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Dynamic Temporal Relations between Anxious and Depressive Symptoms across Adolescence

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Abstract

Symptoms of anxiety and depression are prevalent among adolescents and associated with impairment in multiple domains of functioning. Moreover, anxiety and depression frequently co-occur, with estimated comorbidity rates as high as 75%. Whereas previous research has shown that anxiety symptoms predict increased depressive symptoms over time, the relation between depressive symptoms and later anxiety symptoms has been inconsistent. The present study examined dynamic relations between anxiety and depressive symptoms across adolescence, and explored whether these longitudinal relations were moderated by maternal history of anxiety, family relationship quality, or children's attributional style. Participants included 240 children (M age = 11.86 years; 53.9% female) and their mothers who were assessed annually for six years. Children reported on their depressive symptoms and mothers reported on their child's anxiety symptoms. Dynamic latent change score models indicated that anxiety symptoms predicted subsequent elevations in depressive symptoms over time. Depressive symptoms predicted subsequent elevations in anxiety symptoms among children who had mothers with a history of anxiety, reported low family relationship quality, or had high levels of negative attributions. Thus, whereas anxiety symptoms were a robust predictor of later depressive symptoms during adolescence, contextual and individual factors may be important to consider when examining relations between depressive symptoms and subsequent change in anxiety symptoms.

Keywords

adolescents; anxiety symptoms; depressive symptoms; latent change score models

Symptoms of anxiety and depression are prevalent in children and adolescents and are associated with significant impairment in multiple domains, including social and academic functioning, poor peer and family relationships, alcohol and drug use, suicidal behavior, and risk for adult psychopathology (Axelson & Birmaher, 2001; Birmaher et al., 1996; Costello, Foley, & Angold, 2006). Anxiety and depression frequently co-occur. Approximately 25–50% of children with depression have a comorbid anxiety disorder, and 10–15% of children with anxiety have comorbid depression (Axelson & Birmaher, 2001). Costello and colleagues (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003) estimated that comorbidity rates may be as high as 75%. Thus, epidemiological studies highlight that lifetime and

concurrent comorbidity between symptoms of anxiety and depression are more likely than not. Questions remain, however, regarding how the presence of one disorder is related to the longitudinal course of the other disorder. The present study examined whether symptoms of anxiety and depression predicted subsequent changes in each other across adolescence.

Anxiety typically predates depression in most comorbid cases (e.g., Axelson & Birmaher, 2001; Kovacs, Gatsonis, Paulauskas, & Richards, 1989), leading researchers to posit that anxiety may be a vulnerability factor for developing subsequent depression (Avenevoli, Stolar, Li, Dierker, & Merikangas, 2001; Flannery-Schroeder, 2006; Merikangas, 1990; Seligman & Ollendick, 1998). Specifically, the behavioral and cognitive manifestations of anxiety may develop into patterns of behavior that place children at risk for developing symptoms of depression (Garber, 2007). For example, anxious children may withdraw from others, resulting in greater social isolation, peer rejection, and a decreased sense of self-competence. Impaired social competence, in turn, predicts increased risk for depression (e.g., Cole, Martin, Powers, & Truglio, 1996).

Longitudinal studies also have shown that anxiety predicts *increased* levels of depressive symptoms over time (e.g., Cole, Peeke, Martin, Truglio, & Serocynski, 1998; Keenan, Feng, Hipwell, & Klostermann, 2009; Reinherz et al., 1989; Snyder et al., 2009). Cole et al. (1998) examined longitudinal relations between anxiety and depressive symptoms over a three-year period among children in third and sixth grade. Using an autoregressive cross-lag model, they found that anxiety predicted higher levels of subsequent depressive symptoms, based on both child and parent reports of children's symptoms. In a study using teachers' reports of children's symptoms, Snyder et al. (2009) also found that anxiety symptoms predicted higher levels of depressive symptoms over time. Keenan and colleagues (2009) examined longitudinal relations between depressive symptoms and both social anxiety and generalized anxiety disorders (GAD) in a sample of 6- to 12-year-old girls. They found that changes in depressive symptoms over time were best accounted for by previous levels of depression, but social anxiety and GAD also predicted higher levels of depressive symptoms in the following year.

Evidence of the reverse relation, that is, between depression and later anxiety, has been less consistent. Two large epidemiological studies, the Great Smokey Mountains Study (Costello et al., 2003) and the Dunedin Multidisciplinary Health and Development Study (Moffitt et al., 2007), reported that depression preceded anxiety just as often as anxiety preceded depression. Pine, Cohen, Burley, Brook, and Ma (1998) found that major depression in adolescence predicted an increased likelihood of developing GAD in adulthood even after adjusting for various demographic factors. Cole et al. (1998), however, failed to find a significant relation between child-reported depressive symptoms and subsequent anxiety symptoms. Other studies have found a negative relation between depression and later anxiety over time. Both parent- (Cole et al., 1998) and teacher-reports (Snyder et al., 2009) of children's depressive symptoms have been found to be related to *decreases* in anxiety symptoms over time. In sum, research indicates that whereas anxiety is a robust predictor of subsequent increases in depressive symptoms, the relation of depressive symptoms to later anxiety symptoms is less clear. The first goal of the current prospective study was to test dynamic, temporal relations between anxious and depressive symptoms in adolescents; that

is, how do symptoms of anxiety and depression simultaneously predict changes in each other over time?

Moderators of the Relation between Anxious and Depressive Symptoms

Some of the inconsistency in the literature regarding the longitudinal link between depressive and anxious symptoms may be because other variables that moderate these relations have not been examined. Both anxiety and depression in youth are associated with a variety of contextual and individual factors. In particular, parental psychopathology, the family environment, and cognitive style consistently have been implicated as risks that affect the onset and course of both anxious and depressive symptoms (e.g., Birmaher et al., 1996; Brozina & Abela, 2006; Rapee, 1997; Weissman, Leckman, Merikangas, Gammon, & Prusoff, 1984). Few studies, however, have examined whether such risk factors are related to the temporal associations between anxiety and depression. Moffitt and colleagues (2007) showed that adults with both major depression and generalized anxiety disorder were especially likely to have a family history of psychopathology, been exposed to early childhood adversity, and lower self-esteem. Our selection of moderators was guided not only by the prominence of these variables in the literature, but also because they represent individual- and family-level variables that are malleable and may be amenable to change through intervention. Another purpose of the present study was to examine whether particular correlates of anxious and depressive symptoms – maternal history of psychopathology, family relationship quality, and level of negative attributions -- moderated the strength of the coupling between anxious and depressive symptoms across adolescence.

