hypothalamic-pituitary-adrenal axis dysregulation in dysphoric children and adolescents: Cortisol reactivity to psychosocial stress from preschool through middle adolescence. *Biological Psychiatry, 68*, 484-490.

Hankin, B. L., Mermelstein, R., & Roesch, L. (2007). Sex differences in adolescent depression: Stress exposure and reactivity models. *Child Development, 78*, 279-295.

Hastings, P. D., Zahn-Waxler, C., & Usher, B. (2007). Cardiovascular and affective reactions to social stress in adolescents with internalizing and externalizing problems. *International Journal of Behavioral Development, 31*, 1-11.

Hawton, K., Cole, D., O'Grady, J., & Osborn, M. (1982). Motivational aspects of deliberate self-poisoning in adolescents. *The British Journal of Psychiatry, 141*, 286-291.

Hilt, L. M., Nock, M. K., Lloyd-Richardson, E., & Prinstein, M. J. (2008). Longitudinal study of nonsuicidal self-injury among young adolescents: Rates, correlates, and prelininary test of an interpersonal model. *Journal of Early Adolescence, 28*, 455-469.

Holmbeck, G. N. (1997). Toward terminological, conceptual, and statistical clarity in the study of mediators and moderators: Examples from the child-clinical and pediatric psychology literatures. *Journal of Clinical and Consulting Psychology, 65*, 599-610.

Joiner, T. E., & Rudd, M. D. (1995). Negative attributional style for interpersonal events and the occurrence of severe interpersonal disruptions as predictors of self-reported suicidal ideation. *Suicide and Life-Threatening Behavior, 25*, 297-304.

Keating, D. P., & Bobbitt, B. L. (1978). Individual and developmental differences in cognitive processing components of mental ability. *Child Development, 49*, 155-167.

Kelley, A. E., Schochet, T., & Landry, C. F. (2004). Risk taking and novelty seeking in adolescence: Introduction to part I. *Annals of the New York Academy of Science, 1021*, 27-32.

Kessler, R. C., Berglund, P., Borges, G., Nock, M. K., & Wang, P. S. (2005). Trends in suicide ideation, plans, gestures, and attempts in the United States, 1990-1992 to 2001-2003. *Journal of the American Medical Association, 293*, 2487-2495.

Kessler, R. C., Borges, G., & Walters. E. E. (1999). Prevalence of and risk factors for lifetime suicide attempts in the National Comorbidity Survey. *Archives of General Psychiatry, 56*, 617-626.

Klimes-Dougan, B., Hastings, P. D., Granger, D. A., Usher, B. A., & Zahn-Waxler, C. (2001). Adrenocortical activity in at-risk and normally developing adolescents: Individual differences in salivary cortisol basal levels, diurnal variation, and responses to social challenges. *Developmental Psychopathology, 13*, 695-719.

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

Klonsky, E. D., Oltmanns, T. F., & Turkheimer, E. (2003). Deliberate self-harm in a nonclinical population: Prevalence and psychological correlates. *American Journal of Psychiatry, 160,* 1501-1508.

Larson, R., & Ham, M. (1993). Stress and "storm and stress" in early adolescence: The relationship of negative events with dysphoric affect. *Developmental Psychology, 29,* 130-140.

Lazarus, R. S. (1966). *Psychological stress and the coping process*. New York: McGraw-Hill.

Lazarus, R. S. (1991). *Emotion and adaptation*. New York: Oxford University Press.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.

Lewinsohn, P. M., Rohde, P., & Seeley, J. R. (1994). Psychosocial risk factors for future adolescent suicide attempts. *Journal of Consulting and Clinical Psychology, 62,* 297-305.

Lopez-Duran, N. L., Kovacs, M., & George, C. J. (2009). Hypothalamic-pituitary-adrenal axis dysregulation in depressed children and adolescents: A meta-analysis. *Psychoneuroendocrinology, 34,* 1272-1283.

