

LONGITUDINAL PREDICTION OF ADOLESCENT NONSUICIDAL SELF-INJURY:
EXAMINATION OF A COGNITIVE VULNERABILITY-STRESS MODEL

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ABSTRACT

JOHN D. GUERRY: Longitudinal prediction of adolescent nonsuicidal self-injury:
Examination of a cognitive vulnerability-stress model
(Under the direction of Mitchell Prinstein)

Virtually no research has examined psychological characteristics or events that may lead to adolescent nonsuicidal self-injury (NSSI). While recent work has implicated the role of stressful life events, little is known regarding how stress might combine with other factors to predict NSSI. The present study tested whether a cognitive-vulnerability stress model could predict longitudinal trajectories of NSSI. Adolescent participants (n = 143; 72% female) recruited from a psychiatric inpatient facility reported on measures of NSSI, depression, attributional style, and interpersonal stressors during baseline hospitalization. Levels of NSSI were reassessed at 3, 6, 9, 15, and 18 months post-baseline. Latent growth curve analyses partially supported hypotheses. Although the cognitive vulnerability-stress interaction was not significantly associated with “remission” of NSSI between baseline and 6-month follow-up, this interaction significantly predicted “maintenance” of NSSI between 9, 15, and 18 months post-baseline. These findings further support theoretical models of NSSI which posit emotion regulation functions.

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CHAPTER 1

INTRODUCTION

Nonsuicidal self-injury (NSSI) refers to a broad class of behaviors defined by the direct, deliberate, and socially unacceptable damage to one's own body tissue without suicidal intent. Once considered a behavior restricted to individuals with developmental disabilities or with Borderline Personality Disorder (BPD), NSSI is now recognized as a widespread and pervasive public health problem, occurring at significant rates in both clinical and community samples and spanning a range of diagnostic profiles (Prinstein, Guerry, Browne, & Rancourt, in press). Recent estimates have suggested that NSSI is remarkably prevalent among community-based samples of adults (1-4%; Briere & Gil, 1998; Klonsky, Oltmanns, & Turkheimer, 2003). NSSI has been found to occur at even more alarming rates, however, among preadolescents (7%; Hilt, Nock, Lloyd-Richardson, & Prinstein, 2008) and adolescents (12-15%; Favazza, DeRosear, & Conterio, 1989; Ross & Heath, 2002).

Prevalence estimates from clinical samples, which are notably higher overall, reveal a similar developmental pattern; rates of NSSI are 2-3 times higher among adolescents (40-60%; Darche, 1990; DiClemete, Ponton, & Hartley, 1991) compared to adults (~21%; Briere & Gil, 1998). Some studies have reported that adolescent females engage in NSSI more frequently than males (Ross & Heath, 2002; Bhugra, Thompson, Singh, & Fellow-Smith, 2003). The evidence is conflicted, however, as other investigators have failed to find similar

gender differences (e.g., Hilt et al., 2008; DiClemente et al., 1991; Gratz, Conrad, & Roemer, 2002; Garrison, Addy, McKeown, Cuffe, Jackson, & Waller, 1993).

Despite the striking prevalence of NSSI, as well as some suggestion that its incidence may be increasing (Hawton, Fagg, Simkin, Bale, & Bond, 1997), NSSI research is still in its nascent stages of development. Much of the extant literature has merely provided descriptive data regarding the phenomenon and its psychosocial correlates. While several theoretical models have been proposed to organize clinical descriptions and guide inquiry (e.g., Favazza, 1998; Suyemoto, 1998; Yip, 2005), there is a paucity of research that has either rigorously evaluated theory-based hypotheses or used advanced research or analytic methods. Instead, much of the evidence to date has come from uncontrolled case studies, correlational research, or relied on self-reported measures and cross-sectional methodology (Prinstein et al., in press).

Still fewer studies exist that have examined NSSI using prospective, longitudinal designs. This is a central failing: without establishing its temporal aspects, NSSI's causes, correlates, and consequences cannot be differentiated. Additionally, studies of NSSI most often involve adults, or convenience samples of college-aged students. This is despite the clear research relevance of adolescence, both as the age group during which rates of NSSI are the highest and as the developmental period most associated with the initiation of these behaviors (Favazza & Conterio, 1988). Although such work with adults has offered essential contributions to the literature, its focus has precluded the empirical examination of NSSI through a developmental psychopathology perspective.

For all these reasons, progress toward identifying risk factors for the development of NSSI or the mechanisms by which NSSI is initiated and maintained has been limited. There

is a pressing need for prospective, longitudinal research that specifically targets the development of NSSI during the critical period in which individuals first learn to engage in these dangerous behaviors. Utilizing a sample of clinically-referred adolescents—by far, the group for which NSSI is known to be most prevalent—would constitute a logical, important, and efficient beginning for this line of research.

One promising area of investigation has highlighted the role of stressful life events, particularly those of an interpersonal nature, as common precipitants of NSSI. Retrospective accounts of interpersonal “triggers” immediately preceding episodes of NSSI or suicidal behavior have included intense loneliness, interpersonal rejection or loss, or a recent conflict with a family member, romantic partner, adult authority figure, or peer (e.g., Hawton & Harriss, 2006). In support of this, one study conducted with adolescents in a group treatment home revealed that the frequency of NSSI incidents increased significantly as compared to other time periods during the two weeks prior to the anticipated loss of a staff member (Rosen, Walsh, & Rode, 1990). There also is some evidence to support a more distal association between life stress and individuals’ later engagement in NSSI. For example, Cochrane and Robertson (1975) demonstrated that, as compared to non-self-injuring controls, self-injurers tend to experience far more unpleasant, stressful events in the year preceding incidents of NSSI. These events included a number of interpersonal stressors (e.g., “increases in the number of arguments with spouse”).

The apparent association between life stress and NSSI also is consistent with leading theoretical models. By far, most evidence to date has suggested that individuals engage in NSSI as a strategy, albeit a maladaptive one, to regulate emotional distress (e.g., Chapman, Gratz, & Brown, 2006; Klonsky, 2007; Brown, Comtois, & Linehan, 2002; Nock &

Prinstein, 2004). Specifically, it has been hypothesized that the reduction of tension or more general negative affect serves as a primary motivation for engaging in NSSI (e.g., Chapman, et al., 2006; see also Nock & Prinstein, 2004; 2005; Suyemoto, 1998; Yip, 2005). There also is substantial empirical evidence accumulating to support this theory. For example, Haines and colleagues (1995) found that individuals who had previously engaged in NSSI showed a decrease in psychophysiological arousal and subjective distress during a task that involved NSSI imagery. No such decrease was observed for individuals without a history of self-injury who were administered the same imagery paradigm. From these results, the authors concluded that NSSI is reinforced, at least in part, by its tension-reducing qualities (Haines, Williams, Brain, & Wilson, 1995).

The significance of interpersonal stressors—both as proximal precipitants and more distant correlates or initiators of NSSI—might help to explain the observed developmental variation in the prevalence of NSSI, as well as apparent 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). Corresponding to the emerging salience 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 these interpersonal stressors and report greater emotional reactivity to them, as compared to younger children and adolescent boys (Rudolph, 2002; Rudolph & Hammen, 1999; Hankin, Mermelstein, & Roesch, 2007). These findings parallel commonly cited developmental and gender differences in the prevalence of NSSI.

