Beck’s Cognitive Theory of Depression in Adolescence: Specific Prediction of Depressive Symptoms and Reciprocal Influences in a Multi-Wave Prospective Study

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Little research has investigated Beck’s (1987) cognitive theory of depression among youth, symptom specificity, and potential reciprocal influences over time. Dysfunctional attitudes were assessed at baseline and study’s end among adolescents (N = 350, 6\textsuperscript{th} to 10\textsuperscript{th} grade). Depressive, anxious arousal, and externalizing symptoms and stressors were assessed at four time points over a 5-month period. Hierarchical linear modeling showed that negative events predicted prospective elevations in symptoms. Dysfunctional attitudes interacted with negative events to predict prospective anhedonic depressive symptoms specifically but not general depression, anxious arousal, or externalizing symptoms. Initial depressive symptoms and stressors predicted changes in dysfunctional attitudes, and this reciprocal effect was stronger for girls than boys.

Depression is one of the most commonly diagnosed psychiatric disorders and has been ranked as the fourth leading cause of work disability and premature death worldwide (Murray & Lopez, 1996). Prospective longitudinal studies indicate that rates of depression increase dramatically during adolescence, with adolescents between 15 and 18 years old experiencing a six-fold increase in rates of clinical depression (from 3% to 17%) (Hankin et al., 1998). It is important to identify psychosocial vulnerabilities that contribute to the ontogeny of depression across development. Cognitive theories of depression provide a promising way to understand the developmental etiology and maintenance of depression among adolescents.

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One cognitive theory that has generated a large body of theoretical and empirical support is Beck's (1967, 1983) cognitive theory of depression. Beck's theory (BT) proposes that negative, depressogenic schema, which contain dysfunctional attitudes (i.e., rigid and extreme beliefs about the self and the world that are drawn from certain early experiences in life), influence how an individual interprets, encodes, and retrieves information. Many, but not all (cf., Ingram, Miranda, & Segal, 1998), cognitive theorists have operationalized depressogenic schema as sets of dysfunctional attitudes, and the empirical work has assessed dysfunctional attitudes as the operationalization of latent core depressogenic schema. Given that past work has used self-reported dysfunctional attitudes as the most common method for assessing negative schemas, the present study followed this precedent and focused on self-reported dysfunctional attitudes. Still, it is important to be clear that Beck primarily theorized about and focused on negative schemas, and dysfunctional attitudes are hypothesized to be cognitive products that are accessible to self-reporting and that arise from latent negative schema.

BT contains a vulnerability-stress component, such that dysfunctional attitudes (the vulnerability) are typically latent in vulnerable individuals and must be activated by a relevant negative event (the stressor) in order to contribute to increases in depressive symptoms. For example, the dysfunctional attitude, "If everyone doesn't love me, then I am worthless," can predispose a person to experience depressed affect if he or she perceives social rejection. As such, individuals who possess depressotypic dysfunctional attitudes are theorized to experience increases in depressive symptoms following the occurrence of negative events.

To provide an adequate test of the etiological chain proposed in BT, it is important to conduct longitudinal studies in which initial levels of dysfunctional attitudes are assessed prior to the occurrence of negative events or potential elevations of depressive symptoms. The majority of past longitudinal research that has examined Beck's theory has been conducted with adults and has received broad support (for reviews, see Abramson et al., 2002; Scher, Ingram, & Segal, 2005). However, there is a growing movement to extend this body of research from adults to adolescents. Due to the increase in stressors encountered during adolescence (Ge, Lorenz, & Rand, 1994; Rudolph & Hammern, 1999) that parallels the dramatic increase in depression (Angold, Erkanli, Silberg, Eaves, & Costello, 2002; Hankin et al., 1998), this developmental period provides a rich opportunity to investigate how cognitive vulnerabilities may interact with the increasing trajectory of stressors to predict increases in depressive symptoms (Hankin & Abramson, 2001).

Recent reviews of cognitive theories of depression in adolescents (Abela & Hankin, 2008; Lakdawalla, Hankin, & Merelstein, 2007) reveal that only three longitudinal studies have examined dysfunctional attitudes x stress interactions in adolescent samples. Specifically, Abela and Sullivan (2003) examined BT in a longitudinal study of early adolescents and found that dysfunctional attitudes interacted with stress to predict increases in depressive symptoms across a 6-week period in seventh-grade students possessing high, but not low, levels of social support and self-esteem. Lewinsohn, Joiner, and Rohde (2001) found that dysfunctional attitudes interacted with stressors to predict the onset of major depression in a community sample of adolescents, but only when dysfunctional attitudes exceeded a threshold level. Finally, Abela and Skitch (2007) investigated the vulnerability-stress component of Beck's cognitive theory in a sample of 140 at-risk youth across a 1-year period. Hierarchical linear modeling revealed that youth with both high levels of dysfunctional attitudes and low self-esteem who encountered high levels of stressors demonstrated the greatest...
increase in depressive symptoms. In sum, the scant available research is fairly mixed, so additional research is needed to investigate the basic dysfunctional attitudes x stressors interaction among adolescents.

DEPRESSION CO-OCCURRENCE WITH OTHER EMOTIONAL AND BEHAVIORAL PROBLEMS

Depression and other emotional and behavioral problems, such as anxiety and conduct disorders, commonly co-occur (Angold, Costello, & Erkanli, 1999; Newman, Moffitt, & Caspi, 1996). Comorbid depression is associated with more severe symptoms and correlates as well as worse clinical course and potential treatment outcomes (Birmaher et al., 1996). Investigating patterns of comorbidity can advance knowledge on etiological theories, and demonstrating evidence for predictive specificity provides a critical step to evaluate hypothesized cognitive vulnerability-stress processes.

