Published in final edited form as:
J Abnorm Child Psychol. 2011 May ; 39(4): 489–500. doi:10.1007/s10802-010-9482-2.

Developmental pathways to depressive symptoms in adolescence: A multi-wave prospective study of negative emotionality, stressors, and anxiety

Andrea L. Barrocas[1] and **Benjamin L. Hankin**[1]

[1]Department of Psychology, University of Denver, Denver, Colorado

Abstract

This study examined two potential developmental pathways through which the temperament risk factor of negative emotionality (NE) leads to prospective increases in depressive symptoms through the mediating role of stressors and anxious symptoms in a sample of early to middle adolescents (N=350, 6th–10th graders). The primary hypothesized model was that baseline NE leads to increased stressors, which results in increases in anxious arousal, which culminates with elevated depressive symptoms. An alternate model hypothesized that baseline NE leads to increased anxious arousal, which results in increases in stressors, and this culminates in elevated depressive symptoms. Youth completed self-report measures of NE, stressors, anxious arousal, and depressive symptoms at four time-points. Path analysis supported the primary model and showed that the mediating influence of stressors and anxious arousal explained 78% of the association between NE and prospective elevations in depressive symptoms. The alternate model was not supported. Neither gender nor age were moderators.

Keywords

adolescence; depressive symptoms; temperament; developmental pathways

Depressive disorders increase in prevalence during adolescence (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993) beginning around age 13 (Hankin et al., 1998), and the rate of depression for girls begins to nearly double that of boys starting at about age 13–15 (Hankin et al., 1998; Nolen-Hoeksema & Girgus, 1994). A wealth of research on the risk factors for developing depression has contributed greatly to understanding of the etiology of depression in youth, yet less attention has been given to the temporal ordering of specific risk factors predicting prospective changes in depressive symptoms. Negative emotionality (NE) is a broad temperamental factor that consistently predicts a range of poor psychopathological outcomes and is an especially robust predictor of depression (Lahey, 2009). For example, Krueger (1999) found that a higher level of NE at age 18 predicted an increase in affective

Correspondence concerning this article should be addressed to Andrea L. Barrocas, at the Department of Psychology, University of Denver, 2155 South Race Street, Denver CO 80208. Phone: 303-871-6828. Internet correspondences can be addressed to Andrea.Barrocas@du.edu.

disorder symptoms and anxiety disorder symptoms at age 21 after controlling for initial level of either affective disorder or anxiety disorder symptoms at age 18. Despite several empirical demonstrations that individuals with higher levels of NE are at greater risk for development of depression and greater depressive symptoms, relatively little research has examined mediating processes and developmental pathways that account for the longitudinal association between NE and depressive symptoms.

Many risk factors and processes contributing to the development of depressive symptoms among youth have been identified (Abela & Hankin, 2008). In addition to the temperament trait of NE, both experienced stressful life events and anxious symptoms have been shown to be potent predictors of later depressive symptoms. Stressors tend to precede the onset of a depressive disorder and elevated depressive symptoms during adolescence (see Grant et al., 2003 for a review). Likewise, anxiety is highly comorbid with depression during adolescence (Angold, Costello, & Erkanli, 1999; Steer, Clark, Kumar, & Beck, 2008) and precedes depression in youth (Cole, Peeke, Martin, Truglio, & Seroczynski, 1998; Keenan, Feng, Hipwell, & Klosterman, 2009; Pine, Cohen, Gurley, Brook, & Ma, 1998). Yet to date, no study has incorporated these factors in a developmental trajectory predicting increases in depressive symptoms in adolescence as an outcome.

This study examined two specific pathways (i.e. equifinality) that may explain why higher NE leads to an increase in depressive symptoms through the risk factors of experienced stressors and anxious arousal symptoms. The first specific pathway, the NE-stressors model, posits that subsequent stressors and then anxious arousal lead to increased depressive symptoms. This model is in part conceptually based on Hankin and Abramson's (2001) elaborated cognitive vulnerability-transactional stress theory which articulates one temporal model how certain pre-existing vulnerabilities, such as temperament risk of NE, and other more proximal risk factors, such as stressors and initial negative affect, sequentially unfold over time to contribute to predict prospective elevations in depressive symptoms in adolescence. However, because this theory also introduces cognitive vulnerabilities as an important risk factor for the ontogeny of depression, the NE-stress model tested in this study will be informed by Hankin and Abramson's (2001) theory but will not fully test this model. A second plausible pathway, the NE-anxiety model, is that NE contributes first to increases in subsequent anxious arousal, then level of anxious arousal predicts greater experienced stressors, and finally more stressors lead to increased depressive symptoms. This NE-anxiety model is supported by literature (see below) linking all of the risk factors in a specific direction leading to increases in depressive symptoms. To our knowledge, this is the first study to investigate the temporal ordering of these mediating factors using a multi-wave longitudinal design to differentiate which developmental pathway best accounts for the longitudinal association of NE and later depressive symptoms.