Lovallo W. R. & Thomas T. L. (2000). Stress hormones in psychophysiological research. In J. T. Cacioppo, L. G. Tassinary, & G. G. Bernston (Eds.), *Handbook of Psychophysiology* (pp. 342-67). Cambridge, U.K.: Cambridge University Press.

Luby, J. L., Heffelfinger, A., Mrakotsky, C., Brown, K., Hessler, M., & Spitznagel, E. (2003). Alterations in stress cortisol reactivity in depressed preschoolers relative to psychiatric and no-disorder comparison groups. *Archives of General Psychiatry, 60,* 1248-1255.

Luby, J. L., Mrakotsky, C., Heffelfinger, A., Brown, K., & Spitznagel, E. (2004). Characteristics of depressed preschoolers with and without anhedonia: Evidence for a melancholic depressive subtype in young children. *American Journal of Psychiatry, 161,* 1998-2004.

Mann, J. J., Arango, V. A., Avenevoli, S., Brent, D. A., Champagn, F. A., Clayton, P., Currier, D., Dougherty, D. M., Haghighi, F., Hodge, S. E., Kleinman, J., Lehner, T., McMahon, F., Moscicki, E. K., Oquendo, M. A., Pandey, G. N., Pearson, J., Stanley, B., Terwilliger, & Wenzel, A. (2009). Candidate endophenotypes for genetic studies of suicidal behavior. *Biological Psychiatry, 65,* 556-563.

Maris, R. W. (1981). *Pathways to suicide: A survey of self-destructive behaviors*. Baltimore: The Johns Hopkins University Press.

Mathew, S. J., Coplan, J. D., Goetz, R. R., Feder, A., Greenwald, S., Dahl, R. E., Ryan, N. D., Mann, J. J., & Weissman, M. M. (2003). Differentiating depressed adolescent 24h cortisol secretion in light of their adult clinical outcome. *Neuropsychopharmacology, 28*, 1336-1343.

Metalsky, G. I., Halberstadt, L. J., & Abramson, L. Y. (1987). Vulnerability to depressive mood reactions: Toward a more powerful test of the diathesis-stress and causal mediation components of the reformulated theory of depression. *Journal of Personality and Social Psychology, 52*, 386-393.

McKeown, R. E., Garrison, C. Z., Cuffe, S. P., Waller, J. L., Jackson, K. L., & Addy, C. L. (1998). Incidence and predictors of suicidal behaviors in a longitudinal sample of young adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 37*, 612-619.

Morris, N. M., & Udry, R. J. (1980). Validation of a self-administered instrument to assess stage of adolescent development. *Journal of Youth and Adolescence, 9*, 271-280.

Muehlenkamp, J. J. (2005). Self-injurious behavior as a separate clinical syndrome. *American Journal of Orthopsychiatry, 75*, 324-333.

Negriff, S., Fung, M. T., & Trickett, P. K. (2008). Self-rated pubertal development, depressive symptoms and delinquency: Measurement issues and moderation by gender and maltreatment. *Journal of Youth and Adolescence, 37*, 736-746.

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

Nock, M. K., Joiner, T. E., Gordon, K. H., Lloyd-Richardson, E., & Prinstein, M. J. (2006). Non-suicidal self-injury among adolescents: Diagnostic correlates and relation to suicide attempts. *Psychiatry Research, 144*, 65-72.

Nock, M. K., & Mendes, W. B. (2008). Physiological arousal, distress tolerance, and social problem-solving deficits among adolescent self-injurers. *Journal of Consulting and Clinical Psychology, 76*, 28-38.

Nock, M. K., & Prinstein, M. J. (2004). A functional approach to the assessment of self-mutilative behavior. *Journal of Consulting and Clinical Psychology, 72*, 885-890.

Nock, M. K., & Prinstein, M. J. (2005). Contextual features and behavioral functions of self-mutilation among adolescents. *Journal of Abnormal Psychology, 114*, 140-146.

Nock, M. K., Wedig, M. M., Holmberg, E. B., & Hooley, J. M. (2008a). The emotion reactivity scale: Development, evaluation, and relation to self-injurious thoughts and behaviors. *Behavior therapy, 39*, 107-116.