Still, the mechanisms by which stress contributes to the eventual development of NSSI are poorly understood. Though the increased frequency and intensity of interpersonal stressors may be particularly inherent to the experience of all adolescents, it is obvious that only a minority of these individuals will ever engage in NSSI. At most, extreme and/or chronic distress may be one necessary, but certainly far from sufficient, precondition for the development of NSSI. Research that elucidates the nature of more remote mechanisms in the causal pathways that lead to NSSI would be especially important for the development and testing of prevention and early intervention strategies.

Borrowing from an example of a related research literature may illuminate one such mechanism by which stress contributes to the development of NSSI. Cognitive vulnerability models have proven especially useful for understanding the more remote effects of stress on psychopathological outcomes such as depression (e.g., Abramson, Metalsky, & Alloy, 1989; see Riskind & Alloy, 2006, for a review). Briefly, cognitive theories of depression hypothesize that the ways in which individuals attend to, interpret, and remember life events contribute to the likelihood that they will experience depression.

Cognitive vulnerability-stress models of depression further specify that negative cognitive structures or styles operate within these mental processes and represent a diathesis which, in the presence of stressful life events, confers a vulnerability to development and maintenance of depression. These models have received much theoretical and empirical attention, generally providing support for the longitudinal association between the cognitive vulnerability-stress interaction and future depressive symptoms in adult populations (see Abramson et al., 2002; Ingram, Miranda, & Segal, 1998; Scher, Ingram, & Segal, 2005, for reviews). There also is emerging evidence supporting the capacity of such models to explain

the development of depression among children and adolescents (e.g., Hilsman & Garber, 1995; Lewinsohn, Joiner, & Rohde, 2001; see Lakdawalla, Hankin, & Mermelstein, 2007, for a review).

A similar conceptualization of ‘cognitive vulnerability’ used in these models of depression may likewise prove applicable for the prediction of harmful behaviors such as NSSI. In the case of depression, for example, it has been posited that some individuals reveal this cognitive vulnerability through a tendency to make negative attributions when confronted with stressful life events (e.g., Abramson et al., 1989). 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, individuals who demonstrate a consistent pattern of making stable (as opposed to transient) and global (as opposed to specific) attributions following negative life events are at greater risk of becoming depressed (Abramson et al., 1989).

These considerations provide a powerful rationale for extending this cognitive vulnerability-stress model to the realm of NSSI among adolescents. This leads to the general hypothesis that the same depressogenic tendency to interpret stressful events as due to internal, global, and stable causes may precipitate in some adolescents emotional states that lead to another outcome—the onset or persistence of NSSI. To our knowledge, this idea has not been tested previously.

In the present study, we examine the interaction of attributional style and stressful life events among an inpatient sample of adolescents. We hypothesize that a depressogenic attributional style will be associated longitudinally with increases in NSSI. Specifically, we predict that an interaction between high levels of a depressogenic attributional style and the

occurrence of stressful life events will be associated with increases in incidents of NSSI across an 18-month interval. Given the particular salience of interpersonally-themed stress for adolescents, we will examine this domain of stressful life events specifically. Lastly, because past research suggests a greater vulnerability to interpersonally-themed stress among adolescent girls as opposed to boys (Rudolph, 2002; Rudolph & Hammen, 1999; Hankin et al., 2007), we further hypothesize that gender will act as a moderator within this cognitive vulnerability-stress model; this interaction will be significantly greater among girls.

CHAPTER 2

METHODS

Participants

Participants included 143 adolescents (72% female) between the ages of 12 and 15 years ($M = 13.51$; $SD = .75$), and in grades 7 (20%), 8 (40%) or 9 (40%) at baseline. Although data from this sample were collected previously as part of a prospective, longitudinal study on adolescent suicidal behaviors, the age range of this sample was equally well-suited to examine NSSI. Approximately 75% of participants were White/Caucasian, 4% Latino/a-American, 3% African-American, and 17% Mixed Ethnicity. Approximately 27% of adolescents lived with both biological parents, 29% with their biological mother only, and 15% with their biological mother and a step-parent. The remaining 29% of adolescents lived either with their biological father, extended family members, or in foster or other temporary care. Nineteen percent of mothers reported that they had not obtained a high school diploma, 40% of mothers' highest education was a high school degree, 14% had earned a trade degree, 11% attended some undergraduate college, and 9% had obtained a college degree or higher.

All participants were recruited from a psychiatric inpatient facility in the U.S. Northeast. During the period of recruitment, a total of 246 adolescents matching study inclusion (12-15 years; no past or current psychosis or mental retardation) were admitted to the inpatient unit. At the time of data collection, approximately 40% of all admissions onto

this unit were discharged or transferred within 1-2 days of admission. This length of stay was associated with a variety of factors (e.g., limitations proscribed by insurance carriers, vacancies at local facilities) and was not related to the severity of adolescents' psychological symptoms or adolescents' SES.

Consistent with human subjects regulations, adolescent patients and their parents were approached for study participation only after clinic personnel had gained permission from adolescents' parents/guardians to be contacted about this investigation (typically on the second day following admission). Consent for study participation was subsequently requested from 183 of these eligible adolescents and a total of 162 (88.5%) ultimately provided consent. Of these, 143 (88.3%) were available to be assessed on study measures (19 participants were discharged after consenting but before data could be collected).

Adolescents and their parents initially were assessed during hospitalization (baseline) immediately following consent, typically within 2 to 4 days of admission. Adolescents and parents also completed follow-up assessments at 3, 6, 9, 15, and 18 months post-baseline.

Data were missing for two reasons common to research of this type. First, certain logistical challenges inherent to inpatient data collection (e.g., competing demands for patients' time, unexpected discharge or transfer) yielded missing data on some items or measures within participants. Second, some data were missing due to attrition over various longitudinal intervals (e.g., family relocation, study drop-out, etc.). Many retention strategies were utilized, including frequent phone and mail contact with participants and their network of immediate and extended family members and friends, searches within public access databases for current contact information, and provision of incentives to participants to

encourage completion of follow-up assessments (i.e., \$30 at each follow-up time point for each adolescent and parent participant).

Of the 143 adolescents who completed baseline assessments, 133 (93%) participated in at least one of the follow-up time points, 115 (80%) participated in at least two follow-ups, 106 (74%) participated in at least three, 96 (67%) in four, and 76 (53%) completed every follow-up assessment. A total of 102 adolescents (71%) participated at the final assessment. This retention rate is comparable to prior research on similar populations (e.g., Boergers & Spirito, 2003).

Analyses were conducted to compare adolescents with and without complete longitudinal data on all baseline study variables. Analyses also were conducted to examine adolescents who did and did not participate in the final assessment. In both cases, no significant differences were revealed on any study variables, suggesting no evidence for attrition biases. Missing data analyses indicated that data were missing at random, Little's MCAR $\chi^2(1840) = 1839.57$, NS. To prevent the unnecessary omission of valuable data (cf., listwise deletion), all analyses were conducted using all available data. Analyses using only available data revealed an identical pattern of results.

