

WHY ARE ANXIETY AND DEPRESSIVE SYMPTOMS COMORBID IN YOUTH?  
A MULTI-WAVE, LONGITUDINAL EXAMINATION OF COMPETING  
ETIOLOGICAL MODELS

By

JOSEPH R. COHEN

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Jami F. Young, Ph.D.

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## ABSTRACT OF THE DISSERTATION

Why are Anxiety and Depressive symptoms Comorbid in Youth?

A Multi-Wave, Longitudinal Examination of Competing Etiological Models

By JOSEPH R. COHEN

Dissertation Director:

Jami F. Young, Ph.D.

Comorbidity, defined as the manifestation of multiple disorders within an individual, has become the rule, rather than the exception. In youth, the co-occurrence of depression and anxiety is not only common, but leads to a more severe course of mental illness and poorer treatment response. Thus, researchers have aimed to understand the development of this frequent, and deleterious, relation. A collection of research points to two developmental explanations for why depression and anxiety may relate: A *causal model*, in which anxiety predicts depression, and a *correlated liabilities model*, where shared vulnerability factors predict both anxiety and depression. While promising trends have been identified for both models, a consistent pattern for the development of comorbid symptoms has yet to emerge. The present study sought to clarify past research by introducing a *diathesis-anxiety* approach to understanding comorbidity in youth. Specifically, we predicted that specific cognitive vulnerabilities would interact with anxiety symptoms to predict prospective depressive symptoms. For this study, 678 3<sup>rd</sup> (n=208), 6<sup>th</sup> (n=245), and 9<sup>th</sup> (n=225) grade girls (n=380) and boys (n=298) completed

self-report measures at baseline assessing cognitive vulnerabilities (rumination, negative inferential styles, and self-criticism), stressors, depression, and anxiety. Every 3 months over the next 18 months, youth completed follow-up measures of depression, anxiety, and stressors. Findings supported a *diathesis-anxiety* approach for self-criticism ( $t(2494) = 3.36, p < .001$ ) and rumination ( $t(2505) = 2.40, p = .05$ ). On the other hand, partial support was found for a *correlated liabilities* model, as girls with a negative inferential style were more likely to experience both depressive ( $t(2518) = 2.66, p = 0.008$ ) and anxiety ( $t(1436) = 2.08, p = 0.03$ ) symptoms following a negative event. Finally, no support was found for a *causal* model, as anxiety symptoms did not uniquely predict depressive symptoms ( $p > .05$ ). These results clarify past results concerning comorbidity by suggesting that multiple pathways exist for comorbid depressive and anxiety states, and by introducing *diathesis-anxiety* models as an important and novel explanation for comorbid emotional distress in youth. Clinical implications and future developmental psychopathology research on this important topic are discussed.

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## Introduction

Comorbidity, defined as the manifestation of multiple disorders within the same individual (Seligman & Ollendick, 1998), has become the rule rather than the exception with regard to psychopathology (Angold, Costello, & Erkanli, 1999; Essau & Chang, 2009). The most common combination of disorders in youth is depression and anxiety (Angold et al., 1999; Merikangas et al., 2010), with up to 75% of depressed youth experiencing symptoms of anxiety (Essau & Chang, 2009). Experiencing both forms of emotional distress, as opposed to just one, comes with many complications, including a more severe course of mental illness (Lewinsohn, Rohde, & Seeley, 1995; Mineka, Watson, & Clark, 1998; Rohde, Lewinsohn, & Seeley, 1991; Starr & Davila, 2008) and poorer treatment response (Ollendick, Jarrett, Grillis-Tauechel, Hovey, & Wolff, 2008; Westen, Novotny, & Thompson-Brenner, 2004; Young, Mufson, & Davies, 2006). Because of the frequency and deleterious consequences of experiencing comorbid depression and anxiety, psychologists have aimed to understand why these two forms of psychopathology so commonly co-occur.

Despite increased attention over the past 25 years on the comorbidity between depression and anxiety (Angold & Costello, 1993; Merikangas et al., 2010; Seligman & Ollendick, 1998) there is still a great deal of uncertainty over why these two disorders are so highly comorbid, and what distinguishes youth who develop only one internalizing disorder as opposed to two. Systematic investigations into the relation between the two disorders in recent decades have largely been guided by two influential theories: the tripartite model of anxiety and depression (Clark & Watson, 1991) and the cognitive content-specificity hypothesis (Beck, 1976). Although originally developed and tested

within the context of adulthood, these two theories have been extended downward to youth. However, studies on both the tripartite model (Anderson & Hope, 2008; Laurent & Ettlerson, 2001) and the cognitive content-specificity hypothesis (Epkins, 1996; Garber, Weiss, & Shanley, 1993; Jolly, 1993 Schniering & Rapee, 2004) have produced underwhelming findings concerning comorbid symptoms in children and adolescents. For instance, studies on the tripartite model have found that low positive affect (PA) is not a specific predictor of depressive symptoms, as it also predicts symptoms of social phobia in youth as well (Anderson & Hope, 2008). Furthermore, high negative affect has been shown to relate specifically to Generalized Anxiety Disorder (GAD), but not to other symptoms or diagnoses of anxiety as originally postulated by the theory (Anderson & Hope, 2008). Meanwhile, among the four studies which have examined the cognitive content-specificity hypothesis in youth (Epkins, 1996; Garber, Weiss, & Shanley, 1993; Jolly, 1993; Schniering & Rapee, 2004), no one has been able to replicate the pattern of findings with regard to the content of thoughts distinguishing between depressive and anxiety symptoms in children and adolescents. Given the inconsistent support for these two traditional explanations for comorbidity, alternative theories need to be explored.

In reviewing how two disorders may relate, Neale and Kendler (1995) propose twelve different models which may explain high rates of comorbidity between distinct disorders. This system has been utilized to understand several patterns of comorbidity in psychopathology, including the relation between attention-deficit/hyperactivity disorder and reading disability (Willcutt, Pennington, Olson, Chhabildas, & Hulslander, 2005) and different patterns of comorbidity related to substance abuse (Kendler, Jacobsen, Prescott, & Neale, 2003). Overall, Neale and Kendler's (1995) approach to modeling comorbid

psychopathology has become a recommended methodology to understanding the possible ways two disorders may relate (Krueger & Markon, 2006). In regard to the present research, Neale and Kendler's (1995) model serves as a useful tool in organizing the existing research concerning comorbidity between depression and anxiety in youth, and contextualizing new findings. Utilizing this framework also allows one to test competing hypotheses concerning comorbidity to examine which model best explains the relation between symptoms of the two disorders.

Despite these advantages, few studies have explicitly and adequately tested these different models with regard to depression and anxiety. In order to adequately test the different models posited by Neale and Kendler (1995), data must be collected over time and include multiple follow-ups to understand the causal principles outlined in several of the models (Middeldorp, Cath, Van Dyck, & Boomsma, 2005). To date, 209 empirical articles have cited Neale and Kendler's (1995) article describing the different models of comorbidity. Of these 209 articles, only three studies tested the competing models within a longitudinal framework with regard to depression and anxiety. Cross-referencing these cites led to an additional prospective study which compared different models that map on to Neale and Kendler's (1995) taxonomy, but did not specifically cite the article (Avenvoli, Stolar, Li, Dierker, & Merikangas, 2001). Findings across these studies suggest support for two of Neale and Kendler's (1995) models: The *correlated liabilities model*, in which anxiety and depression are predicted by shared risk factors, and a *causal model* in which anxiety leads to depression. For instance, in a combined longitudinal and family study, Klein, Lewinsohn, Rohde, Seeley, and Shankman (2003) found that the best explanation for comorbidity between anxiety and depression was that both disorders are

caused by non-familial etiological factors. On the other hand, in a similarly designed study, Rice, van den Bree, and Thapar (2004) argued that comorbidity between the internalizing disorders was best explained by shared genetic (familial) vulnerabilities. Therefore, although there was support for the correlated liabilities model from both studies, there was disagreement over which risk factors predicted the manifestation of both forms of symptoms.

On the other hand, the other two studies which tested competing models of depression/anxiety comorbidity have found mixed support for both the correlated liabilities and casual models. Avenvoli and colleagues (2001) suggested that comorbidity between anxiety and depression was partially explained by shared non-familial factors *and* anxiety directly predicting depression. Matthew, Pettit, Lewinsohn, Seeley, and Roberts (2011) added support for Avenvoli and colleagues' (2001) findings by suggesting two distinct etiological models may exist depending on the temporal onset of the disorders. Specifically, when a diagnosis of anxiety preceded depression, the authors found support for a causal model with anxiety presenting a direct pathway to depression. Meanwhile, when a diagnosis of depression preceded an anxiety diagnosis, the authors found support for a correlated liabilities model where both diagnoses were predicted by a similar set of risk factors (e.g., social support, worry). Collectively, these four studies' findings support a collection of other studies that, while not simultaneously testing different models of comorbidity, suggested that comorbidity between depression and anxiety in youth may be explained by a shared set of risk/vulnerability factors and/or anxiety predicting depression (Andrade et al., 2003; Beesdo et al., 2007; Flannery-

Schroeder, 2006; Kendler, Gardner, Gatz, & Pedersen, 2006; Mineka et al., 1998; Seligman & Ollendick, 1998).

The present study sought to build upon this research by utilizing a multi-wave (6 assessments every 3 months), longitudinal (over the course of 18 months) study, which is an optimal design for testing theories related to developmental psychopathology (Willett, Singer, & Martin, 1998). The above studies which simultaneously tested Neale and Kendler's (1995) models were either family or epidemiological studies which are important for showing the course of clinical disorders across the lifespan, but the length of time between follow-ups may make it more difficult to detect subtle changes in symptom fluctuations, important life events, and potential moderators (Abela & Hankin, 2008). Relatedly, the present study utilized a dimensional approach to conceptualizing psychopathology, as opposed to the categorical (diagnostic) approach used in epidemiological/family studies. Utilizing a dimensional approach for depression and anxiety may allow a better understanding of the full spectrum of internalizing symptoms, including the development of sub-threshold symptoms of emotional distress which may be missed using the DSM or other classification systems (Krueger & Finger, 2001). In addition, the present study focused on late childhood and adolescence, which allowed theories related to comorbidity to be tested during a critical period with regard to the emergence of internalizing symptoms (Avenvoli, Knight, Kessler, & Merikangas, 2008; Morris & March, 2004).

Finally, the present study sought to clarify conflicting findings by testing a *diathesis-anxiety* model, which combines the correlated liabilities model and the causal model. This approach is meant to explicitly test whether anxiety symptoms interact with

specific vulnerabilities to predict depression in youth. To date, only two studies have utilized this approach to understand the co-occurrence of depression and anxiety symptoms in youth. In a short-term, multi-wave study, Hankin (2008) found that rumination interacted with anxious arousal symptoms to predict prospective depression symptoms in a sample of early and middle adolescents. Meanwhile, in a multi-wave longitudinal study, Feng, Shaw, and Silk (2008) found that maternal negative control interacted with anxiety symptoms to predict depression during childhood and through adolescence. These findings are congruent with Starr and Davila's recent studies with adults (2012a, 2012b), where the two researchers found that both rumination and a negative attributional style moderated the relation between daily anxious and depressed moods, and that feelings of rejection interacted with daily anxious mood to predict depressed mood in the following days. These preliminary findings suggest that a combination of the causal and correlated liabilities models may be able to resolve conflicting findings concerning the development of comorbid internalizing symptoms during youth. However, it is still unclear whether a diathesis-anxiety approach is a) consistent across different vulnerabilities b) similar for boys and girls, and youth of different ages, and c) whether this approach better explains the co-occurrence of depression and anxiety symptoms compared to the correlated liabilities or causal models. Answers to these questions can highlight important information concerning the development of emotional distress in youth.

