Published in final edited form as:
J Abnorm Child Psychol. 2008 October ; 36(7): 999–1014. doi:10.1007/s10802-008-9228-6.

Cognitive Vulnerability–Stress Model of Depression During Adolescence: Investigating Depressive Symptom Specificity in a Multi-Wave Prospective Study

Benjamin L. Hankin
Department of Psychology, University of South Carolina, Barnwell College, Columbia, SC 29208, USA, e-mail: hankin@sc.edu

Abstract

Depression commonly co-occurs with anxiety and externalizing problems. Etiological factors from a central cognitive theory of depression, the Hopelessness Theory (Abramson et al. *Psychological Review*, *96*, 358–372, 1989), were examined to evaluate whether a negative inferential style about cause, consequence, and self interacted with stressors over time to predict prospective elevations in depressive symptoms specifically compared with typically co-occurring symptoms. Negative inferential style was assessed at baseline in a sample of early and middle adolescents (*N*=350, sixth to tenth graders). Measures of general depressive, anhedonic depressive, anxious arousal, general internalizing, and externalizing symptoms and occurrence of stressors were assessed at four time points over a 5-month period. Results using hierarchical linear modeling show that a negative inferential style interacted with negative events to predict prospective symptoms of general and anhedonic depression specifically but not anxious arousal, general internalizing or externalizing symptoms. Negative events predicted prospective elevations of symptoms of anxious arousal, internalizing, and externalizing problems.

Keywords

Depression; Comorbidity; Cognitive vulnerability; stress; Longitudinal

Introduction

Depression and other emotional and behavioral problems commonly co-occur. This overlap is observed at the level of mood, symptoms, and disorder for many of the most common psychiatric disorders among youth, including anxiety, conduct/oppositional defiant, and attention deficit/hyperactivity (ADHD) (Costello et al. 2003; Lewinsohn et al. 1993; Newman et al. 1998; see Hankin and Abela 2005; Rudolph et al. 2006, for reviews). For example, a meta analysis (Angold et al. 1999) of depression comorbidity with these other symptoms and disorders among community samples revealed that depression was associated with anxiety (median odds ratio=8.2), conduct/oppositional problems (median odds ratio=6.6), and ADHD (median odds ratio=5.5). In addition, comorbid depression is associated with more severe symptoms and correlates as well as worse clinical course and potential treatment outcomes (Birmaher et al. 1996; Lewinsohn et al. 1995). Finally, the developmental epidemiology literature has also identified developmental patterns of sequential comorbidity. Symptoms and a diagnosis of anxiety typically precede the development of depressive symptoms or disorder (Avenevoli et al. 2001; Cohen et al. 1993; Kim-Cohen et al. 2003; Pine et al. 1998), whereas

earlier externalizing problems tend to predict later depressive symptoms (Curran and Bollen 2001; Kim-Cohen et al. 2003). Understanding what factors and processes predict symptoms of depression compared with co-occurring symptoms is important for advancing etiological theories as well as for improving assessment and treatment.

The well-known co-occurrence of symptoms and disorders has been a focus of recent research that seeks to understand the interplay among broad internalizing and externalizing behaviors as well as the more narrow-band syndromes that comprise these higher-order factors. Current theory and research (e.g., Krueger and Markon 2006; Watson 2005) suggests that there is a hierarchical structure to the internalizing distress dimension (see also Krueger et al. 2005 for theory and evidence on a hierarchical structural spectrum model for the externalizing dimension) that includes the broad factor of negative affect at the top and overlapping, but unique, factors of relatively specific anhedonic depression and anxious arousal specific to anxiety/fear problems at the next level (Lahey et al. 2003; Mineka et al. 1998; Watson 2005). This hierarchical model of internalizing, emotional distress symptoms builds upon Clark and Watson's (1991) tripartite theory of anxiety and depression in which anhedonia, or low positive affect, is conceptualized as relatively specific to depression, anxious/physiological arousal is viewed as relatively specific to anxiety, and broad negative affect is non-specifically associated with both depression and anxiety. Despite a great deal of support for this model in adults (see Watson 2005 for a review) and youth (e.g., Chorpita et al. 1998; Joiner et al. 1996; Lonigan et al. 1999), the model was revised (Mineka et al. 1998), largely, to reflect the fact that anxious arousal was not specific to all anxiety problems and disorders, but rather, the physiological arousal component related most strongly to particular anxiety disorders that are characterized by elements of fear, such as panic and phobia disorders. Consistent with this more recent hierarchical conceptualization, factor analytic evidence of common emotional and behavioral disorders among youth, based on diagnostic clinical interviews from a population-based sample of children and parents as informants, showed the expected internalizing and externalizing dimensions at the top of the hierarchy that were comprised of three meaningful factors at the next level, including two internalizing factors that Watson (2005) labeled as "fear" and "distress," and an externalizing factor of conduct and attention problems (Lahey et al. 2003). Symptoms of depression, generalized anxiety disorder and social phobia loaded onto the distress disorder factor, whereas symptoms of specific phobias, obsessive compulsive disorder, and separation anxiety disorder loaded onto the fear disorder factor. Given the importance of understanding specificity of symptoms and the interplay among negative affect, anxiety, depression, and externalizing behaviors, the present study used this theoretically-based and empirically supported hierarchical model of the internalizing spectrum disorders. The different factors from this model were assessed via various measures to assess the particular key aspects of the internalizing dimension in order to examine more clearly the specificity of cognitive vulnerability, stressors, and the cognitive vulnerability–stress interaction predicting these different symptom factors (i.e., broad negative affect and general internalizing problems, depression, and anxious arousal/fear) intended to represent the various aspects of the internalizing spectrum.

Despite the repeated demonstration that depression co-occurs with other emotional and behavioral problems, relatively few studies in the literature have tested specific versus general etiological influences contributing to depression and overlapping anxiety and externalizing symptoms (c.f., Seligman and Ollendick 1998). Past research has identified vulnerability factors for depression (e.g., Garber 2000; Hankin and Abela 2005; Hammen and Rudolph 2003), anxiety (e.g., Albano et al. 2003; Vasey and Ollendick 2000), and externalizing problems (e.g., Hinshaw and Lee 2003; Lahey et al. 2002), yet considerably less research has studied the degree to which these vulnerability factors are specific to depressive symptoms or common to depressive, fear/anxious arousal, and general internalizing and externalizing symptoms. The primary aim of this study is to test whether etiological influences from a central

cognitive theory of depression predict depressive symptoms more specifically compared with anxious arousal symptoms tapping the fear factor, internalizing symptoms characteristic of the broad internalizing dimension, or externalizing behaviors.

Hopelessness Theory of Depression

The Hopelessness Theory (HT; Abramson et al. 1989) is a cognitive vulnerability–stress theory of depression that postulates that a negative inferential style will contribute to depression especially when individuals encounter negative life events. HT posits that a negative inferential style about cause (the tendency to attribute negative events to stable and global causes, also called a negative attributional style), consequences (the propensity to catastrophize the consequences of negative events) and the self (the likelihood of finding negative self-meaning and implications for one's self following the occurrence of negative events) increases the likelihood of an individual developing depressive symptoms. According to the cognitive vulnerability–stress component of HT, this depressogenic inferential style is hypothesized to interact with negative life events to contribute to prospective increases in depressive symptoms. In general, HT has garnered considerable empirical support in children, adolescents, and adults (see Abela and Hankin 2008; Abramson et al. 2002; Hankin and Abela 2005; Lakdawalla et al. 2007; Ingram et al. 1998; Scher et al. 2005 for general reviews), although not all studies among youth have been uniformly supportive (e.g., Abela and Sarin 2002; Hammen et al. 1988).

Originally HT was proposed as an etiological explanation for the ontogeny of depression, so there is reason to believe that its cognitive components may be relatively specific predictors of later depression compared with other psychopathological symptoms. In later extensions of HT, theorists have expanded the theory and logic of HT's causal chain to try to understand the high degree of co-occurrence of depression with anxiety (e.g., the helplessness–hopelessness theory; Alloy et al. 1990). In this model, for example, some cognitive processes (i.e., the combination of both helplessness and hopelessness) would be expected to predict both anxiety and depression, whereas others (i.e., hopelessness without helplessness) would be expected to predict depression specifically. However, little direct empirical support of this model has been obtained as the evidence, predominantly with adults, is equivocal (e.g. Swendsen 1997). Building on these foundational models, Hankin and Abramson (2001) proposed that the interaction of cognitive vulnerability, such as HT's negative inferential style, with negative events would be an etiologically specific risk factor for depression, whereas the occurrence of negative events, without elevated cognitive vulnerability, would contribute to general negative affect and predict symptoms of psychopathology (e.g., externalizing, internalizing, anxious arousal) broadly. Consistent with this notion, prospective research has found that a negative attributional style interacts with stressors to predict future increases in depressive symptoms and disorder among youth (e.g., Abela 2001; Dixon and Ahrens 1992; Hankin et al. 2001; Lewinsohn et al. 2001; Hilsman and Garber 1995; Robinson et al. 1995; see Lakdawalla et al. 2007, for a review). Negative events are broadly associated with anxiety, depression, and externalizing problems (McMahon et al. 2003).

This corpus of research reveals clearly that baseline levels of a negative inferential style about causes (i.e., negative attributional style) interact with later negative life events to predict depression, but these studies have not addressed a key question. Namely, they did not examine whether cognitive vulnerability to depression, by itself as a main effect or in interaction with stressors, represents a specific risk factor predicting later depression. A few studies with youth have examined this issue by investigating cognitive vulnerability as a main effect predictor of depression, but they did not examine the key cognitive vulnerability–stress interaction. These studies provide initial evidence that a negative attributional style is associated with depression more than with anxiety or externalizing problems (Gladstone et al. 1997; Lewinsohn et al.