Maternal history of anxiety and depression

Children of mothers with high levels of psychopathology are at an increased risk for internalizing problems (e.g., Biederman et al., 2006; Hammen, Burge, Burney, & Adrian, 1990; Kovacs et al., 1989; Mufson, Weissman, & Warner, 1992). In particular, offspring of parents with histories of anxiety disorders (e.g., Biederman, Rosenbaum, Bolduc, Faraone, & Hirschfeld, 1991; Hughes, Furr, Sood, Barmish, & Kendall, 2009; Merikangas, Dierker, & Szatmari, 1998), or depressive disorders (Beardslee, Versage, & Gladstone, 1998; Goodman & Gotlib, 1999) are at increased risk for both anxiety and depression. Weissman, and colleagues (1984) found that children whose parents had both depression and an anxiety disorder were more likely to have depression and anxiety themselves as compared to children whose parents had only depression. The current study used a high risk research design (e.g., Beardslee et al., 1998; Hammen, 1991) in which mothers with histories of depressive disorders were oversampled in order to obtain greater variability in the constructs of interest (i.e., children's anxious and depressive symptoms). In addition, we assessed mothers' histories of anxiety disorders, and hypothesized that the relation between anxious and depressive symptoms across time would be stronger for adolescents whose mothers also had a history of anxiety disorders.

Family relationship quality

The quality of the family environment consistently has been linked to children's internalizing symptoms (e.g., Eberhart & Hammen, 2006; Hughes, Hedtke, & Kendall,

2008; Kaslow, Deering, & Racusin, 1994; Lau & Kwok, 2000; Sheeber, Hops, & Davis, 2001). Specifically, high levels of family conflict and low levels of family cohesiveness and expressiveness are related to increased levels of depressive symptoms (e.g., Aydin & Oztütüncü, 2001; Cole & McPherson, 1993; Feldman, Rubinstein, & Rubin, 1988; Mason, Schmidt, Abraham, Walker, & Tercyak, 2009; Sheeber, Hops, Alpert, Davis, & Andrews, 1997), as well as to symptoms of anxiety in children (e.g., Hughes et al., 2008; Pagani, Japel, Vaillancourt, Cote, & Tremblay, 2008). For example, children's trajectories of anxiety symptoms characterized as high were more likely to come from families whose family dysfunction trajectories also were high (Pagani et al., 2008). Additionally, children with comorbid anxiety and depressive disorders have been found to report higher levels of family dysfunction compared to anxious children without comorbid depression (O'Neil, Podell, Benjamin, & Kendall, 2010). We hypothesized that a lower quality family environment (i.e., high levels of conflict and low levels of cohesion and expressiveness) would show a stronger positive longitudinal relation between anxious and depressive symptoms over time.

Negative attributional style

Children's causal attributions for positive and negative events have been linked with their levels of depressive symptoms (e.g., Abela & Hankin, 2008). According to the reformulated learned helplessness model (Abramson, Seligman, & Teasdale, 1978), individuals who attribute negative events to internal, stable, and global causes and positive events to external, unstable, and specific factors are likely to exhibit high levels of depressive symptoms when faced with stress. In a further refinement of the helplessness model, the hopelessness model of depression (Abramson, Metalsky, & Alloy, 1989) proposed that inferential styles about causes, consequences, and the self serve as vulnerabilities to depression in the context of negative events. Relevant to the present study, a pessimistic inferential style about causes was defined as the tendency to attribute negative events to stable and global causes and positive events to unstable and specific causes. Evidence of a significant relation between children's negative attributional style and depressive symptoms has been found repeatedly (e.g., Abela & Hankin, 2008; Zavos, Rijdsdijk, Gergory, & Eley, 2010).

The association between attributional style and anxiety symptoms has been less consistent (e.g., Brozina & Abela, 2006; Rodriguez & Pehi, 1998). For example, Rodriguez and Pehi (1998) found significant cross-sectional correlations between negative attributions and anxiety symptoms, which disappeared once concurrent depression was controlled. In a short-term prospective study, Brozina and Abela (2006) reported that children's inferential style about the causes of negative events interacted with daily hassles to predict increases in depressive but not anxious symptoms. Brozina and Abela, however, did not examine whether the attributional style by stress interaction predicted changes in symptoms of anxiety and depression simultaneously over time. Another goal of the current study was to test whether the strength of the dynamic, longitudinal relations between anxiety and depressive symptoms varied as a function of a pessimistic inferential style about the causes of positive and negative events.

Analyses of the Relations of Anxious and Depressive Symptoms over Time

An overall aim of this study was to examine dynamic, longitudinal relations between symptoms of anxiety and depression during adolescence. Several methodological approaches are available for examining longitudinal change, and each method assumes a different underlying model of developmental processes and change (Ferrer & McArdle, 2003). Each approach, therefore, can yield different conclusions regarding the longitudinal relations between symptoms of anxiety and depression. Typically, studies have used either latent growth curve modeling (parallel process models) or autoregressive cross-lag models; in contrast, the current study used a dynamic latent change score (LCS) modeling approach (McArdle & Hamagami, 2001).

Latent growth curve (LGC) models examine inter-individual differences in intra-individual change (Bollen & Curran, 2006). That is, when participants have been assessed three or more times, using maximum likelihood estimation LGC models estimate the average intercept (e.g., initial level) and average slope (rate of change) among participants. These two latent growth parameters provide information on the average shape of participants' trajectories over time. This model also allows for individual variability in the intercept and slope parameters. LGC models can be extended such that growth curves for two separate phenomena are fit, and correlations among the growth parameters (e.g., correlation among slopes) are estimated. This type of model, called a *parallel process* model, provides information on how change in one variable is correlated with change in another variable. The relation between both variables is not time-dependent, however, and therefore, information about dynamic relations among variables (i.e., time-lagged relations, or how one variable predicts change in the other variable over time) cannot be determined.

In contrast, *autoregressive cross-lag models* can estimate time-dependent relations. Specifically, these models estimate one's score on a variable at a given point in time as a function of their score at the previous time point (autoregressive effect), some proportion of their score on another variable at the previous time point (cross-lag effect), and measurement error. Autoregressive cross-lag models can provide information on interrelations between two variables over time. However, this model does not allow for growth in the variables or for individual differences in growth (Ferrer & McArdle, 2003).

The approach used in the current study, *dynamic latent change score (LCS) modeling*, integrates features of both latent growth curve and autoregressive cross-lag models. LCS models address specific questions about the temporal dynamics (i.e., time-dependent relations) between two variables, referred to as *longitudinal coupling*, while also estimating growth in the variables and allowing for individual differences in change. Coupling parameters can be estimated as time-invariant (i.e., cross-lag paths set equal across time) or time-varying, such that one can test whether the dynamics between two variables change over time. With regard to the longitudinal relations between symptoms of anxiety and depression, the LCS model can capture dynamic, transactional relations over time.

In summary, the current study examined dynamic, temporal relations between symptoms of anxiety and depression across adolescence (i.e., longitudinal coupling), and tested whether

dynamic relations between anxious and depressive symptoms varied as a function of maternal history of anxiety, family relationship quality, or negative attributional style. In addition, to facilitate comparisons with other studies in this literature, we compared the pattern of findings based on LCS models to the results obtained when analyzing these data using the change models common in previous research to demonstrate how methodological choices can affect the observed relations between anxious and depressive symptoms.