The present study sought to test these competing models of comorbidity within a cognitive-vulnerability framework. Past research has shown that several, distinct cognitive vulnerabilities play an important role in predicting both depressive and anxiety

symptoms in youth. Cognitive vulnerabilities are some of the most often studied collection of risk factors for anxiety and depression and have received extensive support as reliable and valid predictors of emotional distress in youth (Abela & Hankin, 2008). In particular, studies have demonstrated that, starting in late childhood, individual differences begin to emerge for rumination (Lopez, Driscoll, & Kistner, 2009), inferential styles (Jacobs, Reinecke, Gollan, & Kane, 2008), and self-criticism (Fichman, Koestner, & Zuroff, 1994), and that these cognitive factors play an important role in predicting emotional distress at a young age. For instance, Rood, Roelofs, Bogels, and Alloy (2010) found an association between styles of rumination and depressive and anxiety symptoms in youth ranging between 10 and 18 years old, while Hankin (2008, 2009) found a prospective association between rumination and depressive and anxiety symptoms in a sample of 6<sup>th</sup> and 10<sup>th</sup> grade adolescents. Meanwhile, Sutton and colleagues (2011) found a correlation between negative inferential styles and depressive and anxiety symptoms in a sample of late adolescents ( $M = 16.9$ ), while Hankin (2009) found that a negative inferential style interacted with negative events to predict anxiety and depressive symptoms in a sample of 6<sup>th</sup> and 10<sup>th</sup> grade adolescents. Finally, cross-sectional and prospective relations between self-criticism and depressive/anxiety symptoms have been found across youth samples as well (Abela & Taylor, 2003; Shahar, Blatt, Zuroff, Kuperminc, & Leadbeater, 2004).

The goal of the present study was to examine these cognitive vulnerabilities in the context of three competing models of comorbidity to provide a more nuanced understanding of comorbid depression and anxiety in youth. The present study had three specific goals. First, we tested whether a correlated liabilities (e.g., a vulnerability-stress



approach; see Gibb & Coles, 2005 for further explanation), causal, or diathesis-anxiety model best explained the relation between anxiety and depression symptoms (see Figure 1 for representations of all three models). Second, we examined whether the explanation for why symptoms co-occur varies within the context of distinct cognitive vulnerabilities. Third, we investigated whether the patterns and developmental pathways concerning comorbidity differ across boys and girls and youth of different ages. While past research has noted comorbidity between depression and anxiety symptoms to be common in youth, limited research exists which tests how sex and age/grade may influence this relation (Essau & Chang, 2009). As important sex and age/grade differences have been found with regard to emotional distress broadly (Hankin & Abramson, 2001; Morris & March, 2004), it seems reasonable to expect that the relation between internalizing symptoms may be sensitive to these demographic factors.

## **Method**

### **Participants**

Data was collected from the *Genetic, Cognitive, and Interpersonal Vulnerabilities to Depression in Youth* project, funded through an R01 grant from NIMH. A multi-site study, data was collected at University of Denver (Denver, CO) and Rutgers University (New Brunswick, NJ). The sample consisted of 678 youth (362 from Denver University; 316 from Rutgers University) who had parental consent and gave verbal assent before baseline assessments. Participants were recruited through community postings and school outreach efforts (e.g., mailing families who had a child in the 3<sup>rd</sup>, 6<sup>th</sup>, or 9<sup>th</sup> grade, attending open houses). The sample was fairly balanced with regard to sex (380 females;

298 males) and consisted of 3<sup>rd</sup> graders (n=208), 6<sup>th</sup> graders (n=245), and 9<sup>th</sup> graders (n=225) at the baseline assessment. Youth and a parent completed evaluations every 3 months for 18-months. Only data collected from youth self-reports were utilized for this study. All aspects of data collection adhere to the APA's code of ethical conduct and were approved by the Institutional Review Board (IRB) at both universities.

## **Procedure**

Only youth who had signed parental consent and gave verbal assent participated in the study. Phase 1 of the study involved an initial laboratory assessment. A research assistant (RA) met with the youth to complete all of the self-report measures described below. For 3<sup>rd</sup> and 6<sup>th</sup> graders, the RAs read out loud the questions to the participants as they completed the forms. For the 9<sup>th</sup> graders, RAs monitored their completion of these questionnaires. Meanwhile, Phase 2 of the study involved a series of 6 telephone follow-up assessments. Assessments occurred every 3 months during the 18 months following the initial assessment. At each assessment, an RA verbally administered the Children's Depressive Inventory (CDI; Kovacs, 1981), the Multidimensional Anxiety Scale for Children (MASC; March, 1997), and the Adolescent Life Events Questionnaire-Revised (ALEQ-R; Hankin & Abramson, 2002). Participants were compensated \$60 at Phase 1, and \$15 for every completed follow-up during Phase 2.

## **Measures**

The *Adolescent Cognitive Style Questionnaire (ACSQ; Hankin & Abramson, 2002)*. The ACSQ is based on the hopelessness theory (Abramson, Metalsky, & Alloy, 1989). It assesses negative inferences about the causes of negative events, their

consequences, and their implications for the self. The questionnaire consists of 12 hypothetical scenarios that may occur in an adolescent's life. Examples of scenarios include: *You take an exam and get a poor grade* and *You want to go to a big party but nobody invites you*. Adolescents are asked to imagine each scenario and write a possible cause of the problem. They are also instructed to indicate to what extent the cause of the event is stable and global (attribution of CAUSES), the likelihood that future negative consequences of the event will occur (attribution of CONSEQUENCES), and the extent to which they believe that what happened shows that they failed as individuals (attribution of SELF). The response scale ranges from 1 to 7, with higher values indicating that the adolescent displays a hopelessness cognitive style. Past research has shown the ACSQ to have high reliability and validity (Hankin, 2008). The ACSQ had a high level of reliability in the present study ( $\alpha = 0.91$ ).

*Children's Response Style Questionnaire* (CRSQ; Abela, Rochon, & Vanderbilt, 2004). The CRSQ is modeled after Nolen-Hoeksema's Response Style Questionnaire (Nolen-Hoeksema & Morrow, 1991). The CRSQ consists of 25 items, each of which describes a particular response to symptoms of depression. The items are grouped into three subscales: Rumination (13 items), Distraction (7 items), and Problem Solving (5 items). Of primary interest to the present study is the Rumination subscale, which assesses one's tendency to focus on negative aspects of oneself. For each item, youth are asked to indicate how often they respond in this way when they are feeling sad, with higher scores indicating higher agreement with the statement. A sample item from this subscale is: *Think about how alone you feel*. Past research has shown the CRSQ to be a reliable and valid measure in youth samples (Abela, Aydin, & Auerbach, 2007; Abela &

Hankin, 2011). The CRSQ had a Cronbach's alpha level of 0.87, indicating a high level of reliability.

*Children's Depressive Experiences Questionnaire Revised, Self-Criticism Subscale* (CDEQR-SC; Abela, 2008). The CDEQR-SC is a 12-item self-report questionnaire used to assess levels of self-criticism in youth. Similar to the 10-item CDEQ-SC (Abela & Taylor, 2003) and 5-item CDEQ-SF-SC (Abela, Fishman, Cohen, & Young, 2012), the CDEQR-SC is an adaptation of the Depressive Experiences Questionnaire (Blatt, D'Afflitti, & Quinlan, 1976), which is a widely used tool to assess self-criticism in adults. The CDEQR-SC was chosen for the present study because the longer version of the inventory was believed to better capture the nature of self-criticism in youth across childhood and adolescence. For each item, the child must choose which of the following options best represents him or her: *not true for me*, *sort of true for me*, or *really true for me*. An example item from the measure is: *If I am not good at everything I do, I get mad at myself*. Item scores range from 0 to 2 and are summed to obtain total scores ranging between 0 to 24; higher scores represent higher levels of self-criticism. Past research has supported the internal consistency, test-retest reliability, and validity of various versions of the CDEQ (Abela, Fishman, et al., 2012; Abela & Taylor, 2003). The CDEQR-SC had adequate internal consistency in the present study ( $\alpha = 0.79$ ), which was more reliable than past versions of the CDEQ (Abela, Fishman, et al., 2012; Abela & Taylor, 2003).

*Adolescent Life Events Questionnaire-Revised* (ALEQ-R; Hankin & Abramson, 2002). The ALEQ-R, in its abbreviated form, consists of 57 potentially negative events that are considered fairly typical of adolescence. The measure assesses the occurrence of

stressors, which are drawn from various life areas including: academics, familial relationships, friendships, and romantic relationships. The participant indicates the frequency of each event during the previous month by selecting a response on a Likert scale from 1 (“never”) to 5 (“always”). The scores range from 57 to 285, with higher scores indicating more frequent stressful life events. Past research has found the ALEQ-R to be a reliable and valid assessment for assessing negative events in youth (Abela & Hankin, 2011; Hankin, 2008; Hankin, Stone, & Wright, 2010)

*Children's Depressive Inventory (CDI; Kovacs, 1981).* The CDI is a 27-item self-report questionnaire that measures the cognitive, affective, and behavioral symptoms of depression. For each item, children are asked which one of three statements best describes how they have been thinking and feeling in the past week. Items are scored from 0 to 2, with a higher score indicating greater symptom severity. For instance, youth are asked to circle which statement describes him or her best: *I am sad once in a while* (0), *I am sad many times* (1), *I am sad all the time* (2). Total CDI scores ranged from 0 to 51. The CDI is the most commonly used and well-studied measure for assessing youth depression (Myers & Winters, 2002). In the present study the coefficient alphas ranged between 0.84 and 0.89 across administrations indicating strong internal consistency.

*Multidimensional Anxiety Scale for Children (MASC; March, 1997).* The MASC is a 39-item measure that assesses the occurrence and intensity of anxiety symptoms. The participant must determine the degree to which each item is true of him or herself on a Likert scale from 0 (“never”) to 3 (“often”), with higher scores indicative of greater levels of anxiety symptoms. The measure may be divided into 4 subscales: (1) physical symptoms (e.g., *I get dizzy or faint feelings*; 12 items), (2) social anxiety (e.g., *I worry*

*about people laughing at me*; 9 items), (3) separation anxiety/panic (e.g., *I avoid going to places without my family*; 9 items), and (4) harm avoidance (e.g., *Bad weather, the dark, heights, animals, or bugs scare me*; 9 items). However, in the present study only the total score was utilized (range = 0 – 117). Past research has found the MASC to be a reliable and valid tool for measuring symptoms of youth anxiety (Alloy et al., 2012; Brozina & Abela, 2006). The present research indicated strong internal consistency with Cronbach's alphas ranging between 0.88 and 0.90 across administrations.

## **Results**

### **Preliminary Analyses**

Preliminary analyses suggested that attributional style, rumination, self-criticism, depressive symptoms and stressors all exhibited significant positive skew requiring these data to be transformed for purposes of normality. For attributional style, rumination, and self-criticism a square root transformation was used, while for stressors and depressive symptoms, a log transformation was necessary. For anxiety symptoms, no transformation was needed.