1997; Weiss et al. 1998). A few prospective vulnerability–stress studies with adults have found that HT's cognitive vulnerability–stress component predicts depressive symptoms more specifically than anxious symptoms (Hankin et al. 2004; Metalsky and Joiner 1992), whereas others have not found such specificity (Luten et al. 1997; Ralph and Mineka 1998). In sum, the empirical evidence concerning symptom specificity from adults is equivocal.

A handful of studies with youth have examined symptom specificity of the cognitive vulnerability–stress component of HT. The available evidence suggests that a negative attributional style interacts with stress to predict depressive symptoms specifically but not externalizing behaviors (Robinson et al. 1995) or anxiety symptoms (Brozina and Abela 2006; Joiner 2000). Lewinsohn et al. (2001) found that a negative attributional style interacted with stress to predict depressive disorder but not non-depressive disorders (e.g., anxiety or behavioral diagnoses), although the form of this interaction was contrary to that postulated by HT (i.e., negative attributional style interacted with low, not high, levels of stressors to predict depression). Finally, only one of these studies (Brozina and Abela 2006) examined the entire construct of negative inferential style according to HT. The others measured only a negative attributional style; negative inferences about consequence and self were not assessed or examined.

In summary, the available evidence suggests a potentially specific association between the main effect of a negative attributional style and depression, and a negative attributional style may interact with stressors to predict depressive symptoms specifically. However, these conclusions are tempered by the limited number of studies, the dearth of studies measuring the full cognitive vulnerability featured in HT, the inconsistency in the past studies, the lack of research investigating specificity of depression compared with co-occurring internalizing and externalizing symptoms, and as introduced next, methodological and design limitations to the extant corpus of research. Thus, both theory as well as the empirical evidence on the essential question of depressive symptom specificity is unclear. To advance knowledge in this area, more rigorous empirical work is needed that can inform the empirical literature and hopefully advance future theory building on this important issue.

Methodological and Design Considerations

Several issues have limited the rigor and power of studies testing vulnerability–stress theories of the development of psychopathology among youth. First, the vast majority of past studies have examined cognitive vulnerabilities and their association with depressive symptoms in either cross-sectional or two-time point prospective designs (e.g., Abela 2001; Gibb and Alloy 2006; Hankin et al. 2001; Lewinsohn et al. 2001; however, see Abela et al. 2006a, b, for recent examples of multi-wave studies). Highlighting the limitations of using cross-sectional or two-time point panel designs, developmental methodologists (e.g., Curran and Willoughby 2003) have cogently and persuasively argued that two-time point prospective studies are not much more informative than simple cross-sectional designs. Further, multi-wave studies (i.e., a minimum of three time points) are needed to test rigorously and accurately longitudinal patterns and developmental processes.

Second, the vast majority of the past studies testing hypotheses from HT (e.g., Abela 2001; Gibb and Alloy 2006; Hankin et al. 2001; Lewinsohn et al. 2001) have used psychometrically inadequate or developmentally non-optimal measures of cognitive vulnerabilities (see Lakdawalla et al. 2007). Poor measurement has limited the accuracy and power of studies evaluating predictions from cognitive vulnerability–stress theories of depression among youth. There is a need for research that uses psychometrically reliable measures of cognitive vulnerabilities to depression that assess the entire theoretical construct of HT's negative inferential style, so theoretical predictions can be more powerfully tested.

Third, many of the past studies have not included sufficiently ethnically diverse samples because most samples were comprised of predominantly middle class, White youth. It is important to investigate etiological theories with diverse samples to enhance generalizability of findings and to explore possible ethnic differences in cognitive vulnerabilities, stressors, and psychopathological symptoms. Moreover, there are well-documented age and sex effects on the development of depression (Hankin et al. 1998; Hankin et al. 2008a; Twenge and Nolen-Hoeksema 2002) such that depressive symptoms and disorder become increasingly more elevated and prevalent throughout adolescence, especially for girls, who begin to become more depressed than boys during early adolescence. The sex difference in depression becomes most dramatic during middle adolescence. Given the general lack of information on how ethnicity may affect etiological factors in depression and the well-known age and sex influences, this study explored whether these demographic factors affected the development of depression and the cognitive vulnerability–stress interaction as a predictor of later depressive and co-occurring symptoms.

The Current Investigation

The current study addresses these limitations and seeks to provide a more powerful test of HT's negative inferential style × stress interaction as a potentially specific predictor of depressive symptoms compared with anxious arousal, general internalizing, and externalizing symptoms. The investigation used a four-wave prospective design with a psychometrically reliable measure of HT's cognitive vulnerability, typically occurring and developmentally relevant stressors among youth, and psychopathological symptoms among a moderately large and ethnically diverse sample of sixth–tenth graders. It was hypothesized that baseline levels of negative inferential style would interact with negative life events assessed over four time points to predict prospective elevations in depressive symptoms more specifically than anxious arousal, general internalizing symptoms, or externalizing behaviors. In contrast, negative life events were hypothesized to nonspecifically predict elevations in psychopathology generally (i.e., anxious arousal, internalizing and externalizing symptoms).

Method

Participants

Participants were youth who were recruited from five Chicago area schools. Schools were selected to represent ethnic and socio-economic diversity typical of the Chicago area. Selected schools included one inner-city private middle school, one affluent private middle school, and three public schools (one middle and two high schools) serving predominantly middle class neighborhoods. Consent forms were passed out during school to the 467 students who were available in the appropriate grades (sixth–tenth) from these selected schools. Parents of 390 youth (83.5%) provided active consent; all 390 youth were willing to participate. 356 youth (91%) completed the baseline questionnaire. The 34 students who were willing to participate but did not complete the baseline visit were sick or absent from school and were unable to reschedule. There were no significant differences in demographic characteristics (age, sex, ethnicity) between the number of available youth in schools (N=467), those who provided consent (N=390), and those who participated (N=356). Data were examined from 350 youth who provided complete data (symptoms and negative cognitive style) at baseline. Rates of participation in the study decreased slightly from baseline: wave 2 (N=303), wave 3 (N=308), and wave 4 (N=345). Age ranged from 11–17 (M=14.5; SD=1.40; there were three 17 year olds); 57% were female; 13% were Latino; 6% were Asian or Pacific Islander, 21% African-American, 53% White, and 7% bi- or multi-racial.

Procedures

Students participated in this study with active parental informed consent. Permission to conduct this investigation was provided by the school districts and their institutional review boards, school principals, the individual classroom teachers, and university institutional review board. Trained research personnel visited classrooms in the schools and briefly described the study to youth, and letters describing the study were sent home to parents. Specifically, students and parents were told that this study was about adolescent mood and experiences, and participation would require completion of questionnaires at four different time points. Students, who agreed to participate and had returned active parental consent, read and signed their own informed consent form after having the opportunity to ask any questions about the study. Youth completed a battery of questionnaires during class time and were debriefed at the end of the study. Participants completed questionnaires at four time points over a 5-month period, with approximately 5 weeks between each time point. Youth were compensated $10 for their participation at each wave in the study, for a possible total of $40 for completing all four assessments.

Measures

Children's Depression Inventory (CDI)—The CDI is a self-report measure that assesses depression in children and adolescents using 27 items (Kovacs 1981). Each item is rated on a scale from 0–2. Reported scores are the average item scores of all items (range 0–2). Higher scores indicate more depression. The CDI has been shown to have good reliability and validity as a measure of general depression in children and adolescents (Klein et al. 2005). Although the CDI is probably the most commonly used measure of assessing depressive symptoms among youth, its construct validity and specificity as a measure of depression has been questioned (e.g., Chorpita et al. 2005; King et al. 1991) given that it appears to contain many items tapping broad negative affect (Chorpita et al. 1998). Thus, although the CDI was intended to assess the depressive symptoms factor of the hierarchical model of internalizing problems (Mineka et al. 1998; Watson 2005) given its wide-spread use, the questionable specificity and construct validity led to the decision to select particular anhedonia items from the CDI, based on past work (e.g., Chorpita et al. 1998; Joiner et al. 1996) in order to examine both the full CDI as the commonly used measure of general depressive symptoms and the relatively more specific anhedonic depressive symptoms based on the CDI. In sum, analyses for depressive symptom specificity are reported for the full CDI to assess general depressive symptoms and for anhedonic CDI items (nos. 4, 12, 15, 20, 21, 22; Chorpita et al. 1998).

Mood and Anxiety Symptom Questionnaire (MASQ)—The MASQ for this study was modified from the original MASQ, which contains 90 items to assess the general distress and specific anxiety and depressive symptoms based on the tripartite theory of anxiety and depression (Clark and Watson 1991; Watson et al. 1995). For this study, only the Anxious Arousal (ANX) subscale was used to assess relatively specific anxious symptoms that are not overly saturated with general negative affect. Given this study's conceptual grounding based on the hierarchical model to represent the broad internalizing symptoms dimension, the ANX was intended to assess the physiological symptoms found to be relatively specific to the fear factor of the hierarchical model. To be clear, the ANX subscale was not intended to represent and assess anxiety symptoms generally because more recent theory and data (Mineka et al. 1998; Watson 2005) show that the anxious arousal scale is associated with particular anxiety disorders, including specific phobias and panic disorder, both of which load onto the fear factor (Lahey et al. 2004) of the hierarchical internalizing model. Youth responded to ten ANX items on a Likert scale from 1 to 5, and reported scores are the average item scores of all items (range 1–5). Reliability and validity of the MASQ has been demonstrated in previous studies with adolescents (e.g., Hankin 2008; Hankin et al. 2008b; Watson et al. 1995). The MASQ was given at all four time points.