The Relationship Among Risk Factors for Depressive Symptoms in Adolescence

Temperament as a Risk Factor

Individual temperamental traits are known to relate to psychopathology and account for some of the individual differences seen among manifestations of psychological disorders and symptoms (see Krueger & Tackett, 2003 for a review; Lahey, 2009). More specifically, underlying temperamental traits, such as NE, have been shown to predict both mood and anxiety disorders over time (Krueger, 1999).[2] NE is conceptualized as a tendency toward experiencing intense discomfort, such as fear and anger, and reacting more easily to stress (see Krueger, 1999; Rothbart, Ahadi, Hershey & Fisher, 2001). Individuals with higher levels of NE may be more likely to attend to and interpret the more negative aspects of their environment and, consequently report more negativity in their surroundings. Prospective studies have shown that adolescents (Wetter & Hankin, 2009; Yang, Chiu, Soong, & Chen, 2008) and adults (Farmer & Seeley, 2009; Kendler, Kuhn, & Prescott, 2004) with higher levels NE also have higher levels of depressive symptoms. Likewise, individuals with high levels of NE are more likely to be anxious (Lonigan et al., 2004). Additionally, other studies using adolescents (Kercher, Rapee, & Schniering, 2009; Wetter & Hankin, 2009) and young adult (Ladawalla & Hankin, 2008; Ormel & Wohlfarth, 1991) samples have found that NE predicts increased depressive symptoms and stressors, and that experiencing more stressors mediates the association between NE and depressive symptoms.

Anxiety as a Risk Factor

Depression and anxiety are highly comorbid (Kessler et al., 2008; see Avenevoli, Knight, Kessler, & Merikangas, 2008; Watson, 2005). Pine and colleagues (1998) also initially showed that childhood anxiety preceded later depression, and many studies since have found that anxiety tends to precede depression over long-term time frames (Avenevoli, Stolar, Li, Dierker, & Merikangas, 2001; Beesdo et al., 2007; Kessler et al., 2008) and follow-ups of shorter time frames (Cole et al., 1998; Hettema, Kuhn, Prescott, & Kendler, 2006). Recent research with girls by Keenan and colleagues (2009) has shown that anxiety disorders most strongly predicted later depressive disorders, although at the symptom level the strongest predictor of depression is prior depression and not prior anxiety. The work in this area establishes the strong relationship between anxiety and depression in children and adolescents and suggests that anxiety precedes depression. However, such developmental epidemiological research does not explain what other factors may play a role in the longitudinal link between depressive and anxious symptoms in these age groups.

Experienced Stressors as a Risk Factor

Stressors play an important role in the onset and course of psychological disorders, especially internalizing symptoms and disorders in youth (Grant et al., 2003). Specifically, stressors lead to increases in depressive symptoms (Eley & Stevenson, 2000; Espejo et al.,

[2]Because research suggests that positive emotionality does not play a substantial role in the causal chain between stressors (Lakdawalla & Hankin, 2008; Wetter & Hankin, 2009) and anxiety (e.g. Lonigan, Vasey, Phillips, & Hazen, 2004), this study only investigated the temperamental trait of NE.

2006; Grant et al., 2003; Landis et al., 2007) and anxious symptoms (Eley & Stevenson, 2000; Rudolph, 2002). Hankin (2008a) found that increases in reported stressors predicted increases in both anxious arousal and depressive symptoms in a sample of early to middle adolescents. Stressors have also been found to lead to greater internalizing symptoms in adolescence (Hammen, 2005; Rudolph, 2002). In support of the NE-anxiety model, recent research on stress generation and anxiety has found that greater anxious styles predicted greater reported stressors in a sample of college students (Riskind, Black, & Sahar, 2010). In addition, there is evidence for stress generation in those with comorbid anxiety and dysthymia (Harkness & Luther, 2001). Yet others studies have not supported stress generation from anxious symptoms (Joiner, Wingate, Gencoz, & Gencoz, 2005; Uhrlass & Gibb, 2007). Consequently, the literature is mixed on whether anxious symptoms lead to stress generation, and this study will add to the literature investigating this process.

