Stress sensitivity interacts with depression history to predict depressive symptoms among youth: Prospective changes following first depression onset

Jessica R. Technow

Nicholas A. Hazel

John R. Z. Abela

Benjamin L. Hankin

Author Note

Jessica R. Technow, Department of Psychology, University of Denver; Nicholas A. Hazel, Department of Psychiatry, University of Colorado Denver; Benjamin L. Hankin, Department of Psychology, University of Denver.

This research was supported in part by research grants from the Social Sciences and Research Council of Canada and the National Alliance for Research on Schizophrenia and Depression awarded to John R.Z. Abela, research grants from the National Institute of Mental Health (R03-MH 066845) and the American Foundation for Suicide Prevention awarded to Benjamin L. Hankin, and T32-MH 015442 from the National Institute of Mental Health supporting Nicholas A. Hazel.

Correspondence concerning this article should be addressed to Jessica Technow, Department of Psychology, Frontier Hall, 2155 S. Race St., University of Denver, Denver, CO 80208.

Email: jessica.technow@du.edu

Abstract

Predictors of depressive symptoms may differ before and after the first onset of major depression due to stress sensitization. Dependent stressors, or those to which characteristics of individuals contribute, have been shown to predict depressive symptoms in youth. The current study sought to clarify how stressors' roles may differ before and after the first depressive episode. Adolescents (N = 382, aged 11 to 15 at baseline) were assessed at baseline and every three months over the course of two years with measures of stressors and depressive symptoms. Semi-structured interviews were conducted every 6 months to assess for clinically significant depressive episodes. Hierarchical linear modeling showed a significant interaction between history of depression and idiographic fluctuations in dependent stressors to predict prospective elevations of symptoms, such that dependent stressors were more predictive of depressive symptoms after onset of disorder. Independent stressors predicted symptoms, but the strength of the association did not vary by depression history. These results suggest a synthesis of stress sensitization and generation processes that might maintain inter-episode depressive symptoms among youth with a history of clinical depression.

Keywords: depression, stress, stress generation, adolescence

Stress sensitivity interacts with depression history to predict depressive symptoms among youth: Prospective changes following first depression onset

Over the course of their lifetimes, 20 to 25% of women and 10-17% of men experience major depression (Kessler, McGonagle, Zhao, & Nelson, 1994). Present estimates suggest that 50-60% of people who experience a first depressive episode go on to have a second one, 70-80% of those with a second episode experience a third, and 90% of individuals who have had three episodes of depression go on to suffer further recurrences (American Psychiatric Association, 2000; Solomon et al., 2000). High depressive symptoms following remission are a known risk factor for future onset (Judd et al., 1998) and thus understanding the factors that maintain elevated symptoms is critical to understanding risk for recurrence. It is well established that stressful life events portend symptom elevations (Carter & Garber, 2011; Grant et al., 2003; Hankin, Mermelstein, & Roesch, 2007) and that rates of stressful life events increase following first onset of depression (Hammen, 1991; 2006, Hammen, Hazel, Brennan, & Najman, 2012). Further, stressful life events may be particularly potent after the first onset of depression (Monroe and Harkness, 2005). Taken together, these factors provide a mechanism by which depressive symptoms may be maintained after first onset, elevating risk for recurrence. However, research has yet to distinguish between stress-symptom relationships before versus after onset of depressive disorder, and thus it is unknown which of these factors are influential in maintaining symptoms after the first onset of depression. Thus, the current study sought to clarify how the prospective relationship between stress and symptoms differs before and after the first onset of depression.

Depression Recurrence

Phenotypically, depression can be either a time-limited, acute disorder or a chronically

recurring, lifelong illness (Monroe & Harkness, 2011). Of adults who experience a first episode of major depression, approximately 60% experience a recurrence (American Psychiatric Association, 2000; Solomon et al., 2000). From a chronic illness perspective, each new instance of depression is thought to arise from underlying vulnerabilities or risk factors, such as biological vulnerabilities, or propensity to generate stress. Thus, it is possible to recover from an episode, but not from the disorder per se, since the underlying vulnerability is present even after the episode remits. The highly recurrent nature of the disorder for individuals who experience more than one episode supports this chronic illness characterization. An acute illness perspective, however, posits that a recurrence is a new instance of the illness, rather than a continuation of previous disorder (Monroe & Harkness, 2011). An individual who experiences only one depressive episode or a few episodes over many years illustrates the acute illness model. At the same time, 40% of individuals who experience a first episode never experience a second (Monroe & Harkness). Presently, few data are available to characterize the risk factors that might distinguish a chronically symptomatic, recurrence-prone individual from an individual who experiences a single lifetime episode or two etiologically distinct episodes.

Critically, the time between first onset and episode recurrence may be marked by elevated subclinical depressive symptoms, and these symptoms may represent a risk for future episodes (Beevers, Rohde, Stice, & Nolen-Hoeksema, 2007). Subclinical symptoms have been found to predict depressive relapse and recurrence (Judd et al., 1998), and residual symptoms after recovery from a depressive episode are associated with increased rates of episode relapse (Faravelli, Amboneti, Palente, & Pazagli, 1988; Simons & Thase, 1992). Judd and colleagues found that after recovery from a depressive episode, those who continued to experience depressive symptoms at weekly follow-ups were more than three times faster to relapse to

another clinically significant episode than individuals who were asymptomatic (median 1.3 years vs. 4.4 years, respectively). Thus, elucidating which factors might contribute to depressive symptom exacerbation following a first depressive episode may help to identify those who may be on their way to developing a chronic lifetime course of depression. Monroe & Harkness (2011) highlight that examining differences between individuals who continue to experience elevated symptoms after a first episode and those who do not are of particular interest and provide an efficient means of studying factors that may put individuals at risk for experiencing persistent impairment. Thus, it is imperative to elucidate risk factors for depressive symptoms in the time after a first episode, and before the recurrence occurs. Clinically, this period represents a critical interval for relapse preventive intervention. If risk factors for symptoms, and thus recurrences, are identified, interventions can be developed to target the processes that ultimately may lead to recurrent, debilitating episodes. If second depressive episodes can be prevented from occurring, a chronic course of depression may be averted.

Mid-adolescence provides an ideal age group to study differences in processes before and after first onsets of depression because it is during this time that many first onsets occur. Rates of depression prior to adolescence are relatively low, affecting only about 1% - 2% of children (Hankin et al., 1998). Thereafter, there is a large six-fold increase in depression prevalence between the ages of 15 and 18 (Costello, Erkanli, & Angold, 2006; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Hankin et al., 1998; Kessler, Avenevoli, & Merikangas, 2001) and prevalence rates remain comparably high throughout adulthood (Kessler, Chiu, Demler, & Walters, 2005). Thus, this age cohort is uniquely informative for examining predictive mechanisms for depressive symptoms in individuals after a first depressive episode, helping to identify who may be on their way to experiencing persistently elevated symptoms. However,

most studies have not differentiated youth with a history of depression from those without. Given that adult depression prevalence rates emerge during adolescence it is likely that the majority of youth in prior studies have not had a history of depression. Additionally, research has shown that adolescent-onset depression may lead to worse outcomes, such as comorbid disorder diagnosis in adulthood, more persistent adult recurrences of depression, and suicidality (Eaton et al., 2008; Jonsson et al., 2011). Thus, the current study utilized a sample of early adolescent youth at baseline (median age of 13) who were repeatedly assessed every 3 months over two years through the transition into middle adolescence when depression rates begin to surge.