Strengths and Difficulties Questionnaire (SDQ)—The SDQ is a brief 25-item questionnaire that assesses general internalizing and externalizing emotional and behavioral problems (Goodman 2001). A five-factor structure, consisting of emotional, conduct, hyperactivity-inattention, peer, and prosocial factors, has been supported in past research with large samples of youth and parents (Goodman 2001). These lower-order five factors can be categorized into two broader general internalizing and externalizing factors that have been shown to be relatively independent of each other (Goodman 2001). The externalizing factor, comprised of conduct and hyperactivity-inattention problems, was used for the present study as a measure of broad externalizing problem behaviors. In addition, the internalizing factor, comprised of the emotional problems factor, was used as a measure of broad negative affect based on the hierarchical internalizing model. In other words, the emotional factor from the SDQ is not hypothesized to be specific to anxiety or depression, and the evidence from factor analytic research (Goodman 2001) shows that this emotional symptoms factor represents broad negative affect, internalizing symptoms. Reported scores are the average item scores of all items (range 0–2). Normative data are available for the SDQ from 9,878 children (ages 4–17). The descriptive statistics (means, SD; See Table 1) from the present sample match the descriptive data from the normative database closely. The SDQ has been shown to be reliable and valid in past research (Goodman 2001). The SDQ was given at all four time points.

Adolescent Cognitive Style Questionnaire (ACSQ)—The ACSQ measures the inferential styles about cause, consequence, and self, as featured in HT (Hankin and Abramson 2002). The ACSQ presents the adolescent with negative hypothetical events in achievement and interpersonal domains and asks the youth to make inferences about the cause (internal–external, stable–unstable, and global–specific), consequences, and characteristics about the self based on the hypothetical event. Only stable and global, not internal, attributions were used in scoring the ACSQ to be consistent with HT's perspective emphasizing stable, global attributions along with negative inferences for the self and consequences. Each item dimension is rated from 1–7. Average item-scores on the total ACSQ range from 1 to 7 with higher scores indicating more negative inferential styles. The ACSQ has demonstrated excellent internal consistency reliability, good test–retest reliability, and factor structure consistent with HT, as a measure of HT's cognitive vulnerability to depression among adolescents. Internal reliability for the ACSQ in this sample was α=0.95 at Time 1.

Adolescent Life Events Questionnaire (ALEQ)—The ALEQ assesses a broad range of negative life events that typically occur among adolescents (Hankin and Abramson 2002). The ALEQ assesses a broad range of life events including school/achievement problems, friendship and romantic difficulties, and family problems. Examples of items from the ALEQ include "got a bad report card" to assess school events, "had an argument with a close friend" for friendship events, "boyfriend/girlfriend broke up with you but you still want to go out with them" for romantic events, and "your parents grounded you" for family events. It consists of 57 different negative life events. Youth were asked to indicate how often [Likert scale ranging from never (0) to always (4)] these different negative events had occurred to them over the past 5 weeks. Responses to the Likert scale were then converted into dichotomous counts of events (never= 0; 1–4 recoded to 1) in order to represent a count of the number of stressors reported over the past 5 weeks between any given follow-up interval. Higher scores indicated exposure to more negative events at a particular time point over the past 5 weeks. The ALEQ was given at all 4 time points.

Results

Preliminary Analyses

Descriptive statistics and intercorrelations for the main variables are presented in Table 1. The baseline measure of negative inferential style was moderately associated with depressive, anxious arousal, general internalizing, and externalizing symptoms as well as stressors both concurrently and prospectively at different waves of data. Stressors were related to depressive, anxious arousal, general internalizing, and externalizing symptoms both concurrently and prospectively across waves of data. There were no ethnic or racial differences (White vs. non-White in one analysis, and White vs. African-American in a second analysis) in any of the variables (all *t*'s<1.50).. Using the different recommended clinical cutoffs for the CDI revealed that 24.3% (CDI cutoff>19; Stark and Laurent 2001) or 32.3% (CDI cutoff>16; Timbremont et al. 2004) of youth were above cut-scores for the CDI. Similarly, for the SDQ, 12.3% of youth were above cut-scores (Goodman 2001) for the internalizing scale and 15.1% for the conduct factor of the SDQ.

Overview of Statistical Approach

Hierarchical linear modeling (HLM; Bryk and Raudenbush 1992; Raudenbush 2000) was used to address the primary hypotheses: (1) Does baseline negative inferential style interact with negative life events over the 4 time points to predict fluctuations of depressive symptoms over time?, and (2) Does the cognitive vulnerability–stress interaction predict depressive symptoms relatively more specifically compared with anxious arousal, internalizing, or externalizing symptoms? HLM is a rigorous approach for approaching these questions because it can represent both change within a person over multiple time points while also ascertaining how individuals may differ from one another in symptom trajectories over time (Bolger et al. 2003; Curran and Willoughby 2003).

The analysis of multiple levels of data is accomplished in HLM 5.04 (Raudenbush et al. 2001) through the construction of levels 1 and 2 equations. At level 1, regression equations are constructed that model separately the variation in the repeated measures (e.g., depressive symptoms, stressors) as a function of time (i.e., the four waves of data). Each equation includes various parameters to capture features of an individual youth's level of symptoms (i.e., depression, anxiety, or externalizing) and stressors over time, such as an *intercept* that describes an individual's average level on the variable across time and a *time-varying covariate* that describes the strength of association between within-person fluctuations in one construct (e.g., symptoms of depression) and within-individual changes in another construct (e.g., stressors) over the four waves of data. At level 2, equations are specified that model individual differences in the level 1 parameters as a function of between-subjects' variables (i.e., HT's negative inferential style). The key cognitive vulnerability–stress interaction is tested by examining the cross-level interaction term representing the effect of a negative inferential style, at level 2, on the slope of within-youth variability in the strength of the relation between stressors and symptoms at level 1.

To test whether the cognitive vulnerability–stress interaction predicts *prospective elevations* in a particular type of symptoms (e.g., depression) over time, lagged analyses were conducted[1]. Symptom scores at time *T* served as the dependent variable in the HLM analysis and time *T*−1 symptom scores were included in the level 1 model along with stressors at time *T* at level 1. Negative cognitive style was entered at level 2 to enable an examination of whether stressors, in interaction with negative cognitive style, were associated with prospective changes in symptom scores between time *T*−1 and time *T*. This approach enables a stringent idiographic examination of the relation between stressors and symptoms for each adolescent along with the essential investigation of cognitive vulnerability, as a level 2 between-subjects factor, as a

moderator of this stressor-symptoms relation (see Gibb et al. 2007 for similar analyses with an adult sample).

A significant advantage of HLM is that it can handle cases with missing data. Random effects models, such as HLM, do not require that every participant provide complete, non-missing data over the 4 time points, so participants with missing data are not eliminated from the data set.

Age and Sex Effects in Symptoms

Age and sex were included in these models at level 2 to investigate whether they influenced prospective changes in symptoms over time. Age significantly predicted overall depression (b=0.04, SE=0.01, t=4.18, p<0.001), anxious arousal (b=0.13, SE=0.02, t=6.79, p<0.001), internalizing (b=0.07, SE=0.01, t=6.23, p<0.001), and externalizing symptoms (b=0.01, SE=0.005, t=1.91, p<0.05) but not anhedonic depression (b=0.12, SE=0.08, t=1.77, p=0.09). Also as expected, the main effect of sex significantly predicted prospective changes in overall depression (b= 0.12, SE=0.02, t=6.08, p<0.001), anxious arousal (b= 0.20, SE=0.04, t=5.38, p<0.001), internalizing (b=0.16, SE=0.02, t=7.99, p<0.001), and externalizing symptoms (b= −0.05, SE=0.02, t=−2.23, p<0.05) but not anhedonic depression (b=0.16, SE=0.20, t=0.81, p>0.25). There was no significant interaction for age × sex effect (all t's<1). Thus, these initial growth curve analyses reveal that older adolescents and girls reported higher levels of overall depressive, internalizing, and anxiety symptoms, whereas older adolescents and boys reported more externalizing symptoms.

The Cognitive Vulnerability–Stress Hypothesis

To test the central hypothesis that HT's negative inferential style would interact with stressors to predict prospective elevations in symptoms over time, HLM was used to examine HT's inferential style interacting with stressors to predict the different dependent variables (i.e., within-youth fluctuations in symptoms of general depression, anhedonic depression, anxious arousal, internalizing, and externalizing behaviors over the four waves of data). The primary predictors (entered simultaneously) of these symptom outcomes were: (1) within-person stressor levels over the four waves of data, (2) HT's negative inferential style, and (3) the cross-level interaction between within-individual stressors and between-youth inferential styles.

Results of these HLM analyses are presented in Table 2 for the various models predicting different symptoms. As seen in Table 2, several significant results emerged across all symptom outcomes examined. First, the main effect of stressors as a within-individual time-varying covariate significantly predicted most symptom trajectories over time, whereas the main effect of cognitive vulnerability marginally predicted only depressive symptoms, not anhedonic depressive, anxious arousal, internalizing or externalizing symptoms. Next, the critical

[1]The equation used for level 1 model for symptoms over four time points:

$$\text{Symptoms}_{tij}=B0_j+B1_j*(\text{Stress})+B2*(\text{Symptoms}_{t-1ij})+R_{ij}$$

Equations for level 2 models:

$$B0_j=G_{00}+G_{01}*(\text{ACSQ})+U_{0j}$$
$$B1_j=G_{10}+G_{11}*(\text{ACSQ})+U_{1j}$$
$$B2_j=G_{20}+U_{2j}$$

cognitive vulnerability × stress interactions (e.g., negative inferential style at level 2 × within-youth stressors over time at level 1) significantly predicted prospective fluctuations in general and anhedonic depressive symptoms specifically, but not anxious arousal, general internalizing, or externalizing symptoms. In order to examine the form of the significant cross-level interaction between negative inferential style and stressors over time predicting depressive symptom elevations over time, the findings from the model in Table 2 were used to calculate predicted scores in general depressive symptoms (i.e., full CDI) for youth with depressogenic or protective inferential styles (plus or minus 1.5 SD on the negative inferential style measures) and those who experienced either low or high stressor levels over time (plus or minus 1.5 SD on the ALEQ across the four data waves). These results are shown in Fig. 1. A more depressogenic inferential style combined with more stressors over time predicted the greatest elevation in depressive symptoms over time. This same pattern was seen for anhedonic depressive symptoms.