Measures

All adolescent questionnaire-based measures were read aloud by a trained research assistant during individual meetings while adolescents privately recorded their responses. This procedure allowed for adequate probing and clarification of study items when necessary, careful monitoring of adolescents' attention and conscientiousness while completing measures, and immediate checking for inconsistencies or omissions in responses.

Nonsuicidal Self-Injury (NSSI). Nonsuicidal self-injury was assessed at baseline and at each follow-up time point using a set of five items adapted from the Suicide Ideation Questionnaire (SIQ; Reynolds, 1985). These items reported the frequency that adolescents engaged in several types of NSSI (i.e., cut/carved skin, hit self on purpose, pulled hair out, burned skin, or other method) without suicidal intent. Respondents were asked to consider the time frame of the past year in answering these items at the first administration of the questionnaire (“baseline”), and then for each subsequent time point (i.e., at the 3-, 6-, 9-, 15-, and 18-month follow-up assessments) they were asked to report on the previous three months. The frequency of engagement in each item was reported on a five point scale (1 = Never; 5 = Almost every day). A mean score across all five items was computed at baseline ($\alpha = .70$).

Attributional Style. Adolescents’ attributional style was assessed at baseline using the revised Children’s Attributional Style Questionnaire (CASQ-R; Kaslow & Nolen-Hoeksema, 1991). The CASQ is a 24-item, forced-choice questionnaire that describes 12 positive and 12 negative hypothetical events. Participants are instructed to imagine each event happening to them and then decide which of the two provided explanations best describes the cause of the event. For example, the item “You get a bad grade in school” lists the following two explanations: “1.) I am not a good student” or “2.) Teachers give hard tests.” Throughout the CASQ-R for a given item, two of the dimensions of attributional style (i.e., internal/external, stable/unstable, global/specific) are held constant while the third is varied. In the example, the locus dimension is varied (internal vs. external), whereas the stability and globility dimensions are held constant.

Composite scores for each of the positive and negative events subscales are calculated by adding together the internal, stable, and global scores across each respective category of items. The overall composite score for the CASQ-R, which is the index utilized in the present study, is derived by subtracting the composite negative event score from the composite positive event score. Scores on this scale range from -12 to +12, with lower scores indicating a more depressogenic attributional style. The psychometric properties of the CASQ-R have been shown to be acceptable, but not strong, with moderate internal consistency for the overall composite score and fair test-retest reliability (Thompson, Kaslow, Weiss, & Nolen Hoeksema, 1998). In this sample, the coefficient alpha was found to be .74, which is consistent with the value found by Thompson et al. (1998; $\alpha = 0.61$).

Interpersonal Life events. Adolescents' experience of life stressors were assessed at baseline using a modified version of the Life Events Checklist (LE-C). The LE-C is a 30-item measure based on several life event inventories developed for use with adolescents (see Coddington, 1971; Compas, Davis, Forsythe, & Wagner, 1987; Johnson & McCutcheon, 1980; Masten, Garmezy, Tellegen, Pellegrini, & Larkin, 1988). Participants were asked whether each of 30 potentially negative life events had happened to them or their families in the past 9 months. Salient points in time such as holidays and school calendar events were discussed with each adolescent to provide referents for the time interval in question. The LE-C includes major life events, such as "Your parents separated or got divorced" and "A family member or close relative died," as well as less severe, often more subjective events, such as "You began dating" and "Your family moved to a new home or apartment."

Due to the previously noted relevance of interpersonally-themed stressors among adolescents, only those items on the LE-C that could be explicitly categorized as stressful

interpersonal life events were included in the foregoing analyses (e.g., “You and your boyfriend/girlfriend had a big fight or broke up”). Adolescents’ scores across this interpersonal domain of 10 items were summed to create an index of interpersonal life stress. Because the scale is a checklist of independent items, it is not appropriate to calculate its internal consistency.

Depression. Adolescents completed the Child Depression Inventory (CDI; Kovacs, 1992) at baseline and again 18 months post-baseline. The CDI, which is a modification of the Beck Depression Inventory designed for use with preadolescent children, consists of 27-items that assess cognitive, affective, and behavioral symptoms of depression, including all but one (psychomotor agitation) of the DSM-IV criteria for a major depressive episode. For each item, children choose among three statements that best describe their level of depressive symptoms in the previous two weeks. Item choices are assigned a numerical value from 0 to 2, with higher scores corresponding to higher levels of depression. A mean score was computed across all items with one exception (i.e., suicidal ideation) to minimize overlap between constructs. The CDI is the most widely used self-reported measure of depressive symptoms in children, with reasonably high levels of internal consistency, test-retest reliability, and convergent validity with other self-reported measures (Saylor, Finch, Spirito, & Bennet, 1984; Carey, Faulstich, Gresham, Ruggiero, & Enyart, 1987; Kazdin, French, Unis, & Esveldt-Dawson, 1983; Saylor, Finch, Baskin, Furey, & Kelly, 1984). The CDI can be used with youths between the ages of 7 and 18 years (Kazdin, 1990). Internal consistency in the present sample as measured at baseline was .88.

Data Analyses

Three sets of analyses were conducted to examine study hypotheses. First, descriptive statistics first were conducted to examine the means and standard deviations on all study variables over the 18-month longitudinal period. As gender was hypothesized to be moderator, separate statistics are reported for adolescent males and females. Correlational analyses also were performed between all study variables.

Second, in order to better understand the course of NSSI over the 18-month follow-up period, an unconditional growth curve model using latent curve analysis was examined. The use of latent curves allows for an 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 performed using AMOS 7.0.

It was anticipated that NSSI slopes may be non-linear, given that for many adolescents NSSI may occur at a high incidence at baseline (i.e., during hospitalization), decrease following discharge, and possibly increase again over the extended longitudinal period. An initial model examined a single latent slope factor. The six measures of NSSI (at baseline, 3, 6, 9, 15, and 18 months post-baseline) were included as observed indicators, with latent intercept and slope factors estimated. A latent intercept factor with paths to all observed indicators set to 1 was modeled. Path weights between the latent slope factor and each observed indicator of NSSI were set to 0, 1, 2, 3, 5, and 6, respectively.

The single slope model then was compared to alternative models examining 1) a piecewise approach (i.e., linear spline), or 2) a curvilinear slope function. The use of the piecewise approach allowed for an examination of two separate slope functions (Bollen & Curran, 2006). Because growth curve modeling requires at least three time points to compute

a slope, the six time points were divided for analyses as follows: a first slope function modeled the curve between baseline, 3, and 6 months post-baseline (i.e., an “NSSI remission” curve), whereas the second slope function modeled the curve between 9, 15, and 18 months post-baseline (i.e., an “NSSI re-emergence” curve). Each linear spline was modeled with two paths fixed (to 0 and 1, respectively) and the third path allowed to freely vary. The curvilinear model required the inclusion of an initial slope function (with paths to observed indicators set to indicate the three month intervals: 0, 1, 2, 3, 5, 6, respectively), and a second slope function with each corresponding path weight squared (Bollen & Curran, 2006).

The best fitting model of those analyses presented above was built upon to examine the central study hypotheses related to the prospective prediction of NSSI. Hypotheses tested a conditional growth curve model. Paths were estimated between exogenous predictors and the latent intercept and slope factors. The following predictors were included: attributional style (CASQ); stressful interpersonal life events (LE-C); the interaction of life events with attributional style; and gender. Additionally, depression (CDI), as measured at baseline and 18 months post-baseline, was included as exogenous predictors to ensure that other variables were not simply serving as a proxy for depression. All predictors in this model were allowed to covary. Model fit will be evaluated by using several indices ($\chi^2 / df < 2.00$; NFI $> .95$; RMSEA $< .05$).