Additionally, as previously mentioned, individuals with higher levels of NE report more experienced stressors (Bolger & Zuckerman, 1995; Wetter & Hankin, 2009; Yang et al., 2008). Longitudinal research with adolescents and adults (Kercher, Rapee, & Schniering, 2009; Lakdawalla & Hankin, 2008; Wetter & Hankin, 2009) suggests that NE predicts generation of additional stressors over time and shows that stressors mediate the relationship between initial NE and prospective elevations in depressive symptoms. However, in these prior studies stressors and depressive symptoms were measured at the same time-point, and the Kercher and colleagues (2009) study sample was limited to girls only. Nonetheless, findings from these studies are consistent with theory hypothesizing that NE prospectively predicts increases in stressors, and in turn, greater experienced stressors predict increases in depressive symptoms in adolescence (Hankin & Abramson, 2001).

Gender and age differences in depressive symptoms in youth

Around middle-adolescence, the rate of depression begins to increase (Hankin et al., 1998; Lewinsohn et al., 1993), and adolescent girls experience more depressive symptoms and depressive disorders than boys (see Hankin, Wetter, & Cheeley, 2008, for a review). Hankin and Abramson (2001) hypothesize that adolescent girls and older youth will exhibit greater levels of each of the risk factors in the developmental pathway than boys and younger youth. As a result, girls and older youth are hypothesized to be more likely to exhibit depressive symptoms. This implies that even though rates of depression among these groups differ, general pathways to depressive symptoms may exist for girls and boys and adolescents of differing ages. In contrast, others have argued that gender specific pathways exist in the emergence of depressive symptoms (e.g. Zahn-Waxler, 2000). According to these models, different groups take divergent paths, such that the processes underlying the prediction of depressive symptoms differ in boys compared with girls. Accordingly, this study will examine gender and age invariance in the two developmental pathways hypothesized to link NE and later depressive symptoms to ascertain whether these mediating pathways apply equally across age and gender as hypothesized by a general depression model, or whether the pathways differ, as implicated in a specific pathway model for different groups.

The Present Study

Prior theoretical evidence suggests a relationship among NE, experienced stressors, anxious arousal symptoms and depressive symptoms, yet an important unresolved question is the temporal ordering through which these risk factors contribute to prospective elevations in depressive symptoms. Similarly, no study has examined the role of gender or age with all four of these factors and differences that may exist in this developmental pathway. Most of the research on NE, stressors, anxious symptoms, and depressive symptoms consists of cross-sectional or only two-time point longitudinal studies (some exceptions include Avenevoli et al., 2001; Cole et al., 1998; Kercher, Rapee, & Schniering, 2009). Although informative, cross sectional and shorter longitudinal designs may not be best for studying developmental trajectory models (Curran & Willoughby, 2003). Curran and Willoughby (2003) suggest the use of multi-wave longitudinal data when using developmental trajectory models to most effectively test for prospective changes over time. The present study advances understanding of how experienced stressors and anxious arousal link NE and depressive symptoms in adolescence by utilizing a multi-wave longitudinal design to test particular theoretically plausible developmental pathways.

This study examined two specific developmental pathways. First, in the NE-stressors model, it was predicted that higher levels of NE predict a higher level of experienced stressors, which in turn then generates increases in anxious arousal, resulting in increases in depressive symptoms (see top portion of Figure 1). Second, in the plausible alternate pathway, the NE-anxiety model, it was predicted that baseline NE would first elevate anxious arousal, which would in turn predict increases in experienced stressors, and this subsequent link would mediate the longitudinal association between baseline NE and later depressive symptoms (see bottom portion of Figure 1). Finally, moderating variables, although not an explanation for difference in depression rates, are important to investigate because they illuminate possible differential pathways to increased depressive symptoms (see Hankin & Abramson, 1999). There may be differential pathways by which girls and older adolescents become more depressed than boys and younger adolescents. Therefore, we explored if gender and/or age moderated either the NE-stressors or the NE-anxiety model.