In sum, consistent with the symptom specificity hypothesis, a more depressogenic inferential style interacted with higher stress levels to predict depressive symptoms (overall and anhedonic depression) specifically but not anxious arousal, general internalizing, and externalizing symptoms. Greater levels of stressors over time were associated with prospective elevations in anxious arousal, general internalizing, and externalizing symptoms over time.

The Cognitive Vulnerability–Stress Hypothesis: Controlling for Co-occurring Symptoms

To provide a more stringent test of the symptom specificity hypothesis, additional HLM analyses were conducted in which anxious arousal and externalizing symptoms, at Time t−1, were included in level 1, along with prior depressive symptoms at Time *t*−1, as time-varying covariates. The other main predictor variables were also included in the model as already described to predict prospective fluctuations in general and anhedonic depressive symptoms. In essence, by including the additional symptom scales in the model as a within-person time varying covariate, the overlap between depressive symptoms and the other co-occurring symptoms is controlled for, and thus, this provides for a more exacting test of the specificity hypothesis (c.f., Seligman and Ollendick 1998). The primary reason for conducting these additional analyses in which co-occurring symptoms were controlled, along with the separate analyses on symptoms just reported, was to more comprehensively examine and report the specificity of cognitive vulnerability–stress interaction effects as the findings could differ depending on whether co-occurring symptoms were controlled or not. Meehl (1977) recommended conducting analyses in which covariates are controlled and analyses in which the covariates are not included because different conceptually meaningful results may obtain that would be obscured by simply controlling for covariates without also examining the results without statistical controls. Moreover, these results provide information about sequential co-occurrence of depressive symptoms with the commonly overlapping symptoms of anxiety and externalizing problems over time as the co-occurring symptoms of anxiety and externalizing problems, in the prior data wave (i.e., Time *t*−1) were investigated. These analyses were conducted one model at a time while including one of the other symptom scales as a covariate (e.g., externalizing first, then anxious arousal symptoms) to predict prospective fluctuations of depressive symptoms.

The results showed that both anxious arousal (b=0.12, SE=0.02, t=4.64, p<0.001) and externalizing behaviors (b=0.06, SE=0.02, t=3.28 p<0.001) as time varying covariates at Time *t*−1 strongly predicted prospective levels of overall depressive symptoms over time. In contrast but consistent with the tripartite model, neither anxious arousal (b=0.00009, SE=0.01, t=0.08, p=0.93) nor externalizing behaviors (b=0.001, SE=0.002, t=0.56, p=0.57) as time varying covariates predicted prospective anhedonic depressive symptoms over time. Most importantly, the central negative inferential style × stress interaction continued to predict prospective

elevations in overall and anhedonic depressive symptoms even after co-occurring symptom fluctuations were statistically covaried. Specifically, a negative inferential style interacted with stressors to predict overall depression after controlling for externalizing behaviors (b=0.007, SE=0.003, t=2.02, p<0.05) and after anxious arousal symptoms (b=0.007, SE=0.003, t=1.95, p<0.05), and predicted anhedonic depression after controlling for externalizing behaviors (b=0.03, SE=0.007, t= 4.84, p<0.001) and after anxious arousal symptoms (b= 0.03, SE=0.002, t=14.89, p<0.001). Thus, the findings presented in Table 2 held even after applying a very conservative test of the symptom specificity hypotheses by controlling for time-varying co-occurring symptoms.

The Cognitive Vulnerability–Stress Hypothesis: Moderation by Age, Ethnicity, or Sex?

Given the rise in depressive symptoms with age, potential race and ethnic differences, and the sex difference found in the epidemiological literature (Avenevoli et al. 2008; Hankin and Abela 2005) as well as the significant age and sex effects for depression seen in this sample, exploratory analyses were conducted to examine whether age, ethnicity (White or non-White to maximize power to examine this question), or sex moderated the cognitive vulnerability–stress interaction effects reported above. Prior research exploring whether age and/or sex moderates the cognitive vulnerability–stress interaction has been inconclusive. Some research has found a significant sex × cognitive vulnerability × stress interaction (e.g., Hankin et al. 2001), whereas other work has not found sex moderation (see Abela and Hankin 2008 for review). Likewise, some research has found a significant age × cognitive vulnerability × stress interaction (e.g., Gibb and Alloy 2006; Nolen-Hoeksema et al. 1992), whereas other work has not found moderation by age (e.g., Dixon and Ahrens 1992; Lewinsohn et al. 2001). In addition to the past equivocal results on possible age and sex moderation as a reason for exploring potential age and sex influences, some developmental theories postulate that age will moderate the cognitive vulnerability–stress interaction such that it will predict depressive symptoms in older, but not younger, youth (Cole and Turner 1993). Likewise, some theoretical models posited to understand the sex difference in depression have postulated sex-specific models in which particular factors and processes are hypothesized to predict depression in girls, but not boys (e.g., Keenan and Hipwell 2005; Zahn-Waxler et al. 2006), whereas other theories specify a general depression model that would apply equally to both boys and girls (e.g., Hankin and Abramson 2001). Thus, it is important to examine possible age and sex moderation given the inconsistent past research and the differing theoretical models.

These analyses were conducted using the same HLM equations with the exception that the main and all interactive effects to examine moderation by age (i.e., age, age × negative inferential style, age × stress, and age × negative inferential style × stress) were included along with the main and interactive effects of stress and negative inferential styles as described above. The analyses examining the potential moderating effects of age on the negative inferential style × stress interaction were all non-significant for predicting depressive (overall and anhedonic), anxious arousal, internalizing, and externalizing symptoms (all t's< 1.5). Similar analyses investigating the potential effect of ethnicity or sex similarly revealed non-significant ethnicity or sex moderation for all symptoms (all t's<1.5).

Discussion

Two main sets of findings emerged from this study. First are the age and sex patterns in depressive, anxious arousal, general internalizing, and externalizing symptoms over time. Second is HT's cognitive vulnerability–stress interaction operating as a specific predictor of depressive symptoms compared with co-occurring anxious arousal, general internalizing, and externalizing symptoms over time.

Sex and Age Differences in the Pattern of Symptoms Over Time

The descriptive analyses examining prospective changes in depressive, anxious arousal, internalizing, and externalizing symptoms over time by age and sex showed results that are consistent with past developmental epidemiological findings (see Avenevoli et al. 2008; Hankin and Abela 2005; Rudolph et al. 2006, for recent reviews). Three sets of findings here are noteworthy.

First, the stability of depressive, anxious arousal, internalizing, and externalizing symptoms across the four waves of data was large and consistent with stability estimates observed in past research. For example, Tram and Cole (2006) recently reported test-retest correlations in depressive symptoms around or above 0.7 over 6 month intervals for a cohort-sequential design of 5th–8th graders followed for four time points over 18 months. The strong continuity of psychopathological symptoms over time is important for at least two reasons. First, it is very difficult to predict prospective changes in symptoms given such high test-retest correlations (cf., Tram and Cole 2006). For example, at least 50% of variance in depressive symptoms is already accounted for by prior symptoms, so this makes it challenging to find significant etiological prediction of future symptoms. Second, it is important to understand and predict prospective elevations in psychopathological symptoms during early and middle adolescence because symptoms become more stable and disorders more likely to recur in late adolescence through adulthood (Rutter et al. 2003). For depression, most individuals experience first onset of depression in middle to late adolescence (Hankin et al. 1998), and those with a past episode are two to seven times more likely to experience a recurrence in adulthood (Rutter et al. 2003).

Second, depressive, anxious arousal, internalizing, and externalizing symptom levels, assessed at baseline, were higher among older compared with younger youth. Third, there were sex differences in baseline symptom levels such that girls exhibited more general depressive, anxious arousal and internalizing symptoms, whereas boys reported more behavioral problems. Past cross-sectional and longitudinal studies show that levels of overall depression (Costello et al. 2003; Hankin et al. 1998; Wade et al. 2002) and anxiety (Costello et al. 2003; Costello and Angold 2006; Lewinsohn et al. 1998) are higher among older compared with younger adolescents and girls compared with boys. Similarly, research shows that externalizing behaviors of ADHD and Conduct Disorder are more prevalent among boys and older youth (Moffitt et al. 2001; Rutter et al. 2003).

Many theorists have proposed different etiological hypotheses that seek to explain these descriptive developmental changes in different symptom levels over time by age and sex. A comprehensive discussion of the various theoretical proposals purported to account for age and sex patterns during early to middle adolescence in different aspects of the development of psychopathology is beyond the scope of this paper (see Bell et al. 2005; Hankin et al. 2008; Rutter et al. 2003; Zahn-Waxler et al. 2006 for some recent examples). It is important to note that the pattern of findings and the theoretical explanations offered to account for the manifestation of symptoms by sex and age are influenced heavily by the developmental period and age range studied. Clearly, the unfolding of overall depressive, anxious arousal, general internalizing and externalizing symptoms over time by age and sex, and the reasons underlying these patterns, will look very different when preadolescent children are studied compared with the present sample of early to middle adolescents given obvious biological, emotional, cognitive, and social developmental changes. The present study focused on one particular theoretically motivated etiological explanation—the cognitive vulnerability–stress component derived from HT, a central cognitive theory of depression.