However, relatively few studies in the literature have tested the specificity of etiological influences contributing to depression and overlapping anxiety and externalizing symptoms in general. Many vulnerability factors for depression (e.g., Hankin & Abela, 2005), anxiety (e.g., Albano, Chorpita, & Barlow, 2003), and externalizing problems (e.g., Hinshaw & Lee, 2003) have been identified, yet considerably less research has studied the degree to which these vulnerability factors are specific to depressive symptoms or common to depressive, anxious, and externalizing symptoms. With respect to the specificity of BT's in depression, only one study (Lewinsohn, Joiner, & Rohde, 2001) reported symptom specificity. They found that the dysfunctional attitudes x stress interaction predicted onset of depressive disorders specifically, yet this study is limited in that all other co-occurring psychiatric disorders (e.g., anxiety and externalizing problems) were grouped together. Thus, the particular specificity of depression versus anxiety, as co-occurring internalizing syndromes, versus externalizing problems, is unknown in adolescence.

Indeed, past research has not considered and examined different facets of negative affect and emotional distress from a theoretically guided, empirically supported framework. In particular, structural models of psychopathology (e.g., Lahey, Applegate, Waldman, Hankin, & Rick, 2004; Watson, 2005) can guide predictions concerning hypothesized specificity and when etiological factors should be expected to relate to psychopathology broadly versus a particular disorder, such as depression, specifically. A well-studied structural model for internalizing problems, the tripartite theory of anxiety and depression (Clark & Watson, 1991), specifies that both depression and anxiety are comprised of general negative affect (e.g., symptoms of worry, poor concentration, etc.). Anxiety can be differentiated from general negative affect and depression by focusing on and assessing the more specific physiological symptoms of anxious arousal (e.g., heart palpitations, shortness of breath, etc.), and depression may be characterized by low positive affect (i.e., anhedonia). Research with youth (e.g., Chorpita, Albano, & Barlow, 1998; Joiner & Lonigan, 2000; Lonigan, Phillips, & Hope, 2003) has tested and supported the tripartite theory of anxiety and depression. In the present study, we assessed four dependent symptom measures based on and guided by these structural models of psychopathology—general depressive symptoms, specific anhedonic depressive symptoms, anxious arousal symptoms, and general externalizing problems—to examine whether the key dysfunctional attitudes x stress interaction
predicts depressive symptoms (general depression and/or anhedonia) more specifically than anxious arousal or externalizing symptoms.

SEX AND ETHNIC DIFFERENCES IN DEPRESSION

Most of the past studies have not sufficiently included 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 dysfunctional attitudes, stressors, and psychopathological symptoms. Moreover, there are well-documented sex effects on the development of depression (Hankin et al., 1998; Hankin, Wetter, & Checley, 2008; Twenge & Nolen-Hoeksema, 2002), such that depressive symptoms and disorder become increasingly more elevated and prevalent throughout adolescence, especially for girls. 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 sex differences, this study explored whether these demographic factors moderated the dysfunctional attitudes-stress interaction as a predictor of later depressive and co-occurring symptoms.

POTENTIAL TRANSACTIONAL PROCESSES IN BECK’S THEORY

In order to understand better the role of dysfunctional attitudes in depression in youth, a developmental psychopathological perspective that incorporates transactional models can inform an understanding of the development and maintenance of maladaptive psychological functioning (Cicchetti, 2006). Transactional models are a process-oriented approach that consider the dynamic and reciprocal interactions between a person and his/her environment (Sameroff & Mackenzie, 2003) and may be useful in understanding the ontogeny and chronicity of depressive symptoms in adolescence. Sameroff and Chandler (1975) proposed that a transactional model is an improvement over traditional “cause-effect” models that specify only one single main cause of developmental outcomes. Central to the transactional model is that effects of the individual and the environment are bidirectional, and outcomes can neither be attributed to the environment alone nor to characteristics of the individual alone. Furthermore, transactional processes can be amplifying (Sameroff & Mackenzie, 2003), such that positive feedback loops bring about escalating, cyclical effects. Transactional models can be used to explain the exacerbation or maintenance of symptoms. For example, Cicchetti and Schneider-Rosen (1984) theorized that a depressive episode may serve as a vulnerability factor for later depression in youth, and a transactional approach might be useful in explaining this phenomenon.

Consistent with this idea, Hankin and Abramson (2001) have incorporated transactional processes into the traditional cognitive vulnerability-stress model to demonstrate how certain factors, such as depressive symptoms or other distal risks (e.g., genetics, temperament, maltreatment), can initiate a causal chain that leads to more stressors, may worsen cognitive vulnerability, and ultimately contribute to even greater
increases in depressive symptoms over time. To date, little research has examined these reciprocal and potentially transactional processes. In an important study providing support for a vulnerability-transactional stress model, Gibb and Alloy (2006) found evidence for reciprocal associations among cognitive vulnerabilities, negative events, and depressive symptoms among youth. Specifically, verbal victimization prospectively predicted negative attributional style (Abramson, Seligman, & Teasdale, 1978) and depressive symptoms in fourth and fifth graders, and a negative attributional style mediated the relation between verbal victimization and depressive symptoms. In addition, depressive symptoms prospectively predicted verbal victimization and negative attributional style. These findings can be interpreted within, but do not prove, a transactional model of depression in which experiencing verbal abuse leads to negative attributional style and depressed mood, which in turn leads to additional experiences of verbal abuse and worsening of negative attributional style, bringing about increases in depressive symptoms. Thus, reciprocal relations among risk factors of depression, both within the environment and individual, and depression itself, can lead to a vicious cycle that maintains and intensifies depression.