Stress and Depression

Stressors play a critical role in the onset and course of depression (Grant et al., 2003; Hammen, 2006). Stressors prospectively predict increases in depressive symptoms (Eley & Stevenson, 2000; Grant et al., 2003; Hankin, 2008; Landis et al., 2007), first onset of depressive disorders (Lewinsohn, Allen, Seeley, & Gotlib, 1999), and depression relapse (Backs-McDermott, Dobson, & Jones, 2010; Tennant, 2002). The processes by which stress leads to depressive symptoms are thus likely important for understanding post-morbid symptom maintenance and risk for a more chronic course of disorder.

This is particularly true in that the contribution of life stress to depression changes with repeated episodes. Several theoretical models and empirical findings suggest that with each new depressive episode, individuals become susceptible to the onset of a new episode at increasingly lower levels of stress (stress sensitization, as reviewed by Monroe & Harkness, 2005). For example, Post (1994) posited that prior experiences of depression are encoded in the brain as "memory traces", which heighten reactivity of the stress response system, increasing the

likelihood of episodes at lower levels of stress. At a symptomatic level, this model would be consistent with a stronger predictive relationship between stress and symptoms following a depressive episode than before the first episode.

Different types of stress may also operate differently across the course of depression. Dependent stressors, such as a relationship problems or failing a test, are those events that are due, at least in part, to an individual's characteristics or behaviors, and they have been shown to be more predictive of depression than independent ("fateful"; e.g. a death in the family) stressors (Kendler, Karkowski, & Prescott, 1999). Those who have experienced an episode of depression report more dependent stressors compared to those that have not, a process known as stress generation (Hammen, 1991; Hammen, 2006). Previous studies have shown support for the stress generation model predicting depressive symptoms in children and adolescents (Clements, Aber, & Seidman, 2008; Hankin, et al., 2007; Kercher & Rapee, 2009; Kercher, Rapee, & Schniering, 2009; Shih, Abela, & Starrs, 2009; for a review of the evidence for stress generation, see Liu & Alloy, 2010). Thus, it is likely that dependent stressors play an essential role in the maintenance and exacerbation of depressive symptoms (Hammen, 1991; Joiner, Wingate, & Otamendi, 2005), and in turn may affect the course of major depression (Monroe & Harkness, 2005). Liu and Alloy (2010) further hypothesized an additional link between stress generation and stress sensitization. Specifically, following a depressive episode, individuals may become more sensitized to dependent stressors than independent stressors. Such a relationship would further enlarge the role of dependent stressors in promoting recurrence, both through greater numbers of events and through individuals' greater sensitivity to those events.

Taken together, previous research suggests that stressors likely precipitate first onsets, and stress generation processes thereafter work to maintain chronically higher levels of

dependent stress in previously depressed individuals. Moreover, the stress sensitization model would suggest that these higher rates of dependent stress would in turn lead to higher rates of depressive symptoms following first onset. Thus, both stress generation and sensitization processes likely play critical roles in determining the course of major depressive illness.

Methodological Considerations

Several methodological considerations have affected previous research in this area. Critically, most prior studies of stress and depression in youth have relied on cross-sectional or two time point panel designs. Two time point designs do not allow for rigorous study of prospective change in symptoms over time (Curran & Willoughby, 2003). Multi-wave prospective designs, though, allow for more accurate assessment of depressive symptoms that cannot be ascertained as precisely in cross-sectional or two time point designs, which require retrospective recall and can skew prevalence rates. Multi-wave prospective assessments with relatively short (e.g., six months or less) time frames are needed in order to maximize accuracy of diagnostic data (Costello, Erkanli, & Angold, 2006; Moffit et al., 2010). Furthermore, Monroe and Harkness (2011) noted that the median time to recurrence after a first lifetime episode of depression is approximately 12 months. The current study utilized a nine-wave design with follow-ups three months apart to maximize opportunities for examining post-episode fluctuations in symptoms during a time in which an individual is at a particularly high risk for recurrence. This further allowed for within-person comparisons before and after the first onset of depression, increasing power and clarifying the nature of effects.

In addition, we utilized an idiographic, as opposed to nomothetic, approach to examine the relationship between fluctuations in stressors and depressive symptoms (see Abela & Hankin, 2008; Abela, Zuroff, Ho, Adams, & Hankin, 2006). An idiographic perspective predicts that a

youth would experience increased depressive symptoms when he or she is experiencing a level of stress that is higher than his or her average, habitual level. Thus, the idiographic approach takes the context of stressor experience into account. An idiographic approach is likely to be particularly important for predicting prospective changes in depressive symptoms post-depressive onset in the context of stress sensitization. In addition to one's threshold to respond to stress being lowered after a depressive episode, there is heightened reactivity within an individual's stress response system (Post, 1994), which is most consistent with an idiographic perspective on stress.

The Current Study

The current study sought to understand how stress sensitivity predicts depressive episodes before and after the first onset of depression. Stress generation processes are likely to contribute additional dependent stressors following a depressive onset, which are in turn likely to be particularly depressogenic at that time (Liu & Alloy, 2010). The current study sought to answer these questions by investigating the interaction of history of depression and stressors (both dependent and independent).

Adolescents were recruited from two North American cities and assessed for stress and depressive symptoms every three months for two years (i.e., 9 waves of data) during the transition from early to middle adolescence when rates of clinical depression surge and stressors increase. We hypothesized that there would be an interaction between history of depression and dependent stress to predict depressive symptoms in youth, such that a history of depression would moderate and strengthen the impact of fluctuations in dependent stressors on depressive symptoms in youth. We further hypothesized that independent stressors would predict

depressive symptoms both before and after first onset, but that their effect would not be moderated by history of depression.

Method

Participants

Participants were recruited for the current study in Montreal, Quebec, Canada, and Chicago, Illinois, United States. Participants were recruited through advertisements in local newspapers and throughout the greater community at the two sites seeking participants for a study of adolescent development. The final sample consisted of 382 adolescents (225 girls) along with one of their parents (300 mothers), with 68.3 percent of participants identifying as non-Hispanic Caucasian. Abela and Hankin (2011) reported further demographic details of the two sites. Adolescents' ages at baseline ranged from 11-15 years with a median age of 13. The Montreal and Chicago samples were comparable in terms of adolescent gender composition, $\chi^2(1) = 0.42$, *ns*; adolescent age, $t(380) = 0.62$, *ns*; adolescent grade, $t(380) = 0.47$, *ns*; highest level of education completed by mother, $F(1, 380) = 2.10$, *ns*; highest level of education completed by father, $F(1, 330) = 0.62$, *ns*; and family income, $F(1, 380) = 0.24$, *ns*. However, the Chicago sample consisted of a greater proportion of ethnic minority youth, $\chi^2(1) = 17.36$, $p < .001$, and youth from single-parent households, $\chi^2(1) = 8.84$, $p < .01$. At the initial assessment, 58 adolescents met diagnostic criteria for a lifetime history of a clinically significant depressive episode. There have been only three other publications from the current data set (Abela & Hankin, 2011; Hankin & Abela, 2011; Stone, Hankin, Gibb, & Abela, 2011), which examined rumination, nonsuicidal self-injury, and corumination respectively. Thus, none of the prior publications examined the interaction presented in the current study.

Procedure

Phase one of the study involved a baseline laboratory assessment. Two research assistants met with one adolescent-parent pair at a time. Parents completed a consent process and adolescents provided their assent. Parents also completed a demographics questionnaire, which included a question on ethnicity, and were asked to report their annual familial income on a scale of one to eight in $15,000 increments. A research assistant verbally administered the Children's Depression Inventory (CDI; Kovacs, 2003) and Adolescent Life Events Questionnaire (ALEQ; Hankin & Abramson, 2002) aloud to the adolescents while the adolescent responded using his/her own copies. A trained diagnostician interviewed the child and parent separately to ascertain the adolescent's current and past depressive symptoms and depressive episode occurrence using the Schedule for Affective Disorders and Schizophrenia in School-Age Children (K-SADS; Kaufman, Birmaher, Brent, Rao, & Ryan, 1996).