Affective Symptom Specificity of Ht's Cognitive Vulnerability–Stress Interaction

The second main finding was that HT's negative inferential style, assessed at baseline, interacted with within-youth changes in stressors over time to predict prospective fluctuations in general and anhedonic depressive symptoms specifically. In addition, occurrence of stressors over time was associated generally with psychopathological symptoms—anxious arousal, internalizing and externalizing behaviors in this study. These findings are consistent with Hankin and Abramson's (2001) elaborated cognitive vulnerability–transactional stress theory of depression and other extensions of cognitive vulnerability depression theories (e.g., Alloy et al. 1990; Seligman and Ollendick 1998). This pattern of HT's negative inferential style × stress interaction predicting depressive symptoms in particular was maintained even after co-occurring anxious and externalizing over time were controlled. This is a very stringent test of the affective symptom specificity hypothesis (Seligman and Ollendick 1998), so these results provide particularly compelling evidence that HT's cognitive vulnerability–stress interaction is an etiologically specific process contributing to elevations in depressive symptoms, not general symptoms of psychopathology, over time among youth.

These results replicate a growing body of studies from youth and adults suggesting that HT's negative inferential style, especially in interaction with stressors, may be a specific risk process for the development of depressive symptoms. The extant research with youth supports this specificity when depressive and externalizing symptoms are examined (e.g., Robinson et al. 1995) and when anxiety and depressive symptoms are compared (e.g., Brozina and Abela 2006; Joiner 2000). The preponderance of adult research supports the specificity of HT's cognitive vulnerability–stress interaction for depression versus anxiety (e.g., Hankin et al. 2004, Metalsky and Joiner 1992).

Given the descriptive analyses found in this study and reported in the literature, age, ethnicity, and sex were examined as potential moderators of the central cognitive vulnerability–stress interaction. Results showed that sex, ethnicity, and age did not interact with HT's negative inferential style × stress interaction to predict any symptoms. These findings reveal that the basic cognitive vulnerability–stress interaction predicting depressive symptoms applies equally well to boys and girls, youth of different ethnicities, and to early as well as middle adolescents. Clearly, caution is needed in interpreting these age, ethnicity, and sex moderation results so that they are not over-generalized beyond the age range and sample examined in this study because there may be sex, ethnic, or age moderation found in other samples. For example, some research has found that sex moderates the negative attributional style × stress interaction among middle to late adolescence (e.g., Hankin et al. 2001) and in early adolescence and children (e.g., Abela and Sullivan 2003), although other studies have not found moderation by sex (see Abela and Hankin 2008, for a review), so a clear, replicable pattern of sex moderation across studies has not been found.

Strengths and Limitations

The present results need to be interpreted with certain limitations in mind. First, all of the data come from youth who self-reported symptom levels, stressor occurrence, and negative inferential style. Clearly, given the likely mono-operation bias of same informant and method for assessing the central constructs in this study, use of multiple methods (e.g., information processing paradigms to assess cognitive vulnerability; Joormann et al. 2007) and multiple informants (e.g., parents, teachers) would be an important next step in future research for developmental psychopathology and cognitive vulnerability theory research to take. Second, this study did not investigate clinical levels of anxiety, depression, or externalizing problems through structured diagnostic interviews, so it is unclear whether the present findings of affective symptom specificity will generalize to more severe levels of psychopathology. Most research suggests that anxiety, depression, and externalizing problems can be represented and

conceptualized best as dimensional continua, rather than discrete categories (e.g., Fergusson et al. 2005; Hankin et al. 2005; Osgood et al. 2002; Rivas-Vazquez et al. 2004; Vollebergh et al. 2001), so it is most likely that the etiological factors that contribute to subclinical levels of psychopathology may also predict clinical levels as well (c.f., Gotlib et al. 1995). Use of structured diagnostic interviews in future research can address this issue. Third, self-report of stressors has been criticized because depressed mood and/or cognitive vulnerability may bias assessment of stressors (e.g., Cohen and Cohen 1984; Simons et al. 1993). Even though prior levels of symptoms were controlled for in lagged analyses and this likely removes potential depressive symptom bias, future research with use of contextual stress interviews to assess for more objective stressors in difference thematic domains (e.g., achievement and interpersonal) and stressor types (e.g., independent and dependent; Hammen 1991) would be helpful (Monroe and Roberts 1990; Monroe and Simons 1991). Also, the measure of stressors is most accurately considered as a count of the number of stressors reported by youth in the follow-up interval, and as such, the severity or impact of particular events was not investigated (see Hammen 2005, for discussion of stress measurement). Future vulnerability–stress research can examine whether the degree of stressor threat (e.g., major stressor versus hassle) affects prediction of depression from the cognitive vulnerability–stress interaction. Finally, this study did not assess nor test for etiologically specific risk factors for anxiety (e.g., anxiety sensitivity) or externalizing problems (e.g., lack of constraint, or excessive impulsivity). These and other potentially specific vulnerabilities to psychopathology can be examined in the future.

Despite these various limitations, the present research had several strengths, including a multi-wave assessment of various commonly occurring psychopathological symptoms, use of a psychometrically reliable and valid measure of HT's negative inferential style, and data analytic methods best suited for multi-level longitudinal repeated measures data. Also, a modestly large sample of early and middle adolescence youth was assessed, so there was sufficient power to test hypotheses. The sample was relatively racially and ethnically diverse and represented a wide socio-economic range, as opposed to predominantly White, middle-class samples used in much past research. Finally, the sample was recruited from the community, as opposed to outpatient or inpatient clinics, so the results should be more generalizable, less prone to Berkson's bias, and the effect sizes should be more appropriately and accurately estimated as compared with a purely clinic-referred sample (Cohen and Cohen 1984). Still, replication of these results using an enriched clinic sample with diagnoses would help to confirm whether these findings apply to more severe, comorbid, clinically referred youth.

In sum, results from this multi-wave prospective study of early and middle adolescents showed that HT's negative inferential style X stress interaction specifically predicted prospective elevations in general and specific anhedonic depressive symptoms, but not co-occurring anxious arousal, general internalizing, or externalizing symptoms. In contrast, stressors predicted anxious arousal, general internalizing, and externalizing behaviors over time. These findings advance knowledge on specific and general risk processes that predict depressive and co-occurring anxiety and externalizing symptoms.

References

Abela JRZ. The hopelessness theory of depression: A test of the diathesis-stress and causal mediation components in third and seventh grade children. Journal of Abnormal Child Psychology 2001;29:241–254. [PubMed: 11411786]

Abela JRZ, Adams P, Hankin BL. The timing of parent and child depression: A Hopelessness Theory perspective. Journal of Clinical Child and Adolescent Psychology 2006a;35:253–263. [PubMed: 16597221]

Abela, JRZ.; Hankin, BL. Cognitive vulnerability to depression in children and adolescence: A developmental psychopathology approach. In: Abela, JRZ.; Hankin, BL., editors. Handbook of child and adolescent depression. NY: Guilford Press; 2008. p. 35-78.

Abela JRZ, Sarin S. Cognitive vulnerability to hopelessness deppression: A chain is only as strong as its weakest link. Cognitive Therapy and Research 2002;26:811–829.

Abela JRZ, Sullivan C. A test of Beck's cognitive diathesis-stress theory of depression in early adolescents. Journal of Early Adolescence 2003;23:384–404.

Abela JRZ, Zuroff DC, Ho R, Adams P, Hankin BL. Excessive reassurance seeking, hassles, and depressive symptoms in children of affectively-ill parents: A multi-wave longitudinal study. Journal of Abnormal Child Psychology 2006b;34:171–188. [PubMed: 16555142]

Abramson, LY.; Alloy, LB.; Hankin, BL.; Haeffel, GJ.; MacCoon, D.; Gibb, BE. Cognitive vulnerability–stress models of depression in a self-regulatory and psychobiological context. In: Gotlib, IH.; Hammen, C., editors. Handbook of depression. New York: The Guilford Press; 2002. p. 295-313.

Abramson LY, Metalsky GI, Alloy LB. Hopelessness depression: A theory-based subtype of depression. Psychological Review 1989;96:358–372.

Albano, AM.; Chorpita, BF.; Barlow, D. Childhood anxiety disorders. In: Mash, EJ.; Barkley, RA., editors. Child psychopathology. Vol. 2. NY: Guilford Press; 2003. p. 279-329.

Alloy, LB.; Kelly, KA.; Mineka, S. Comorbidity of anxiety and depressive disorders: A helplessness-hopelessness perspective. In: Maser, JD.; Cloninger, RC., editors. Comorbidity of mood and anxiety disorders. Washington, DC, US: American Psychiatric Association; 1990. p. 499-543.

Angold A, Costello EJ, Erkanli A. Comorbidity. Journal of Child Psychology and Psychiatry and Allied Disciplines 1999;40:57–87.

Avenevoli, S.; Knight, E.; Kessler, RC.; Merikangas, KR. The epidemiology of depression in children and adolescents. In: Abela, JRZ.; Hankin, BL., editors. Handbook of child and adolescent depression. NY: Guilford; 2008. p. 6-34.

Avenevoli S, Stolar M, Li J, Dierker L, Merikangags K. Comorbidity of depression in children and adolescents: Models and evidence from a prospective high-risk family study. Biological Psychiatry 2001;49:1071–1081. [PubMed: 11430849]

Bell, DJ.; Foster, SL.; Mash, EJ. Understanding behavioral and emotional problems in girls. In: Bell, DJ.; Foster, SL.; Mash, EJ., editors. Handbook of behavioral and emotional problems in girls. New York, NY, US: Kluwer Academic/Plenum Publisher; 2005. p. 1-24.