Means, standard deviations, and correlations of all baseline measures, prior to transformations, can be found in Table 1. Of note, the small to medium significant associations between internalizing symptoms and stressors (Grant & Compas, 1995; Muris, Merckelbach, Ollendick, King, & Bogie, 2002), and internalizing symptoms and cognitive vulnerabilities (Garnefski, Legerstee, Kraaij, Kommer, & Teerds, 2002; Lakdawalla, Hankin, & Mermelstein, 2007) are similar to past community research which investigated these constructs in youth. Meanwhile, means and standard deviations for the follow-up measures can be found in Table 2.

Prior to testing the study's hypotheses, confirmatory factor analyses (CFAs) were conducted to investigate whether self-report measures used in the present study were able to adequately distinguish between a) depressive and anxiety symptoms and b) forms of anxiety (e.g., separation anxiety, social anxiety, physical symptoms of anxiety, and harm avoidance). All CFAs were tested using AMOS 20 software. With regard to depressive and anxiety symptoms, an excellent fit (Hu & Bentler, 1999) was demonstrated for a two-factor model ( $\chi^2 = 0.779$ ,  $p = .377$ ), CFI = 1.00, RMSEA = .00 (90% CI: .00 to .14); AIC = 53.10), and there was little support for a one-factor solution ( $\chi^2 = 303.96$ ,  $p < .001$ ), CFI = 0.51, RMSEA = .68 (90% CI: .62 to .75); AIC = 424.48). Of note, a chi-square difference test further demonstrated the superiority of a two-factor, opposed to one-factor, solution ( $\Delta\chi^2 = 373.38$ ,  $p < .001$ ). On the other hand, little support was found for a four factor model which distinguished between the MASC subscales ( $\chi^2 = 249.514$ ,  $p < .001$ , CFI = 0.754, RMSEA = .163 (90% CI: .145 to .182); AIC = 325.38). Taken together, results of the CFAs indicated that we were able to adequately distinguish between depressive and anxiety symptoms, but not differentiate between forms of anxiety symptoms. Thus, consistent with past research (O'Neil & Kendall, 2012; Storch et al., 2011), the hypotheses were tested using the MASC total anxiety score.

Next, as missing data is common in multi-wave longitudinal data, it was examined if participants who missed follow-ups differed systematically from those who had better or perfect completion rates during the course of the study. For the present study, 63.8% of participants completed the Time 1 and all six follow-up assessments, with 19.4% of the sample missing 1 follow-up, 5.7% of the sample missing 2 follow-ups, and 11% of the sample missing 3 or more follow-ups. Consistent with other multi-wave,

longitudinal studies, there was a negative relation between the amount of follow-ups completed by participants and prospective depressive symptoms and follow-up stressors (see Twenge & Nolen-Hoeksema, 2002 for a discussion of this issue). In response, an approach described by Hedeker and Gibbons (1997) was used to see if the number of follow-ups completed by participants influenced any of the hypothesized relations in our study. Overall, no significance was found for follow-ups interacting with any hypothesized vulnerabilities to predict prospective internalizing symptoms ( $p > .05$ ). Thus, it was concluded that data were missing at random (MAR).

### **Data Analytic Approach**

Analyses were carried out using the SAS (version 9.2) MIXED procedure for maximum likelihood estimation. All cognitive vulnerabilities were entered into all models as time-invariant, between-subject, Level 2 variables. Meanwhile, stressors, anxiety symptoms, and depressive symptoms were entered as time-varying, within-subject, Level 1 variables. All Level 2 variables were group mean centered to increase the interpretability of various parameters in our models (Muller, Judd, & Yzerbyt, 2005), and all Level 1 predictors were centered at each participant's mean so that scores reflect upwards or downwards fluctuations in an individual's reported occurrence of stressors or symptoms as compared to his or her mean level. Finally, a time-lagged data-analysis approach was utilized when investigating all multi-level models as symptom outcomes at Time  $T-1$  were entered as a time-varying covariate when predicting symptom outcomes at Time  $T$ . This time lagged analysis tests prospective changes in symptoms from Time  $T-1$  to Time  $T$  across each of the successive waves of the multi-wave follow-up, to ensure that baseline vulnerabilities are predicting differences in symptoms above and beyond the



previous follow-up. In addition, anxiety symptoms were entered at time  $T-1$  and depressive symptoms were entered at time  $T$  for all *causal* and *diathesis-anxiety* models tested. Reverse models with depressive symptoms predicting anxiety symptoms were also tested within this time-lagged framework.

For all analyses, three additional fixed effects and three additional random effects were included in all of the statistical models. With regard to fixed effects, preliminary analyses revealed that girls experienced higher depressive symptoms over time compared to boys ( $t(4097) = 3.35, p = .001, r_{\text{effect size}} = .05$ ), and that older youth experienced elevated symptoms of depression compared to younger youth ( $t(4097) = 10.09, p < .001, r_{\text{effect size}} = .16$ ). Therefore, both sex and grade were entered as covariates in all analyses. In addition, because of the high rates of comorbidity between internalizing symptoms (Angold et al., 1999), it is important to account for anxiety symptoms when utilizing depressive symptoms as an outcome, and depressive symptoms when using anxiety symptoms as an outcome. At the same time, automatically controlling for comorbid symptoms may lead to misleading findings because the constructs are correlated (Miller & Chapman, 2001; Schwartz, Susser, Morabia, & Bromet, 2006). Therefore, models for depressive and anxiety symptoms were initially tested independently, and if a model was found to be significant, analyses were then conducted that also controlled for the comorbid symptoms. Findings in the present manuscript were only considered significant if the pattern of findings were similar under both conditions. As controlling for concurrent symptoms is considered a more stringent model, all findings reported below reflect estimates while accounting for concurrent symptoms. With regard to random effects, a random intercept and slope were tested in the models. Both the random slope

for stressors ( $p < .001$ ) and random intercept ( $p < .001$ ) were significant in all analyses for depressive and anxiety symptoms, so these random effects were retained in subsequent analyses. In addition, an appropriate covariance structure was selected for analyses by fitting the model with the structure which provided the ‘best’ fit based on Akaike information criterion (AIC and AICC) and Schwarz Bayesian criterion (BIC; see Littell, Pendergast, & Natarajan, 2000 for explanation of different covariance structures and selection rules). For all analyses, the heterogeneous autoregressive structure (ARH) was significant ( $p < .001$ ) and demonstrated the best fit to the data. Finally, effect sizes using the  $r$  statistic (see Rosnow, Rosenthal, & Rubin, 2000 for explanation of statistic; see Rice & Harris, 2005 for comparisons to other effect size statistics) were calculated for all results. We believed this allowed for a greater understanding of the clinical impact of our findings, and for comparisons between the different models tested.

### **Causal Model**

It was first examined whether anxiety symptoms (at Time  $T-1$ ) predicted depressive symptoms (at Time  $T$ ). Higher-ordered interactions were examined to see if this relation varied as a function of age and/or sex. No significance was found for a three-way interaction between Grade  $\times$  Sex  $\times$  Anxiety Symptoms ( $b = 0.000$ ;  $SE = 0.004$ ;  $t(2548) = 0.10$ ,  $p = 0.92$ ,  $r_{\text{effect size}} = .00$ ), nor two-way interactions between Sex  $\times$  Anxiety Symptoms ( $b = -0.014$ ;  $SE = 0.010$ ;  $t(2550) = -1.43$ ,  $p = 0.15$ ,  $r_{\text{effect size}} = .03$ ) and Grade  $\times$  Anxiety Symptoms ( $b = -0.003$ ;  $SE = 0.002$ ;  $t(2550) = -1.70$ ,  $p = 0.09$ ,  $r_{\text{effect size}} = .03$ ). There was also no significance found for anxiety symptoms directly predicting prospective depressive symptoms ( $b = 0.009$ ;  $SE = 0.005$ ;  $t(2551) = 1.82$ ,  $p = 0.07$ ,  $r_{\text{effect size}} = .04$ ).

The reverse relation was also tested, to see if depressive symptoms (at Time  $T-1$ ) predicted symptoms of anxiety (at Time  $T$ ). As before, it was tested whether any significant interactions emerged with depressive symptoms predicting anxiety symptoms. However, the three-way interaction Grade  $\times$  Sex  $\times$  Depressive Symptoms ( $b = -0.262$ ;  $SE = 0.152$ ;  $t(2548) = -1.73$ ,  $p = 0.08$ ,  $r_{\text{effect size}} = .03$ ), and two-way interactions between Sex  $\times$  Depressive Symptoms ( $b = 0.271$ ;  $SE = 0.364$ ;  $t(2550) = 0.74$ ,  $p = 0.46$ ,  $r_{\text{effect size}} = .01$ ) and Grade  $\times$  Depressive Symptoms ( $b = -0.021$ ;  $SE = 0.077$ ;  $t(2550) = -0.28$ ,  $p = 0.78$ ,  $r_{\text{effect size}} = .01$ ) were not significant. With regard to depressive symptoms directly predicting anxiety symptoms in the sample as a whole, inconsistent support emerged with depressive symptoms predicting anxiety symptoms when controlling for concurrent depressive symptoms ( $b = 0.790$ ;  $SE = 0.191$ ;  $t(2551) = 4.14$ ,  $p < .001$ ,  $r_{\text{effect size}} = .08$ ), but insignificant when omitting this covariate ( $b = 0.336$ ;  $SE = 0.190$ ;  $t(2580) = 1.77$ ,  $p = 0.09$ ,  $r_{\text{effect size}} = .03$ ). Thus, conclusive statements concerning a causal relation between depressive symptoms predicting anxiety symptoms could not be made.

### **Correlated Liabilities Models**

Next, it was tested whether specific cognitive vulnerabilities interacted with stressors to predict prospective depressive and anxiety symptoms. It was first tested whether gender and/or grade moderated any of the potential *correlated liabilities models*. However, no significant interactions emerged ( $p > .10$ ), suggesting that the vulnerability-stress models did not vary as a function of grade and/or sex. With regard to depressive symptoms, no support for rumination ( $b = 0.009$ ;  $SE = 0.005$ ;  $t(2495) = 1.61$ ,  $p = 0.10$ ,  $r_{\text{effect size}} = .03$ ) or self-criticism ( $b = 0.010$ ;  $SE = 0.005$ ;  $t(2484) = 1.84$ ,  $p = 0.07$ ,  $r_{\text{effect size}} = .04$ ) interacting with stressors to predict depressive symptoms was found. However,

support for a negative inferential style interacting with stressors to predict prospective increases in depressive symptoms ( $b = 0.014$ ;  $SE = 0.005$ ;  $t(2518) = 2.66$ ,  $p = 0.008$ ,  $r_{\text{effect size}} = .05$ ) was established.

To examine the form of this interaction, the predicted CDI scores for children possessing either a negative or non-negative inferential style (plus or minus 1.0 SD above/below the group mean) and who reported either low or high ALEQ scores in comparison to their own average ALEQ score (plus or minus 1.0 SD) were calculated. As both CDI and ALEQ are within-subject variables centered at each participant's mean, slopes are interpreted as the increase in a child's CDI score that would be expected given he or she scored one standard deviation higher on the ALEQ. The results of such calculations are presented in Figure 1 and, as can be seen, individuals with a negative inferential style who experience high levels of stressors were most at risk for experiencing elevated depressive symptoms over time.