Phase two of the study involved a series of eight telephone follow-up assessments, which occurred every three months for the two years following the initial assessment. At each time point, a research assistant verbally administered the CDI and the ALEQ to adolescents. At the six-, 12-, 18-, and 24-month follow-up assessments, a diagnostician obtained information regarding the adolescent's depressive symptoms during the past six months from both the parent and adolescent using the K-SADS. If a parent-adolescent pair missed one of the follow-up assessments in which the K-SADS was administered, information pertaining to that time interval was obtained in the subsequent K-SADS administration. Parents and adolescents were compensated $200 for participating in the study. The average number of follow-up assessments completed by participants was 6.74 (*SD* = 1.61). The number of follow-up assessments completed by participants was distributed as follows: .8% (*n* = 3) none, 1.6% (*n* = 6) one, 1.8%

(*n* = 7) two, 1.0% (*n* = 4) three, 3.1% (*n* = 12) four, 6.0% (*n* = 23) five, 15.7% (*n* = 60) six, 30.4% (*n* = 116) seven, and 39.5% (*n* = 151) eight. The number of follow-up assessments completed was not significantly associated with any of the following variables at Time 1: age (*r* = -.07, *p* = .17), gender (*r* = -.03, *p* = .57), and depressive symptoms (*r* = - .09, *p* = .07).

Measures

Schedule for Affective Disorders and Schizophrenia for School-Age Children, Present Version (K-SADS, Kaufman et al., 1996). The K-SADS is a semi-structured clinical interview designed to arrive at DSM-IV (American Psychiatric Association, 2000) diagnoses. The K-SADS was administered separately to the adolescent and the parent and was used to obtain information about depressive episode experience. A severity score was computed by summing the severity ratings (1 to 3, with 3 indicating clinical level severity) for each symptom. A best estimate diagnosis was based on sets of information from both the parent and youth interviews. The interviewer interpreted the adolescent and parent reports and arrived at an estimate that best captured the adolescent's psychopathology. The K-SADS has been shown to yield reliable diagnoses of depressive disorders (Chambers et al., 1985) and is frequently used in clinical studies of depression in youth. In the current study, current and past histories of depressive episodes were assessed, and a positive history of depression indicated best estimate report of a definite or probable major depressive episode or a definite or probable minor depressive episode. Diagnostic interviewers completed an intensive training program for administering the K-SADS and for assigning DSM-IV diagnoses, which consisted of approximately 80 hours of didactic instruction, listening to audio taped interviews, conducting practice interviews, and passing regular exams (85% or above). The principal investigators at

each site held weekly supervision sessions for the interviewers and reviewed interviewers' notes and tapes.

Child Depression Inventory (CDI; Kovacs, 2003). The CDI is a 27-item self-report questionnaire that measures the cognitive, affective, and behavioral symptoms of depression. Adolescents report on depressive symptom presence and severity for the past week. Items are scored from 0-2 with higher scores reflecting greater symptom severity. Total scores range from 0 to 54. The CDI has a high level of internal consistency (alpha from .87 to .91 across administrations for the current study) and can distinguish youth with major depressive disorder from non-depressed youth (Saylor, Finch, Spirito, & Bennet, 1984).

Adolescent Life Events Questionnaire (ALEQ; Hankin & Abramson, 2002). The ALEQ assesses the occurrence of a broad range of negative events typically reported by adolescents including school problems (e.g., *You got a bad grade on a progress report*), relationship difficulties (e.g., *You found out your boyfriend/girlfriend was cheating on you*), and family problems (e.g., *You had an argument with a close family member (parent, sibling)*). Each of the 57 events is rated for occurrence during the previous three months on a Likert scale ranging from "never" to "always". Reliability and validity for the ALEQ have been established (Auerbach, Abela, Zhu, & Yao, 2010; Hankin, 2008; Hankin, Stone, & Wright, 2010). For the current study, the ALEQ was broken down into scales of independent and dependent events. Two raters trained in coding dependence of stressors using the UCLA Life Stress Interview (Hammen et al., 1987) independently coded each item on the ALEQ as likely dependent or independent. The measure was thus broken down into two separate scales of independent ($ALEQ_{ind}$; 20 items; e.g. *Your parent lost a job*) and dependent ($ALEQ_{dep}$; 33 items; e.g. *Getting punished by parents*) stressors. Initial rater agreement for dependence of items was good (κ =

.89) and disagreements were resolved by consensus. This classification of ALEQ items into dependent and independent events has been used previously (e.g., Auerbach & Ringo-Ho, 2012; Hankin et al., 2010).

Results

Statistical Approach

Hierarchical linear modeling was used to investigate the main hypothesis that there would be an interaction between history of depression and within-youth fluctuations in dependent stressors to predict depressive symptoms in adolescents. CDI was the criterion variable. A person-mean $ALEQ_{dep}$ variable was created by calculating each participant's $ALEQ_{dep}$ mean across all available waves of data. The $ALEQ_{dep}$ predictor variable was created by centering each participant's $ALEQ_{dep}$ scores at their individual person-mean to examine the effects of idiographic fluctuations in stressors. Corresponding $ALEQ_{ind}$ variables were also created. A lagged CDI score was also created as the CDI score at the most recently available time point prior to the current time point. To covary for ethnicity, a dummy variable was used to indicate whether the parent reported the child's ethnicity as non-Hispanic Caucasian. There was not enough power to examine ethnicity in more detail.

The history of depression variable was coded as a dichotomous time-varying variable. Youth received a score of 0 for history of depression when no past or current clinically significant episode had been reported. Youth continued to have a score of 0 for history of depression until an episode was reported. If the best estimate report of depression revealed a clinically significant episode, youth were assigned a 1 for the history of depression variable beginning at that time point. From that time point forward, youth continued to have a 1 for

history of depression. The 58 youth who had histories of depression at baseline had a score of 1 for the entire study.

All analyses were conducted in HLM 7.0 using restricted maximum likelihood estimation and unstructured effect covariance matrices. At level 1, depressive symptoms were modeled as a function of person-centered stressors ($ALEQ_{dep}$, $ALEQ_{ind}$) and history of a depressive episode (Hx), and their interaction. All analyses also controlled for the previous time point's CDI score in order to evaluate the role of stressors on strictly prospective changes in CDI scores. For example:

$$CDI_{ti} = \beta_{0i} + \beta_{1i}(ALEQ_{depti}) + \beta_{2i}(Hx_{ti}) + \beta_{3i}(ALEQ_{dep} \times Hx_{ti}) + \beta_{4i}(LagCDI) + e_{ti}$$

At level 2, mean levels of depressive symptoms were predicted by sex (1 = female; 0 = male), person-mean levels of stress, family income, and self-reported ethnicity. For example:

$$\beta_{0i} = \gamma_{00} + \gamma_{01}\,(Sex) + \gamma_{02}(Person\text{-}mean\ ALEQ_{dep}) + \gamma_{03}\,(Income) + \gamma_{04}\,(Ethnicity) + r_{0i}$$

$$\beta_{1i} = \gamma_{10} + r_{1i}$$

…

$$\beta_{4i} = \gamma_{40} + r_{4i}$$

Descriptive Statistics

Of the 382 adolescents, 115 (30.1%) had an onset of at least one depressive episode during the course of the study. This is commensurate with epidemiological estimates of depression incidence in that age range (Kessler et al., 2005; 1994). Figure 1 illustrates the distribution of depressive symptoms, stressors, and cumulative depression prevalence across the study. Dependent and independent stressors were correlated over time; *r*'s ranged from .65 to .71, consistent with prior research using contextual stress interviews (e.g. Conway, Hammen, & Brennan, 2012).