CHAPTER 3

RESULTS

Descriptive Statistics

Table 1 presents the means and standard deviations for all study variables, as well as the results of *t* tests examining gender differences. To allow for a more complete exploration of the course of NSSI across the 18-month interval, four separate indices were derived from the same data (i.e., the set of five NSSI items on the SIQ). These indices, which each highlight a unique dimension of NSSI, were calculated for each time point as follows: 1) the number (and percent) of individuals engaging in any form of NSSI; 2) a composite mean representing the overall mean of the frequency of NSSI for individuals across all five methods of self-injury; 3) the mean number of different methods utilized; and 4) the sample mean frequency of the most commonly used method of self-injury for each individual (i.e., an average was computed across the entire sample that, for each individual, only considered data from the most frequently reported method).

Results indicated that over two-thirds of the full sample (a total of 95 adolescents) reported that they had engaged in some form of NSSI during the year prior to hospitalization. At all time points subsequent to the baseline assessment, however, the numbers of individuals reporting such behaviors over each preceding 3-month period were markedly decreased from baseline (all *ps* < .001). These numbers remained relatively stable across the extended follow-up period, ranging from 23 adolescents (22.8% of the follow-up sample)

reporting any form of NSSI at 15 months post-baseline to 34 individuals (34% of the follow-up sample) at 9 months post-baseline. A similar longitudinal pattern was observed when considering other indices. For example, when the frequency of NSSI behaviors was examined over time as a composite mean across all methods, adolescents reportedly engaged in NSSI most often in the year prior to baseline hospitalization ($M = 1.54$, $SD = .62$; approximately between “never” and “a few times”). As with the number of self-injurers, the overall frequency of NSSI declined considerably following hospital discharge and remained relatively low across the 18-month follow-up period.

Gender differences were observed consistently across all longitudinal indices of NSSI. At baseline hospitalization, a significantly greater proportion of adolescent girls reported that they had engaged in some form of NSSI over the previous year than did boys [48.7% vs. 75.2%; $\chi^2(1) = 9.08$, $p < .01$]. Although a higher proportion of girls engaged in NSSI at every follow-up time point, this difference only reached statistical significance at 6-month follow-up [12.1% vs. 36.5%; $\chi^2(1) = 6.58$, $p < .05$]. Similarly, relative to adolescent boys at all six time points, a trend was found whereby adolescent girls reportedly engaged in NSSI more frequently—both when calculated as an overall mean and when only taking into account the most commonly used method—and girls tended to utilize more numerous methods of self-injury. However, this pattern of gender differences in favor of girls only reached statistical significance at baseline, 6 months, and 15 months post-baseline (all $ps < .05$, $ds = .40$, $.53$, $.45$, respectively).

In general, results from descriptive analyses for the remaining study variables were in line with expectations and consistent with past work. The results from the CASQ-R, our measure of cognitive vulnerability, indicated that adolescent girls tended to make somewhat

more depressogenic attributions at baseline than did boys. This was observed as a trend, albeit failing to reach statistical significance [$t(130) = 1.41, p = .16$]. Results from the LE-C indicated that adolescent girls reported a significantly greater number of interpersonal stressors ($M = .37, SD = .17$) than did boys ($M = .29, SD = .17$) over the 9-month period preceding hospitalization [$t(107) = -2.19, p < .05, d = .47$]. Finally, results from the CDI revealed that girls tended to report significantly higher symptoms of depression at both baseline [$t(142) = -2.01, p < .05, d = .36$] and 18-month follow-up time points [$t(88.38) = -4.24, p < .001, d = .72$].

Intercorrelations between all study variables are presented in Table 2a (and are displayed separately by gender in Table 2b). For the most part, results from correlational analyses were as expected. For example, there was a negative correlation between attributional style (CASQ-R) and NSSI (composite mean) over time such that a more depressogenic attributional style tended to be associated with a higher occurrence of NSSI. This negative correlation reached significance for NSSI measured at baseline, 3-month, and 18-month follow-ups (all $ps < .001$). Also as expected, both baseline and 18-month follow-up measures of depression (CDI) were significantly and positively correlated with NSSI measured at every interval (all $ps < .05$) and significantly and negatively correlated with baseline (“adaptive”) attributional style (all $ps < .001$).

Course of NSSI Over Time

The analysis of unconditional growth curve models began with an examination of a one slope model including baseline, 3-, 6-, 9-, 15-, and 18-month measures of NSSI. The model was a poor fit (see Table 3). This one slope model was then compared to a piecewise,

linear spline model with a first latent slope factor representing the slope between baseline, 3-, and 6-month time points, and a second slope factor representing changes between 9, 15, and 18 months. Path weights for the first latent slope factor were set to 0 at baseline, were allowed to freely vary at 3 months, and were set to 1 at 6 months (additional time point paths set to 1). For the second slope factor, path weights were allowed to freely vary at both 9 and 15 months but were set to 1 at 18 months (additional time point paths set to 0). This model yielded a good fit, $\chi^2(9) = 9.83$, NS; $\chi^2/df = 1.09$; CFI = .99; NFI = .94; RMSEA = .03; AIC = 45.83, and was a better fit to the data than was the single slope model. A third model with a quadratic slope factor also was modeled. This curvilinear model included an initial slope function with paths to baseline, 3-, 6-, 9-, 15-, and 18-month measures of NSSI set to indicate the three month intervals (i.e., 0, 1, 2, 3, 5, and 6, respectively) and a second slope function with paths to each corresponding time point squared (i.e., 0, 2, 4, 9, 25, and 36, respectively). The fit for the quadratic model, $\chi^2(12) = 30.98$, $p < .01$; $\chi^2/df = 2.58$; CFI = .86; NFI = .81; RMSEA = .10; AIC = 60.98, was not better than the initial slope model, and fit substantially worse than did the piecewise model (see Table 3 for comparison of all model fits).

Because of its good fit, the piecewise model was used as the starting point upon which all analyses listed below were built. The estimated unstandardized path weight for NSSI at 3 months post-baseline on the first slope factor was .96, $p < .001$, and for NSSI at 9 and 15 months post-baseline on the second slope factor were 1.56 and -4.65, respectively (NS each). Estimated intercept parameters indicated that NSSI was significantly greater than 0 at baseline ($M = 1.53$, $p < .001$; variance = 1.25, NS). Estimated parameters for the first slope factor ($M = -.37$, $p < .001$; variance = 1.11, NS) indicate declining levels of NSSI between baseline, 3, and 6 months post-baseline (i.e., an NSSI remission slope). However,

estimated parameters for the second slope factor for NSSI between 9, 15, and 18 months post-baseline were not significant, indicating, on average, consistent levels of NSSI across this time period ($M = .02$, NS; variance = .00, NS; i.e., an NSSI maintenance slope).

Although gender differences were hypothesized in the trajectories of NSSI over time, a multiple group analysis was not possible given the relatively small number of boys. Likewise, due to insufficient power and the number of variables to be examined, gender could not be included as an exogenous predictor in the analyses below.