In line with a *correlated liabilities model*, it was next examined whether a negative inferential style interacted with stressors to predict anxiety symptoms as well. As the other vulnerabilities failed to interact with stressors to predict depressive symptoms, only a negative inferential style could be considered for the *correlated liabilities model*. As with depressive symptoms, it was first tested if any potential moderating effects concerning sex and grade existed. Results suggested that a significant interaction existed with Sex (e.g., Sex  $\times$  Negative Inferential Style  $\times$  Stressors;  $b = 1.118$ ;  $SE = 0.420$ ;  $t(2516) = 2.65$ ,  $p = 0.008$ ,  $r_{\text{effect size}} = .05$ ). Once again, a simple slope post-hoc approach was utilized to better understand the nature of this interaction. Findings from these analyses demonstrated that the interaction between stressors and a negative

inferential style was significant for girls ( $b = 0.552$ ;  $SE = 0.266$ ;  $t(1436) = 2.08$ ,  $p = 0.03$ ,  $r_{\text{effect size}} = .05$ ), but not for boys ( $b = -0.581$ ;  $SE = 0.327$ ;  $t(1075) = -1.78$ ,  $p = 0.08$ ,  $r_{\text{effect size}} = .05$ ). Results from these analyses are displayed in Figure 2. As shown, girls who experienced elevated levels of stressors (e.g., plus 1 SD above his or her mean) and had a negative inferential style (1 SD above the group mean) experienced the highest level of anxiety symptoms.

### **Diathesis-Anxiety Models**

It was first tested whether gender and/or grade moderated any of the proposed diathesis-anxiety models. However, no significant four-way or three-way relations emerged ( $p > .10$ ). In addition, no support for a diathesis-anxiety model with regard to a negative inferential style ( $b = 0.008$ ;  $SE = 0.005$ ;  $t(2529) = 1.66$ ,  $p = .10$ ,  $r_{\text{effect size}} = .03$ ) was found. Next, it was tested whether self-criticism and rumination significantly interacted with anxiety symptoms to predict prospective depressive symptoms. Results suggested that both self-criticism ( $b = 0.016$ ;  $SE = 0.005$ ;  $t(2494) = 3.36$ ,  $p < .001$ ,  $r_{\text{effect size}} = .07$ ) and rumination ( $b = 0.012$ ;  $SE = 0.005$ ;  $t(2505) = 2.40$ ,  $p = .02$ ,  $r_{\text{effect size}} = .05$ ) interacted with anxiety symptoms to predict prospective depressive symptoms. Similar to the correlated-liabilities models, post-hoc simple slope analyses were conducted to understand the nature of these interactions. Findings from these analyses are depicted in Figure 3 and indicate that youth high in self-criticism (1 SD above the group mean) and rumination (1 SD above the group mean) who also experienced elevated levels of anxiety (1 SD above his or her mean) were projected to experience the highest level of prospective depressive symptoms. To provide a more stringent test, it was examined whether the diathesis-anxiety models remained significant even when including the

traditional vulnerability-stress interactions. The pattern of findings remained similar for both rumination ( $b = 0.124$ ;  $SE = 0.005$ ;  $t(2505) = 2.58$ ,  $p = .009$ ,  $r_{\text{effect size}} = .05$ ) and self-criticism ( $b = 0.016$ ;  $SE = 0.005$ ;  $t(2494) = 3.40$ ,  $p < .001$ ,  $r_{\text{effect size}} = .07$ ) interacting with anxiety symptoms to predict depressive symptoms. Finally, it was tested whether the reverse relation was also significant by specifically examining if these vulnerabilities interacted with depressive symptoms to predict prospective anxiety symptoms. Findings for both rumination ( $b = 0.241$ ;  $SE = 0.182$ ;  $t(2495) = 1.32$ ,  $p = 0.19$ ,  $r_{\text{effect size}} = .03$ ) and self-criticism ( $b = 0.106$ ;  $SE = 0.179$ ;  $t(2484) = 0.59$ ,  $p = 0.55$ ,  $r_{\text{effect size}} = .01$ ) in these models were insignificant, suggesting that a *diathesis-anxiety*, but not a *diathesis-depression* model, may be a valid explanation for comorbid depressive and anxiety symptoms.

## Discussion

The goal of the present study was to add new knowledge to the ongoing debate concerning the etiological origins of comorbid depressive and anxiety symptoms in youth. Findings from this research support other recent findings which found that multiple pathways may exist as to why individuals experience comorbid depressive and anxiety symptoms (Matthew et al., 2011). Specifically, this study found that individual differences in various cognitive vulnerabilities may lead to differing explanations for comorbid symptoms. While no support for anxiety directly predicting depressive symptoms was found, a correlated liabilities model was supported. Specifically, a negative attributional style interacted with stressors to predict prospective depressive and anxiety symptoms in girls. Of note, a negative attributional style interacted with stressors to uniquely predict depressive, but not anxiety, symptoms in boys. Meanwhile, for youth

who tend to ruminate or be self-critical, the diathesis-anxiety model seems to be the approach which best explains how elevated depressive and comorbid anxiety symptoms may develop together within an individual. Not only novel, these findings provide impactful insight into the development of emotional distress in youth, which may be translated into future clinical and research endeavors.

A traditional explanation for comorbid symptoms states that anxiety directly predicts the onset of depressive symptoms (Cole, Peeke, Martin, Truglio, & Seroczynski, 1998; Flannery-Schroeder, 2006). However, the present study is more consistent with recent research which suggested that this causal model may not be the best explanation for why comorbid depressive and anxiety symptoms exist. For instance, when Moffitt and colleagues (2008) investigated the relation between depressive and anxiety symptoms prospectively, they found a reciprocal relation between the forms of emotional distress, with depression predicting anxiety almost as often as depression predicting anxiety. The authors suggested that past findings supporting anxiety directly predict depression may be due to an overreliance on retrospective studies in which participants may underreport earlier episodes of depressive episodes. Similarly, Rice and colleagues (2004) also found little support for anxiety uniquely predicting the onset of depression in youth. Instead, they offered that the occurrence of anxiety before depression represents an earlier expression of psychopathology from a shared vulnerability, and that same vulnerability will predict depressive symptoms at a later date. Our research is partially congruent with findings by Rice and colleagues (2004) that other models of comorbidity may provide more valid explanations as to why depressive and anxiety symptoms co-occur. However,

unlike these studies, findings from the present study suggest that anxiety symptoms do play an important role in the development of depressive symptoms.

The emergence of diathesis-anxiety models is consistent with other past research in youth (Feng et al., 2008; Hankin, 2008) and adults (Starr & Davila, 2012a, 2012b) and helps introduce a new explanation for comorbid emotional distress in youth. The present study found that anxiety symptoms interacted with rumination and self-criticism to predict prospective depressive symptoms in children and adolescents. Importantly, support for the reverse model of depressive symptoms interacting with any cognitive vulnerabilities to predict prospective anxiety symptoms was not found. These findings suggest that there is something unique about anxiety which makes it a vulnerability for prospective depressive symptoms. There are several possible explanations which may illustrate how this process plays out. Recent research has shown that rumination and self-blame (a byproduct of self-criticism; Dunkley, Zuroff, & Blankenstein, 2003) are closely related to symptoms of anxiety in children (Legerstee, Garnefski, Jellesma, Verhulst, & Utens, 2010) and adolescents (Legerstee, Garnefski, Verhulst, & Utens, 2011). While these cognitive coping mechanisms are typically conceptualized as vulnerabilities for anxiety symptoms (Lewis, Byrd, & Ollendick, 2012), other research which found that anxiety symptoms predict maladaptive coping mechanisms, such as rumination (Starr & Davila, 2012a), suggests that the relation is more reciprocal in nature. In other words, as one's symptoms of anxiety increase, maladaptive coping mechanisms such as rumination and self-criticism increase, which in turn leads to more anxiety. While this reciprocal relation has not been explicitly tested with regard to anxiety in children and adolescents, past research with both self-criticism (Shahar et al., 2004) and rumination (Nolen-



Hoeksema, Stice, Wade, & Bohon, 2007) have demonstrated this cyclical pattern in other contexts. The present study suggests that an outcome of this interaction is not only elevated levels of anxiety and maladaptive coping, but also a distinct form of emotional distress in the form of depressive symptoms. Thus, while a direct causal relation between anxiety and depression has been inconsistently reported in the literature (Cole et al., 1998; Moffitt et al., 2008; Rice et al., 2004), the present study suggests that by examining how these symptoms interact with specific cognitive constructs, a clearer picture of the relation between anxiety and prospective depressive symptoms emerges.

An added strength of the present study was that it directly tested diathesis-anxiety interactions alongside traditional, diathesis-stress interactions. The present study was the first to provide a simultaneous comparison between the two types of interactions. While past research has shown that the interactions between rumination and stressors (Abela & Hankin, 2011; Abela, Hankin, Sheshko, Fishman, & Stolow, 2012) and self-criticism and stressors (Abela, Sakellaropoulo, & Taxel, 2007; Abela & Taylor, 2003) predict depressive symptoms in youth samples, findings from the present study offer an alternative explanation as to how depressive symptoms may emerge in children and adolescents high in rumination and/or self-criticism. Of note, the present study's findings are not necessarily contradictory with past vulnerability-stress research, as extensive research has shown that anxiety symptoms and elevated levels of stressors are highly correlated (Lewis et al., 2012). Instead, findings from the present research suggest that focusing on the interaction between these specific vulnerabilities and anxiety symptoms, as opposed to stressors, may provide an even stronger explanation as to how these vulnerabilities go on to predict depressive symptoms.

While the diathesis-anxiety approach provided insight as to why children and adolescents high in rumination and self-criticism may experience comorbid emotional distress, it did not explain the pattern of findings across the cognitive vulnerabilities. Instead, the present study found that the best explanation for comorbid depressive and anxiety symptoms for youth who have a negative attributional style was a correlated liabilities model, with stressors interacting with the cognitive vulnerability to predict both depressive and anxiety symptoms in girls. Of note, the correlated liabilities model only served as an explanation for girls with a negative inferential style, which is contrary to past research which found that a negative inferential style was predictive of both types of symptoms in girls and boys (Alloy et al., 2012; Brozina & Abela, 2006; Hankin, 2009). However, it is important to emphasize that the findings were similar for both groups even though a negative inferential style was significant for girls ( $p = .03$ ;  $r_{\text{effect size}} = .05$ ), but not boys ( $p = .08$ ;  $r_{\text{effect size}} = .05$ ). Therefore, a more accurate statement from our findings may be that a negative inferential style impacts both depressive and anxious symptoms in girls and boys, but is a slightly stronger predictor of emotional distress in girls, which is similar to a conclusion drawn by Hankin (2009).

Evidence for a correlated liabilities model concerning a negative inferential style is congruent with past vulnerability research which suggested that comorbidity between depression and anxiety is best explained through a shared pattern of risk factors (Beesdo et al., 2007; Rice et al., 2004). There may be a strong theoretical reason as to why a negative inferential style interacted with stressors, as opposed to anxiety symptoms, to predict prospective symptoms in the present study. According to The Hopelessness Theory (HT; Abramson et al., 1989), negative inferences are made following a negative

life event when the causes, consequences, and information about the self are interpreted in a biased manner. Therefore, according to HT, there is a need for external events to activate these negative inferences, which then go on to predict emotional distress. In other words, anxiety symptoms or other internal feedback may not be enough to “trigger” the negative inferential style.