Additionally, analyses were conducted to ensure that symptom scores predict depressive episodes in the current sample. Children's Depression Inventory scores lagged from the previous time point predicted major depressive episodes (b= .01, p < .004), minor depressive episodes (b = .004, p = .014), as well as a composite of both major and minor depressive episodes (b = .076, p < .001).

Stressor Occurrence Before and After First Onset of Depression

Average pre-morbid dependent and independent stressor scores were calculated for individuals who experienced a first onset of depression during the course of the study. These scores were compared to average stress scores of individuals who had no history of depression at the end of the study, controlling for gender (which was not a significant predictor in either analysis, p's > .12). Individuals who had a depressive episode had, on average, 6.1 more dependent events at each time point prior to onset than individuals who completed the study without an episode (SE = 1.34, p < .01). Individuals who experienced a first onset also had nearly two more independent events at each pre-morbid time point (b = 1.96, SE = 0.58, p < .01) than was the average amongst individuals who finished the study without an episode.

Consistent with the stress generation perspective, separate mixed effects models revealed that, after controlling for participant sex, a positive history of diagnosis predicted higher rates of dependent stressors (b = 1.47, SE = 0.49, p < .01), but not independent stressors (b = 0.21, SE = 0.20, p = .29).

Stressor Impact as a Function of Depression History

Table 1 shows the results of three mixed models in which prospective increases in youth depressive symptoms were predicted by person-centered stressors, history of depression, and their interactions, while controlling for demographic variables, prior CDI, and average levels of

stressors. The first column presents the results using both dependent and independent stressors. Lower family income (γ_{04}) and higher average levels of dependent stressors (γ_{02}) were associated with greater rates of depressive symptoms. History of depression was a significant predictor of higher depressive symptoms ($\gamma_{10} = 1.30$, $p < .01$), though the random effect suggests nearly significant individual differences in mean differences with or without a history of depression ($r_1 = 10.44$, $p = .09$). Critically, history of depression moderated the effect of dependent stress ($\gamma_{40} = 0.20$, $p = .03$), such that individuals who had experienced a history of depression showed greater increases in depressive symptoms following dependent stressors. This effect is illustrated in Figure 2. On average, the depressogenic effects of dependent stressors were twice as high for those with a history of depression ($\gamma_{20} + \gamma_{40} = 0.38$) as for those without ($\gamma_{20}$). Moreover, the nearly significant random effect for the interaction term ($r_4 = 0.49$, $p = .09$) suggests the presence of significant unaccounted for variance in the moderating influence of depression history.

History of depression did not moderate independent stress ($\gamma_{50} = 0.06$, $p = .73$), and there was not a significant difference in the extent to which depression moderated each type of stress ($\gamma_{40} - \gamma_{50} = .14$, $\chi^2(1) = 0.40$, $p > .50$). Notably, after controlling for the other effects in the model, there was not a significant main effect for independent stressors ($\gamma_{30} > -0.01$, $p = .95$).

Because rates of dependent and independent stressor exposure are correlated, the second and third models in Table 1 present separate models using only dependent stressors or independent stressors. As in the joint model, fluctuations in dependent stressors were a significant predictor of depressive symptoms (γ_{20}), and their effects were moderated by history of depression (γ_{40}). In the rightmost column of Table 1, idiographic fluctuations in independent stressors did not interact with history of depression ($\gamma_{50} = 0.14$, $p = .35$), as in the joint model.

But unlike in the joint model, independent stressors were significant predictors of depressive symptoms ($\gamma_{30} = 0.20$, $p < .01$).

Stressor Impact Pre- and Post-First Onset

To clarify whether the model was strictly relevant to within-person changes in stress reactivity, the model was fit to the subset of participants who had their first onset of depression during the course of the study. Of those individuals who entered the study without a history of depression, 57 had first onsets of depression during the two years of the study and were missing no data at level 2. As in the whole sample, three models were estimated. Joint analyses that included both dependent and independent stressors are shown in the first column of Table 2 and were largely consistent with the model in the whole sample. Unlike in the whole sample, history of depression did not significantly predict higher levels of symptoms ($\gamma_{10} = -0.76$, $p = .17$). As in the whole sample, however, fluctuations in dependent stressors significantly predicted symptoms ($\gamma_{20} = 0.21$, $p < .01$), whereas independent stressors did not ($\gamma_{30} = 0.11$, $p = .47$), though the difference between the two effects was not significant ($\gamma_{20} - \gamma_{30} = .10$, $\chi^2(1) = 0.25$, $p > .50$). Most importantly, onset of depressive episodes during the two years of the study moderated the effects of dependent ($\gamma_{40} = 0.29$, $p = .04$), but not independent ($\gamma_{50} = 0.05$, $p = .84$), stressors, but the difference between the two moderating effects was not significant ($\gamma_{40} - \gamma_{50} = .24$, $\chi^2(1) = 0.44$, $p > .50$). There was little individual variation left unexplained for any level-1 predictors.

Separate analyses of dependent and independent stressors in this subsample largely confirmed the findings of previous analyses. Dependent stressors were significant predictors of depressive symptoms before ($\gamma_{20} = 0.23$, $p < .01$) first onset, and their association with symptoms became stronger after first onset ($\gamma_{30} = 0.29$, $p < .01$). By contrast, the results for independent stressors are displayed in the last columns of Table 3. As in the whole sample, the impact of

independent stressors was significant (γ_{30} = 0.33, p = .04), but not significantly different before and after the first onset of depression (γ_{50} = 0.29, p = .25).

Discussion

The current study sought to determine whether the processes by which stress promotes depressive symptoms differ before and after the first onset of major depression by following youth for two years through the transition from early into middle adolescence when many individuals experience their first depressive episode. Specifically, we hypothesized that there would be an interaction between history of depression and dependent stressors to predict prospective changes in depressive symptoms in youth. Results supported this hypothesis, such that youth with a history of a depressive episode showed a stronger association between person-centered dependent stressor fluctuations and depressive symptoms. This effect was not found for independent stressors.

Importantly, the major findings within the full sample held within the subsample of youth who did not enter the study with a history of depression, but who experienced a first depressive episode by the study's end. Examining the hypothesis that prior depression interacts with dependent stressors to predict later symptoms with this more homogenous sample of youth who developed a first depressive episode during the two years of the study follow-up provided a stringent test of this interaction. This finding adds to the growing literature showing that dependent stressors are more predictive of depressive symptoms and major depressive episodes than independent stressors (Kendler et al., 1999). The present findings extends this prior knowledge in new ways by demonstrating that this predictive ability is even greater among adolescents who have had an onset of major depression. Taken together, findings suggest that idiographic changes in dependent stressors play a substantial role in maintaining post-episode

depressive symptoms. Importantly, all analyses in the present study controlled for the effects of the previous measure of depressive symptoms, resulting in a strictly prospective test.

The current study extends the literature on the role of dependent stress by noting that history of depression increases the strength of the relationship between dependent stress and prospective change in depressive symptoms. Results suggest that the experience of a depressive episode alters the way youth react to dependent stressors, such that a lower number of stressors result in symptom elevations over time after onset of disorder. This finding is consistent with the stress sensitization hypothesis (Monroe & Harkness, 2005), which posits that after an episode, an individual can succumb to a recurrent depressive episode from lower severity levels of stress. The high rate of dependent stressors after a depressive episode and the increased reactivity to those stressors points to an increased liability for the production of depressive symptoms. Interestingly, there was significant variability in the random effect after depressive episode onset, underscoring that there is considerable between-persons variation in depressive symptoms following a first depressive episode.