Gibb and Alloy’s (2006) findings contribute to an emerging body of research that provides support for potentially transactional and reciprocal associations among depressive symptoms and cognitive vulnerabilities (see Nolen-Hoeksema, Stice, Wade, & Bohon, 2007, for an example with rumination), yet there is still a paucity of research in this area. Furthermore, although Beck theorized bidirectional effects between symptoms of depression and dysfunctional attitudes (Beck, 1967; Beck & Weishaar, 2005), no study to date has specifically examined reciprocal, bi-directional effects among depressive symptoms, stressors, and dysfunctional attitudes among youth.

THE CURRENT INVESTIGATION

The current study aims to provide a more powerful test of BT’s dysfunctional attitudes x stress interaction as a possibly specific predictor of depressive symptoms compared with anxious arousal and externalizing symptoms. In addition, potential transactional, bi-directional changes in dysfunctional attitudes were investigated. The study used a four-wave prospective design among a moderately large and ethnically diverse sample of sixth to tenth graders. The majority of past research in cognitive theories of depression has used two-time point designs to test hypotheses, yet developmental methodologists have noted that multi-wave designs are more powerful for examining developmental changes and prospective prediction of symptoms (Curran & Willoughby, 2003). Cross-sectional research cannot disentangle whether cognitive factors are a cause, consequence, or correlate of emotional distress (Barnett & Gotlib, 1988; Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). Multi-wave studies (i.e., a minimum of three time points) are needed to test rigorously and accurately longitudinal patterns and developmental processes, such as prediction of symptoms over time. We hypothesized that baseline dysfunctional attitudes would interact with stressors over time to predict prospective elevations in depressive symptoms specifically, but not externalizing behaviors or anxious arousal symptoms, and that initial depressive symptoms and stressors would predict prospective changes in dysfunctional attitudes.
METHOD

Participants

Participants were youth who were recruited from five Chicago area schools. Schools were selected to represent ethnic and socioeconomic 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. At baseline, 467 students were available in the appropriate grades (6th-10th) from these selected schools and were invited to participate. Parents of 390 youth (83.5%) provided active consent; all 390 youth were willing to participate. A total of 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, stressors, and dysfunctional attitudes) at baseline. Rates of participation in the study decreased slightly at each wave of follow-up: wave 2 \(N = 303\), wave 3 \(N = 308\), and wave 4 \(N = 345\). Age ranged from 11-17 years \(M = 14.5; \text{SD} = 1.40\); 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 returned active parental consent, read and signed an informed child consent form after asking 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 questionnaire packets at four time points over a 5-month period, with approximately five weeks between each time point. The spacing for the follow-up intervals was chosen based on past research (e.g., Hankin, Abramson, & Siler, 2001) that found cognitive vulnerability predicted prospective depressive symptoms using a 5-week follow-up. Also, because understanding the prospective dynamics among co-occurring symptoms of depression, anxiety, and externalizing problems was a key aspect of the study, a relatively short time frame was used to provide enhanced, accurate recall of symptoms (see Costello, Erkanli, & Angold, 2006, for evidence that shorter time frames provide more accurate, less biased findings). Youth were compensated
$10 for participation at each wave in the study, for a possible total of $40 for completing all four assessments.

Measures

*Children's Depression Inventory (CDI; Kovacs, 1985).* The CDI is a self-report measure that assesses depression in children and adolescents using 27 items. 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, Dougherty, & Olino, 2005). The range of CDI scores from this sample (total score mean CDI was 13.06; SD = 8.69) was comparable to published norms (Kovacs, 2001). Although the CDI is one of the most commonly used measure of assessing depressive symptoms among youth (Fristad, Emery, & Beck, 1997), its construct validity and specificity as a measure of depression has been questioned (e.g., Chorpita, Moffitt, & Gray, 2005; King, Ollendick, & Gullone, 1991), given that it appears to contain many items tapping broad negative affect (Chorpita et al., 1998). Thus, although the CDI is often and widely used to assess general depressive symptoms, 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, Catanaro, & Laurent, 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. In sum, analyses for depressive symptom specificity are reported for the full CDI to assess general depressive symptoms and for anhedonic CDI items (#4, 12, 15, 20, 21, 22; Chorpita et al., 1998).

*Mood and Anxiety Symptom Questionnaire (MASQ; Watson et al., 1995)*. 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 & Watson, 1991). 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. To be clear, the ANX subscale was not intended to represent and assess anxiety symptoms generally because more recent theory and data (Mineka, Watson, & Clarks, 1998; Watson, 2005) show that the anxious arousal scale is associated with particular anxiety disorders, including specific phobias and panic disorder. Youth responded to 10 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, in press-a, b; Watson et al., 1995). The MASQ was given at all 4 time points.