While the present study was similar with other research which tested a correlated liabilities model, it was inconsistent with recent findings put forth by Starr and Davila (2012a, 2012b) in which a negative attributional style interacted with anxiety symptoms to predict depressive symptoms. There are several possibilities why findings concerning a negative attributional style were different between the studies. First, Starr and Davila’s studies were assessed cross-sectionally (2012a) or over the course of three weeks (2012b), while the present study took place over the course of 18 months. Thus, a diathesis-anxiety approach may describe the concurrent or short-term relation between a negative attributional style and emotional distress, but in the long-term a correlated liabilities model may provide the best explanation. Second, the Starr and Davila studies focused on an older, college-aged and adult population, while the present study’s participants were children and adolescents. Past research has shown that the structure and meaning of a negative attributional style may change over time, and that the relation between a negative attributional style (and cognitive vulnerabilities in general) and depressive symptoms may differ based on age (Cohen, Young, & Abela, 2011; Jacobs et al., 2008). Therefore, in adulthood, a diathesis-anxiety model may provide the best explanation for comorbid symptoms, while a correlated liabilities model may provide the best approach to understanding this pattern of symptoms in youth. Finally, differences

between the studies may be the result of different measures for negative attributional style. Starr and Davila's studies (2012a, 2012b) utilized a measure which specifically assessed inferences about one's own internal state, and the control he or she has over anxiety symptoms. On the other hand, the ACSQ assesses a more global negative inferential style across situations. As important differences have been found within types of inferential styles (e.g., Brozina & Abela, 2006; Cohen et al., 2011) and emotional distress, future studies may want to investigate whether the best explanation for comorbid symptoms varies as a function of the type of inferential style.

Within the context of the present study, evidence for a correlated liabilities model and diathesis-anxiety model supports Matthew and colleagues (2011) notion that different etiological roots may exist for comorbid depressive and anxiety conditions. While Matthew and colleagues (2011) suggested that the temporal order between depressive episodes and anxiety disorders may differentiate between causal and correlated liability models, the present research suggests that specific vulnerabilities may lead to different explanations as to why one may develop comorbid conditions. Considering different pathways for comorbid conditions is an important step in comorbidity research.

Traditional theories, such as the tripartite model (Anderson & Hope, 2008; Clark & Watson, 1991) and the cognitive content-specificity theory (Beck, 1976; Garber et al., 1993; Schniering & Rapee, 2004), attempted to be complete explanations as to why depressive and anxiety symptoms co-exist. While accommodations were made to these theories (e.g., Ollendick, Seligman, Goza, Byrd, & Singh, 2003 accounted for how different forms of anxiety symptoms may relate to depressive symptoms within the context of the tripartite theory), there has been little movement away from a "one size fits

all” approach to comorbidity. However, the present research, coupled with that of Matthew and colleagues (2011), suggests that the long-standing pattern of inconsistencies concerning the origins of comorbid conditions may result from inconsistent patterns as to how one develops comorbid conditions. Thus, by utilizing a framework which tests competing explanations of comorbidity (e.g., Neale & Kendler, 1995) across distinct psychological conditions (e.g., temporal ordering of symptoms, specific vulnerabilities) a more nuanced understanding of the etiological underpinnings of different comorbid conditions is possible.

While the large sample, multi-wave, longitudinal design, rigorous statistical analyses, and dimensional approach to psychopathology were all strengths of the study, there are some notable limitations which need to be addressed in future research. First, findings from the study are based solely on self-report measures. While reliable and valid measures were used for each construct, and theoretically these are constructs which can be measured through self-report techniques (Haefel & Howard, 2010), future studies should use a multi-method approach when investigating these questions. For instance, computer-based tasks have been shown to provide objective information concerning cognitive vulnerability (Beevers, 2005; Gibb, Beevers, & McGeary, 2013), and semi-structured interviews may provide better insight into the nature of stressors (Rudolph, Hammen, Burge, Lindberg, Herzberg, & Daley, 2000), depressive symptoms (Ingram & Siegle, 2002), and anxiety symptoms (Velting, Setzer, & Albano, 2004). Relatedly, future studies may want to include assessments of depressive and anxiety symptoms across multiple informants (e.g., parent, teacher, and self) to provide a more reliable and valid understanding of a given youth’s emotional distress (De Los Reyes & Kazdin, 2005).

Second, the present study utilized a community sample to make inferences on comorbid clinical conditions. Therefore, findings concerning the different models for comorbidity should be seen as exploratory until it has been tested whether these models are able to predict diagnostic status as assessed by a clinical interview. Third, the present study was unable to distinguish between forms of anxiety symptoms, and instead treated it as a unitary construct. Given the important differences in the relations between distinct forms of anxiety and depressive symptoms (Kaufman & Charney, 2000), and cognitive vulnerabilities (Ferreri, Lapp, & Peretti, 2011), future studies should use methods which can test whether the present study's findings are consistent across different types of anxiety. Finally, although we found statistical significance for our findings, the effect sizes were in the small range. While past research has noted that small effect sizes are expected when conducting non-experimental field research (McClelland & Judd, 1993), interpretations from these findings should be tempered until other studies have replicated these findings.

Findings from the present study not only provide important etiological findings concerning the development of emotional distress, but may also be translated into important clinical implications. For instance, support for a diathesis-anxiety model demonstrates that although anxiety symptoms do not directly predict depressive symptoms, they still play a role in the development of depressive symptoms. Therefore, findings from the present study support others who have advocated for targeting anxiety symptoms within the context of depression prevention and treatment programs (Flannery-Schroeder, 2006; Young et al., 2006). In addition, findings for distinct etiological pathways between rumination/self-criticism and a negative attributional style further

demonstrates the need for clinicians and intervention developers to understand the unique vulnerability profile of his or her patients. For instance, findings from the present study suggest that a program for adolescents with a negative inferential style should focus on the inferences and the stressors in that patient's life which may be activating the negative inferential style. This can be done through a variety of empirically validated approaches which challenge these inferences (e.g., see Garber, 2006 for examples), or through problem-solving stressful adolescent contexts such as problematic family or peer relationships (e.g., Mufson, Dorta, Moreau, & Weissman, 2004). On the other hand, clinicians treating patients high in rumination and self-criticism, may similarly target the problematic cognitive style (e.g., see Garber, 2006), but also use specific techniques to decrease symptoms of anxiety which are activating these specific cognitive styles (e.g., Conrad & Roth, 2007). Because findings from this paper are preliminary, these translated implications are speculative at this point. However, continued research on the etiology of comorbid conditions may lead to more successful treatment of an impaired youth population (Lewinsohn, Rohde, & Seeley, 1995; Mineka et al., 1998; Starr & Davila, 2008), which has proven difficult to treat (Ollendick et al., 2008; Young et al., 2006).

## References

- Abela, J. R. Z. (2008). *Children's Depressive Experiences Questionnaire-Revised*. Unpublished Measure, Rutgers University, New Brunswick, NJ.
- Abela, J. R. Z., Aydin, C. M., & Auerbach, R. P. (2007). Responses to depression in children: Reconceptualizing the relation among response styles. *Journal of Abnormal Child Psychology*, *35*, 913-927.
- Abela, J. R. Z., Fishman, M. B., Cohen, J. R., & Young, J. F. (2012). Personality predispositions to depression in children of affectively-ill parents: The buffering role of self-esteem. *Journal of Clinical Child & Adolescent Psychology*, *41*, 391-401.
- Abela, J. R. Z. & Hankin, B. L. (2008). Cognitive vulnerability to depression in children and adolescents. In J. R. Z. Abela & B. L. Hankin (Eds.) *Handbook of depression in children and adolescents* (pp. 35-78). New York, NY: The Guilford Press.
- Abela, J. R. Z. & Hankin, B. L. (2011). Rumination as a vulnerability factor to depression during the transition from early to middle adolescence: A multiwave longitudinal study. *Journal of Abnormal Psychology*, *120*, 259-271.
- Abela, J. R., Hankin, B. L., Sheshko, D. M., Fishman, M. B., & Stolow, D. (2012). Multi-wave prospective examination of the stress-reactivity extension of response styles theory of depression in high-risk children and early adolescents. *Journal of abnormal child psychology*, *40*, 277-287.
- Abela, J. R. Z., Rochon, A. & Vanderbilt, E. (2004). A test of the integration of the response styles and social support theories of depression in third and seventh grade children. *Journal of Social and Clinical Psychology*, *23*, 653-674.
- Abela, J.R.Z., Sakellaropoulo, M., & Taxel, E. (2007). Integrating two subtypes of depression: Psychodynamic theory and its relation to hopelessness depression in schoolchildren. *Journal of Early Adolescence*, *27*, 363-385.
- Abela, J.R.Z. & Taylor, G. (2003). Specific vulnerability to depressive mood reactions in schoolchildren: The moderating role of self-esteem. *Journal of Clinical Child and Adolescent Psychology*, *32*, 408-418.
- Abramson, L. Y., Metalsky, G. I. & Alloy, L. B. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological Review*, *96*, 358-372.
- Alloy, L. B., Black, S. K., Young, M. E., Goldstein, K. E., Shapero, B. G...Moore, L. C. (2012). Cognitive vulnerabilities and depression versus other psychopathology symptoms and diagnoses in early adolescence. *Journal of Clinical Child & Adolescent Psychology*, *41*, 539-560.
- Anderson, E. R. & Hope, D. A. (2008). A review of the tripartite model for understanding the link between anxiety and depression in youth. *Clinical Psychology Review*, *28*, 275-287.



- Andrade, L., Craveo-Anduaga, J. J., Berglund, P., Bijl, R. V., De Graaf, R., Vollebergh, W...Wittchen, H. (2003). The epidemiology of major depressive episodes: Results from the International Consortium of Psychiatric Epidemiology (ICPE) surveys. *International Journal of Methods in Psychiatric Research, 12*, 3-21.
- Angold, A. & Costello, E. J. (1993). Depressive comorbidity in children and adolescents: Empirical, theoretical, and methodological issues. *Annual Progress in Child Psychiatry and Child Development, 150*, 1779-1791.
- Angold, A., Costello, E. J., & Erkanli, A. (1999). Comorbidity. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 40*, 57-88.
- Avenevoli, S., Knight, E., Kessler, R. C. & Merikangas, K. R. (2008). Epidemiology of depression in children and adolescents. In J. R. Z. Abela and B. L. Hankin (Eds.), *Handbook of child and adolescent depression* (pp. 6-34). New York: The Guilford Press.
- Avenevoli, S., Stolar, M., Li, J., Dierker, L., & Merikangas, K. R. (2001). Comorbidity of depression in children and adolescents: Models and evidence from a prospective high-risk family study. *Society of Biological Psychiatry, 1071-1081*.
- Beck, A. T. (1976). *Cognitive therapy and the emotional disorders*. Oxford: International Universities Press.
- Beck, R. & Perkins, T. S. (2001). Cognitive content-specificity for anxiety and depression: A meta-analysis. *Cognitive Therapy and Research, 25*, 651-663.
- Beesdo, K., Bittner, A., Pine, D. S., Murray, B., Stein, M. D., Hofler, M... Wittchen, H. (2007). Incidence of social anxiety disorder and the consistent risk for secondary depression in the first three decades of life. *Archives of General Psychiatry, 64*, 903-912.
- Beevers, C. G. (2005). Cognitive vulnerability to depression: A dual process model. *Clinical Psychology Review, 25*, 975-1002.
- Blatt, S. J., D'Afflitti, J. P., & Quinlan, D. M. (1976). Experiences of depression in normal young adults. *Journal of Abnormal Psychology, 95*, 383-389.
- Brozina, K. & Abela, J. R. Z. (2006). Symptoms of depression and anxiety in children: Specificity of the hopelessness theory. *Journal of Clinical Child & Adolescent Psychology, 35*, 515-527.
- Clark, L. A. & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology, 100*, 316-336.
- Cohen, J.R., Young, J.F., & Abela, J.R.Z. (2011). Cognitive vulnerability to depression in children: An idiographic, longitudinal examination of inferential styles. *Cognitive Therapy and Research, 36*, 643-654.