Method

Participants

Participants were 240 mothers and children (54% female) who were part of a longitudinal study of children at risk for psychopathology. Children were first assessed in sixth grade (M age = 11.86 years, $SD = 0.56$) and then annually through grade 12. The ethnic composition of the sample was 81.5% Caucasian, 14.8% African American, and the remaining 3.7% were Hispanic, Native American, or reported "Other." Families were predominantly working class (e.g., nurse's aide, salesclerk) to middle class (e.g., store manager, teacher) with a mean socioeconomic status (Hollingshead, 1975) of 41.67 ($SD = 13.29$).

Procedure

Parents of fifth grade children from metropolitan public schools were invited to participate in a study about parents and children. A brief health history questionnaire comprised of 24 medical conditions (e.g., diabetes, heart disease, depression) and 34 medications (e.g., fluoxetine, amitriptyline) and a letter describing the project were sent to over 3,500 families. Of the 1,495 mothers who indicated an interest in participating, the 587 who had endorsed either a history of depression, use of antidepressants, or no history of psychopathology were interviewed further by telephone. The remaining families were excluded because the mother either did not indicate depression or reported other serious health problems (e.g., cancer, multiple sclerosis). Of the 587 screened, 349 mothers reported either a history of depression or no history of psychiatric problems. The remaining 238 families were excluded because the mothers did not indicate sufficient symptoms to meet criteria for a depressive disorder (38%), had other psychiatric disorders that did not also include a depressive disorder (19%), were no longer interested (21%), they or the target child had a serious medical condition (14%), the target child was in the wrong grade (6%), or the family had moved out of the area (2%). The Structured Clinical Interview for *Diagnostic and Statistical Manual of Mental Disorders* diagnoses (SCID; Spitzer, Williams, Gibbon, & First, 1990) was then conducted with the 349 mothers who had a history of some depression or had had no psychiatric problems. Inter-rater reliability was calculated on a random subset of 25% of these interviews. There was 94% agreement ($\kappa = .88$) for diagnoses of depressive disorders. The final sample of 240 families consisted of 185 mothers who had a history of a mood disorder (high-risk group) and 55 mothers who were lifetime free of psychopathology (low-risk group).

Children were first assessed in sixth grade (Time 1) and then yearly through 12th grade (Times 2 through 7). A research assistant, unaware of the mother's psychiatric history, interviewed and administered a battery of questionnaires separately to the mother and

adolescent. The study procedures met the approval of the institutional review board for the protection of human subjects. Mothers provided informed consent, and children completed an assent form. Only those measures relevant to the current study are described below.

Measures

Depressive symptoms—Children reported annually on their own depressive symptoms with the Children's Depression Inventory (CDI; Kovacs, 1981). The CDI consists of 27-items that measure cognitive, affective, and behavioral symptoms of depression. Children chose one of three sentences that best described them during the past two weeks (e.g., "I am sad once in a while, I am sad many times, I am sad all the time"). Items were scored 0 to 2 and were summed to create a total score (range 0 to 54). The CDI has good internal consistency, test-retest reliability, and convergent validity with other self-report measures (Sitarenios & Kovacs, 1999). In the current sample, coefficient alphas ranged from .80 to .90 across time.

Anxiety symptoms—Mothers reported about their children's symptoms and behaviors on the Child Behavior Checklist (CBCL; Achenbach, 1991). Mothers rated how true a statement was about her child using a scale ranging from 0 (*not true*) to 2 (*very true or often true*). The anxiety symptoms composite (e.g., Snyder et al., 2009) was used as the measure of children's anxiety symptoms and was created by taking the mean of the following items: clings to adults or too dependent; feels he/she has to be perfect; nervous, high strung, or tense; too fearful or anxious; self-conscious or easily embarrassed; shy or timid; and worries (possible range for composite score was 0 to 2). Cronbach's alphas ranged from .58 to .75 in the current sample across the time points.

Moderators

Mothers' history of anxiety—Mothers' psychopathology at Time 1 was assessed with The Structured Clinical Interview for *Diagnostic and Statistical Manual of Mental Disorders* diagnoses (SCID; Spitzer et al., 1990). Mothers' anxiety history was coded as 1 (*any history*) if mothers reported current or lifetime panic disorder, social anxiety disorder, obsessive-compulsive disorder, or generalized anxiety disorder, and as 0 if no current or lifetime history of an anxiety disorder was reported.

Family relationship quality—Children completed the Family Environment Scale (FES; Moos & Moos, 1994), a self-report measure of the social environment of the family. Children rated 36 statements as true or false about their family (e.g., "We fight a lot in our family," "There is a feeling of togetherness in our family.") The subscales of Cohesion, Expressiveness, and Conflict assess the degree of commitment and support among family members, the degree to which family members are encouraged to express their feelings, and to openly express conflict, respectively. These three subscales comprised the Family Relationship Index (FRI), which was created by summing the cohesion and expressiveness subscales, and subtracting the conflict subscale. Children's FRI scores at grade 6 were used in the current study; higher scores reflected better family relationship quality. Cronbach's alpha of the FRI index was .78.

Negative attributional style—The *Children's Attributional Style Questionnaire* (CASQ; Seligman et al., 1984) measures attribution dimensions derived from the reformulated learned helplessness and hopeless models (Abramson et al., 1989; Abramson et al., 1978). The original CASQ contains 48 forced-choice items describing 24 positive and 24 negative events. In the current study, we used the revised CASQ (CASQ-R; Thompson, Kaslow, Weiss, & Nolen-Hoeksema, 1998) containing 12 positive and 12 negative items, and included the additional 12 negative items from the original CASQ. Children choose among two explanations for why an event occurred [e.g., “You get a bad grade in school,” with the two explanations being (a) “I am not a good student,” and (b) “Teachers give hard tests.”]. The explanations assess children's attributions with regard to internal, stable, and global causes of positive and negative events. The current study used the globality/stability index (Abramson et al., 1989), which was created by subtracting children's global and stable attributions for negative events from their global and stable attributions for positive events; lower scores reflected higher levels of negative attributions. Cronbach's alpha of the composite was .62 in the current sample. Scores at grade 6 were used in the current study.

Analysis Plan

To assess dynamic, longitudinal associations between anxiety and depressive symptoms, we fit a dynamic bivariate latent change score (LCS) model (McArdle & Hamagami, 2001), controlling for children's risk status (i.e., maternal history of depression). We also compared the bivariate LCS results to those from two traditional change models that typically have been used in previous research: (a) parallel process model, and (b) autoregressive cross-lag model.

LCS models are an extension of latent growth curve models and incorporate features of autoregressive cross-lag models. As a preliminary step, univariate LCS models were fit to examine latent change in anxious and depressive symptoms, respectively (e.g., Hawley, Ho, Zuroff, & Blatt, 2006; Kouros & Garber, 2010). Specifically, LCS analyses begin by partitioning observed scores into true scores (i.e., latent scores) and error variances. These latent scores predict the latent score at the next time point, and the residual represents latent change. The purpose of an LCS model is to predict these latent change scores across time. When latent change in one variable is modeled, there are several different change models, or *univariate LCS models*, that can be tested. Four possible univariate change models were considered, including (a) a *no change* model, in which previous scores perfectly predicted scores at the subsequent time point; (b) a *proportional change* model, in which change was proportional to one's score at the previous time point; (c) a *constant change* model, in which change occurred systematically as a function of time (e.g., linear growth curve model); and (d) a *dual change model* with time-varying proportional coefficients, in which latent change was predicted by some proportion of one's previous score plus systematic change that occurs over time.