Nock, M. K., Wedig, M. M., Janis, I. B., & Deliberto, T. L. (2008b). Self-injurious thoughts and behaviors. In J. Hunsley & E. J. Mash (Eds.), *A Guide to Assessments that Work* (pp. 158-177). New York: Oxford University Press.

Najmi, S., Wegner, D. M., Nock, M. K. (2007). Thought suppression and self-injurious thoughts and behaviors. *Behaviour Research and Therapy, 45*, 1957-1965.

Oquendo, M. A., Malone, K. M., & Mann, J. J. (1997). Suicide: Risk factors and prevention in refractory major depression. *Depression and Anxiety, 5*, 202-211.

Peterson, C., Semmel, A., von Baeyer, C., Abramson, L. Y., Metalsky, G. I., & Seligman, M. E. P. (1982). The Attributional Style Questionnaire. *Cognitive Therapy and Research, 6*, 287-300.

Priester, M. J., & Clum, G. A. (1992). Attributional style as a diathesis in predicting depression, hopelessness, and suicide ideation in college students. *Journal of Psychopathology and Behavioral Assessment, 14*, 111-122.

Prinstein, M. J., Nock, M. K., Simon, V., Aikins, J. W., Cheah, C. S. L., & Spirito, A. (2008). Longitudinal trajectories and predictors of adolescent suicidal ideation and attempts following inpatient hospitalization. *Journal of Clinical and Consulting Psychology, 76*, 92-103.

Rao, U., Hammen, C., Ortiz, L. R., Chen, L., & Poland, R. E. (2008). Effects of early and recent adverse experiences on adrenal response to psychosocial stress in depressed adolescents. *Biological Psychiatry, 64*, 521-526.

Reinherz, H. Z., Tanner, J. L., Berger, S. R., Beardslee, W. R., & Fitzmaurice, G. M. (2006). Adolescent suicidal ideation as predictive of psychopathology, suicidal behavior, and compromised functioning at age 30. *American Journal of Psychiatry, 163*, 1226-1232.

Roberts, R. E., Roberts, C. R., & Xing, Y. (2010). One-year incidence of suicide attempts and associated risk and protective factors among adolescents. *Archives of Suicide Research, 14*, 66-78.

Robins, C. J., Block, P., & Peselow, P. (1989). Relations of sociotropic and autonomous personality characteristics to specific symptoms in depressed patients. *Journal of Abnormal Psychology, 98*, 86-88.

Rogers, J. R. (2001a). Theoretical grounding: The “missing link” in suicide research. *Journal of Counseling and Development, 79*, 365-372.

Rogers, J. R. (2001b). Psychological research into suicide: Past, present, and future. In D. Lester (Ed.), *Suicide: Resources for the new millennium* (pp. 31-44). Philadelphia: Brunner-Routledge.

Rogers, J. R. (2003). The anatomy of suicidology: A psychological perspective on the status of suicide research. *Suicide and Life-Threatening Behavior, 33*, 9-19.

Ross, S., & Heath, N. (2002). A study of the frequency of self-mutilation in a community sample of adolescents. *Journal of Youth and Adolescence, 311*, 67-77.

Rudd, M. D., Berman, A. L., Joiner, T. E., Nock, M. K., Silverman, M. M., Mandrusiak, M., Van Orden, K., & Witte, T. (2006). Warning signs for suicide: Theory, research, and clinical applications. *Suicide and Life-Threatening Behavior, 36*, 255-262.

Rudolph, K. D. (2002). Gender differences in emotional responses to interpersonal stress during adolescence. *Journal of Adolescent Health, 30*, 3-13.

Rudolph, K. D., & Asher, S. R. (2000). Adaptation and maladaptation in the peer system: Developmental processes and outcomes. In A. J. Sameroff & M. Lewis (Eds.), *Handbook of developmental psychopathology* (2nd ed., pp. 157 – 175). Dordrecht, the Netherlands: Kluwer Academic Publishers.