*Strengths and Difficulties Questionnaire (SDQ; Goodman, 2001).* The SDQ is a brief 25-item questionnaire that assesses general internalizing/emotional and externalizing/behavioral problems. A five-factor structure, consisting of emotional, conduct, hyperactivity—inattentive, peer, and prosocial factors, has been supported in past research with large samples of youth and parents (Goodman, 2001). The externalizing factor, comprised of conduct problems, was used for the present study. Normative data are available for the SDQ from a large national sample of children (Goodman, 2001). The descriptive statistics (means, standard deviation; see Table 1) from the present sample matched the descriptive data from the normative database closely. The SDQ has been shown to
be reliable and valid in past research (Goodman, 2001). Reported scores are the average item scores of all items (range 0-2). The SDQ was given at all 4 time points.

Dysfunctional Attitudes Scale (DAS; Lewinsohn et al., 2001). The DAS is a 9-item scale adapted from the adult version of the Dysfunctional Attitudes Scale (Weissman & Beck, 1978). The DAS assesses adolescents’ propensity to endorse dysfunctional attitudes, the cognitive vulnerability emphasized in BT. Andrews, Lewinsohn, Hops, and Roberts (1993) reported that the 9 items in the DAS loaded most highly onto a general dysfunctional attitudes factor, and these 9 items correlated highly with a full version of the DAS. Moderate test-retest reliability and good validity was reported among high school students (Lewinsohn et al., 2001). Internal consistency was \( \alpha = .70 \) at Time 1, and test-retest reliability from T1 to T4 in this sample was \( r = .22 \) (see Hankin, 2008, for stability of dysfunctional attitudes over time among youth). Adolescents rated the items on a 5-point Likert scale, with higher scores indicating greater levels of dysfunctional attitudes.

Adolescent Life Events Questionnaire (ALEQ; Hankin & Abramson, 2002). The ALEQ assesses a broad range of negative life events that typically occur among adolescents, 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. Youth were asked to indicate how often (Likert scale ranging from never (0) to always (4)) these negative events had occurred to them over the past 5 weeks. These ratings were then transformed into dichotomous count of stressors (0 = no event; 1 = event occurred). Average item-scores on the dichotomized ALEQ were used to assess a count of stressors (range from 0 to 1.0). Higher scores indicate more exposure to negative events over the past 5 weeks.

RESULTS

Preliminary Analyses

Descriptive statistics and intercorrelations for the main variables are presented in Table 1. The baseline measure of dysfunctional attitudes was moderately associated with depressive symptoms, but only weakly with anxious arousal and externalizing symptoms both concurrently and prospectively at different waves of data. The different psychopathological symptoms (i.e., depressive, anxious arousal, and externalizing) were associated with the other co-occurring symptoms, concurrently and prospectively across waves of data as expected.

A between-subjects t-test showed that there was a significant sex difference in dysfunctional attitude levels, \( t(1, 348) = 2.72, p < .01 \). Boys (\( M = 25.32, SD = 5.50 \)) exhibited higher levels of dysfunctional attitudes than girls (\( M = 23.64, SD = 5.94 \)). Age was not associated with dysfunctional attitudes (\( r = .01 \)). The Age x Sex interaction, tested via ANOVA, was not significant. Age and sex differences in symptom measures and stressors are reported elsewhere (see Hankin, in press-c). Briefly, girls
### TABLE 1. Descriptive Statistics and Correlations Among Main Measures at Baseline and Over Time

|                | 1    | 2    | 3    | 4    | 5    | 6    | 7    | 8    | 9    | 10   | 11   | 12   | 13   | 14   | 15   | 16   | 17   | 18   | 19   |
|----------------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|------|
| 1. DAS         | .28  |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |
| 2. CDI1        |      | .08  |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |
| 3. ANX1        |      |      | .63  |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |
| 4. EXTERN1     |      |      |      | .46  |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |
| 5. CDI2        |      |      |      |      | .23  |      |      |      |      |      |      |      |      |      |      |      |      |      |      |
| 6. ANX2        |      |      |      |      |      | .74  |      |      |      |      |      |      |      |      |      |      |      |      |      |
| 7. EXTERN2     |      |      |      |      |      |      | .56  |      |      |      |      |      |      |      |      |      |      |      |      |
| 8. CDI3        |      |      |      |      |      |      |      | .61  |      |      |      |      |      |      |      |      |      |      |      |
| 9. ANX3        |      |      |      |      |      |      |      |      | .67  |      |      |      |      |      |      |      |      |      |      |
| 10. EXTERN3    |      |      |      |      |      |      |      |      |      | .67  |      |      |      |      |      |      |      |      |      |
| 11. CDI4       |      |      |      |      |      |      |      |      |      |      | .68  |      |      |      |      |      |      |      |      |
| 12. ANX4       |      |      |      |      |      |      |      |      |      |      |      | .64  |      |      |      |      |      |      |      |
| 13. EXTERN4    |      |      |      |      |      |      |      |      |      |      |      |      | .63  |      |      |      |      |      |      |
| 14. ALEQ1      |      |      |      |      |      |      |      |      |      |      |      |      |      | .63  |      |      |      |      |      |
| 15. ALEQ2      |      |      |      |      |      |      |      |      |      |      |      |      |      |      | .43  |      |      |      |      |
| 16. ALEQ3      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      | .42  |      |      |      |
| 17. ALEQ4      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      | .41  |      |      |      |
| 18. AGE        |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      | .66  |      |      |
| 19. SEX        |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      | .75  |
| Mean           |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |
| SD             |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |      |

**Note.** DAS = Dysfunctional Attitudes Scale; CDI = Children's Depression Inventory; ANX = Anxious Arousal; EXTERN = Externalizing Behaviors; ALEQ = stressors. All correlations above .10 are significant at $p < .05$, correlations above .16 are significant at $p < .01$, and correlations above .18 are significant at $p < .001$. 