Next, we tested a *bivariate LCS model*, in which anxious and depressive symptoms were modeled together and each variable simultaneously predicted latent change in the other variable. For example, latent change in depressive symptoms was modeled as a function of (a) systematic change that occurred over time (e.g., linear slope), (b) one's level of

depressive symptoms at the previous time point, referred to as a proportional effect, and (c) one's level of anxiety symptoms at the previous time point, referred to as a coupling effect. Latent change in anxiety symptoms was simultaneously modeled as a function of (a) systematic change that occurred over time, (b) one's levels of anxiety symptoms at the previous time point, and (c) one's level of depressive symptoms at the previous time point. Thus,

$$\begin{aligned}\Delta \text{ Depressive Symptoms} &= S_D + \beta_D (\text{previous depressive symptoms}) + \gamma_D (\text{previous anxiety symptoms}) \\ \Delta \text{ Anxiety Symptoms} &= S_A + \beta_A (\text{previous anxiety symptoms}) + \gamma_A (\text{previous depressive symptoms})\end{aligned}$$

Figure 1 presents a bivariate LCS model, where β_D and β_A represent the proportional effects for depressive symptoms and anxiety symptoms, respectively, and the parameters γ_{DA} and γ_{AD} represent longitudinal coupling between depressive symptoms and anxiety symptoms. If γ_{DA} is significant, this would indicate that one's level of depressive symptoms predicted subsequent change in anxiety symptoms. If γ_{AD} is significant, this would indicate that one's anxiety symptoms predicted subsequent change in depressive symptoms. When both γ_{DA} and γ_{AD} are significant, this indicates *bidirectional longitudinal coupling* between depressive symptoms and anxiety symptoms; if neither coupling parameter is significant, this would indicate that depressive symptoms and anxiety symptoms are not dynamically related over time.

Models were fit using AMOS statistical software, version 17.0 (Arbuckle, 2007). Full information maximum likelihood (FIML) estimation was used to handle missing data. The fit of a particular model was assessed using several fit indices, including the relative χ^2 index (χ^2/df), (Bollen, 1989), the comparative fit index (CFI; Bentler, 1990), and the root mean square error of approximation (RMSEA; Browne & Cudeck, 1993).

Results

Descriptive Statistics

Means, standard deviations, and bivariate correlations among the study variables are presented in Table 1. Across all time points, scores on the CDI ranged from 0 to 25–31. The percent of adolescents with a cutoff score at or above 13 after Time 1 were 5.6%, 6.6%, 9.6%, 9.3%, 7.4%, and 14.2%, respectively, which is consistent with what would be expected based on comparisons with the norms for this age range (Kovacs, 1992). Anxiety and depressive symptom scores were positively correlated at every grade (except grade 8), and positively correlated across time. There were no significant differences in boys' and girls' mean depressive or anxiety symptom scores at any time point.

Preliminary Analyses: Univariate LCS Models of Depressive and Anxious Symptoms

Univariate LCS analyses, controlling for risk, indicated that the dual change model with time-varying proportional effects best represented change in both depressive and anxious symptoms. Specifically, for depressive symptoms, the dual change model provided the best fit to the data [χ^2 (28, $N = 240$) = 50.91, $\chi^2/df = 1.82$, CFI = .95, RMSEA = .06, AIC =

82.91] compared to the no change [$\chi^2 (38, N = 240) = 137.98$, AIC = 149.98], proportional change, [$\chi^2 (37, N = 240) = 128.26$, AIC = 142.26], and constant change, [$\chi^2 (34, N = 240) = 76.28$, AIC = 96.28] models. Parameter estimates indicated that, on average, depressive symptom scores linearly declined over time [$b = -.75$, $SE = .38$, $p < .05$], and scores at grades 6 and 10 predicted subsequent declines in scores at the next time point [$\beta_{D1} = -.38$, $SE = .09$, $p < .01$; $\beta_{D5} = -.25$, $SE = .10$, $p < .01$]. Moreover, there was significant variability in both the intercept [$\sigma^2 = 12.44$, $p < .01$] and slope [$\sigma^2 = 1.33$, $p < .05$] parameters, indicating individual differences in change over time. The dual change model with time-varying proportional effects also best represented change in anxiety symptoms [$\chi^2 (28, N = 240) = 53.78$, $\chi^2/df = 1.92$, CFI = .96, RMSEA = .06, AIC = 85.78] as compared to the no change [$\chi^2 (38, N = 240) = 160.44$, AIC = 172.44], proportional change [$\chi^2 (37, N = 240) = 112.64$, AIC = 126.64], and constant change [$\chi^2 (34, N = 240) = 76.44$, AIC = 96.44] models. The average growth parameters in this model, however, were not significant. Accounting for time-varying proportional effects, there was significant individual variability in the intercept [$\sigma^2 = .06$, $p < .01$], but not the slope [$\sigma^2 = .001$, $p = .14$]. There were not significant differences between boys and girls in the intercept or slope growth parameters for anxious or depressive symptoms. Based on these preliminary univariate models, dual change score models of anxiety and depressive symptoms were used in subsequent analyses of the dynamic relation between these two constructs.

Dynamic Relations between Anxious and Depressive Symptoms: Bivariate LCS Model

A bivariate LCS model was fit to examine dynamic, longitudinal relations between anxious and depressive symptoms, controlling for risk. Results from the bivariate LCS model are presented in Table 2. This model provided a good fit to the sample data, [$\chi^2 (100, N = 240) = 161.06$, $\chi^2/df = 1.61$, CFI = .95, RMSEA = .05], and was more parsimonious compared to a model in which the dynamics between anxious and depressive symptoms (i.e., γ_{DA} and γ_{AD}) were allowed to vary over time [$\chi^2 (10, N = 240) = 13.12$].

Parameter estimates indicated longitudinal coupling between anxiety and depressive symptoms, such that symptoms of anxiety predicted subsequent elevations in depressive symptoms over time [$\gamma_{AD} = 2.92$, $SE = 1.49$, $p < .05$]. Expected trajectories for children with average levels of depressive symptoms at grade 6, based on all parameter estimates (slope, proportional, and coupling estimates) are depicted in Figure 2, Panel A. Notably, trajectories of depressive symptoms differed for children depending on their level of anxiety symptoms. Children with low levels of anxiety at grade 6 showed decreasing symptoms of depression through grade 12; whereas children with high levels of anxiety at grade 6 had an overall increase in depressive symptoms through grade 12. At the same time, depressive symptoms predicted subsequent elevations in anxiety symptoms at the trend level [$\gamma_{DA} = .01$, $SE = .01$, $p = .051$] (see Figure 2, Panel B).