- Cole, D. A., Peeke, L. G., Martin, J. M., Truglio, R., & Seroczynski, A. D. (1998). A longitudinal look at the relation between depression and anxiety in children and adolescents. *Journal of Consulting and Clinical Psychology, 66*, 451-460.
- Conrad, A., & Roth, W. T. (2007). Muscle relaxation therapy for anxiety disorders: It works but how? *Journal of Anxiety Disorders, 21*, 243-264.
- De Los Reyes, A., & Kazdin, A. E. (2005). Informant discrepancies in the assessment of childhood psychopathology: a critical review, theoretical framework, and recommendations for further study. *Psychological Bulletin, 131*, 483-509.
- Dunkley, D. M., Zuroff, D. C., & Blankstein, K. R. (2003). Self-critical perfectionism and daily affect: Dispositional and situational influences on stress and coping. *Journal of Personality and Social Psychology, 84*, 234-252.
- Epkins, C. C. (1996). Cognitive specificity and affective confounding in social anxiety and dysphoria in children: *Journal of Psychopathology and Behavioral Assessment, 18*, 83-101.
- Essau, C. A. & Chang, W. C. (2009). Epidemiology, comorbidity, and course of adolescent depression. In C. A. Essau (Ed.) *Treatment of adolescent depression: Theory and practice*. Oxford: Oxford University Press.
- Feng, X., Shaw, D. S., & Silk, J. S. (2008). Developmental trajectories of anxiety symptoms among boys across early and middle childhood. *Journal of Abnormal Psychology, 117*, 32-47.
- Ferreri, F., Lapp, L. K., & Peretti, C. S. (2011). Current research on cognitive aspects of anxiety disorders. *Current Opinion in Psychiatry, 24*, 49-54.
- Fichman, L., Koestner, R., & Zuroff, D. C. (1994). Dependency, self-criticism, and perceptions of inferiority at summer camp: I'm even worse than you think. *Journal of Youth and Adolescence, 25*, 113-126.
- Flannery-Schroeder, E. C. (2006). Reducing anxiety to prevent depression. *American Journal of Preventative Medicine, 31*, 136-142.
- Garber, J. (2006). Depression in children and adolescents: linking risk research and prevention. *American Journal of Preventive Medicine, 31*, 104-125.
- Garber, J., Weiss, B. & Shanley, N. (1993). Cognitions, depressive symptoms, and development in adolescents. *Journal of Abnormal Psychology, 102*, 47-57.
- Garnefski, N., Legerstee, J., Kraaij, V., van den Kommer, T., & Teerds, J. A. N. (2002). Cognitive coping strategies and symptoms of depression and anxiety: A comparison between adolescents and adults. *Journal of Adolescence, 25*, 603-611.
- Gibb, B. E., Beevers, C. G., & McGeary, J. E. (2013). Toward an integration of cognitive and genetic models of risk for depression. *Cognition & Emotion, 27*, 193-216.

- Gibb, B.E., & Coles, M. (2005). Cognitive vulnerability–stress models of psychopathology: A developmental perspective. In B.L. Hankin & J.R.Z. Abela (Eds.), *Development of psychopathology: A vulnerability–stress perspective* (pp. 104–135). Thousand Oaks, CA: Sage.
- Grant, K. E., & Compas, B. E. (1995). Stress and anxious-depressed symptoms among adolescents: Searching for mechanisms of risk. *Journal of Consulting and Clinical Psychology, 63*, 1015-1021.
- Haefffel, G. J. & Howard, G. S. (2010). Self-report: Psychology’s four-letter word. *American Journal of Psychology, 123*, 181-188.
- Hankin, B. L. (2008). Cognitive vulnerability-stress model of depression during adolescence: Investigating depressive symptom specificity in a multi-wave prospective study. *Journal of Abnormal Child Psychology, 36*, 999-1014.
- Hankin, B. L. (2009). Development of sex differences in depressive and co-occurring anxious symptoms during adolescence: Descriptive trajectories and potential explanations in a multiwave prospective study. *Journal of Clinical Child & Adolescent Psychology, 38*, 460-472.
- Hankin, B. L. & Abramson, L. Y. (2001). Development of gender differences in depression: An elaborated cognitive vulnerability-transactional stress theory. *Psychological Bulletin, 127*, 773-796.
- Hankin, B.L. & Abramson, L.Y. (2002). Measuring cognitive vulnerability to depression in adolescence: Reliability, validity, and gender differences. *Journal of Clinical Child and Adolescent Psychology, 31*, 491-504.
- Hankin, B. L., Stone, L., Wright, P. A. (2010). Corumination, interpersonal stress generation, and internalizing symptoms: Accumulating effects and transactional influences in a multiwave study of adolescents. *Development and Psychopathology, 22*, 217-235.
- Hedeker, D. & Gibbons, R. D. (1997). *Longitudinal Data Analysis*. Hoboken, NJ: John Wiley & Sons, Inc.
- Hu, L. & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal, 6*, 1-55.
- Ingram R.E., & Siegle G.J. (2002) Contemporary methodological issues in the study of depression: Not your father's Oldsmobile. In I.H. Gotlib & C.L. Hammen (Eds.) *Handbook of depression* (pp. 86-114). New York, NY: Guilford Press.
- Jacobs, R. H., Reinecke, M. A., Gollan, J. K., & Kane, P. (2008). Empirical evidence of cognitive vulnerability for depression among children and adolescents: A cognitive science and developmental perspective. *Clinical Psychology Review, 28*, 759-782.

- Jolly, J. B. (1993). A multi-method test of the cognitive-specificity hypothesis in young adolescents. *Journal of Anxiety Disorders, 7*, 223-233.
- Kaufman, J. & Charney, D. (2000). Comorbidity of mood and anxiety disorders. *Depression and Anxiety, 12*, 79-86.
- Kendler, K. S., Gardner, C. O., Gatz, M., & Pedersen, N. L. (2006). A Swedish national twin study of lifetime major depression. *The American Journal of Psychiatry, 163*, 109-114.
- Kendler, K. S., Jacobson, K. C., Prescott, C. A., & Neale, M. C. (2003). Specificity of genetic and environmental risk factors for use and abuse/dependence of cannabis, cocaine, hallucinogens, sedatives, stimulants, and opiates in male twins. *American Journal of Psychiatry, 160*, 687-695.
- Klein, D. N., Lewinsohn, P. M., Rohde, P., Seeley, J. R., & Shankman, S. A. (2003). Family study of co-morbidity between major depressive disorder and anxiety disorders. *Psychological Medicine, 33*, 703-714.
- Kovacs, M. (1981). Rating scales to assess depression in school-aged children. *Acta Paedopsychiatrica: International Journal of Child & Adolescent Psychiatry, 46*, 305-315.
- Krueger, R. F. & Finger, M. S. (2001). Using item response theory to understand comorbidity among anxiety and unipolar mood disorders. *Psychological Assessment, 13*, 140-151.
- Krueger, R. F. & Markon, K. E. (2006). Reinterpreting comorbidity: A model-based approach to understanding and classifying psychopathology. *Annual Review of Clinical Psychology, 2*, 111-133.
- Lakdawalla, Z., Hankin, B. L., & Mermelstein, R. (2007). Cognitive theories of depression in children and adolescents: A conceptual and quantitative review. *Clinical Child and Family Psychology Review, 10*, 1-24.
- Laurent, J. & Ettelson, R. (2000). An examination of the tripartite model of anxiety and depression and its application to youth. *Clinical Child and Family Psychology Review, 4*, 209-229.
- Legerstee, J. S., Garnefski, N., Jellesma, F. C., Verhulst, F. C., & Utens, E. M. W. J. (2010). Cognitive coping and childhood anxiety disorders. *European Child and Adolescent Psychiatry, 19*, 143-150.
- Legerstee, J. S., Garnefski, N., Verhulst, F. C., & Utens, E.M.W.J. (2011). Cognitive coping in anxiety-disordered adolescents. *Journal of Adolescence, 34*, 319-326.
- Lewinsohn, P.M., Rohde, P., & Seeley, J.R. (1995). Adolescent psychopathology: III. The clinical consequences of comorbidity. *Journal of the American Academy of Child and Adolescent Psychiatry, 34*, 510-519.

- Lewis, K. M., Byrd, D. A., & Ollendick, T. H. (2012). Anxiety symptoms in African-American and Caucasian youth: Relations to negative life events, social support, and coping. *Journal of Anxiety Disorders, 26*, 32-39.
- Littell, R. C., Pendergast, J., & Natarajan, R. (2000). Tutorial in biostatistics: Modeling covariance structure in the analysis of repeated measures data. *Statistics in Medicine, 19*, 1793-1819.
- Lopez, C. M., Driscoll, K. A., & Kistner, J. A. (2009). Sex differences and response styles: Subtypes of rumination and associations with depressive symptoms. *Journal of Clinical Child & Adolescent Psychology, 38*, 27-35.
- March, J. S. (1997). *Manual for the Multidimensional Anxiety Scale for Children (MASC)*. Toronto: Multi-Health Systems.
- Matthew, A.R., Petit, J.W., Lewinsohn, P.M., Seeley, J.R., & Roberts, R.E. (2011). Comorbidity between major depressive disorder and anxiety disorders: Shared etiology or direct causation? *Psychological Medicine, 41*, 2023-2034.
- McClelland, G. H., & Judd, C. M. (1993). Statistical difficulties of detecting interactions and moderator effects. *Psychological Bulletin, 114*, 376-390.
- Merikangas, K. R., He, J., Burstein, M., Swanson, S. A., Avenevoli, S., Cui, L...Swendson, J. (2010). Lifetime prevalence of mental disorders in U.S. adolescents: Results from the National Comorbidity Survey Replication-Adolescent Supplement. *Journal of the American Academy of Child and Adolescent Psychiatry, 49*, 980-989.
- Middeldorp, C. M., Cath, D. C., Van Dyck, R., & Boomsma, D. I. (2005). The comorbidity of anxiety and depression in the perspective of genetic epidemiology: A review of twin and family studies. *Psychological Medicine, 35*, 611-624.
- Miller, G. A., & Chapman, J. P. (2001). Misunderstanding analysis of covariance. *Journal of Abnormal Psychology, 110*, 40-48.
- Mineka, S., Watson, D., & Clark, L. A. (1998). Comorbidity of anxiety and unipolar mood disorders. *Annual Review of Psychology, 49*, 377-412.
- Moffitt, T. E., Harrington, H., Caspi, A., Kim-Cohen, J., Goldberg, D., Gregory, A. M., & Poutlon, R. (2008). Depression and generalized anxiety disorder: Cumulative and sequential comorbidity in a birth cohort followed prospectively to age 32 years. *Archives of General Psychiatry, 64*, 651-660.
- Morris, T. L. & March J. S. (2004). *Anxiety disorders in children and adolescents, 2<sup>nd</sup> Edition*. New York: Guilford Press.
- Mufson, L., Dorta, K. P., Moreau, D., & Weissman, M. W. (2004). *Interpersonal Psychotherapy for depressed adolescents, 2<sup>nd</sup> Edition*. New York: Guilford Press.