Strengths and Limitations

Several strengths of the current study should be noted. First, the multi-wave design allowed for more accurate assessment of depressive symptoms and episodes. The current study assessed youth depressive symptoms every three months and depressive episode occurrence every six months, allowing for more accurate and precise measurement of youth depression experience (Costello et al., 2006). Additionally, use of the K-SADS to assess diagnostically significant episodes of depression assures accuracy of measurement of episodes meeting diagnostic criteria. Finally, the use of idiographically measured stressors resulted in a person-focused analysis that hews closely to common sense notions of the role of stress in symptom

formation, in which symptom fluctuations are more closely tied to increases and decreases in stress across time, rather than absolute levels.

Several limitations of the current study should be noted. First, self-report measures were used to assess stressors. Self-report measures, however, are not without worth, as individuals are capable of reporting on their personal histories and, thus, stressor experience (Haeffel & Howard, 2010). This is particularly true in regard to ALEQ, which was designed to assess relatively objective, discrete events with a high degree of reliability. Additionally, there was a significant correlation between dependent and independent stressors. This same correlation has been observed when coding checklists elsewhere (e.g., daily events in Sahl, Cohen, & Dasch, 2009) or when using contextual stress interviews (e.g., with dependent interpersonal events in Conway et al., 2012), but is not necessarily observed in interviews over longer time periods (e.g., the UCLA Contextual Stress Interview over one year in Rudolph & Hammen, 1999, or with non-interpersonal dependent events in Conway et al., 2012). There are several possible reasons for this correlation, including a possible reporting bias or the fact that a lack of context could lead to an inflation of the correlation. It is also theoretically likely that during times of high independent stress, a person may engage in behaviors that generate more dependent stressors (e.g., an adolescent engages in more conflict with their parents when physically ill). Notably, such linkages of independent and dependent stress would be particularly pronounced over short time periods (as in the current study) that allows for tighter temporal coupling of stressors, but less noticeable over longer time periods (such as those commonly assessed by interviews) due to averaging over time. Nonetheless, contextual stress interviews remain the gold standard of stress assessment (Monroe, 2008), especially with regard to stressor timing and distinguishing acute stressors from ongoing conditions.

A second limitation is that the data extracted from the interview measures and the analytic methods cannot perfectly, clearly define when depressive episodes ended, and hence post-onset time points may include data collected while an adolescent was currently depressed or just briefly in remission from an episode. Yet this possible limitation seems mostly mitigated by prior research showing that the median duration of adolescent depressive episodes is approximately 8 weeks in community samples (Lewinsohn, Clarke, Seeley, Rohde, 1994; Rao, Hammen, Daley, 1999), so most episodes would be expected to have resolved by the next data collection point (i.e., 6 months between interview assessment periods).

Finally, episodes of probable minor depression were included in the depression history variable, as minor depressive episodes have been demonstrated to cause impairment in functioning in youth (Gonzalez-Tejera et al., 2005; Sihvola et al., 2007). This may suggest that the current findings are not strictly limited to diagnoses of major depression, per se. Also, the current study also did not address dysthymia, in which depressive symptoms do not necessarily portend a depressive episode or recurrence. Future research should consider the mechanisms of sensitization in probing the diagnostic boundaries of the effect. Lastly, the current study examined depressive symptoms before and after onset of disorder, but did not address how symptoms lead to a recurrence of depression. Adequately powered future studies should extend these findings to incorporate heightened symptoms' role in future episodes.

Conclusion

The current study examined how experiencing a depressive episode alters predictive mechanisms of youth depressive symptoms. Specifically, the current study demonstrated that the association between idiographic dependent stressor fluctuations and depressive symptoms increases in strength following a first episode. The current study underscores the importance of

distinguishing symptom prediction before or after onset of disorder, as the predictive mechanisms clearly change over the course of the first episode. Through better attention to these processes we will hopefully better understand how depressive symptoms are maintained, creating risk for future episode onset. Future research needs to examine how risk factors for a first episode may differ from those that put youth at risk for a more chronic and debilitating course of disorder, as there are important clinical implications for identifying these differing risk factors.

References

Abela, J.R.Z., & Hankin, B.L. (2008). Cognitive vulnerability to depression in children and adolescents. In J.R.Z. Abela & B.L. Hankin (Eds.), *Handbook of depression in children and adolescents* (pp. 35-78). New York, NY: Guilford Press.

Abela, J. Z., & Hankin, B. L. (2011). Rumination as a vulnerability factor to depression during the transition from early to middle adolescence: A multiwave longitudinal study. *Journal of Abnormal Psychology, 120*(2), 259-271. doi:10.1037/a0022796

Abela, J. Z., Zuroff, D. C., Ho, M. R., Adams, P., & Hankin, B. L. (2006). Excessive Reassurance Seeking, Hassles, and Depressive Symptoms in Children of Affectively Ill Parents: A Multiwave Longitudinal Study. *Journal of Abnormal Child Psychology, 34*(2), 171-187. doi:10.1007/s10802-005-9011-x

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., txt. rev.). Washington, DC: Author.

Auerbach, R.P., Abela, J.R.Z., Zhu, X., & Yao, S. (2010). Understanding the role of coping in the development of depressive symptoms: Symptom specificity, gender differences, and cross-cultural applicability. *British Journal of Clinical Psychology, 49*(4), 547-561. doi:10.1348/014466509X479681

Auerbach, R. P., & Ringo-Ho, M. H. (2012). A cognitive-interpersonal model of adolescent depression: The impact of family conflict and depressogenic cognitive styles. *Journal of Clinical Child and Adolescent Psychology, 41*(6), 792-802. doi:10.1080/15374416.2012.727760

Backs-Dermott, B. J., Dobson, K. S., & Jones, S. L. (2010). An evaluation of an integrated model of relapse in depression. *Journal of Affective Disorders, 124*(1-2), 60-67.

doi:10.1016/j.jad.2009.11.015

Beevers, C. G., Rohde, P., Stice, E., & Nolen-Hoeksema, S. (2007). Recovery from major depressive disorder among female adolescents: A prospective test of the scar hypothesis. *Journal of Consulting and Clinical Psychology, 75*(6), 888-900. doi:10.1037/0022-006X.75.6.888

Carter, J., & Garber, J. (2011). Predictors of the first onset of a major depressive episode and changes in depressive symptoms across adolescence: Stress and negative cognitions. *Journal of Abnormal Psychology, 120*(4), 779-796. doi:10.1037/a0025441

Chambers, W.J., Puig-Antich, J., Hirsch, M., Paez, P., Ambrosini, P., Tabrizi, M., & Davies, M. (1985). The assessment of affective disorders in children and adolescents by semi-structured interview: Test-retest reliability of the Schedule for Affective Disorders and Schizophrenia for School-Age Children, Present Episode Version. *Archives of General Psychiatry, 42,* 696-702.