Moderators of Coupling Effects

Next, multigroup bivariate LCS analyses were run to examine whether the dynamic relation between anxious and depressive symptoms differed by (a) mothers' history of anxiety, (b) quality of family relationships, and (c) children's global/stable attributional style. Multi-group analyses in SEM compare a series of increasingly restricted models to test whether a

parameter(s) is equivalent or varies between groups. Specifically, a model in which parameters are freely estimated for each group is compared to a model in which certain parameters are constrained to be equal between groups. A nonsignificant chi-square difference test would indicate that the constrained parameter(s) is equivalent across groups, whereas a significant chi-square difference test would indicate that the parameter(s) differs between groups. Once a parameter is found to be equivalent across groups, that parameter is constrained to be equal between groups as additional parameters are tested.

Mothers' history of anxiety—Multi-group bivariate LCS analyses examined whether longitudinal coupling between symptoms of depression and anxiety differed for children whose mothers had a history of any anxiety disorder compared to children whose mothers did not have a history of anxiety, controlling for risk (i.e., mothers' history of depression) (Table 3). The final model provided a good fit to the sample data [χ^2 (213, $N = 240$) = 316.41, $\chi^2/df = 1.49$, CFI = .91, RMSEA = .05]. The relation of anxiety symptoms to change in depressive symptoms was equivalent between groups; that is, anxiety symptoms predicted subsequent elevations in depressive symptoms among children regardless of their mothers' history of anxiety [$\gamma_{AD} = 3.25$, $SE = 1.36$, $p < .05$]. In contrast, a significant group difference was found for γ_{DA} , indicating that depressive symptoms predicted subsequent elevations in anxiety symptoms for children whose mothers had a history of anxiety [$\gamma_{DA} = .01$, $SE = .01$, $p < .01$], but not for children whose mothers did not have a history of anxiety (Figure 3). Thus, bidirectional, longitudinal coupling was found for children whose mothers had a history of an anxiety disorder.

Family relationship quality—Two groups were created based on a median split to examine whether the dynamic, longitudinal relation between depressive and anxious symptoms differed for children reporting low ($M = 13.86$; $SD = 4.02$) as compared to high ($M = 21.21$; $SD = 1.72$) levels of family relationship quality, controlling for risk (Table 3). The final model provided a good fit to the sample data [χ^2 (212, $N = 240$) = 298.35, $\chi^2/df = 1.41$, CFI = .92, RMSEA = .04]. The coupling parameter γ_{AD} did not differ between groups, indicating that anxiety symptoms predicted subsequent elevations in depressive symptoms for children reporting both low and high levels of family relationship quality [$\gamma_{AD} = 3.19$, $SE = 1.11$, $p < .01$]. Group differences were found in γ_{DA} , indicating that depressive symptoms predicted subsequent elevations in anxiety symptoms for children with low family relationship quality, $\gamma_{DA} = .04$, $SE = .01$, $p < .01$, but not for children with high family relationship quality. Thus, evidence of bidirectional, longitudinal coupling between anxiety and depressive symptoms was found for children with low family relationship quality.

Negative attributional style—Two groups were created based on a median split to examine whether the dynamic, longitudinal relations between depressive and anxious symptoms differed for children with low ($M = 1.42$; $SD = .28$) and high ($M = .44$; $SD = .40$) levels of negative attributions, controlling for risk (Table 4). The final model provided a good fit to the sample data, χ^2 (215, $N = 240$) = 313.96, $\chi^2/df = 1.46$, CFI = .91, RMSEA = .04. The coupling parameter representing the effect of anxiety symptoms on subsequent change in depressive symptoms did not differ between groups, indicating that anxiety

symptoms were related to subsequent elevations in depressive symptoms for children with both low and high levels of negative attributions, $\gamma_{AD} = 4.10$, $SE = 1.10$, $p < .01$. In contrast, group differences were found in γ_{DA} indicating that depressive symptoms predicted subsequent elevations in anxiety symptoms for children with high levels of negative attributions, $\gamma_{DA} = .01$, $SE = .01$, $p < .01$, but not for children with low levels of negative attributions. In sum, bidirectional longitudinal coupling between depressive and anxious symptoms was found for children with high levels of negative global/stable attributions.

Alternative Models of Changes

Finally, we compared the results from the bivariate LCS analysis to results based on a parallel process model and an autoregressive cross-lag model similar to what has been used by others in this literature (e.g., Cole et al., 1998; Snyder et al., 2009). These analyses allowed us to explore whether the different methods for modeling change yielded similar or different findings, which may account for some of the inconsistencies across studies.

Parallel process model—A parallel process model was fit to examine whether children's anxiety and depressive symptom slopes were correlated over time, controlling for risk. First, separate growth models were fit for anxious symptoms and depressive symptoms, respectively.

A linear model provided a more parsimonious fit to the data compared to a quadratic growth model for depressive symptoms, $\chi^2(3) = 7.38$, $p = .06$. Although the linear slope was not significant, there was significant individual variability in the slope parameter, $\sigma^2 = .54$, $\chi^2(230) = 448.05$, $p < .01$. For anxiety symptoms, a quadratic model provided a better fit compared to the linear growth model, $\chi^2(3) = 14.31$, $p < .01$. Anxiety symptoms decreased linearly over time, $b = -.03$, $SE = .01$, $p < .01$, although the quadratic slope was not significant. There was significant individual variability in children's linear, $\sigma^2 = .01$, $\chi^2(220) = 284.97$, $p < .01$, and quadratic, $\sigma^2 = .0001$, $\chi^2(220) = 276.57$, $p < .01$, growth.

Next, we fit a parallel process model to examine whether linear changes in depressive symptoms were correlated with linear and/or quadratic growth in anxiety symptoms. This model provided a good fit to the sample data, $\chi^2(97) = 155.88$, $\chi^2/df = 1.60$, CFI = .95, RMSEA = .05. Results indicated that although the intercepts for anxious and depressive symptoms were significantly correlated ($r = .23$, $p < .05$), the linear slopes were not significantly correlated ($r = -.03$, $p = .86$). Additionally, the linear slope for depressive symptoms was not correlated with the quadratic slope for anxious symptoms ($r = .15$, $p = .35$). Thus, results of the parallel process analysis indicated that changes in anxious and depressive symptoms were not related.

Autoregressive cross-lag model—An autoregressive cross-lag model was fit to the data (e.g., Keenan et al., 2009), controlling for risk. Although several cross-lag paths were significant, this model provided a poor fit to the sample data, $\chi^2(77, N = 240) = 355.72$, $\chi^2/df = 4.62$, CFI = .79, RMSEA = .12 (results available upon request). Therefore, support for longitudinal relations between anxious and depressive symptoms was not found using this analytic approach.

Discussion

The current longitudinal study examined the dynamic temporal associations between anxious and depressive symptoms in youth followed from early through middle adolescence. Consistent with previous research (e.g., Cole et al., 1998; Snyder et al., 2009), we found that symptoms of anxiety were a robust predictor of subsequent elevations in depressive symptoms over time in adolescents. This relation was not trivial; for example, a child with high levels of anxiety in grade 6 was predicted to increase 2.42 points on the measure of depressive symptoms (i.e., CDI), or roughly half a standard deviation, during the important 6-year developmental period of adolescence.