Birmaher B, Ryan ND, Williamson DE, Brent DA, Kaufman J, Dahl RE, et al. Childhood and adolescent depression: A review of the past 10 years. Part I. Journal of American Academy of Child and Adolescent Psychiatry 1996;35:1427–1439.

Bolger N, Davis A, Rafaeli E. Diary methods: Capturing life as it is lived. Annual Review of Psychology 2003;54:579–616.

Brozina K, Abela JRZ. Symptoms of depression and anxiety in children: Specificity of the hopelessness theory. Journal of Clinical Child and Adolescent Psychology 2006;35:515–527. [PubMed: 17007597]

Bryk, AS.; Raudenbush, SW. Hierarchical linear models: Applications and data analysis methods. Thousand Oaks, CA, US: Sage Publications, Inc; 1992.

Chorpita BF, Albano AM, Barlow DH. The structure of negative emotions in a clinical sample of children and adolescents. Journal of Abnormal Psychology 1998;107:74–85. [PubMed: 9505040]

Chorpita BF, Moffitt CE, Gray J. Psychometric properties of the Revised Child Anxiety and Depression Scale in a clinical sample. Behaviour Research and Therapy 2005;43:309–322. [PubMed: 15680928]

Clark LA, Watson D. Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. Journal of Abnormal Psychology 1991;100:316–336. [PubMed: 1918611]

Cohen P, Cohen J. The clinician's illusion. Archives of General Psychiatry 1984;41:1178–1182. [PubMed: 6334503]

Cohen P, Cohen J, Kasen S, Velez CN. An epidemiological study of disorders in late childhood and adolescence: I. Age and gender specific prevalence. Journal of Child Psychology and Psychiatry and Allied Disciplines 1993;34:851–867.

Cole DA, Turner JE. Models of cognitive mediation and moderation in child depression. Journal of Abnormal Psychology 1993;102:271–281. [PubMed: 8315139]

Costello, EJ.; Angold, A. Developmental epidemiology. In: Cicchetti, D.; Cohen, DJ., editors. Develomental psychopathology. Vol. 2. NY: Wiley; 2006. p. 41-75.

Costello EJ, Mustillo S, Erkanli A, Keeler G, Angold A. Prevalence and development of psychiatric disorders in childhood and adolescence. Archives of General Psychiatry 2003;60:837–844. [PubMed: 12912767]

Curran, PJ.; Bollen, KA. The best of both worlds: Combining autoregressive and latent curve models. In: Collins, LM.; Sayer, AG., editors. New methods for the analysis of change: Decade of behavior. Washington, D.C: APA; 2001. p. 107-135.

Curran PJ, Willoughby MT. Implications of latent trajectory models for the study of developmental psychopathology. Development And Psychopathology 2003;15(3):581–612. [PubMed: 14582933]

Dixon JF, Ahrens AH. Stress and attributional style as predictors of self-reported depression in children. Cognitive Therapy & Research 1992;16:623–634.

Fergusson DM, Horwood LJ, Ridder EM. Subthreshold Depression in Adolescence and Mental Health Outcomes in Adulthood. Archives of General Psychiatry 2005;62:66–72. [PubMed: 15630074]

Garber, J. Development and depression. In: Sameroff, AJ.; Lewis, M.; Miller, SM., editors. Handbook of developmental psychopathology. Vol. 2. New York: luwer Academic/Plenum; 2000. p. 467-490.

Gibb BE, Alloy LB. A prospective test of the hopelessness theory of depression in children. Journal of Clinical Child and Adolescent Psychology 2006;35:4–274.

Gibb BE, Beevers CG, Andover MS. The hopelessness theory of depression: A prospective multi-wave test of the vulnerability–stress hypothesis. Cognitive Therapy and Research 2007;30:763–772.

Gladstone TRG, Kaslow NJ, Seeley JR, Lewinsohn PM. Sex differences, attributional style, and depressive symptoms among adolescents. Journal of Abnormal Child Psychology 1997;25:297–305. [PubMed: 9304446]

Goodman R. Psychometric properties of the Strengths and Difficulties Questionnaire (SDQ). Journal of the American Academy of Child and Adolescent Psychiatry 2001;40:1337–1345. [PubMed: 11699809]

Gotlib IH, Lewinsohn PM, Seeley JR. Symptoms versus a diagnosis of depression: Differences in psychosocial functioning. Journal of Consulting and Clinical Psychology 1995;63:90–100. [PubMed: 7896995]

Hammen C. The generation of stress in the course of unipolar depression. Journal of Abnormal Psychology 1991;100:555–561. [PubMed: 1757669]

Hammen C. Stress and depression. Annual Review of Clinical Psychology 2005;1:293–319.

Hammen C, Adrian C, Hiroto D. A longitudinal test of the attributional vulnerability model in children at risk for depression. British Journal of Clinical Psychology 1988;27:37–46. [PubMed: 3355906]

Hammen, C.; Rudolph, KD. Childhood mood disorders. In: Mash, EJ.; Barkley, RA., editors. Child psychopathology. Vol. 2. NY: Guilford Press; 2003. p. 233-278.

Hankin BL. Response styles theory and symptom specificity in adolescents: A multi-wave prospective study. Journal of Clinical Child and Adolescent Psychology. 2008in press

Hankin, BL.; Abela, JRZ. Depression from childhood through adolescence and adulthood: A developmental vulnerability–stress perspective. In: Hankin, BL.; Abela, JRZ., editors. Development of psychopathology: A vulnerability–stress perspective. Thousand Oaks, California: Sage Publications; 2005. p. 245-288.

Hankin BL, Abramson LY. Development of gender differences in depression: An elaborated cognitive vulnerability–transactional stress theory. Psychological Bulletin 2001;127:773–796. [PubMed: 11726071]

Hankin BL, Abramson LY. Measuring cognitive vulnerability to depression in adolescence: Reliability, validity, and gender differences. Journal of Child and Adolescent Clinical Psychology 2002;31:491–504.

Hankin BL, Abramson LY, Miller N, Haeffel GJ. Cognitive vulnerability–stress theories of depression: Examining affective specificity in the prediction of depression versus anxiety in three prospective studies. Cognitive Therapy and Research 2004;28:309–345.

Hankin BL, Abramson LY, Moffitt TE, McGee R, Silva PA, Angell KE. Development of depression from preadolescence to young adulthood: Emerging gender differences in a 10-year longitudinal study. Journal of Abnormal Psychology 1998;107:128–140. [PubMed: 9505045]

Hankin BL, Abramson LY, Siler M. A prospective test of the hopelessness theory of depression in adolescence. Cognitive Therapy and Research 2001;25:607–632.

Hankin BL, Fraley RC, Lahey BB, Waldman I. Is youth depressive disorder best viewed as a continuum or discrete category? A taxometric analysis of childhood and adolescent depression in a population-based sample. Journal of Abnormal Psychology 2005;114:96–110. [PubMed: 15709816]

Hankin, BL.; Wetter, E.; Cheely, CA. Sex differences in child and adolescent depression: A developmental psychopathological approach. In: Abela, JRZ.; Hankin, BL., editors. Handbook of child and adolescent depression. New York, NY: Guilford Press; 2008a. p. 377-414.

Hankin BL, Wetter E, Cheely C, Oppenheimer C. Beck's cognitive theory of depression in adolescence: Prospective prediction of depressive symptoms and transactional influences in a multi-wave study. International Journal of Cognitive Therapy. 2008b(in press)

Hilsman R, Garber J. A test of the cognitive diathesis-stress model of depression in children: Academic stressors, attributional style, perceived competence, and control. Journal of Personality & Social Psychology 1995;69:370–380. [PubMed: 7643310]

Hinshaw, SP.; Lee, SP. Conduct and oppositional defiant disorders. In: Mash, EJ.; Barkley, RA., editors. Child psychopathology. Vol. 2. NY: Guilford Press; 2003. p. 144-198.

Ingram, RE.; Miranda, J.; Segal, ZV. Cognitive vulnerability to depression. New York, NY: Guilford Press; 1998.

Joiner TE. A test of the hopelessness theory of depression in youth psychiatric inpatients. Journal of Clinical Child Psychology 2000;29:167–176. [PubMed: 10802826]

Joiner TE Jr, Catanzaro SJ, Laurent J. Tripartite structure of positive and negative affect, depression, and anxiety in child and adolescent psychiatric inpatients. Journal of Abnormal Psychology 1996;105:401–409. [PubMed: 8772010]

Joormann J, Talbot L, Gotlib IH. Biased processing of emotional information in girls at risk for depression. Journal of Abnormal Psychology 2007;116:135–143. [PubMed: 17324024]

Keenan K, Hipwell AE. Preadolescent clues to understanding depression in girls. Clinical Child and Family Psychology Review 2005;8:89–105. [PubMed: 15984082]

Kim-Cohen J, Caspi A, Moffitt TE, Harrington H, Milne BJ, Poulton R. Prior juvenile diagnoses in adults with mental disorder: Developmental follow-back of a prospective-longitudinal cohort. Archives of General Psychiatry 2003;60:709–717. [PubMed: 12860775]

King NJ, Ollendick TH, Gullone E. Test anxiety in children and adolescents. Australian Psychologist 1991;26:25–32.