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reported more general depressive symptoms, anxious arousal symptoms, and stressors, whereas boys reported more externalizing problems.

Overview of Statistical Approach

The analysis of multiple levels of data was accomplished in HLM 5.04 (Raudenbush, Bryk, Cheong, & Congdon, 2001) through the construction of Level 1 and 2 equations. At level 1, regression equations were constructed that model separately the variation in the repeated measures (e.g., depressive symptoms, stressors) as a function of time (i.e., the 4 waves of data). Each equation included 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 4 waves of data. At Level 2, equations were specified that model individual differences in the Level 1 parameters as a function of between-subjects variables (i.e., BT’s dysfunctional attitudes). The key cognitive vulnerability-stress interaction was tested by examining the cross-level interaction term representing the effect of dysfunctional attitudes, at Level 2, on the slope of within-youth variability in the strength of the relation between stressors and symptoms at Level 1. 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.

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. 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. Dysfunctional attitudes were entered at Level 2 to enable an examination of whether stressors, in interaction with dysfunctional attitudes, 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, Beevers, Andover, & Holleran, 2006, for similar analyses with an adult sample).

The equations used to test the hypotheses were as follows:

For Level 1 model for symptoms over 4 time points:

\[
\text{Symptoms}_{ij} = B_0 + B_1 \times (\text{Stress}) + B_2 \times (\text{Symptoms}_{t-1}) + R_{ij}
\]

For Level 2 models:

\[
B_0 = G_{00} + G_{01} \times (\text{DAS}) + U_{0i}
\]

\[
B_1 = G_{10} + G_{11} \times (\text{DAS}) + U_{1i}
\]

\[
B_2 = G_{20} + U_{2i}
\]
TABLE 2. Dysfunctional Attitudes X Stress Interactions Predicting Prospective Fluctuations of General Depressive
Anhedonic Depressive, Anxious Arousal, and Conduct Symptoms.

<table>
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<th>df</th>
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<td>.009</td>
<td>2.71</td>
<td>1,347**</td>
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<tr>
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<td>DAS x STRESS</td>
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<td>.007</td>
<td>1.30</td>
<td>1,347</td>
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<td><strong>Anxious Arousal Symptoms</strong></td>
<td></td>
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<tr>
<td>ANX-1</td>
<td>.08</td>
<td>.01</td>
<td>4.86</td>
<td>1,347***</td>
</tr>
<tr>
<td>DAS</td>
<td>.002</td>
<td>.03</td>
<td>.07</td>
<td>1,347</td>
</tr>
<tr>
<td>STRESS</td>
<td>.79</td>
<td>.29</td>
<td>2.66</td>
<td>1,347**</td>
</tr>
<tr>
<td>DAS x STRESS</td>
<td>.01</td>
<td>.01</td>
<td>1.08</td>
<td>1,347</td>
</tr>
<tr>
<td><strong>Externalizing Symptoms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SDQ-EXTERNALIZING-1</td>
<td>.07</td>
<td>.02</td>
<td>3.89</td>
<td>1,347***</td>
</tr>
<tr>
<td>DAS</td>
<td>.01</td>
<td>.03</td>
<td>.35</td>
<td>1,347*</td>
</tr>
<tr>
<td>STRESS</td>
<td>.79</td>
<td>.34</td>
<td>2.34</td>
<td>1,347**</td>
</tr>
<tr>
<td>DAS x STRESS</td>
<td>.01</td>
<td>.01</td>
<td>1.01</td>
<td>1,347</td>
</tr>
</tbody>
</table>

Note. N = 250. DAS = Children's Dysfunctional Attitudes Scale; CDI = Children's Depression Inventory; ANH-CDI = Anhedonic symptoms of CDI; ANX = Anxious Arousal Symptoms; SDQ = Strengths and Difficulties Questionnaire. *p < .05; **p < .01; ***p < .001.

Thus, this analytic strategy enables a strong test of BT's cognitive vulnerability-stress interaction because prospective elevations in symptoms can be examined beyond the effect of prior symptoms, given the strong continuity in symptom levels over time during adolescence (c.f., Tram & Cole, 2006; see Table 1 for strong continuity in symptoms).

The Cognitive Vulnerability-Stress Hypothesis

To test the central hypothesis that BT's dysfunctional attitudes would interact with stressors to predict prospective elevations in symptoms over time, HLM was used to examine BT's dysfunctional attitudes interacting with stressors to predict the different dependent variables (i.e., within-youth fluctuations in symptoms of general depression, anhedonic depression, anxious arousal, and externalizing behaviors over the 4 waves of data). The primary predictors of these symptom outcomes were: 1) within-person prior levels of symptoms to enable examination of prospective elevations of symptoms over time, (2) within-person stressor levels over the 4 waves of data, (3) BT's dysfunctional attitudes, and (4) the cross-level interaction between within-individual stressors and between-youth dysfunctional attitudes. Results of these HLM analyses
are presented in Table 2 for the various models predicting different symptoms. We first present the findings for depressive (both general and specific anhedonic) symptoms and then for co-occurring symptoms of anxious arousal and externalizing. It is worth noting that for all symptoms, except anhedonic depression, prior symptoms predicted prospective fluctuations in symptoms at the next wave over time.