Baseline Cognitive Vulnerability-Stress Interaction as a Predictor of NSSI Trajectories

The next goal of analyses was to build upon the unconditional growth curve model listed above to examine central study hypotheses related to the prospective prediction of NSSI trajectories. Three exogenous predictors were added to the model listed above: 1) attributional style (CASQ-R); 2) interpersonal life events (derived from the LE-C); and 3) the interaction between interpersonal life events and attributional style. Depression (CDI), as measured at baseline and 18 months post-baseline, also was included as exogenous predictors as a rigorous control (i.e., to ensure that other variables were not simply serving as a proxy for depression). Paths were estimated between all predictors and the latent intercept and both NSSI slopes were estimated. All predictors were allowed to covary. The fit of this model was adequate, $\chi^2(24) = 31.99$, NS; $\chi^2/df = 1.33$; CFI = .98; RMSEA = .05. All standardized path weights are listed in Table 4.

Several associations among exogenous predictors were revealed. First, higher levels of depression reported at baseline and 18-month follow-up were each associated with higher levels of baseline NSSI (i.e., intercept). No other baseline measure emerged as a significant

predictor of baseline NSSI. Higher levels of depression also were associated with a lower NSSI “remission slope” (i.e., slope 1) during the first 6 months of follow-up, above and beyond all other estimated associations. This indicated that higher levels of baseline depression were associated with attenuated NSSI recovery over this longitudinal interval. Finally, after accounting for the associations between all other exogenous predictors and the NSSI “maintenance slope” (i.e., slope 2), the interaction between negative attributional style and stressful life events emerged as the only significant predictor of NSSI between 9, 15, and 18 months.

Taken together, these results suggested that individuals who possessed a more depressogenic attributional style in conjunction with the experience of a greater number of stressful interpersonal life events tended to engage in NSSI more frequently between 9- and 18-month follow-ups than those who reported a more “adaptive” attributional style and/or fewer interpersonal stressors. (See Figure 1 for a graphical depiction of the conditional growth curve model with significant paths displayed for exogenous predictors). Although all of the preceding analyses were conducted using the NSSI composite mean, the same pattern of results were found using the two other calculated indices of NSSI (i.e., the number of discrete methods utilized and the frequency of the most commonly used method).

CHAPTER 4

DISCUSSION

NSSI is becoming increasingly recognized as a significant public health problem, occurring at surprisingly high rates both within community and clinical samples. Engagement in NSSI has been correlated with a number of other serious health risk behaviors and a variety of mental health problems, including anxiety, depression, and suicidality (Andover, Pepper, Ryabchenko, Orrico, & Gibb, 2005; Klonsky et al., 2003). Some have suggested (e.g., Joiner, 2005) that the repetitive engagement in NSSI may even serve as “suicide training,” desensitizing an individual to physical and psychological repercussions of suicide.

Compounding an already considerable level of concern, some evidence is accumulating to suggest that rates of NSSI—both in terms of lifetime prevalence and the repetition of these behaviors—have been increasing dramatically in recent years, particularly among young people (Briere & Gil, 1998; Hawton et al., 1997; Soloff, Lis, Kelly, Cornelius, & Ulrich, 1994; Zlotnick, Mattia, & Zimmerman, 1999). Research roads appear unerringly to lead to adolescence as the developmental period associated with the highest rates of NSSI and the age group in which these behaviors are most commonly initiated (Favazza & Conterio, 1988). Despite this general consensus, there is a dearth of research that has examined this phenomenon among adolescent populations. Further, virtually no research has been conducted to investigate the development of adolescent NSSI using prospective,

longitudinal designs. Preliminary research that aims to identify certain longitudinally distal predictors and mechanisms through which adolescent NSSI is initiated or maintained would be especially useful to inform prevention and early intervention strategies.

The widely cited association between stress and NSSI may constitute the most logical beginning for such a line of research. Indeed, leading theoretical models of NSSI (namely, those that hypothesize an emotion regulation function; e.g., Chapman et al., 2006) and the extant empirical literature are in accord with respect to the importance of stressful life events, both as immediate precipitants of NSSI and as more temporally upstream correlates. Currently, however, the mechanisms by which life stress contributes to either the eventual development of NSSI or the recurrence of these behaviors are poorly understood.

The present study aimed to address at least preliminarily many of these basic limitations by examining the longitudinal course of NSSI within a preexisting clinical sample of adolescents. As cognitive vulnerability-stress models have proved particularly useful in explaining the relationship between stress and later psychopathology (c.f., the onset and maintenance of adult and adolescent depression; see Abramson et al., 2002, for a review), we tested the hypothesis that such a model would aid in the prediction NSSI across an 18-month follow-up period. Given that interpersonal stressors are a particularly salient and frequent experience for adolescents, interpersonally-themed stress was measured specifically.

Descriptive analyses revealed several findings. First, NSSI was found to be remarkably prevalent in this sample; slightly over two-thirds of adolescents reported that they had engaged in some form of NSSI over the year preceding baseline hospital admission. Comparably high rates have been found in studies of NSSI among similar inpatient samples of adolescents (e.g., ~61%; DiClemete et al., 1991). At 3-months subsequent to discharge,

however, the reported prevalence of NSSI declined sharply to approximately one-third of the sample and then remained relatively stable over the extended 18-month follow-up period. The same longitudinal pattern was observed when the frequency of engagement in NSSI was considered, whether this variable was calculated as the overall mean across multiple methods (i.e., the study's primary outcome variable), the number of discrete methods utilized, or the frequency of the most commonly used method of self-injury.

The marked decrease from baseline levels of NSSI at follow-up could be expected given that adolescents were admitted to the hospital during the peak of psychiatric crisis when the incidence of NSSI would likely be at its highest. Presumably, these patients would thereafter be discharged only after this crisis had abated (i.e., following a course of inpatient treatment, after which they were determined to no longer be of imminent harm to themselves, etc.). Mirroring the NSSI longitudinal drop-off and providing further support for the notion of general improvement following hospital discharge, adolescents reported significantly lower levels of depression at 18-month follow-up than they had at baseline.

Consistent with some previous work (e.g., Bhugra et al., 2003; Ross & Heath, 2002), a number of gender differences in NSSI were found in favor of adolescent girls. For example, relative to boys at baseline and at 6-month follow-up, a significantly greater proportion of girls reported that they had engaged in some form of NSSI over the year prior to hospitalization. While this gender difference failed to reach statistical significance at other time points, a consistently higher proportion of girls than boys engaged in NSSI throughout the duration of the study. Similarly, adolescent girls tended to engage in NSSI more frequently—both when calculated as the overall mean or as the mean of the most commonly used method—and girls tended to use a greater variety of methods of self-injury.

Other gender differences were found across study variables that were consistent with expectations and in line with previous work. First, as with research conducted by Rudolph and colleagues (e.g., Rudolph, 2002; Rudolph & Hammen, 1999), adolescent females in this sample reported experiencing a higher number of interpersonal stressors at baseline hospitalization than did adolescent boys. Second, the well-documented disparity between the sexes in the experience of depression (e.g., Hankin & Abramson, 2001) was replicated here; compared to adolescent boys, adolescent girls reported higher levels of depression on the CDI during baseline hospitalization and at 18-month follow-up. Relatedly, there appeared to be a trend whereby adolescent girls made more depressogenic attributions on the CASQ-R (i.e., the study's measure of cognitive vulnerability) than did adolescent boys.