Several explanations have been proposed for why anxiety predicts later depression. In particular, negative sequelae of anxiety can confer increased risk of developing or exacerbating already present symptoms of depression (Flannery-Schroeder, 2006). For example, socially anxious children may try to avoid negative evaluations from others by withdrawing or disengaging. Such dysfunctional behaviors may actually increase the likelihood of the very rejection they are trying to avert, which then may result in feelings of sadness, loneliness, and low self-worth (e.g., Alden & Taylor, 2004; Gazelle & Ladd, 2003). Thus, the emotional, cognitive, and behavioral manifestations of anxiety may create the very conditions that are likely to contribute to elevated levels of depressive symptoms.

The present study found a robust relation between prior symptoms of anxiety and subsequent symptoms of depression, over and above prior depressive symptoms. Moreover, this relation was operative among children at both high and low risk with regard to several individual and contextual factors. Further exploration of the precise mechanisms intervening between anxiety and depression is needed to inform the construction of interventions aimed at preventing the emergence of depression in anxious children (Garber & Weersing, 2010).

In contrast to the often replicated finding that anxiety precedes depression, results regarding the reverse relation, that is, between prior depressive symptoms and subsequent anxiety, have been more complex (Cole et al., 1998; Costello et al., 2003; Moffitt et al., 2007). The current study showed that depressive symptoms were followed by elevations in anxious symptoms for a subset of youth who had mothers with a history of anxiety, reported low family relationship quality, or had a more negative attributional style. Notably, the correlations among these moderating variables were small to modest. Thus, these three factors may make relatively independent contributions to the longitudinal association between children's depressive symptoms and subsequent anxiety symptoms.

Children are likely to encounter multiple risk processes, however. For example, the cumulative risk of being exposed to both parental psychopathology and poor family relationship quality might further exacerbate the anxiety-depression relation (e.g., Forehand, Biggar, & Kotchick, 1998). Alternatively, these factors also might interact, conferring greater coupling between anxiety and depressive symptoms at high levels of both moderators. For example, Kouros, Merrilees, and Cummings (2008) found that the robust negative association between interparental conflict and children's emotional security about the family was further strengthened in the context of high paternal depressive symptoms.

Given the modest size of the current sample, however, we did not explore all possible interactions of the multiple risk factors on the longitudinal relation between anxiety and depressive symptoms. Further research is needed to examine how various risk factors work together, in either a cumulative or interactive way, to predict children's mental health, thereby providing an ecologically valid model of children's risk experiences.

One potential explanation for the depression to anxiety link is that in the context of adverse conditions such as poor family relationships or maternal anxiety, depressed adolescents may lack adequate resources and sufficient social support to help them deal with the stressors they encounter. Such depressed adolescents also may have acquired from their anxious mothers the use of less adaptive responses to stress such as rumination, excessive worry, and heightened physiological reactivity. These uncontrolled responses to stress (e.g., involuntary disengagement; Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001) may heighten and exacerbate their symptoms of anxiety, particularly if uncertainty about how to handle ongoing or new stressors persists. Additionally, adolescents with more pessimistic explanatory styles tend to attribute negative events and their associated depressive symptoms to global/stable causes that they expect will persist. Under these conditions, adolescents with depressive symptoms may become increasingly anxious realizing that they have neither sufficient support nor adequate skills to ameliorate their distress or the circumstances surrounding it.

Thus, contextual and individual factors may impact the strength and direction of the association between depressive and anxious symptoms. Moreover, the fact that depressive symptoms predicted changes in anxiety symptoms for only a subset of children whereas anxiety predicted later depression regardless of the moderating factors suggests that the processes by which depressive symptoms predict anxiety likely differ from those by which anxiety predicts later depression. Therefore, potential moderators as well as mediators of these relations should be examined separately in future prospective investigations.

This study made several notable contributions to our understanding of children's depressive and anxious symptom trajectories. Whereas previous research has focused on samples of children in middle childhood, this study examined dynamic relations between symptoms of anxiety and depression across adolescence, an important developmental period when such internalizing symptoms are increasing (e.g., Axelson & Birmaher, 2001; Hankin et al., 1998). Second, this study provides some clues for understanding the inconsistent findings regarding the relation of depressive symptoms to later anxiety that have been reported in the literature; that is, we showed that when accounting for certain individual and contextual risk factors, children with depressive symptoms *do* have subsequent elevations in symptoms of anxiety during adolescence.

Another strength of the current study was the use of a bivariate LCS model to examine longitudinal coupling between symptoms of anxiety and depression over time. The use of different change models yielded different results with regard to these longitudinal relations (Ferrer & McArdle, 2010). Similarly, Snyder et al. (2009) found a positive correlation between parents' and teachers' reports of children's anxious and depressive symptoms, yet when the autoregressive cross-lag paths were added to the model, a negative relation

between depression and later anxiety was found. The LCS models better captured the dynamic relations between symptoms of anxiety and depression that would have been missed by using the other change models. In the current study, the dynamic relation between symptoms of anxiety and depression would not have been detected if we had only relied on a parallel process or autoregressive cross-lag model.

Ultimately, the choice of which change model to use depends on one's theory about how symptoms are expected to be related over time. For example, the parallel process model would assume that the rates of change in anxious and depressive symptoms are related prospectively, but would not account for any time-dependent relations between symptoms over time. The autoregressive cross-lag model would account for time-dependent relations over time, but not for systematic growth or individual differences in change in anxious and depressive symptoms across time. The LCS model used here more accurately represented the theoretical perspective on how symptoms of anxiety and depression are longitudinally coupled over time.

Limitations of this study provide directions for future research. First, the extent to which the results of this study generalize to children with clinical diagnoses of anxiety or depressive disorders is not known. Replication of the current study with a clinical sample is needed to determine whether the relations observed in the present high-risk, community sample would be the same in youth with more severe forms of these disorders. Relatedly, the measures of anxiety and depression in the current study were questionnaires rather than clinical interviews. Nevertheless, the use of different informants about adolescents' depressive and anxious symptoms reduced the possibility that the correlations between these constructs were primarily due to shared method. Similarly, the measure of anxiety symptoms did not include overlapping items with depressive symptoms, which also reduced the likelihood that the correlations between these variables were spurious. Third, the measure of anxiety assessed an array of symptoms that cut across a range of anxiety disorders. The relations between depressive symptoms and each type of anxiety disorder may differ, however. For example, whereas many anxiety disorders typically occur prior to depression, panic disorder and OCD are less likely to precede depression (Lewinsohn, Zinbarg, Seeley, Lewinsohn, & Sack, 1997).

Fourth, maternal history of anxiety, family relationship quality, and adolescents' negative attributions were tested separately as moderators, but they do not occur in isolation. That is, children are likely exposed to multiple risk factors. Although the current sample was not large enough to consider the moderators simultaneously, future research should explore the cumulative risk to which children are exposed and how this may weaken or exacerbate the longitudinal relations between symptoms of anxiety and depression.