The critical cognitive vulnerability x stress interactions (dysfunctional attitudes at Level 2 x within-youth stressors over time at Level 1) significantly predicted prospective fluctuations in anhedonic depressive symptoms specifically, but not general depressive symptoms (i.e., the whole CDI). The main effect of stressors, as a within-individual time-varying covariate, significantly predicted prospective changes for general depressive symptoms. In order to examine the form of the significant cross-level interaction between dysfunctional attitudes and stressors over time predicting anhedonic depressive symptom elevations over time, the findings from the model in Table 2 were used to calculate predicted scores in these symptoms for youth with high or low dysfunctional attitudes (plus or minus 1.5 SD on DAS) and those who experienced either low or high stressor levels over time (plus or minus 1.5 SD on the ALEQ across the 4 data waves). These results are shown in Figure 1. Greater dysfunctional attitudes combined with more stressors over time predicted the greatest prospective elevation in anhedonic depressive symptoms over time.

In contrast and consistent with the specificity hypothesis, the dysfunctional attitudes x stress interaction did not predict prospective changes in anxious arousal or externalizing symptoms. However, the main effect of stressors, as a within-individual time-varying covariate, significantly predicted prospective symptom changes over time for anxious arousal and externalizing symptoms. The main effect of BT’s dysfunctional attitudes did not predict changes in these symptoms over time.

In sum, consistent with the symptom specificity hypothesis, more dysfunctional attitudes interacted with higher stress levels to predict anhedonic depressive symptoms specifically but not general depressive, anxious arousal, or externalizing symptoms.
Greater levels of stressors over time were associated with prospective elevations in general depressive, anxious arousal, and externalizing symptoms over time.

**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, Knight, Kessler, & Merikangas, 2008; Hankin et al., 2008), 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. 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 x dysfunctional attitudes, age x stress, and age x dysfunctional attitudes x stress) were included along with the main and interactive effects of stress and dysfunctional attitudes as described above. The analyses examining the potential moderating effects of age on dysfunctional attitudes x stress interaction were all non-significant for predicting anhedonic, general depressive, anxious arousal, 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). Thus, the basic cognitive vulnerability-stress interaction predicting prospective fluctuations in anhedonic depressive symptoms over time appears to hold equally across sexes, ethnicities, and ages.

**Bi-Directional Effects Among Dysfunctional Attitudes, Stressors, and Symptoms?**

The final hypothesis examined whether changes in dysfunctional attitudes from Time 1 to Time 4 could be predicted by initial levels of stressors and/or symptoms given our hypothesis that a bi-directional, reciprocal process may occur over time among youth such that initial dysfunctional attitudes would interact with ongoing stressors to predict prospective changes in depressive symptoms and that later changes in more depressogenic dysfunctional attitudes might be predicted by initial symptom levels, especially depression and stressors. Hierarchical regression analyses were performed in which dysfunctional attitudes at Time 1 and symptoms or stressors at Time 1 were entered as predictors of residual change scores in dysfunctional attitudes from Time 1 to Time 4. As an initial examination, each predictor variable was entered one at a time to examine the independent prediction of residual change in dysfunctional attitudes after controlling for T1 DAS. Time 4DAS was predicted by T1 CDI (β = .37, t = 6.89, p < .001), anxious arousal (β = .21, t = 3.74, p < .001), conduct problems (β = .21, t = 4.00, p < .001), and stressors (β = .23, t = 4.45, p < .001). Table 3 shows the results when all baseline predictors were entered together in the same analysis to control for overlapping predictor variance. Consistent with hypothesis, prospective changes in dysfunctional attitudes were predicted predominantly by initial levels of general depressive symptoms.
TABLE 3. Hierarchical Regression Analysis Testing Prospective Changes in Dysfunctional Attitudes at Time 4 as a Function of Baseline Symptoms and Stressors

<table>
<thead>
<tr>
<th>Predictor</th>
<th>( \beta )</th>
<th>t</th>
<th>Change in ( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time 1 DAS</td>
<td>.24</td>
<td>4.5***</td>
<td></td>
</tr>
<tr>
<td>Step 2:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time 1 CDI</td>
<td>.33</td>
<td>5.00***</td>
<td></td>
</tr>
<tr>
<td>Time 1 ANX</td>
<td>.00</td>
<td>.06</td>
<td></td>
</tr>
<tr>
<td>Time 1 SDQ-Externalizing</td>
<td>.10</td>
<td>1.92</td>
<td></td>
</tr>
<tr>
<td>Time 1 Stressors</td>
<td>.09</td>
<td>1.56</td>
<td></td>
</tr>
</tbody>
</table>

Note. \( N = 345 \). DAS = Dysfunctional Attitudes Scale; CDI = Children’s Depression Inventory; ANX = Anxious Arousal Symptoms; SDQ = Strengths and Difficulties Questionnaire. *p < .05; **p < .01; ***p < .001.