- Muller, D., Judd, C. M., & Yzerbyt, V. Y. (2005). When moderation is mediated and mediation is moderated. *Journal of Personality and Social Psychology*, *89*, 852-863.
- Muris, P., Merckelbach, H., Ollendick, T., King, N., & Bogie, N. (2002). Three traditional and three new childhood anxiety questionnaires: Their reliability and validity in a normal adolescent sample. *Behaviour Research and Therapy*, *40*, 753-772.
- Myers, K. & Winters, N. C. (2002). Ten-year review of rating scales. II: Scales for internalizing disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, *41*, 634-659.
- Neale, M. C. & Kendler, K. S. (1995). Models of comorbidity for multifactorial disorders. *American Journal of Human Genetics*, *57*, 935-953.
- Nolen-Hoeksema, S., & Morrow, J. (1991). A prospective study of depression and posttraumatic stress symptoms after a natural disaster: the 1989 Loma Prieta Earthquake. *Journal of Personality and Social Psychology*, *61*, 115-121.
- Nolen-Hoeksema, S., Stice, E., Wade, E., & Bohon, C. (2007). Reciprocal relations between rumination and bulimic, substance abuse, and depressive symptoms in female adolescents. *Journal of Abnormal Psychology*, *116*, 198-207.
- Ollendick, T.H., Jarrett, M.A., Grills-Taquechel, A.E., Hovey, L.D., & Wolff, J.C. (2008). Comorbidity as a predictor and moderator of treatment outcome in youth with anxiety, affective, attention, deficit/hyperactivity disorder, and oppositional/conduct disorders. *Clinical Psychology Review*, *28*, 1447-1471.
- Ollendick, T. H., Seligman, L. D., Goza, A. B., Byrd, D. A., & Singh, K. (2003). Anxiety and depression in children and adolescents: a factor-analytic examination of the tripartite model. *Journal of Child and Family Studies*, *12*, 157-170.
- O'Neil, K. A., & Kendall, P. C. (2012). Role of comorbid depression and co-occurring depressive symptoms in outcomes for anxiety-disordered youth treated with cognitive-behavioral therapy. *Child & Family Behavior Therapy*, *34*, 197-209.
- Rice, M. E. & Harris, G. T. (2005). Comparing effect sizes in follow-up studies: ROC Area, Cohen's d, and r. *Law and Human Behavior*, *29*, 615-620.
- Rice, F., van den Bree, M. B. M., & Thapar, A. (2004). A population-based study of anxiety as a precursor for depression in childhood and adolescence. *BMC Psychiatry*, *4*, doi: 10.1186/1471-244X/4/43.
- Rohde, P., Lewinsohn, P. M., & Seeley, J. R. (1991). Comorbidity of unipolar depression II: Comorbidity in adolescents and adults. *Journal of Abnormal Psychology*, *100*, 214-222.

- Rood, L., Roelofs, J., Bogels, S. M., Bogels, S. M., & Alloy, L. B. (2010). Dimensions of negative thinking and the relations with symptoms of depression and anxiety in children and adolescents. *Cognitive Therapy and Research, 34*, 333-342.
- Rosnow, R. L., Rosenthal, R., & Rubin, D. B. (2000). Contrasts and correlations in effect size estimation. *Psychological Science, 11*, 446-453.
- Rudolph, K. D., Hammen, C., Burge, D., Lindberg, N., Herzberg, D., & Daley, S. E. (2000). Toward an interpersonal life-stress model of depression: The developmental context of stress generation. *Development and Psychopathology, 12*, 215-234.
- Schniering, C. A. & Rapee, R. M. (2004). The relationship between automatic thoughts and negative emotions in children and adolescents: A test of the cognitive content-specificity hypothesis. *Journal of Abnormal Psychology, 113*, 464-470.
- Schwartz, S., Susser, E., Morabia, A., & Bromet, E. (2006). What is a cause. In S. S. E. Susser, A. Morabia, & E. J. Bromet (Eds.), *Psychiatric epidemiology: Searching for the causes of mental disorders* (pp. 33-42). New York, NY: Oxford University Press.
- Seligman, L. D. & Ollendick, T. H. (1998). Comorbidity of anxiety and depression in children and adolescents: An integrative review. *Clinical Child and Family Psychology Review, 1*, 125-144.
- Shahar, G., Blatt, S. J., Zuroff, D. C., Kuperminc, G., & Leadbeater, B. J. (2004). Reciprocal relations between depressive symptoms and self-criticism (but not dependency) among early adolescent girls (but not boys). *Cognitive Therapy and Research, 28*, 85-103.
- Starr, L. R. & Davila, J. (2008). Differentiating interpersonal correlates of depressive symptoms and social anxiety in adolescence: Implications for models of comorbidity. *Journal of Clinical Child & Adolescent Psychology, 37*, 337-349.
- Starr, L. R. & Davila, J. (2012a). Responding to anxiety with rumination and hopelessness: Mechanism of anxiety-depression symptoms co-occurrence? *Cognitive Therapy and Research, 36*, 321-337.
- Starr, L. R. & Davila, J. (2012b). Cognitive and interpersonal moderators of daily co-occurrence of anxious and depressed moods in generalized anxiety disorder. *Cognitive Therapy and Research, 36*, 655-669.
- Storch, E. A., Caporino, N. E., Morgan, J. R., Lewin, A. B., Rojas, A., Brauer, L... & Murphy, T. K. (2011). Preliminary investigation of web-camera delivered cognitive-behavioral therapy for youth with obsessive-compulsive disorder. *Psychiatry Research, 189*, 407-412.
- Sutton, J. M., Mineka, S., Zinbarg, R. E., Craske, M. G., Griffith, J. W., Rose, R. D...Mor, N. (2011). The relationship of personality and cognitive styles with self-

- reported symptoms of depression and anxiety. *Cognitive Therapy and Research*, 35, 381-393.
- Twenge, J. M., & Nolen-Hoeksema, S. (2002). Age, gender, race, socioeconomic status, and birth cohort difference on the children's depression inventory: A meta-analysis. *Journal of abnormal psychology*, 111, 578.
- Velting, O. N., Setzer, N. J., & Albano, A. M. (2004). Update on and advances in assessment and cognitive-behavioral treatment of anxiety disorders in children and adolescents. *Professional Psychology: Research and Practice*, 35, 42-54.
- Westen, D., Novotny, C. M., & Thompson-Brenner, H. (2004). The empirical status of empirically supported psychotherapies: Assumptions, findings, and reporting in controlled clinical trials. *Psychological Bulletin*, 130, 631-663.
- Willcutt, E. G., Pennington, B. F., Olson, R. K., Nomita, C., & Hulslander, J. (2005). Neuropsychological analyses of comorbidity between reading disability and attention deficit hyperactivity disorder: In search of the common deficit. *Developmental Neuropsychology*, 27, 35-78.
- Willett, J. B., Singer, J. D., & Martin, N. C. (1998). The design and analysis of longitudinal studies of development and psychopathology in context: Statistical models and methodological recommendations. *Development and Psychopathology*, 10, 395-426.
- Young, J. F., Mufson, L., & Davies, M. (2006). Impact of comorbid anxiety in an effectiveness study of interpersonal psychotherapy for depressed adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, 45, 904-912.



Table 1.

*Baseline Means, Standard Deviations, and Correlations*

Measures	Mean	SD	1	2	3	4	5
1. CRSQ	26.23	7.66					
2. ACSQ	2.91	0.90	.40**				
3. CDEQR-SC	19.27	4.29	.51**	.47**			
4. ALEQ	64.53	17.09	.38**	.35**	.38**		
5. CDI	7.02	5.84	.48**	.42**	.42**	.52**	
6. MASC	41.92	15.66	.36**	.31**	.34**	.28**	.28**

*Note:* CRSQ = Baseline scores on the Children's Response Style Questionnaire; ACSQ = Baseline scores on the Attributional Cognitive Style Questionnaire; SPCA-Acad. = Baseline scores on the Self-Perception for Children and Adolescents, Academic Subscale; SPCA-Soc. = Baseline scores on the Self-Perception for Children and Adolescents, Social Subscale; CDEQR-SC = Baselines scores on the Children's Depressive Experiences Questionnaire, Self-Criticism Subscale; ALEQ = Baseline scores on the Adolescent Life Events Questionnaires; CDI = Baseline scores on the Children's Depressive Inventory; MASC = Baseline scores on the Multidimensional Anxiety Scale for Children. \* =  $p < .05$ ; \*\* =  $p < .01$

Table 2.

*Means and Standard Deviations for all Follow-up Measures*

Measures	Mean	SD	N
<i>CDI</i>			
FU1	5.36	5.34	605
FU2	4.33	4.52	585
FU3	4.76	4.94	588
FU4	3.86	4.46	592
FU5	4.17	4.75	571
FU6	5.32	5.94	552
<i>MASC</i>			
FU1	42.69	15.27	603
FU2	42.17	14.50	583
FU3	41.59	15.53	593
FU4	40.94	14.56	590
FU5	41.26	15.18	568
FU6	34.75	15.58	556
<i>ALEQ</i>			
FU1	13.85	7.64	609
FU2	13.21	7.81	587
FU3	13.10	7.96	592
FU4	13.99	7.83	590
FU5	13.98	8.01	577
FU6	14.87	8.16	560

*Note:* CDI = Scores on the Children's Depressive Inventory; MASC = Scores on the Multidimensional Anxiety Scale for Children; ALEQ = Scores on the Adolescent Life Events Questionnaire; FU = Follow-up Assessment

Table 3.

*Diathesis-Anxiety Models for Self-Criticism and Rumination*

Model for Rumination				
	<i>B</i>	<i>SE</i>	<i>T</i>	<i>Df</i>
Grade	0.025	0.005	5.43***	621
Gender	-0.013	0.022	-0.61	621
Anxiety	0.049	0.005	9.79***	2505
Depress_Lag	-0.071	0.005	-14.29***	2505
Anxiety_Lag	0.010	0.005	1.90	2505
Rumination	0.137	0.011	12.45***	621
Rumination x Anxiety_Lag	0.012	0.005	2.40*	2505
Model for Self-Criticism				
Grade	0.020	0.005	4.04***	618
Gender	0.010	0.023	0.44	618
Anxiety	0.048	0.005	9.67***	2494
Depress_Lag	-0.071	0.005	-14.42***	2494
Anxiety_Lag	0.010	0.005	1.85	2494
Self-Criticism	0.102	0.012	8.86***	618
Self-Criticism x Anxiety_Lag	0.016	0.004	3.36***	2494

*Note:* Grade = Child's grade in school; Gender = Child's gender (0 = Boy; 1 = Girl); Anxiety = Scores on the Multidimensional Anxiety Scale for Children over time; Depress\_Lag = Scores on the Children's Depressive Inventory at Time ( $n-1$ ); Scores on the Multidimensional Anxiety Scale for Children over time at Time ( $n-1$ ); Rumination = Baseline scores on the Children's Response Style Questionnaire; Self-Criticism = Children's Depressive Experience Questionnaire, Self-Criticism subscale. \* =  $p < .05$ ; \*\*  $p < .01$ ; \*\*\*  $p < .001$ .

Figure 1.