Clements, M., Aber, J., & Seidman, E. (2008). The dynamics of life stressors and depressive symptoms in early adolescence: A test of six theoretical models. *Child Development, 79*(4), 1168-1182. doi:10.1111/j.1467-8624.2008.01182.x

Conway, C. C., Hammen, C., & Brennan, P. A. (2012). Expanding stress generation theory: Test of a transdiagnostic model. *Journal of Abnormal Psychology, 121*(3), 754-766. doi:10.1037/a0027457

Costello, E.J., Erkanli, A., & Angold, A. (2006). Is there an epidemic of child or adolescent depression?. *Journal of Child Psychology and Psychiatry, 47*(12), 1263-1271. doi:10.1111/j.1469-7610.2006.01682.x

Costello, E.J., Mustillo, S., Erkanli, A., Keeler, G., & Angold, A. (2003). Prevalence and development of psychiatric disorders in childhood and adolescence. *Archives of General Psychiatry, 60*(8), 837-844. doi:10.1001/archpsyc.60.8.837

Curran, P.J., & Willoughby, M.T. (2003). Implications of latent trajectory models for the study of developmental psychopathology. *Development and Psychopathology*, *15*(3), 581-612. doi:10.1017.S0954579403000300

Eaton, W. W., Shao, H., Nestadt, G., Lee, B., Bienvenu, O., & Zandi, P. (2008). Population-based study of first onset and chronicity in major depressive disorder. *Archives of General Psychiatry, 65*(5), 513-520. doi:10.1001/archpsyc.65.5.513

Eley, T. C., & Stevenson, J. (2000). Specific life events and chronic experiences differentially associated with depression and anxiety in young twins. *Journal of Abnormal Child Psychology, 28*(4), 383-394. doi:10.1023/A:1005173127117

Faravelli, C., Amboneti, A., Palente, S., Pazagli, A., (1988). Depressive relapse and incomplete recovery from index episode. *American Journal of Psychiatry, 143,* 886-891.

Grant, K. E., Compas, B. E., Stuhlmacher, A. F., Thurm, A. E., McMahon, S. D., & Halpert, J. A. (2003). Stressors and child and adolescent psychopathology: Moving from markers to mechanisms of risk. *Psychological Bulletin, 129*(3), 447-466. doi:10.1037/0033-2909.129.3.447

González-Tejera, G., Canino, G., Ramirez, R., Chávez, L., Shrout, P., Bird, H., ... & Bauermeister, J. (2005). Examining minor and major depression in adolescents. *Journal of child Psychology and Psychiatry*, *46*(8), 888-899.

Haeffel, G.J., & Howard, G.S. (2010). Self-report: Psychology's four-letter word. *The American Journal of Psychology, 123*(2), 181-188.

Hammen, C. (1991). Generation of stress in the course of unipolar depression. *Journal of Abnormal Psychology, 100*(4), 555-561. doi:10.1037/0021-843X.100.4.555

Hammen, C. (2006). Stress Generation in Depression: Reflections on Origins, Research, and Future Directions. *Journal Of Clinical Psychology, 62*(9), 1065-1082. doi:10.1002/jclp.20293

Hammen, C., Adrian, C., Gordon, D., Burge, D., Jaenicke, C., & Hiroto, D. (1987). Children of depressed mothers: Maternal strain and symptom predictors of dysfunction. *Journal of Abnormal Psychology, 96*(3), 190-198. doi:10.1037/0021-843X.96.3.190

Hammen, C. C., Hazel, N. A., Brennan, P. A., & Najman, J. J. (2012). Intergenerational transmission and continuity of stress and depression: Depressed women and their offspring in 20 years of follow-up. *Psychological Medicine, 42*(5), 931-942. doi:10.1017/S0033291711001978

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*(7), 999-1014. doi:10.1007/s10802-008-9228-6

Hankin, B. L., & Abela, J. Z. (2011). Nonsuicidal self-injury in adolescence: Prospective rates and risk factors in a 2 ½year longitudinal study. *Psychiatry Research, 186*(1), 65-70. doi:10.1016/j.psychres.2010.07.056

Hankin, B.L., & Abramson, L.Y. (2002). Measuring cognitive vulnerability to depression in adolescence: Reliability, validity, and gender differences. *Journal of Clinical Child and Adolescent Psychology, 31,* 491-504. doi:10.1207/153744202320802160

Hankin, B.L., Abramson, L.Y., Moffitt, T.E., Silva, P.A., McGee, R., & Angell, K.E. (1998). Development of depression from adolescence to young adulthood: Emerging gender

differences in a 10-year longitudinal study. *Journal of Abnormal Psychology, 107*(1), 128-140.

Hankin, B.L., Mermelstein, R., & Roesch, L. (2007). Sex differences in adolescent depression: stress exposure and reactivity models. *Child Development, 78*(1), 279-295. doi:10.1111/j.1467-8624.2007.00997.x

Hankin, B.L., Stone, L., & Wright, P.A. (2010). Corumination, interpersonal stress generation, and internalizing symptoms: Accumulating effects and transactional influences in a multiwave study of adolescents. *Development and Psychopathology, 22*(1), 217-235. doi:10.1017/S0954579409990368

Joiner, T. R., Wingate, L. R., & Otamendi, A. (2005). An interpersonal addendum to the hopelessness theory of depression: Hopelessness as a stress and depression generator. *Journal of Social and Clinical Psychology, 24*(5), 649-664. doi:10.1521/jscp.2005.24.5.649

Jonsson, U. U., Bohman, H. H., von Knorring, L. L., Olsson, G. G., Paaren, A. A., & von Knorring, A. (2011). Mental health outcome of long-term and episodic adolescent depression: 15-year follow-up of a community sample. *Journal of Affective Disorders, 130*(3), 395-404. doi:10.1016/j.jad.2010.10.046

Judd, L. L., Akiskal, H. S., Maser, J. D., Zeller, P. J., Endicott, J., Coryell, W., & ... Keller, M. B. (1998). Major depressive disorder: A prospective study of residual subthreshold depressive symptoms as predictor of rapid relapse. *Journal of Affective Disorders, 50*(2-3), 97-108. doi:10.1016/S0165-0327(98)00138-4

Kaufman, J., Birmaher, B., Brent, B., Rao, U., & Ryan, N. (1996). The Schedule for Affective Disorders for School-Age Children. University of Pittsburgh Medical Center.

Kendler, K. S., Karkowski, L. M., & Prescott, C. A. (1999). Causal relationship between stressful life events and the onset of major depression. *The American Journal of Psychiatry, 156*(6), 837-848.

Kercher, A., & Rapee, R. M. (2009). A test of a cognitive diathesis—Stress generation pathway in early adolescent depression. *Journal of Abnormal Child Psychology, 37*(6), 845-855. doi:10.1007/s10802-009-9315-3

Kercher, A. J., Rapee, R. M., & Schniering, C. A. (2009). Neuroticism, life events and negative thoughts in the development of depression in adolescent girls. *Journal of Abnormal Child Psychology, 37*(7), 903-915. doi:10.1007/s10802-009-9325-1

Kessler, R.C., Avenevoli, S., & Merikangas, K.R. (2001). Mood disorders in children and adolescents: An epidemiological perspective. *Biological Psychiatry, 49*(12), 1002-1014. doi: 10.1016/S0006-3223(01)01129-5

Kessler, R.C., Chiu, W.T., Demler, O., & Walters, E.E. (2005). Prevalence, severity, & comorbidity of 12-month DSM-IV disorders in the National Comorbidity Study replication. *Archives of General Psychiatry, 62*(6), 617-627. doi:10.1001/archpsyc.62.6.617

Kessler, R. C., McGonagle, K. A., Zhao, S., & Nelson, C. B. (1994). Lifetime and 12-month prevalence of DSM-III—R psychiatric disorders in the United States: Results from the National Comorbidity Study. *Archives of General Psychiatry, 51*(1), 8-19.

Kovacs, M. (2003). Children's Depression Inventory: Technical Manual. Toronto: Multi-Health Systems.