Last, given well-known sex differences in depressive symptoms and stressors (e.g., Hankin, Mermelstein, & Roesch, 2007), we finally explored whether a sex x stressors interaction and/or a sex x depressive symptoms interaction predicted change in dysfunctional attitudes from Time 1 to Time 4. Similar hierarchical regressions were conducted one at a time in that the sex x initial stressor interaction was included in one model and the sex x initial depression interaction was included in the second model. Results showed that both sex moderation models significantly predicted change in T4 dysfunctional attitudes: sex x stress (\( \beta = .29, t = 1.98, p < .05 \)) and sex x initial depression (\( \beta = .31, t = 2.21, p < .001 \)). Follow-up analyses, in which regressions were conducted separately among boys only and then girls only, showed that initial depressive symptoms and stressors both predicted change in dysfunctional attitudes more strongly among girls compared with boys. Specifically, initial stressors predicted residual change in DAS for girls (\( \beta = .30, t = 4.52, p < .001 \)) more so than for boys (\( \beta = .17, t = 2.16, p < .05 \)), and a similar pattern was seen in that initial depressive symptoms predicted residual change in DAS more for girls (\( \beta = .49, t = 7.58, p < .001 \)) than boys (\( \beta = .37, t = 4.77, p < .001 \)).

DISCUSSION

It is important to be able to understand and predict prospective elevations in depressive symptoms during early and middle adolescence because the prevalence of depressive disorders increases (Hankin et al., 1998), symptoms stabilize (Tram & Cole, 2006), and adolescent-onset depression is more likely to recur in late adolescence through adulthood (Rutter, Kim-Cohen, & Maughan, 2006). Thus, studying etiological risk factors for depression and their affective specificity during adolescence can advance knowledge on depression onset, maintenance, and co-occurrence with other syndromes. BT was examined in this study as one possible etiological mechanism that has been supported in past research, primarily with adults (Abramson et al., 2002) and to some extent with youth (Abela & Hankin, 2008; Lakdawalla et al., 2007).

Results from the current study provided support for BT and showed that initial dysfunctional attitudes interacted with negative life events across the 4 time points to predict later fluctuations in only anhedonic depressive symptoms over time but not general depressive, anxious arousal, or externalizing symptoms. This interaction suggests that higher levels of dysfunctional attitudes, combined with more stressors over
time, predicted the greatest elevations in prospective anhedonic depressive symptoms specifically. That the dysfunctional attitudes x stress interaction only predicted anhedonic, but not general, depressive symptoms is intriguing, and, pending replication in future research, provides support for the validity of the tripartite model of anxiety and depression and other recent structural models of psychopathology (Clark, 2005; Lahey et al., 2004; Watson, 2005). Several studies have noted that the overall CDI, generally used by many researchers as a measure of depressive symptoms, may be a nonspecific measure of general distress and negative affect (Chorpita et al., 1998), rather than a specific assessment of depression, as it contains items assessing nonaffective features, including social and behavioral problems. Given the heterogeneous nature of depression, especially among youth, additional conceptual and psychometric work is needed to continue to clarify the fundamental nature of depression and ways to assess the core latent construct of depression. This basic and fundamental research on conceptualization and measurement of depression will likely yield stronger tests of the specificity of etiological depression theories, such as BT.

Of interest, the dysfunctional attitudes x stress interaction predicting anhedonic depressive symptoms was not moderated by sex, ethnicity, or age. With the well-known age and sex effects observed in depression and other psychopathological symptoms (Zahn-Waxler, Crick, Shirk, Shircliff, & Woods, 2006), the finding that dysfunctional attitudes interacted with stressors to predict anhedonic depressive symptoms equally for boys and girls (as well as early and middle adolescents and youth of different ethnicities) suggests that this cognitive vulnerability-stress component is not a sex-specific pathway contributing to depression (see Hankin et al., 2008, for a discussion of general depression models versus sex-specific etiological models). That boys reported more depressogenic dysfunctional attitudes than girls is consistent with past research with adults (Haefel et al., 2003). Although dysfunctional attitudes cannot explain why girls exhibit more depressive symptoms than boys, other research shows that other cognitive vulnerabilities, including a negative cognitive style (Abramson, Metalsky, & Alloy, 1989) in interaction with stressors and the main effect of rumination (Nolen-Hoeksema, 1991), partially explain the sex difference in depressive symptoms (Hankin, in press).

Another goal of this study was to examine whether a reciprocal relationship among dysfunctional attitudes, stressors, and symptoms might exist, such that changes in dysfunctional attitudes over time could be explained by initial levels of stressors or symptoms (i.e., depressive, anxious arousal, or externalizing). Results, in which initial levels of symptoms and stressors were entered as predictors of change in dysfunctional attitudes over time, provided initial evidence for a reciprocal, potentially transactional relationship between depressive symptoms and dysfunctional attitudes after controlling for other baseline factors. However, this bi-directional effect was moderated by sex in that initial depressive symptoms and stressors predicted prospective changes in dysfunctional attitudes over time more strongly for girls than boys. That this bi-directional finding was stronger for girls than boys is particularly interesting in light of the fact that boys reported more dysfunctional attitudes than girls and the well-known sex difference in depression. One possibility is that the sex difference found in this sample of early and middle adolescence for dysfunctional attitudes may change over time and development, as girls experience more stressors and depressive symptoms and, in turn, may exhibit more dysfunctional attitudes. However, this potentially transactional process hypothesis is counter to one study of young adults showing that men report more dysfunctional attitudes than women (Haefel et al., 2003). Thus, additional research
is needed to replicate this initial and suggestive bi-directional finding and moderation by sex. Finally, these results reinforce the need for researchers to always investigate sex differences in psychopathology research (Hankin et al., 2008) and social/emotional development (Rose & Rudolph, 2006), as some developmental pathways and results may apply more strongly to one sex than the other.