Klein DN, Dougherty LR, Olino TM. Added toward guidelines for evidence-based assessment of depression in children and adolescents. Journal of Clinical Child and Adolescent Psychology 2005;34:412–432. [PubMed: 16026212]

Kovacs M. Rating scales to assess depression in school children. Acta Paedopsychiatrica 1981;46:305–315. [PubMed: 7025571]

Krueger RF, Markon KE. Understanding psychopathology: Melding behavior genetics, personality, and quantitative psychology to develop an empirically based model. Current Directions in Psychological Science 2006;15:113–117. [PubMed: 18392116]

Krueger RF, Markon KE, Patrick CJ, Iacono WG. Externalizing psychopathology in adulthood: A dimensional spectrum conceptualization and its implications for DSM-V. Journal of Abnormal Psychology 2005;114:537–550. [PubMed: 16351376]

Lahey BB, Applegate B, Waldman I, Hankin BL, Rick J. The Structure of Child and Adolescent Psychopathology: Generating New Hypothesis. Journal of Abnormal Psychology 2004;113:358–385. [PubMed: 15311983]

Lahey, BB.; Moffitt, TE.; Caspi, A. Causes of conduct disorder and juvenile delinquency. New York: Guilford; 2003.

Lahey BB, Loeber R, Burke J, Rathouz PJ, McBurnett K. Waxing and waning in concert: Dynamic comorbidity of conduct disorder with other disruptive and emotional problems over 17 years among clinic-referred boys. Journal of Abnormal Psychology 2002;111:556–567. [PubMed: 12428769]

Lakdawalla Z, Hankin BL, Mermelstein R. Cognitive theories of depression in children and adolescents: A conceptual and quantitative review. Child Clinical and Family Psychology Review 2007;10:1–24.

Lewinsohn PM, Gotlib IH, Lewinsohn M, Seeley JR, Allen NB. Gender differences in anxiety disorders and anxiety symptoms in adolescents. Journal of Abnormal Psychology 1998;107:109–118. [PubMed: 9505043]

Lewinsohn PM, Hops H, Roberts RE, Seeley JR, Andrews JA. Adolescent psychopathology:1. Prevalence and incidence of depression and other DSM-III-R disorders in high school students. Journal of Abnormal Psychology 1993;102:133–144. [PubMed: 8436689]

Lewinsohn PM, Joiner TE, Rohde P. Evaluation of cognitive diathesis-stress models in predicting major depressive disorder in adolescents. Journal of Abnormal Psychology 2001;110:203–215. [PubMed: 11368074]

Lewinsohn PM, Rohde P, Seeley JR. Adolescent psychopathology: III. The clinical consequences of comorbidity. Journal of the American Academy of Child & Adolescent Psychiatry 1995;34:510–519. [PubMed: 7751265]

Lewinsohn PM, Seeley JR, Gotlib IH. Depression related psychosocial variables: Are they specific to depression in adolescents? Journal of Abnormal Psychology 1997;106:365–375. [PubMed: 9241938]

Lonigan CJ, Hooe ES, David CF, Kistner JA. Positive and negative affectivity in children: Confirmatory factor analysis of a two-factor model and its relation to symptoms of anxiety and depression. Journal of Consulting and Clinical Psychology 1999;67:374–386. [PubMed: 10369058]

Luten A, Ralph J, Mineka S. Depressive attributional style: Is it specific to depression vs. anxiety vs. negative affect? Behavior Research and Therapy 1997;8:703–720.

McMahon SD, Grant KE, Compas BE, Thurm AE, Ey S. Stress and psychopathology in children and adolescents: Is there evidence of specificity? Journal of Child Psychology and Psychiatry 2003;44:107–133. [PubMed: 12553415]

Meehl, PE. Psychodiagnosis: Selected papers. Oxford, England: U. Minnesota Press; 1977.

Metalsky GI, Joiner TE Jr. Vulnerability to depressive symptomatology: A prospective test of the diathesis- stress and causal mediation components of the hopelessness theory of depression. Journal of Personality and Social Psychology 1992;63:667–675. [PubMed: 1447690]

Mineka S, Watson D, Clark LA. Comorbidity of anxiety and unipolar mood disorders. Annual Review of Psychology 1998;49:377–412.

Moffitt, TE.; Caspi, A.; Rutter, M.; Silva, PA. Sex differences in antisocial behaviour. Cambridge, England: Cambridge University Press; 2001.

Monroe SM, Roberts JE. Conceptualizing and measuring life stress: Problems, principles. procedures, progress. Stress Medicine, 6, Special issue: IIV. Advances in measuring life stress 1990:209–216.

Monroe SM, Simons AD. Diathesis-stress theories in the context of life stress research: Implications for the depressive disorders. Psychological Bulletin 1991;110:406–425. [PubMed: 1758917]

Newman DL, Moffitt TE, Caspi A, Silva PA. Comorbid mental disorders: Implications for treatment and sample selection. Journal of Abnormal Psychology 1998;107:305–311. [PubMed: 9604559]

Nolen-Hoeksema S, Girgus JS, Seligman MEP. Predictors and consequences of childhood depressive symptoms: A 5-year longitudinal study. Journal of Abnormal Psychology 1992;101:405–422. [PubMed: 1500598]

Osgood DW, McMorris BJ, Potenza MT. Analyzing multiple-item measures of crime and deviance I: Item response theory scaling. Journal of Quantitative Criminology 2002;18:267–296.

Pine DS, Cohen P, Gurley D, Brook J, Ma Y. The risk for early-adulthood anxiety and depressive disorders in adolescents with anxiety and depressive disorders. Archives of General Psychiatry 1998;55:56–64. [PubMed: 9435761]

Ralph J, Mineka S. Attributional style and self esteem: The prediction of emotional distress following a midterm exam. Journal of Abnormal Psychology 1998;107:203–215. [PubMed: 9604550]

Raudenbush SW. Comparing personal trajectories and drawing causal inferences from longitudinal data. Annual Review of Psychology 2000;52:501–525.

Raudenbush, SW.; Bryk, A.; Cheong, YF.; Congdon, R. Hierarchical linear and nonlinear modeling 5.04. Lincolnwood, IL: Scientific Software International; 2001.

Rivas-Vazquez RA, Saffa-Biller D, Ruiz I. Current Issues in Anxiety and Depression: Comorbid, Mixed, and Subthreshold Disorders. Professional Psychology: Research and Practice 2004;34:74–83.

Robinson NS, Garber J, Hilsman R. Cognitions and stress: Direct and moderating effects on depressive versus externalizing symptoms during the junior high transition. Journal of Abnormal Psychology 1995;104:453–463. [PubMed: 7673569]

Rudolph, KD.; Hammen, C.; Daley, SE. Mood disorders. In: Wolfe, DA.; Mash, EJ., editors. Behavioral and emotional disorders in adolescents: Nature, assessment, and treatment. New York, NY, US: Guilford Publications; 2006. p. 300-342.

Rutter M, Caspi A, Moffitt TE. Using sex differences in psychopathology to study causal mechanisms: Unifying issues and research. Journal of Child Psychiatry and Psychology 2003;44:1092–1115.

Scher CD, Ingram RE, Segal ZV. Cognitive reactivity and vulnerability: Empirical evaluation of construct activation and cognitive diatheses in unipolar depression. Clinical Psychology Review 2005;25:487–510. [PubMed: 15914266]

Seligman LD, Ollendick TH. Comorbidity of anxiety and depression in children and adolescents: An integrative review. Clinical Child and Family Psychology Review 1998;1:125–144. [PubMed: 11324302]

Simons AD, Angell KL, Monroe SM. Cognition and life stress in depression: Cognitive factors and the definition, rating, and generation of negative life events. Journal of Abnormal Psychology 1993;102:584–591. [PubMed: 8282928]

Stark KD, Laurent J. Joint factor analysis of the Children's Depression Inventory and the Revised Children's Manifest Anxiety Scale. Journal of Clinical Child Psychology 2001;30:552–567. [PubMed: 11708242]

Swendsen JD. Anxiety, depression, and their comorbidity: An experience sampling test of the Helplessness-Hopelessness Theory. Cognitive Therapy & Research 1997;21:97–114.

Timbremont B, Braet C, Dreessen L. Assessing depression in youth: relation between the Children's Depression Inventory and a structured interview. Journal of Clinical Child and Adolescent Psychology 2004;33:149–157. [PubMed: 15028549]

Tram JM, Cole DA. A multimethod examination of the stability of depressive symptoms in childhood and adolescence. Journal of Abnormal Psychology 2006;115:674–686. [PubMed: 17100525]

Twenge JM, Nolen-Hoeksema S. Age, gender, race, socioeconomic status, and birth cohort difference on the children's depression inventory: A meta-analysis. Journal of Abnormal Psychology 2002;111:578–588. [PubMed: 12428771]

Vasey, MW.; Ollendick, TH. Anxiety. In: Sameroff, AJ.; Lewis, M.; Miller, S., editors. Handbook of developmental psychopathology. Vol. 2. Dordrecht, Netherlands: Kluwer Academic Publishers; 2000. p. 511-529.

Wade TJ, Cairney J, Pevalin DJ. Emergence of gender differences in depression during adolescence: National panel results from three countries. Journal of the American Academy of Child and Adolescent Psychiatry 2002;41(2):190–198. [PubMed: 11837409]

Watson D. Rethinking the mood and anxiety disorders: A quantitative hierarchical model for DSM-V. Journal of Abnormal Psychology 2005;114:522–536. [PubMed: 16351375]

Watson D, Weber K, Assenheimer JS, Clark LA, Strauss ME, McCormick RA. Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression scales. Journal of Abnormal Psychology 1995;104:3–14. [PubMed: 7897050]

Weiss B, Susser K, Catron T. Common and specific features of childhood psychopathology. Journal of Abnormal Psychology 1998;107:118–128. [PubMed: 9505044]

Vollebergh WAM, Iedema J, Bijl RV, de Graaf R, Smit F, Ormel J. The structure and stability of common mental disorders: The NEMESIS study. Archives of General Psychiatry 2001;58:597–603. [PubMed: 11386990]

Zahn-Waxler, C.; Crick, NR.; Shirtcliff, EA.; Woods, KE. The origins and development of psychopathology in females and males. In: Cicchetti, D.; Cohen, DJ., editors. Developmental psychopathology, Vol 1. Theory and method. Vol. 2. New Jersey: John Wiley & Sons; 2006. p. 76-138.