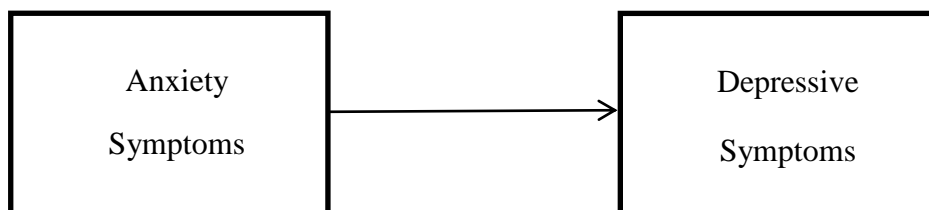
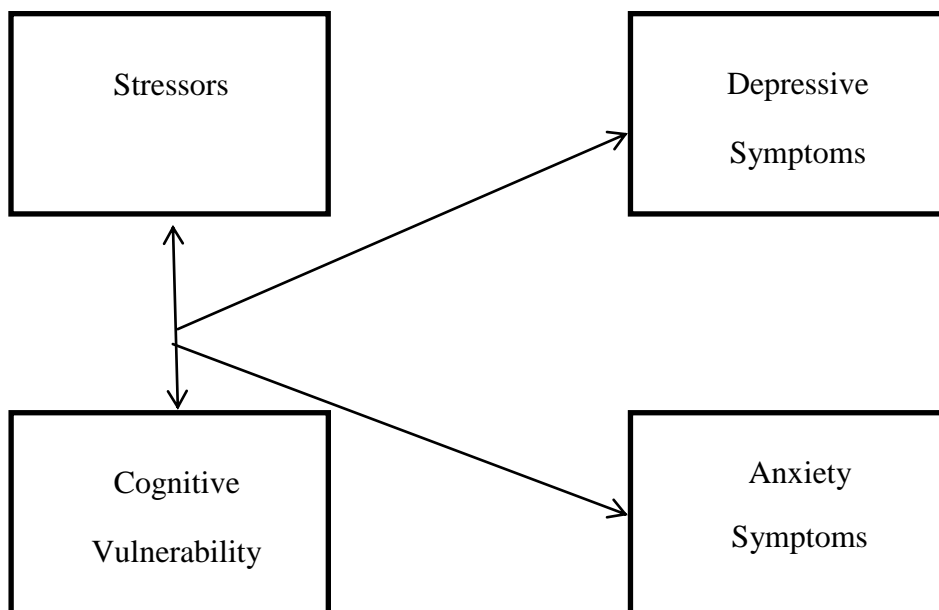
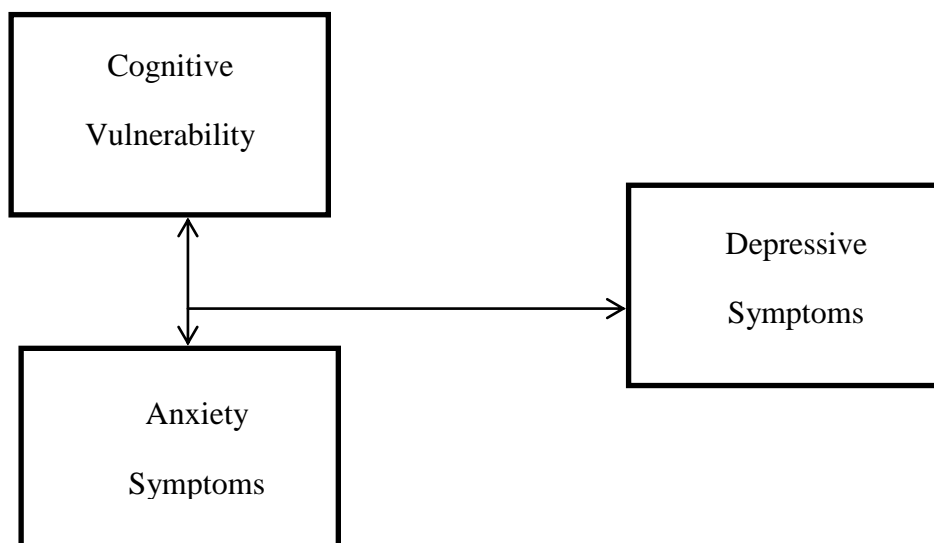
*The Three Competing Comorbidity Models for Depression and Anxiety Symptoms in Youth***Causal Model****Correlated Liabilities Model****Diathesis-Anxiety Model**

Figure 2.

*Predicted slope of the relation between negative events and depressive symptoms for children possessing high (pessimistic) and low (optimistic) negative inferential styles (Correlated Liabilities Model).*

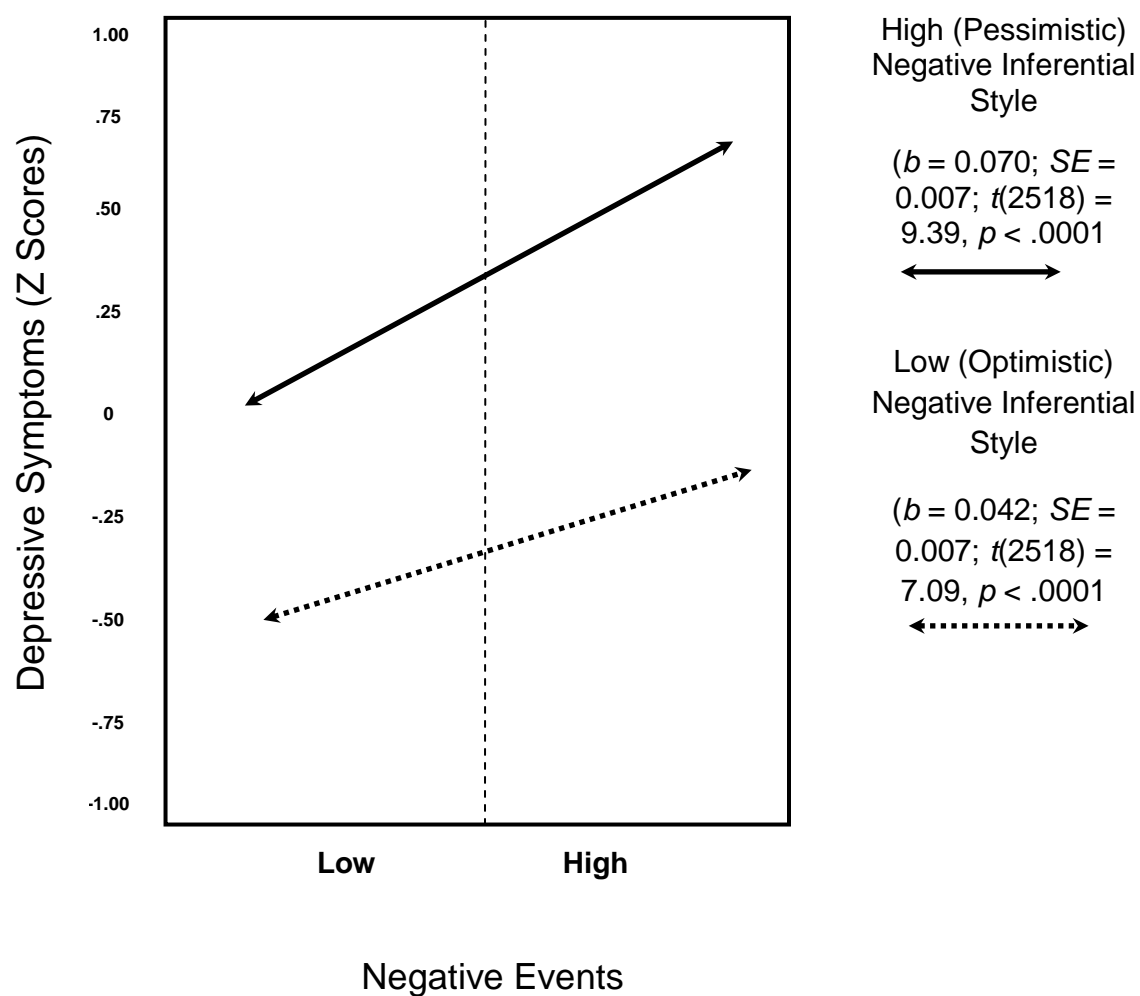


Figure 3.

*Predicted slope of the relation between negative events and anxiety symptoms for girls possessing high (pessimistic) and low (optimistic) negative inferential styles (Correlated Liabilities Model).*

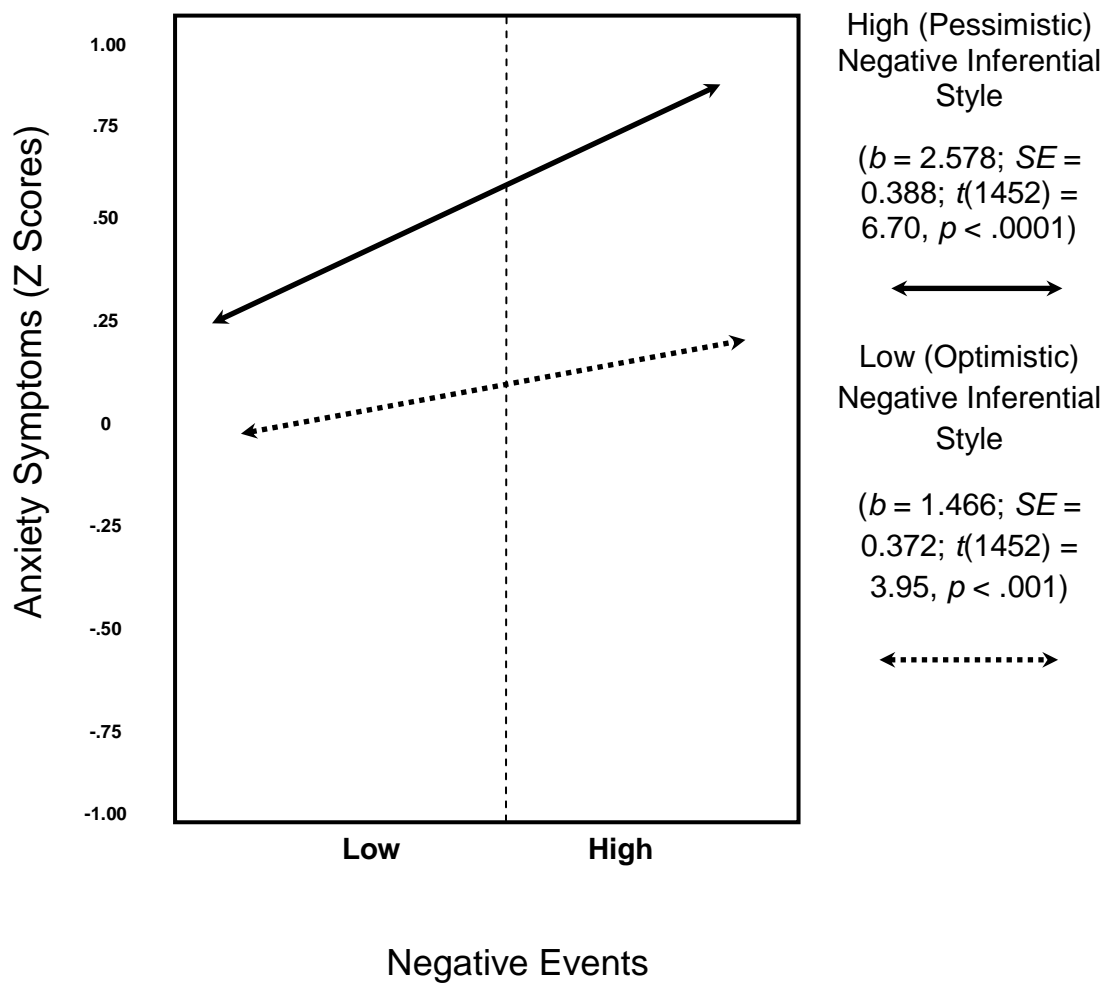


Figure 4.

*Predicted slope of the relation between anxiety symptoms (T-1) and depressive symptoms (T) possessing high or low cognitive vulnerabilities (Diathesis-Anxiety Models).*