Perhaps of greatest importance, this study afforded the first opportunity to closely examine the trajectories of NSSI following inpatient hospitalization and whether certain factors might aid in the longitudinal prediction of these behaviors. We initially examined concurrent associations between baseline NSSI (intercept) and other study variables. Analyses revealed that the only variable to be significantly associated with baseline ("past year") NSSI as reported during hospitalization was the level of depression, measured both concurrently and at 18-month follow-up. The positive direction of this association was as expected; higher levels of depression were related to more frequent engagement in NSSI over the year preceding hospital admission. Next, our analyses of the longitudinal data indicated that the average course of NSSI in this sample included a period of substantial NSSI remission during the first 6 months following hospitalization (i.e., an NSSI "remission slope"), followed by a year in which NSSI remained stable and relatively infrequent (i.e., an NSSI "maintenance slope").

Although the *average* incidence trajectory of NSSI in the entire sample was not found to re-emerge over the extended follow-up period, it is nonetheless important to understand factors that might be significantly associated with certain *individual* trajectories of NSSI. More specifically, the ability to predict either the persistent, reemerging, or increased engagement in NSSI over time relative to individuals who cease or greatly reduce these behaviors would be especially critical towards identifying high-risk adolescents. As such, the primary goal of this study involved an examination of whether certain baseline factors would predict later NSSI trajectories, namely, depression, cognitive vulnerability, interpersonal stress, and the cognitive vulnerability-stress interaction. Of these, baseline depression emerged as the only significant predictor of the overall remission in NSSI during the first 6 months following hospital discharge. Predictably, higher levels of depression were associated with an attenuated decline in NSSI over this longitudinal interval. In other words, although the overall occurrence of NSSI in this sample declined sharply during the 6 months subsequent to hospital discharge, individuals who reported more symptoms of depression during hospitalization tended to continue to engage in NSSI with more frequency than did their less depressed peers.

More directly relevant to long-term prediction of NSSI trajectories, only one baseline factor emerged as a significant predictor of the change in NSSI between 9- and 18-month follow-ups. While interpersonal life events and attributional style alone were not significantly related to later NSSI, the cognitive vulnerability-stress interaction between these two variables emerged as the only significant predictor of this second slope of NSSI (i.e., the NSSI “maintenance slope”). Thus, consistent with our primary hypothesis, individuals with

more depressogenic attributional styles in conjunction with the experience of a greater number of stressful interpersonal life events tended to engage in more long-term NSSI.

Although the size of this effect may seem marginal, it is particularly impressive when considered in context. First, the substantial length of the longitudinal interval provides a rigorous test of the cognitive vulnerability-stress interaction. It is remarkable that the interaction of the single baseline measures of depressogenic attributional style and stressful interpersonal life events remains a significant predictor of engagement in NSSI 1.5 years later. Second, this effect is significant above and beyond that accounted for by depression as measured at both baseline and 18-month time points. It follows from this that the power of the cognitive vulnerability-stress interaction to predict future NSSI is not simply serving as a proxy for the effects of depression.

Moreover, we have reason to believe that the true size of such an interaction effect may be considerably underestimated in this study due to certain limitations related to the operationalization and measurement of study constructs. Perhaps the most obvious of these is the use of the CASQ-R as our measure of “cognitive vulnerability.” To begin, there are two concerns related to the theoretical fidelity of the CASQ-R. First, referencing the cognitive vulnerability to depression literature, some have argued that the CASQ-R is an incomplete measure of negative attributional style in children and adolescents (e.g., Lakdawalla et al., 2007). Although the questionnaire assesses attributions made to the *causes* of events, it does not address the two other principal facets of hopelessness depression theory (i.e., attributions made as to the consequences of the event as well as attributions made about the self with respect to the event). In this way, it is possible that the CASQ-R lacks sufficient

adherence to theoretically-prescribed face validity and is therefore an inadequate test of cognitive vulnerability.

The second theoretical limitation of the CASQ-R is perhaps more germane to the present study. Major theories of depression have argued for an important corollary of cognitive vulnerability-stress models of depression, the “specific vulnerabilities” hypothesis (see Abramson et al., 1989; Beck, 1987). This hypothesis maintains that an individual may possess one or more “specific vulnerabilities” (e.g., an achievement-related vulnerability vs. an interpersonal vulnerability) that typically remain latent until activated or “triggered” by a relevant stressor (e.g., “I failed a test” vs. “I broke up with my boyfriend”, respectively). Thus, in pairing the CASQ-R—which was designed to measure only a general attributional style across multiple domains of hypothetical stressors—with a relatively specific measure of interpersonal “stress,” it is possible we have greatly attenuated their resultant interaction effect.

Finally, there are concerns regarding the psychometric performance of the CASQ-R. As many have noted previously, the questionnaire has demonstrated poor internal consistency (α 's typically ranging from 0.4 to 0.6; Gladstone & Kalsow, 1995; Thompson et al., 1998). Although the coefficient alpha found in our sample (0.74) is a notable improvement, this reliability is unsatisfactory. In addition, while adolescent attributional style is considered to be a fairly stable construct, particularly over a short period of time (cf. Nolen-Hoeksema, Girgus, & Seligman, 1992), the test-retest reliability of the CASQ-R has been found to be only fair (e.g., 0.53 at 6-month retest; Thompson et al., 1998).

This potential for measurement error may be especially problematic given that our one-time administration of the CASQ-R at baseline was used to predict NSSI 18 months

later. Utilizing a questionnaire with poor psychometric properties as the sole measure of attributional style may curtail the power of our study to detect the significant effect of cognitive vulnerability to predict later NSSI in adolescents. More importantly, as has been noted elsewhere (Lakdawalla et al., 2007), the low reliability of the CASQ-R would likely underestimate effect sizes for attributional style as well as effects sizes for the corresponding cognitive vulnerability-stress interaction. Therefore, considering the combined shortcomings of the CASQ-R, a future study to better test the hypotheses addressed here will benefit from substituting for this instrument a measure of cognitive vulnerability that is better grounded in theory, has demonstrably improved psychometric properties, and makes a more explicit attempt to assess specific cognitive vulnerabilities that could then directly map onto the domains of stress hypothesized to be relevant for those particular vulnerabilities.

A second study limitation relates to our measurement of the construct of interpersonal stress. As with corresponding studies testing the cognitive vulnerability-stress interaction to predict depression in children and adolescents (e.g., Lewinsohn et al., 2001; Robinson, Garber, & Hilsman, 1995; Hankin, Abramson, & Siler, 2001; Nolen-Hoeksema et al., 1992), our measure of interpersonal life events consisted of a binary, “yes/no” checklist that simply recorded whether 10 objectively stressful life events or daily hassles had occurred in the year preceding hospitalization. Future research might improve upon these checklists by not only increasing the number of items assessed but also taking into account the emotional degree to which an individual experienced an event or whether the event was personally significant.