Landis, D., Gaylord-Harden, N. K., Malinowski, S. L., Grant, K. E., Carleton, R. A., & Ford, R. E. (2007). Urban adolescent stress and hopelessness. *Journal of Adolescence, 30*(6), 1051-1070. doi:10.1016/j.adolescence.2007.02.001

Lewinsohn, P. M., Allen, N. B., Seeley, J. R., & Gotlib, I. H. (1999). First onset versus recurrence of depression: Differential processes of psychosocial risk. *Journal of Abnormal Psychology, 108*(3), 483-489. doi:10.1037/0021-843X.108.3.483

Lewinsohn, P. M., Clarke, G. N., Seeley, J. R., & Rohde, P. (1994). Major depression in community adolescents: Age at onset, episode duration, and time to recurrence. *Journal of the American Academy of Child & Adolescent Psychiatry, 33*(6), 809-818. doi:10.1097/00004583-199407000-00006

Liu, R. T., & Alloy, L. B. (2010). Stress generation in depression: A systematic review of the empirical literature and recommendations for future study. *Clinical Psychology Review, 30*(5), 582-593. doi:10.1016/j.cpr.2010.04.010

Moffit, T.E., Caspi, A., Taylor, A., Kokaua, J., Milne, B.J., Polanczyk, G., & Poulton, R. (2010). How common are common mental disorders? Evidence that lifetime prevalence rates are doubled by prospective versus retrospective ascertainment. *Psychological Medicine, 40,* 899-909. doi:10.1017/S0033291709991036

Monroe, S. M. (2008). Modern approaches to conceptualizing and measuring human life stress. *Annual Review Of Clinical Psychology*, 433-52. doi:10.1146/annurev.clinpsy.4.022007.141207

Monroe, S. M., & Harkness, K. L. (2005). Life stress, the 'kindling' hypothesis, and the recurrence of depression: Considerations from a life stress perspective. *Psychological Review, 112*(2), 417-445. doi:10.1037/0033-295X.112.2.417

Monroe, S. M., & Harkness, K. L. (2011). Recurrence in major depression: A conceptual analysis. *Psychological Review, 118*(4), 655-674. doi:10.1037/a0025190

Post, R. M. (1994). Mechanisms underlying the evolution of affective disorders: Implications for long-term treatment. In L. Grunhaus & J. F. Greden. Eds., *Severe depressive disorders. Progress in psychiatry* (pp. 23– 65). Washington, DC: American Psychiatric Association.

Rao, U., Hammen, C., & Daley, S. E. (1999). Continuity of depression during the transition to adulthood: A 5-year longitudinal study of young women. *Journal of the American Academy of Child & Adolescent Psychiatry, 38*(7), 908-915. doi:10.1097/00004583-199907000-00022

Rudolph, K. D., & Hammen, C. (1999). Personality and social development: Age and gender as determinants of stress exposure, generation, and reactions in youngsters: A transactional perspective. *Child Development, 70*(3), 660-677.

Sahl, J. C., Cohen, L. H., & Dasch, K. B. (2009). Hostility, interpersonal competence, and daily dependent stress: A daily model of stress generation. *Cognitive Therapy Research, 33,* 199-210. doi:10.1007/s10608-007-9175-5

Saylor, C.F., Finch, A.J., Spirito, A., & Bennett, B. (1984). The Children's Depression Inventory: A systematic evaluation of psychometric properties. *Journal of Consulting and Clinical Psychology, 52,* 955-967.

Shih, J. H., Abela, J. R. Z., & Starrs, C. (2009). Cognitive and interpersonal predictors of stress generation in children of affectively ill parents. *Journal of Abnormal Child Psychology, 37,* 195-208. doi:10.1007/s10802-008-9267-z

Sihvola, E., Keski-Rahkonen, A., Dick, D. M., Pulkkinen, L., Rose, R. J., Marttunin, M., & Kaprio, J. (2007). Minor depression in adolescence: Phenomenology and clinical correlates. *Journal of Affective Disorders, 97,* 211-218. doi:10.1016/j.jad.2006.06.019

Simons, A. D., & Thase, M. E. (1992). Biological markers, treatment outcome, and 1-year follow-up in endogenous depression: Electroencephalographic sleep studies and response to cognitive therapy. *Journal of Consulting and Clinical Psychology, 60*(3), 392-401. doi:10.1037/0022-006X.60.3.392

Solomon, D. A., Keller, M. B., Leon, A. C., Mueller, T. I., Lavori, P. W., Shea, M., & ... Endicott, J. (2000). Multiple recurrences of major depressive disorder. *The American Journal of Psychiatry, 157*(2), 229-233.

Stone, L. B., Hankin, B. L., Gibb, B. E., & Abela, J. Z. (2011). Co-rumination predicts the onset of depressive disorders during adolescence. *Journal Of Abnormal Psychology*, *120*(3), 752-757. doi:10.1037/a0023384

Tennant, C. (2002). Life events, stress and depression: A review of the findings. *Australian and New Zealand Journal of Psychiatry, 36*(2), 173-182. doi:10.1046/j.1440-1614.2002.01007.x

Table 1

Hierarchical Linear Models of Children's Depression Inventory scores in the Whole Sample (n = 325)

	Both dependent and independent stressors	Only dependent stressors	Only independent stressors
	Estimate (*SE*)	Estimate (*SE*)	Estimate (*SE*)
		Fixed Effects	
Intercept, γ_{00}	3.85* (0.49)	3.85* (0.49)	5.04* (0.38)
Sex, γ_{01}	0.27 (0.24)	0.30 (0.25)	0.39 (0.24)
Person-mean dependent stressors, γ_{02}	0.15* (0.06)	0.13* (0.04)	
Person-mean independent stressors, γ_{03}	-0.06 (0.13)		0.37* (0.09)
Family income, γ_{04}	-0.16* (0.06)	-0.15* (0.06)	-0.09 (0.06)
Youth self-reported ethnicity, γ_{05}	-0.26 (0.28)	-0.27 (0.27)	-0.27 (0.28)
History of depression, γ_{10}	1.30* (0.41)	1.28* (0.43)	1.65* (0.42)
Dependent stressors, γ_{20}	0.18* (0.04)	0.18* (0.03)	
Independent stressors, γ_{30}	0.00 (0.06)		0.20* (0.05)
History of depression × dependent stressors, γ_{40}	0.20* (0.09)	0.19* (0.07)	
History of depression × independent stressors, γ_{50}	0.06 (0.16)		0.14 (0.15)
Lagged CDI, γ_{60}	0.33* (0.03)	0.34* (0.03)	0.44* (0.03)
		Random Effects	
Intercept, r_0	1.29*	1.09	1.46
History of Depression, r_1	10.44	11.09*	10.08
Dependent stressors, r_2	0.03	0.02	
Independent stressors, r_3	0.09		0.05
History of depression × dependent stressors, r_4	0.49	0.15	
History of depression × independent stressors, r_5	1.30		0.47
Lagged CDI, r_6	0.05	0.04*	0.05
Level-1, ε	9.34	10.03	11.69

Note: *: $p < .05$. CDI: Children's Depression Inventory. Sex is coded female = 1, male = 0. Ethnicity is coded 0 = non-Hispanic White, 1 = otherwise. History of depression is coded 1 = positive history, 0 = no history.