Rudolph, K. D., & Hammen, C. (1999). Age and gender as determinants of stress exposure, generation, and reactions in youngsters: A transactional perspective. *Child Development, 70*, 660-677.

Schaffer, D., Fisher, P., Lucas, C. P., Dulcan, M. K., & Schwab-Stone, M. E. (2000). NIMH Diagnostic Interview Schedule for Children Version IV (NIMH DISC-IV): Description, differences from previous versions, and reliability of some common diagnoses. *Journal of the American Academy of Child and Adolescent Psychiatry, 39*, 28-38.

Schwartz, E. B., Granger, D. A., Susman, E. J., Gunnar, M. R., Laird, B. (1998). Assessing salivary cortisol in studies of child development. *Child Development, 69*, 1503-1513.

Smith, J. M., Alloy, L. B., & Abramson, L. Y. (2006). Cognitive vulnerability to depression, rumination, hopelessness, and suicide ideation: Multiple pathways to self-injurious thinking. *Suicide and Life-Threatening Behavior, 36*, 443-454.

Smyth, J., Ockenfels, M. C., Porter, L., Kirschbaum, C., Hellhammer, D. H., & Stone, A. A. (1998). Stressors and mood measured on a momentary basis are associated with salivary cortisol secretion. *Psychoneuroendocrinology, 23*, 353-370.

Steinberg, L., Dahl, R., Keating, D., Kupfer, D. J., Masten, A. S., & Pine, D. S. (2006). The study of developmental psychopathology in adolescence: Integrating affective neuroscience with the study of context. In D. Cicchetti & D. J. (Eds.), *Developmental psychopathology, Vol. 2: Developmental neuroscience (2nd ed.)* (pp. 710-741). Hoboken, NJ: Wiley.

Stern, R. M., Ray, W. J., & Quigley, K. S. (2001). *Psychophysiological recording*. New York: Oxford.

Stroud, L. R., Foster, E., Papandonatos, G. D., Handwerger, K., Granger, D. A., Kivlighan, K. T., & Niaura, R. (2009). Stress response and the adolescent transition: Performance versus peer rejection stressors. *Development and Psychopathology, 21*, 47-68.

Stroud, L. R., Tanofsky-Kraff, M., Wilfley, D. E., & Salovey, P. (2000). The Yale Interpresonal Stressor (YIPS): Affective, physiological, and behavioral responses to a novel interpersonal rejection paradigm. *Annals of Behavioral Medicine, 22*, 204-213.

Suyemoto, K. L. (1998). The functions of self-mutilation. *Clinical Psychology Review, 18*, 531-554.

U. S. Department of Health and Human Services, P. H. S. (2001). *National strategy for suicide prevention: Goals and objectives for action*. Rockville, MD: U.S. Department of Health and Human Services.

Wagner, B. M. (2009). Suicidal behavior in children and adolescents. New Haven: Yale University Press.

Walsh, B. W. (2006). *Treating self-injury: A practical guide*. New York: Guilford Press.

Weishaar, M. E. (1996). Cognitive risk factors in suicide. In P. M. Salkovskis (Ed.), *Frontiers of cognitive therapy* (pp. 226-249). New York: Guilford.

WHO. (2010). *World Health Organization: Suicide Prevention*. Retrieved 2010 July 14, from http://www.who.int/mental_health/prevention/suicide/suicideprevent/en/

Willoughby, M., Vandergrift, N., Blair, C., & Granger, D. A. (2007). A structural equation modeling approach for the analysis of cortisol data collected using pre-post-post designs. *Structural Equation Modeling, 14*, 125-145.

Wood, A., Kroll, L., Moore, A., & Harrington, R. (1995). Properties of the Mood and Feelings Questionnaire in adolescent psychiatric outpatients: A research note. *Journal of Child Psychology and Psychiatry, 36*, 327-334.

Yip, K. (2005). A multi-dimensional perspective of adolescents' self-cutting. *Child and Adolescent Mental Health, 10*, 80-86.