Thus, it is remarkable that a baseline interaction between negative attributional style and stressful interpersonal life events remains a significant predictor of long-term adolescent NSSI. The finding of a significant cognitive vulnerability-stress interaction reveals an

important mechanism through which the often-cited, distal experience of stressful life events could eventually lead to either the first-time engagement or the perpetuation of adolescent NSSI. Although our findings are preliminary and in need of replication, we believe that the current research—which is one of the first of its kind to examine adolescent NSSI longitudinally—has highlighted a potentially fruitful and important avenue for research into the development of a dangerous and persistent self-injurious behavior.

Further, the finding of a significant cognitive vulnerability-stress interaction lends additional empirical support to leading theoretical models of NSSI. These models have hypothesized that such behaviors most commonly serve an emotion regulation function (e.g., Chapman et al., 2006; Klonsky, 2007; Brown et al., 2002; Nock & Prinstein, 2004). More specifically, data has been accumulating to suggest that the majority of individuals engage in NSSI to alleviate acute negative affect (i.e., an automatic negative reinforcement function; e.g., Nock & Prinstein, 2004). The present study thus adds important dimensions to preliminary empirical evidence in support of a direct link between preceding negative internal states and NSSI. Our rigorous control for the effects of depression suggests that the interaction between negative attributional style and interpersonal stress is a unique predictor of adolescent NSSI.

The importance of such a conclusion can not be overstated and its implications are far-reaching. It is possible that individuals who engage in NSSI or those predisposed to such behaviors may suffer from a certain kind of emotion dysregulation, beyond that which could be simply explained by symptoms of depression. Indeed, very recent research would seem to support just such a general hypothesis. The results of several recent studies, which incorporated measures of subjective distress and physiological arousal, indicate that

adolescents and young adults who engage in NSSI experience both higher levels of negative affect and exhibit significantly lower levels of distress tolerance than those without histories of NSSI (Armev & Crowther, 2008; Crowell et al., 2008; Klonsky & Olino, 2008; Nock & Mendes, 2008).

Our data suggest that prediction and prevention of NSSI are possible. If so, then NSSI prevention and intervention efforts can be thought of in two related ways: 1) at-risk individuals or individuals with a history of NSSI could be taught to replace habitual, self-destructive behavior with healthier, more adaptive strategies when faced with the experience of overwhelming negative affect; and 2) strategies could be targeted in the first place towards preventing these individuals from reaching some critical threshold of stress. The results of the present study have a more direct bearing on the latter, prevention-based model of NSSI. The importance of identifying specific cognitive vulnerabilities within individuals that interact with relevant, activating domains of stress and lead to NSSI among individuals is patent. As with the successful treatment of depression, cognitive behavioral therapies could be targeted to correct maladaptive attributions—whether they are the same or unique—that confer greater risk for the development of adolescent NSSI.

Table 1. Descriptive Statistics for Primary Study Variables and Tests for Gender Differences

	Total	Boys	Girls	Statistic
NSSI, <i>N</i> (%) reporting any behavior				
Baseline ¹ (<i>n</i> = 140)	95 (67.9%)	19 (48.7%)	76 (75.2%)	$\chi^2(1) = 9.08^{\Psi}$
3 Months (<i>n</i> = 101)	33 (32.7%)	8 (24.2%)	25 (36.8%)	$\chi^2(1) = 1.58$, NS
6 Months (<i>n</i> = 107)	31 (29.0%)	4 (12.1%)	27 (36.5%)	$\chi^2(1) = 6.58^*$
9 Months (<i>n</i> = 100)	34 (34.0%)	6 (20.0%)	28 (40.0%)	$\chi^2(1) = 3.74$, NS
15 Months (<i>n</i> = 101)	23 (22.8%)	3 (10.3%)	20 (27.8%)	$\chi^2(1) = 3.57$, NS
18 Months (<i>n</i> = 102)	29 (28.4%)	5 (16.7%)	24 (33.3%)	$\chi^2(1) = 2.89$, NS
NSSI (composite mean), <i>M</i> (<i>SD</i>)				
Baseline ¹ (<i>n</i> = 140)	1.54 (.62)	1.36 (.57)	1.61 (.63)	$t(138) = -2.19^*$
3 Months (<i>n</i> = 101)	1.21 (.41)	1.17 (.39)	1.23 (.42)	$t(99) = -.72$
6 Months (<i>n</i> = 107)	1.16 (.34)	1.03 (.09)	1.21 (.40)	$t(87.69)^2 = -3.77^+$
9 Months (<i>n</i> = 100)	1.19 (.39)	1.11 (.30)	1.22 (.42)	$t(98) = -1.33$
15 Months (<i>n</i> = 101)	1.08 (.20)	1.02 (.06)	1.11 (.23)	$t(91.09)^2 = -2.95^{\Psi}$
18 Months (<i>n</i> = 102)	1.18 (.42)	1.10 (.27)	1.21 (.48)	$t(100) = -1.16$
NSSI (# of methods), <i>M</i> (<i>SD</i>)				
Baseline ¹ (<i>n</i> = 140)	1.65 (1.52)	1.08 (1.38)	1.87 (1.51)	$t(138) = -2.85^{\Psi}$
3 Months (<i>n</i> = 101)	.55 (.94)	.42 (.87)	.62 (.98)	$t(99) = -.97$
6 Months (<i>n</i> = 107)	.44 (.81)	.12 (.33)	.58 (.92)	$t(101.92)^2 = -3.78^+$
9 Months (<i>n</i> = 100)	.62 (1.06)	.37 (1.00)	.72 (1.08)	$t(98) = -1.57$
15 Months (<i>n</i> = 101)	.29 (.57)	.10 (.31)	.36 (.63)	$t(95.28)^2 = -2.73^{\Psi}$
18 Months (<i>n</i> = 102)	.49 (.99)	.30 (.84)	.57 (1.05)	$t(100) = -1.25$
NSSI (maximum frequency), <i>M</i> (<i>SD</i>)				
Baseline ¹ (<i>n</i> = 140)	2.29 (1.26)	1.77 (1.01)	2.50 (1.29)	$t(87.23)^2 = -3.52^{\Psi}$
3 Months (<i>n</i> = 101)	1.70 (1.20)	1.52 (1.03)	1.79 (1.28)	$t(99) = -1.09$

6 Months (<i>n</i> = 107)	1.58 (1.12)	1.15 (.44)	1.77 (1.28)	$t(100.86)^2 = -3.70^+$
9 Months (<i>n</i> = 100)	1.57 (.99)	1.37 (.85)	1.66 (1.03)	$t(98) = -1.35$
15 Months (<i>n</i> = 101)	1.34 (.75)	1.10 (.31)	1.43 (.85)	$t(98.37)^2 = -2.82^\Psi$
18 Months (<i>n</i> = 102)	1.54 (1.03)	1.33 (.88)	1.63 (1.08)	$t(100) = -1.31$
CASQ-R, <i>M</i> (<i>SD</i>)				
Baseline (<i>n</i> = 132)	2.86 (4.27)	3.70 (4.18)	2.54 (4.28)	$t(130) = 1.41$
Life events ^{3,4} , <i>M</i> (<i>SD</i>)				
Interpersonal (<i>n</i> = 109)	.34 (.17)	.29 (.17)	.37 (.17)	$t(107) = -2.19^*$
Depression (CDI), <i>M</i> (<i>SD</i>)				
Baseline (<i>n</i> = 144)	.72 (.36)	.63 (.37)	.76 (.35)	$t(142) = -2.01^*$
18 Months (<i>n</i> = 101)	.49 (.30)	.34 (.19)	.55 (.32)	$t(88.38)^2 = -4.24^+$

* $p < .05$; $^\Psi p < .01$; $^+ p < .001$. 1. Past year. 2. Equal variances not assumed. 3. Measured at baseline. 4. Past 9 months.