Table 2
Hierarchical Linear Models Of Children's Depression Inventory Scores in Only Those Participants Who Experienced a First-Onset of Depression During the Two Years of the Study (n = 57)

	Both dependent and independent stressors	Only dependent stressors	Only independent stressors
	Estimate (*SE*)	Estimate (*SE*)	Estimate (*SE*)
		Fixed Effects	
Intercept, γ_{00}	5.35* (0.91)	5.25* (1.04)	6.46* (0.91)
Sex, γ_{01}	-0.70 (0.57)	-0.07 (0.70)	-0.19 (0.66)
Person-mean dependent stressors, γ_{02}	0.33* (0.09)	0.21* (0.07)	
Person-mean independent stressors, γ_{03}	-0.56 (0.29)		0.19 (0.23)
Family income, γ_{04}	-0.26 (0.13)	-0.11 (0.14)	-0.05 (0.18)
Youth self-reported ethnicity, γ_{05}	0.72 (0.76)	0.82 (0.81)	1.90* (0.84)
History of depression, γ_{10}	-0.76 (0.55)	-0.49 (0.62)	-0.16 (0.61)
Dependent stressors, γ_{20}	0.21* (0.07)	0.23* (0.07)	
Independent stressors, γ_{30}	0.11 (0.15)		0.33* (0.15)
History of depression × dependent stressors, γ_{40}	0.29* (0.14)	0.29* (0.10)	
History of depression × independent stressors, γ_{50}	0.05 (0.26)		0.29 (0.25)
Lagged CDI, γ_{60}	0.26* (0.05)	0.31* (0.06)	0.41* (0.05)
		Random Effects	
Intercept, r_0	3.92*	6.34	1.01
History of Depression, r_1	5.60	8.37*	6.49
Dependent stressors, r_2	0.04	0.03	
Independent stressors, r_3	0.36		0.21
History of depression × dependent stressors, r_4	0.34	0.06	
History of depression × independent stressors, r_5	0.58		0.19
Lagged CDI, r_6	0.05	0.05*	0.05
Level-1, ε	10.73	12.19	15.04

Note: *: $p < .05$. CDI: Children's Depression Inventory. Sex is coded female = 1, male = 0. Ethnicity is coded 0 = non-Hispanic White, 1 = otherwise. History of depression is coded 1 = positive history, 0 = no history.

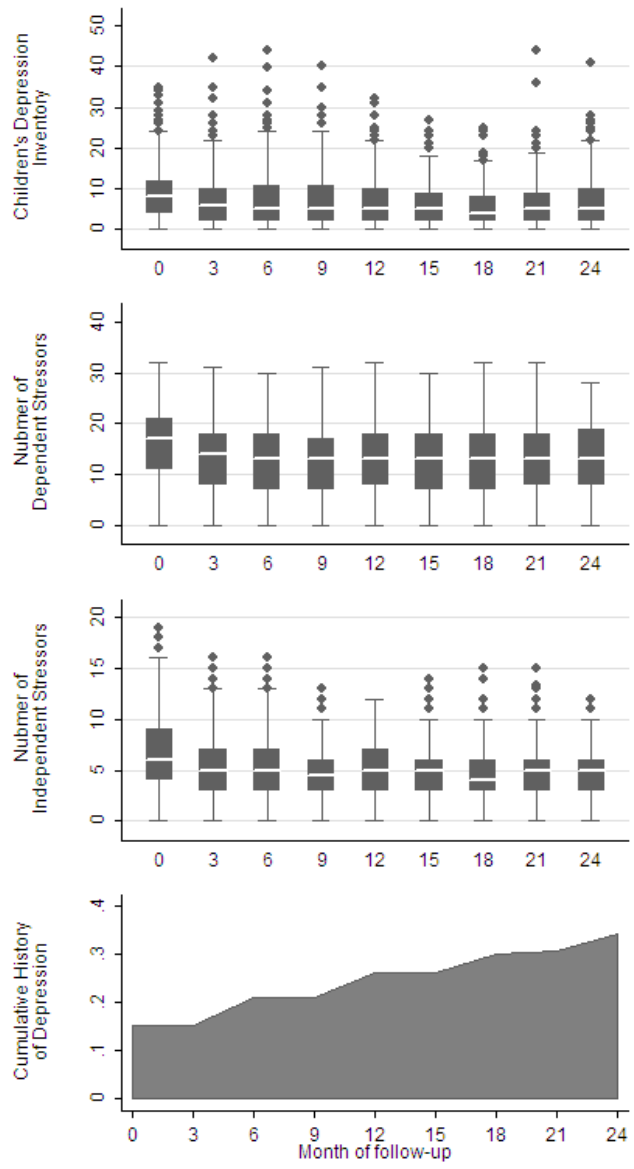

Figure 1. Upper panels show distribution of depressive symptoms, dependent stressors, and independent stressors over time. The bottom panel describes the cumulative lifetime prevalence of depressive episodes over the course of the study.

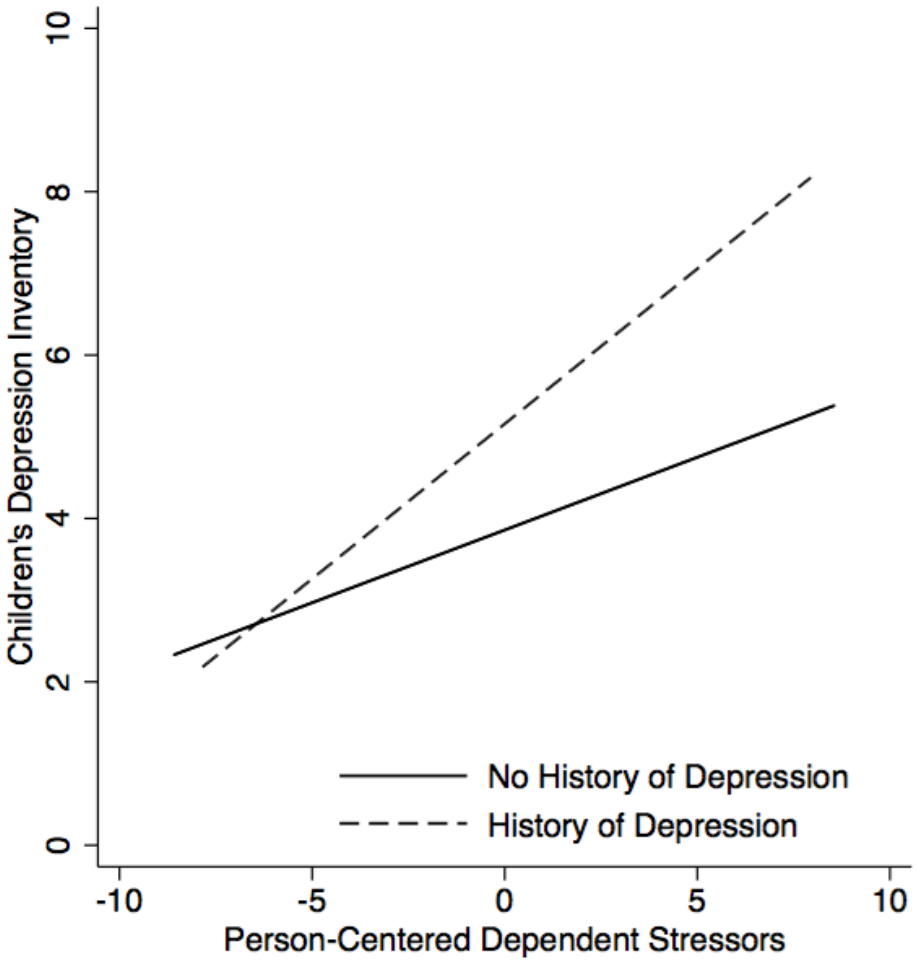

Figure 2. Interaction of history of depression and idiographic fluctuations in dependent stress within the entire sample. All covariates were held at 0.