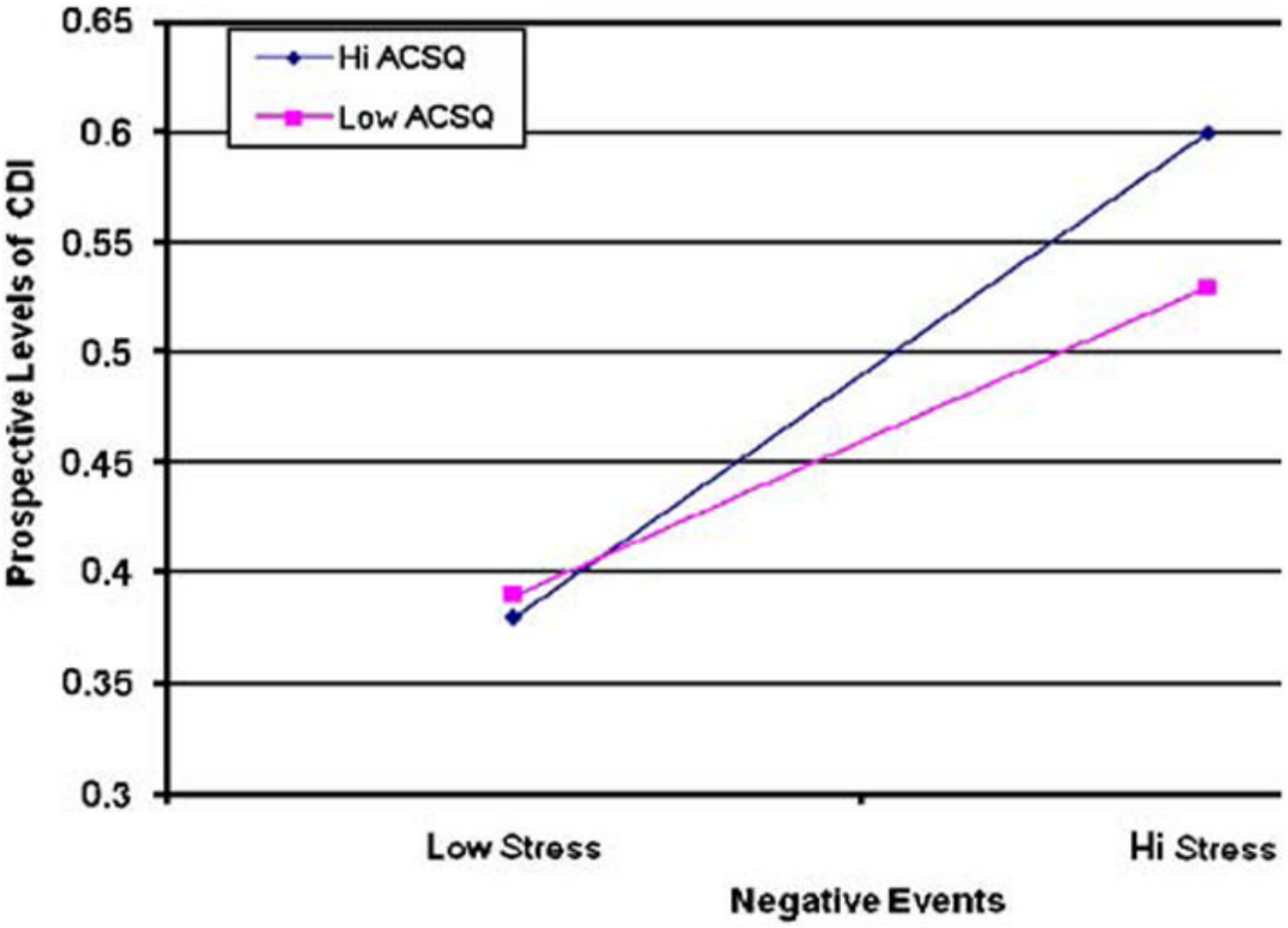

Fig. 1.
Interaction of baseline negative inferential style by within-person stressors over time predicting prospective fluctuations in depressive symptoms after controlling for the prior data wave of depressive symptoms. *ACSQ* HT's negative inferential style

Table 1

Descriptive Statistics and Correlations Among Main Measures at Baseline and Over Time

Parameter	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
ACSQ																					
INTERN1	0.34																				
CDI1	0.42	0.65																			
ANX1	0.41	0.62	0.63																		
EXTERN1	0.33	0.36	0.46	0.37																	
INTERN2	0.38	0.55	0.60	0.71	0.22																
CDI2	0.42	0.54	0.74	0.56	0.35	0.79															
ANX2	0.42	0.48	0.61	0.56	0.33	0.71	0.83														
EXTERN2	0.33	0.40	0.57	0.47	0.45	0.59	0.74	0.64													
INTERN3	0.41	0.50	0.57	0.51	0.27	0.59	0.61	0.53	0.47												
CDI3	0.40	0.50	0.67	0.55	0.30	0.58	0.69	0.57	0.51	0.83											
ANX3	0.36	0.51	0.58	0.58	0.29	0.56	0.63	0.57	0.49	0.85	0.87										
EXTERN3	0.31	0.29	0.36	0.29	0.40	0.23	0.37	0.30	0.45	0.46	0.53	0.54									
INTERN4	0.33	0.57	0.57	0.62	0.56	0.63	0.64	0.50	0.48	0.65	0.65	0.62	0.33								
CDI4	0.33	0.56	0.70	0.56	0.37	0.59	0.68	0.52	0.51	0.63	0.70	0.64	0.39	0.89							
ANX4	0.39	0.53	0.64	0.56	0.27	0.59	0.64	0.53	0.47	0.61	0.65	0.63	0.30	0.88	0.85						
EXTERN4	0.32	0.37	0.48	0.38	0.35	0.33	0.45	0.34	0.46	0.41	0.43	0.41	0.51	0.65	0.67	0.63					
ALEQ1	0.23	0.32	0.33	0.43	0.37	0.25	0.29	0.28	0.32	0.24	0.22	0.26	0.29	0.42	0.41	0.41	0.51				
ALEQ2	0.33	0.35	0.44	0.41	0.35	0.54	0.59	0.60	0.60	0.41	0.45	0.44	0.40	0.44	0.46	0.45	0.38	0.33			
ALEQ3	0.21	0.35	0.39	0.35	0.36	0.36	0.41	0.34	0.39	0.53	0.50	0.56	0.56	0.42	0.45	0.36	0.44	0.21	0.31		
ALEQ4	0.25	0.38	0.47	0.39	0.36	0.38	0.44	0.37	0.45	0.40	0.42	0.41	0.41	0.65	0.66	0.69	0.75	0.25	0.40	0.42	
Mean	3.17	0.75	0.47	2.20	0.52	0.67	0.45	2.2	0.45	0.76	0.45	2.21	0.47	0.77	0.56	2.22	0.55	0.54	0.47	0.47	0.46
SD	1.12	0.47	0.32	0.75	0.39	0.38	0.34	0.73	0.32	0.39	0.34	0.72	0.28	0.46	0.48	0.76	0.30	0.21	0.21	0.22	0.26
Coefficient α	0.80	0.67	0.90	0.86	0.75	0.63	0.91	0.85	0.70	0.72	0.91	0.83	0.63	0.58	0.90	0.85	0.68	–	–	–	

All correlations above 0.16 are significant at $p<0.01$ and correlations above 0.18 are significant at $p<0.001$

ACSQ Negative inferential style, *ALEQ* Adolescent Life Events Questionnaire, *CDI* children's depression inventory, *ANX* anxious arousal, *EXTERN* externalizing behaviors, *INTERN* general internalizing symptoms

J Abnorm Child Psychol. Author manuscript; available in PMC 2009 October 19.

Table 2

Negative Inferential Style × Stress Interactions Predicting Prospective Trajectories of Symptoms of Depression, Anhedonic Depression, Anxious Arousal, General Internalizing, and Externalizing Behaviors

Predictor	*b*	SE	*tdf*
Depressive symptoms			
CDI-1	0.02	0.009	2.671, 347**
ACSQ	0.12	0.05	2.371, 347**
Stress	0.84	0.06	13.451, 347***
ACSQ × stress	0.01	0.003	2.061, 347*
Anhedonic depressive symptoms			
ANH-CDI-1	0.003	0.004	0.641, 347
ACSQ	0.05	0.03	1.481, 347
Stress	0.08	0.03	2.121, 347*
ACSQ × Stress	0.04	0.002	16.391, 347***
Anxious arousal symptoms			
ANX-1	0.08	0.01	4.861, 347***
ACSQ	0.06	0.04	1.391, 347
STRESS	1.65	0.57	2.891, 347***
ACSQ × stress	0.17	0.11	1.571, 347
General internalizing symptoms			
SDQ—INTERNALIZING-1	0.12	0.06	2.091, 347*
ACSQ	0.11	0.21	0.551, 347
Stress	3.82	0.1.62	2.361, 347**
ACSQ × stress	0.06	0.37	0.161, 347
Externalizing symptoms			
SDQ—externalizing-1	0.07	0.02	3.891, 347***
ACSQ	0.13	0.06	2.091, 347*
Stress	1.65	0.57	2.861, 347***
ACSQ × stress	0.02	0.08	0.331, 347