Incorporating reciprocal and potentially transactional processes into research on the development and maintenance of depressive symptoms may allow researchers to begin to explain the course of depression in adolescents. For example, it is well demonstrated that middle to late adolescence is a time when most youth become depressed for the first time (Hankin et al., 1998), and that after exposure to their first episode of depression, youth are substantially more likely to have a recurrence in adulthood (Rutter et al. 2006). Although the analyses from the present study were not able to test comprehensively the full range of potentially transactional processes that may occur among adolescents, these results provide preliminary support for a bi-directional relationship between depressive symptoms and cognitive vulnerability factors, such as dysfunctional attitudes, that may play an important role in explaining the course, recurrence, and maintenance of depressive symptoms. Future research is needed to replicate this pattern of findings and would require multi-wave data in which simultaneous examination of the bi-directional associations among all variables over time are examined (see Sameroff & Mackenzi, 2003, for discussion of the challenges to testing transactional processes). Pending such replication, additional research is needed to examine potential processes and mechanisms that may account for the likely reciprocal, transactional relationship found among symptoms, stressors, and dysfunctional attitudes over time (see Gibb & Alloy, 2006, for a similar set of reciprocal findings with a negative attributional style). For example, gene-environment interplay, such as evocative and active person-environment correlations (Rutter, Moffitt, & Caspi, 2006), and interpersonal processes, such as excessive reassurance seeking and impaired interpersonal relationships (Rudolph, Flynn, & Abaied, 2008) may be some potential mechanisms.

Some of the strengths of the present work include a prospective, multi-wave design that allowed for testing longitudinal patterns and processes. Second, this study is one of the first to examine dysfunctional attitudes as specific predictors of anhedonic depressive symptoms, as opposed to anxious arousal or externalizing symptoms. Little past research had tested for this predictive specificity of the dysfunctional attitudes-stress interaction among adolescents (c.f., Lewinsohn et al., 2001), and that work was limited by collapsing non-depressive disorders together into one category. Thus, we were able to advance knowledge regarding potentially common predictors of general child/adolescent psychopathology compared with anhedonic depression specifically. Third, this study took an initial step by testing potentially transactional and reciprocal relationships among dysfunctional attitudes, stressors, and symptoms and moderation of these influences by sex. Incorporating bi-directional influences into a developmental psychopathological perspective allows researchers to account for and test potentially dynamic interactions between a person and his/her environment. Finally, this study tested these aims on a relatively large, moderately ethnically diverse sample that enabled tests of potential moderation by age, sex, and ethnicity. The cognitive vulnerability-stress interaction predicted fluctuations in depressive symptoms equivalently across sexes, ethnicity, and ages, although the bi-directional prediction of later dysfunctional attitudes by initial depressive symptoms and stressors was stronger for girls than boys.
While this study makes important advances in this area of research, there are several limitations to the current work. First, while the hierarchical regression analyses testing that initial depressive symptoms predict prospective changes in dysfunctional attitudes provide preliminary support for a bi-directional, potentially transactional relationship between dysfunctional attitudes and depressive symptoms, these analyses were not able to test the full range of a transactional relationship, such as potentially cyclical or cascading effects. Future work is needed in which cognitive vulnerabilities, such as dysfunctional attitudes, are assessed and analyzed over multiple time points. Second, all of the data come from youth who self-reported symptom levels, stressor occurrence, and dysfunctional attitudes. 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, diagnostic interviews) and multiple informants (e.g., parents, peers) would be an important next step for future research in this area. A potentially specific concern and limitation is the assessment of dysfunctional attitudes using the DAS (Lewinsohn et al., 2001), as the relatively poor test-retest reliability and equivocal stability over time (see Hankin, 2008) suggests that additional psychometric research is needed to improve the assessment of dysfunctional attitudes among youth (e.g., see Abela & Skitch, 2007; Abela & Sullivan, 2003, for a potentially more reliable and valid self-report measure of dysfunctional attitudes in youth). Given the potential psychometric concerns of the DAS used in this study, replication of the findings is needed using alternative measurement strategies. Still, it is worth pointing out the predictive validity of the DAS as the vulnerability-stress interaction predicted prospective anhedonic depressive symptoms. Third, 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 would generalize to more severe levels of psychopathology. Use of structured diagnostic interviews in future research can address this issue. Finally, the specificity findings, in which the dysfunctional attitudes x stressors interaction predicted only anhedonic depressive symptoms, needs to be replicated in other research with youth in which general depressive and co-occurring anxiety and externalizing symptoms are assessed with other measures and other informants. Although the symptom measures used in this study have demonstrated reasonable reliability and validity in research with youth, it is possible that the lack of findings for general depression, anxiety, and externalizing problems are particular to the measures used in this study. Relatedly, the anhedonic depression measure was pulled from items on the CDI, rather than a measure specifically designed and validated for assessing anhedonia (e.g., Watson et al., 1995). This may be problematic as seen in the results in which anhedonic depressive symptoms at Time T-1 were not associated with symptoms at Time T in the HLH analyses, so there may be measurement concerns that should be pursued in future replications.

In the future, expanding on the present work will enable researchers to begin to understand the complex course of depression as well as the causes, consequences, and correlates of depression in adolescents and more clearly understand the relationships among cognitive vulnerabilities, stressors, and symptoms. With statistical and methodological advances, it will be important to harness these techniques and assess for more complex transactional relationships and examine the processes underlying these associations.
REFERENCES


Developmental Psychology, 30, 467-483.