Fifth, the moderators were assessed in grade 6, reflecting preadolescent exposure to these risk factors. However, these individual and contextual factors likely were also changing over time. Taking into account the time-varying nature of family and individual risk processes will allow for a more complete understanding of children's symptom trajectories (Kouros, Cummings, & Davies, 2010).

Finally, although a strength of this study is the prospective longitudinal design and the data analytic technique accounted for baseline correlations allowing for more cogent statements regarding temporal relations, there may be unmeasured third variables, such as shared genetic vulnerability (e.g., Zavos et al., 2010), that account for the longitudinal coupling between symptoms of anxiety and depression observed in this study. Finally, clinical implications of these findings are that symptoms of anxiety and depression can predict the longitudinal course of each other across adolescence, and therefore accounting for levels of both types of symptoms is important for charting children's mental health trajectories. The robust predictive nature of anxiety for later depressive symptoms underscores the importance of early preventive efforts for children exhibiting anxious symptoms to ameliorate possible negative consequences on children's depressive symptoms (Garber & Weersing, 2010). Additionally, among children with depressive symptoms, targeted interventions for children whose mothers have a history of anxiety and depression, children from families with poor relationship quality, or children with pessimistic attributional styles may help reduce the likelihood that these children will experience subsequent elevations in anxiety symptoms.

Whereas the findings of the present study suggest *who* to target for interventions, questions still remain about *what* to target in the interventions. We posited that lack of social support and maladaptive responses to stress may potentially explain how depressive symptoms predict later anxiety; a test of these mediating processes, however, is warranted. Similarly, interventions targeted at addressing social withdrawal and low self-esteem among anxious children may provide clues into the mechanisms by which anxiety predicts later depressive symptoms. In order to refine existing prevention programs as well as develop new interventions for effectively reducing symptoms of anxiety and depression, a key direction for future research would be to examine the specific mechanisms by which symptoms of one disorder (e.g., anxiety) are related to changes in the other disorder (e.g., depression) over time.

Biography

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Figure 1.

Bivariate latent change score (LCS) model of dynamic relations between symptoms of depression and anxiety.

Note. DEP = depressive symptoms, ANX = anxiety symptoms. Unlabeled one-arrow paths were set equal to 1. Error variances of observed variables were set equal across time for depressive and anxious symptoms, respectively, and the error variances of depressive and anxious symptoms within each time point were correlated and set equal over time (not shown in model). * indicates the latent variable score; Δ indicates the latent change score (i.e., latent change), β_D and β_A represent time-varying proportional coefficients for depressive and anxious symptoms, respectively; γ_{DA} represents unidirectional longitudinal coupling from depressive symptoms to anxious symptoms, such that depressive symptoms predict subsequent change in anxious symptoms over time; γ_{AD} represents unidirectional longitudinal coupling from anxious to depressive symptoms, such that anxious symptoms predict subsequent change in depressive symptoms over time.

Figure 2.

Panel A Expected change in children's depressive symptoms over time.

Note. Y-axis has been truncated in the figure. Trajectories plotted for children with average level of depressive symptoms at grade 6. Low Anxiety = Time 1 (grade 6) CBCL Anxiety score was less than 1 *SD* below mean. High Anxiety = Time 1 (grade 6) CBCL Anxiety score more than 1 *SD* above mean.

Panel B. Expected change in children's anxiety symptoms over time. *Note.* Y-axis has been truncated in the figure. Trajectories plotted for children with average level of anxiety symptoms at grade 6. CDI = Children's Depression Inventory. Low CDI = Time 1 (grade 6) CDI score was less than 1 *SD* below mean. High CDI = Time 1 (grade 6) CDI score more than 1 *SD* above mean.

Figure 3.

Expected change in children's anxiety symptoms over time, as moderated by mothers' history of anxiety.

Note. Y-axis has been truncated in the figure. Trajectories plotted for children with average levels of anxiety symptoms at grade 6. CDI = Children's Depression Inventory. Low CDI = Time 1 (grade 6) CDI score was less than 1 *SD* below mean. High CDI = Time 1 (grade 6) CDI score more than 1 *SD* above mean

Table 1

Means, Standard Deviations, and Inter-correlations among Study Variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.
1. Risk ^a	--																		
2. Child Sex	.06	--																	
3. Grade 6 Depressive Symptoms	.19**	-.07	--																
4. Grade 6 Anxious Symptoms	.26**	-.05	.14*	--															
5. Grade 7 Depressive Symptoms	.21**	-.03	.50***	.19***	--														
6. Grade 7 Anxious Symptoms	.18**	.01	.09	.63***	.14*	--													
7. Grade 8 Depressive Symptoms	.20**	-.05	.41**	.26**	.60***	.12	--												
8. Grade 8 Anxious Symptoms	.22**	-.12 [†]	.01	.55***	.14 [†]	.57***	.11	--											
9. Grade 9 Depressive Symptoms	.14*	.06	.29***	.28**	.44***	.18*	.48**	.19*	--										
10. Grade 9 Anxious Symptoms	.17*	.04	.09	.60***	.25***	.57***	.18*	.63**	.36**	--									
11. Grade 10 Depressive Symptoms	.19*	.05	.29***	.23**	.33***	.15 [†]	.36**	.14	.50**	.33**	--								
12. Grade 10 Anxious Symptoms	.26**	.01	.09	.56***	.23***	.50***	.19*	.56**	.35**	.66***	.28***	--							
13. Grade 11 Depressive Symptoms	.23**	.08	.28**	.25***	.42***	.19*	.46**	.07	.57**	.32**	.52**	.21*	--						
14. Grade 11 Anxious Symptoms	.28**	-.11	.03	.51***	.15*	.51***	.13 [†]	.50**	.20*	.56***	.14	.65***	.24**	--					

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.
15. Grade 12 Depressive Symptoms	.15*	.12	.26**	.25**	.49**	.12	.49**	.09	.54**	.22**	.50**	.30**	.56**	.29**	--				
16. Grade 12 Anxious Symptoms	.24**	.03	.20**	.52**	.30**	.44**	.32**	.36**	.30**	.55**	.29**	.62**	.30**	.30**	.43**	--			
<i>Moderators^b</i>																			
17. Mother Anxiety History ^c	.38**	-.02	.21**	.20**	.27**	.21**	.25**	.20**	.11	.15*	.08	.12	.23**	.23**	.25**	.17**	--		
18. Negative Attributional Style	-.15*	.17	-.54**	-.11 [†]	-.42**	-.05	-.30**	-.06	-.22**	-.05	-.24**	-.08	-.13 [†]	-.05	-.17*	-.08	-.19*	--	
19. Family Relationship Index	-.21**	.17*	-.55	-.11 [†]	-.50**	-.01	-.38**	-.01	-.26**	-.13 [†]	-.15 [†]	-.09	-.23**	-.23**	-.25**	-.07	-.26**	.47**	--
Mean	--	--	6.11	.34	4.76	.38	4.68	.31	4.88	.28	5.74	.24	5.17	.27	5.72	.24	--	.98	17.70
SD	--	--	4.99	.33	4.45	.33	5.06	.28	5.22	.29	5.50	.24	5.40	.32	6.39	.29	--	.56	4.77

Note. *N* = 240 at Grade 6.