Note. NSSI = Nonsuicidal self-injury; CASQ-R = Children's Attributional Style Questionnaire – Revised; CDI = Child Depression Inventory.

Table 2a. Pearson Correlations among Primary Study Variables (Overall)

Variable	NSSI 3 months	NSSI 6 months	NSSI 9 months	NSSI 15 months	NSSI 18 months	CASQ-R	LE Interpersonal	CDI baseline	CDI 18 months
NSSI – Baseline	.46***	.51***	.23*	.46***	.47***	-.38***	.06	.50***	.50***
– 3 months	–	.36**	.13	.31**	.32**	-.39***	.01	.35***	.34**
– 6 months		–	.30**	.59***	.54***	-.17	.06	.27**	.43***
– 9 months			–	.28**	.32**	-.11	-.23*	.21*	.30**
– 15 months				–	.39***	-.20	.02	.34***	.52***
– 18 months					–	-.37***	.02	.37***	.48***
CASQ-R						–	-.01	-.54***	-.46***
LE – Interpersonal							–	-.05	.10
CDI – Baseline								–	.58***
– 18 months									–

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

Note. NSSI = Nonsuicidal self-injury; CASQ-R = Children’s Attributional Style Questionnaire – Revised

LE-Interpersonal = interpersonal life events; CDI = Child Depression Inventory.

Table 2b. Pearson Correlations among Primary Study Variables by Gender
(Boys above diagonal, girls below)

Variable	NSSI Baseline	NSSI 3 months	NSSI 6 months	NSSI 9 months	NSSI 15 months	NSSI 18 months	CASQ-R Baseline	LE Interpersonal	CDI baseline	CDI 18 months
NSSI – Baseline	–	.67***	.51**	.12	.10	.52**	-.38*	-.18	.52**	.42*
– 3 months	.40**	–	.64***	.11	.24	.53**	-.42*	.08	.37*	.28
– 6 months	.50***	.35**	–	.24	.61**	.24	.04	.22	.19	.20
– 9 months	.22	.12	.27*	–	.16	.15	.06	-.34	.38*	.02
– 15 months	.49***	.35**	.56***	.28*	–	.12	.28	.12	.08	.00
– 18 months	.45***	.27*	.57***	.34**	.42***	–	-.59**	-.02	.51**	.60**
CASQ-R	-.36***	-.38**	-.16	-.14	-.25*	-.33**	–	.07	-.46**	-.51**
LE – Interpersonal	.11	-.04	.01	-.26	-.06	-.02	.02	–	-.30	-.32
CDI – Baseline	.48***	.34**	.22	.12	.36**	.33**	-.57***	.02	–	.49**
– 18 months	.46***	.37**	.39**	.32**	.54***	.45***	-.44***	.11	.56***	–

*p < .05; **p < .01; ***p < .001.

Note. NSSI = Nonsuicidal self-injury; CASQ-R = Children’s Attributional Style Questionnaire – Revised

LE-Interpersonal = interpersonal life events; CDI = Child Depression Inventory.

Table 3. Unconditional growth curve model fits

	χ^2	DF	χ^2/DF	CFI	NFI	RMSEA	AIC
Single Slope	62.11**	12	5.18	.65	.62	.17	92.11
Curvilinear Slope	30.98*	12	2.58	.86	.81	.10	60.98
Piecewise (Linear Spline)	9.83	9	1.09	.99	.94	.03	45.83

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

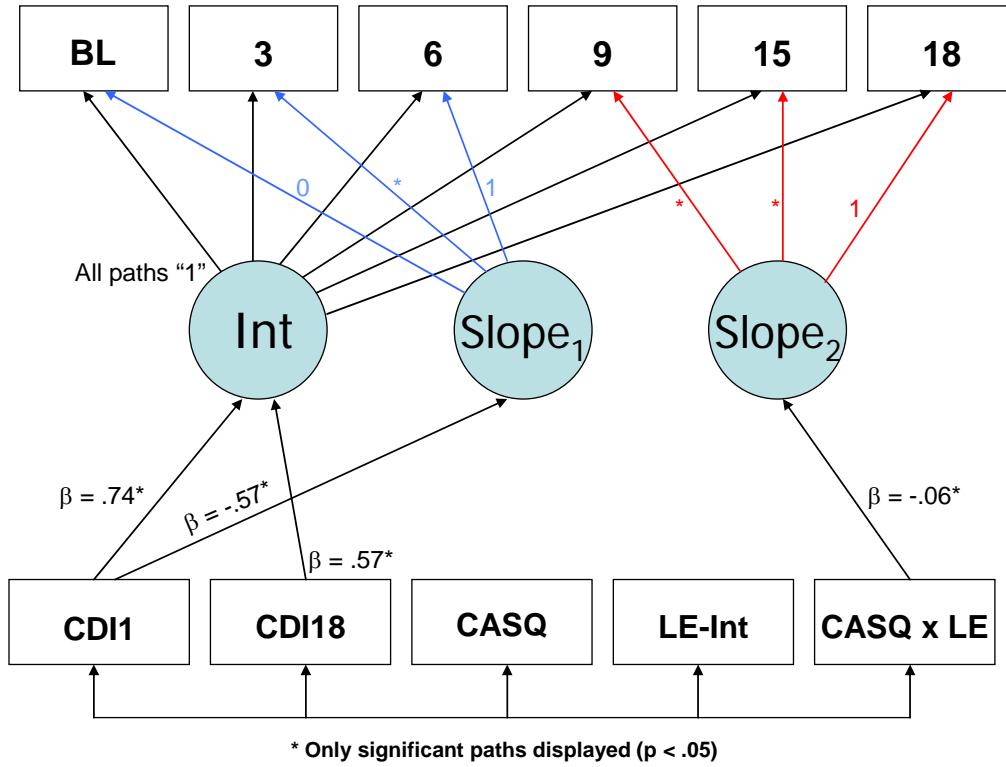
Table 4. Prediction of NSSI from exogenous predictors; Standardized Regression Weights (and Standard Errors)

	NSSI		
	Intercept	Remission	Maintenance
		Slope	Slope
Interpersonal life events (LE-int)	.03 (.05)	-.04 (.05)	-.02 (.02)
Attributional style (CASQ-R)	-.06 (.06)	.06 (.06)	-.04 (.03)
CASQ-R x LE-int	-.08 (.05)	.00 (.05)	-.06 (.03)*
Depression (CDI)			
Baseline	.74 (.20)***	-.57 (.18)**	.02 (.08)
18-months	.57 (.22)**	-.10 (.21)	.07 (.09)

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

Note. NSSI = Nonsuicidal self-injury; CASQ-R = Children's Attributional Style Questionnaire – Revised; LE-int = interpersonal life events; CDI = Child Depression Inventory.

Figure 1. Conditional growth curve model



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