^aRisk = Mothers' history of a depressive disorder coded 0 = no history of depression (low risk) and 1 = history of depression (high risk); Depressive Symptoms assessed with the Children's Depression Inventory; Anxious Symptoms assessed with the Anxiety subscale of Child Behavior Checklist.

^bLevel of moderators at grade 6 used in analyses, median split created for LCS analyses.

^cMothers' anxiety history coded such that 0 = no history of anxiety and 1 = any history of anxiety (social, panic, OCD, and/or GAD).

[†]*p* < .10;

* *p* < .05,

** *p* < .01

Table 2

Parameter Estimates from Bivariate LCS Analysis Examining Dynamic Relations between Depressive and Anxiety Symptoms

Parameter Estimates	<u>Depressive Symptoms ↔ Anxiety Symptoms</u>	
	Depressive Symptoms	Anxiety Symptoms
<u>Intercept</u>		
Mean	4.35 (.63)**	.22 (.04)**
Variance	12.06 (2.15)**	.06 (.01)**
<u>Linear Slope</u>		
Mean	.42 (.42)	-.04 (.02) [†]
Variance	1.50 (.74)	.00 (.00)
<u>Proportional Coefficients</u>		
β_1	-.42 (.10)**	.01 (.09)
β_2	-.29 (.14)*	-.22 (.08)**
β_3	-.23 (.12) [†]	-.11 (.09)
β_4	-.16 (.12)	-.23 (.10) [†]
β_5	-.25 (.10)*	.04 (.13)
β_6	-.16 (.10)	-.21 (.10)*
<u>Coupling Parameters</u>		
$\gamma_{\text{ANXIETY} \rightarrow \text{DEPRESSION}}$	2.92 (1.49)*	
$\gamma_{\text{DEPRESSION} \rightarrow \text{ANXIETY}}$		.01 (.01) [†]

Note. Unstandardized parameter estimates reported (with standard errors in parentheses).

Analyses controlled for risk.

[†] $p < .10$,

* $p < .05$,

** $p < .01$

Table 3
Results from Multigroup Bivariate LCS Analyses: Mothers' History of Anxiety and Family Relationship Quality as Moderators

Parameters	Mothers' History of Anxiety			Family Relationship Quality					
	No History			Positive History			Quality Low		
	DEP	ANX		DEP	ANX		DEP	ANX	ANX
Intercept									
Mean	4.19 (.61)**	.23 ^a (.04)**	5.56 (.90)**	.23 ^a (.04)**	7.16 (.65)**	.22 ^b (.04)**	2.58 (.46)**	.22 ^b (.04)**	
Variance	11.86 ^a (2.08)**	.06 ^b (.01)**	11.86 ^a (2.08)**	.06 ^b (.01)**	13.12 (3.08)**	.07 ^c (.10)*	2.08 (.32)	.07 ^c (.10)*	
Slope									
Mean	.14 (.38)	-.02 ^a (.02)	1.04 (.53) [†]	-.02 ^a (.02)	.02 ^b (.34)	-.03 ^c (.02)	.02 ^b (.34)	-.03 ^c (.02)	
Variance	1.18 (.56)*	.001 (.00)**	3.00 (1.25)*	.01 (.01)	1.23 ^a (.40)**	.03 (.01)*	1.23 ^a (.40)**	.002 (.001)*	
Proportional Coefficients									
β ₁	-.48 ^a (.09)**	.07 (.09)	-.48 ^a (.09)**	-.15 (.12)	-.38 ^b (.06)**	-.74 (.17)**	-.38 ^b (.06)**	.21 (.09)*	
β ₂	-.35 ^a (.13)**	-.12 (.08)	-.35 ^a (.13)**	-.40 (.10)**	-.21 ^b (.09)*	-.75 (.15)**	-.21 ^b (.09)*	-.11 (.08)	
β ₃	.004 (.15)	-.03 (.09)	-.48 (.12)**	-.33 (.14)*	-.17 ^a (.08)*	-.68 (.17)**	-.17 ^a (.08)*	-.00 (.10)	
β ₄	-.13 (.11)	-.07 (.11)	-.28 (.15) [†]	-.48 (.16)**	-.18 (.08)*	-.87 (.17)**	.02 (.11)	.01 (.12)	
β ₅	-.30 ^a (.09)**	.01 ^b (.12)	-.30 ^a (.09)**	.01 ^b (.12)	-.17 ^c (.07)*	-.66 (.21)**	-.17 ^c (.07)*	.21 (.15)	
β ₆	-.18 ^a (.10)+	-.05 (.11)	-.18 ^a (.10) [†]	-.46 (.13)**	.04 (.10)	-.85 (.17)**	-.31 (.07)**	.05 (.12)	
Coupling Parameters									
γ _{AD}	3.25 ^a (1.36)*		3.25 ^a (1.36)**		3.19 ^a (1.11)**		3.19 ^a (1.11)**		
γ _{DA}		.00 (.01)		.02 (.01)**		.04 (.01)**		-.00 (.01)	

Note. Unstandardized parameter estimates (with standard errors in parentheses) presented. Similar subscripts in the same row indicate parameter that is equivalent between groups.

[†] $p < .10$,
* $p < .05$,
** $p < .01$

Table 4

Results from Multigroup Bivariate LCS Analyses: Negative Attributions as a Moderator

Negative Attributions				
		Low		High
Parameters		DEP	ANX	ANX
<u>Intercept</u>				
Mean	2.69 (.52)	.19 (.04)**	7.24 (.65)**	.30 (.04)**
Variance	1.79 (1.38)	.05 (.01)**	11.81 (3.40)**	.09 (.02)**
<u>Slope</u>				
Mean	.39 ^b (.41)	-.02 (.03)	.39 ^b (.41)	-.04 (.04)
Variance	2.09 ^a (.75)**	.01 ^b (.003) [†]	2.09 ^a (.75)**	.01 ^b (.003) [†]
<u>Proportional Coefficients</u>				
β_1	-.50 ^a (.08)**	.56 (.13)**	.50 ^a (.08)**	-.30 (.11)**
β_2	-.38 ^a (.11)**	-.05 (.09)	-.38 ^a (.11)**	-.29 (.11)*
β_3	-.29 ^a (.10)**	.24 (.14) [†]	-.29 ^a (.10)**	-.23 (.13) [†]
β_4	.20 ^a (.10)*	.23 (.15)	.20 ^a (.10)*	-.41 (.13)**
β_5	-.29 ^a (.08)**	.50 (.18)**	-.29 ^a (.08)**	-.14 (.17)
β_6	-.24 ^a (.08)**	.19 (.14)	-.24 ^a (.08)**	-.36 (.14)**
<u>Coupling Parameters</u>				
γ_{AD}	4.10 ^a (1.44)**		4.10 ^a (1.44)**	
γ_{DA}			-.02 (.01) [†]	.02 (.01)**

Note. Unstandardized parameter estimates (with standard errors in parentheses) presented. Similar subscripts in the same row indicate parameter that is equivalent between groups.

[†] $p < .10$,

* $p < .05$,

** $p < .01$