Method

Participants

Participants were adolescents in the 6^{th}–10^{th} grade who were recruited from five Chicago area schools. Schools were selected to represent the ethnic and socio-economic diversity of the Chicago area. Consent forms were given to 467 youth at the five schools, in the appropriate grades, and 390 of these youth (83.5%) had parents who provided active consent and were also interested in participating. Present analyses are based on 350 students who provided complete data at baseline. Students ranged in age from 11–17 (M=14.5; SD=1.40; 36% age 11–14 and 64% age 15–17), 57% were female, 53% identified as white, 21% African American, 13% Latino/a, 6% Asian or Pacific Islander, and 7% identified as bi- or multi-racial. There were no significant demographic difference (gender, age, ethnicity) between those originally available at the schools (N=467) and those used for present

analyses (N=350). Rates of participation decreased slightly from baseline: wave 2 (N=303), wave 3 (N=308), and wave 4 (N=345).

Procedures

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

Measures

Depressive Symptoms—The Children's Depression Inventory (CDI, Kovacs, 1981) is a 27-item self-report measure that assesses depressive symptoms in youth. Each item is rated on a 0–2 Likert scale with item scores being added together. Scores for the current study ranged from 0 to 51. Higher score indicating greater symptom severity and a score of 13 in clinical samples and 20 in population-based samples is considered the clinical cut-off (Kovacs, 1992). The CDI has been shown to have good reliability (alphas for the current study ≥ .90) and validity (Kazdin, French, & Unis, 1983) as a measure of general depressive symptoms in youth. The CDI was given at waves one and four.

Negative Emotionality—The Dimensions of Temperament Scale-Revised (DOTS-R, Wills, Windle, & Cleary, 1998) is a 54-item self-report measure that assesses 10 dimensions of temperament. For this study, 11 items from the Negative Emotionality subscale were used to assess adolescent NE. Adolescents responded to these items on a 4-point Likert scale ranging from "not at all" to "very much." Means were computed for each participant and scores ranged from 1 to 4. The DOTS-R has good reliability (alpha for current study = .84). The DOTS-R was given at wave one.

Anxious Arousal Symptoms—The Mood and Anxiety Symptom Questionnaire (MASQ) used in this study was modified from the original MASQ (Watson et al., 1995), which is a 90-item self-report measure that assesses the general distress and specific anxious symptoms and depressive symptoms based on the tripartite model of depression and anxiety (Clark & Watson, 1991). For this study, only the Anxious Arousal (ANX) subscale was used to assess relatively specific anxious symptoms that are not overly saturated with negative affect. Adolescents responded to 10 ANX items on a 1–5 Likert scale. Examples of items from the ANX scale of the MASQ are "hands were cold or sweaty" and "muscles twitched

or trembled." Means were computed for each participant, and scores for the current study ranged from 1 to 5. A higher score indicates greater symptom severity. Reliability (alphas for the current study ≥ .83) and validity of the MASQ have been demonstrated in previous studies with adolescents (e.g., Hankin, 2008b; Watson et al., 1995). The MASQ was given at waves one, two and three.

Experienced Stressors—The Adolescent Life Events Questionnaire (ALEQ, Hankin & Abramson, 2002) is a 57–item self-report measure that assesses a broad range of negative life events that typically occur among adolescents. The ALEQ assesses life events including school/achievement problems, friendship and romantic difficulties, and family problems. Examples of items from the ALEQ include "got a bad grade on a report card" to assess school events, "had an argument with a close friend" for friendship events, "boyfriend/girlfriend broke up with you" for romantic events, and "your parents grounded you" for family events. Adolescents indicate how often these events have happened to them over the past 5 weeks using a 0–4 Likert scale, ranging from never (0) to always (4). Means were computed for each participant, and scores for this study ranged from 0 to 4. Higher scores indicated report of more experienced negative events at any given time over the 5 weeks. Test-retest reliability is good, with *r*'s ranging from .42 over 5 weeks to .35 over 2 years (see Wetter & Hankin, 2009). The ALEQ was given at waves one, two, and three.

Results

Preliminary Analyses

Descriptive statistics of all variables used in analyses (means, standard deviations, and correlations) are presented in Table 1. All variables were significantly positively associated with one another.

Before examining the sequential mediation models, we examined whether T1 NE led to T4 depressive symptoms using hierarchical regression. T1 NE, in fact, significantly predicted elevations in T4 depressive symptoms ($\beta = .16$, $t = 3.47$, $p < .01$) after controlling for T1 depressive symptoms.

Overview of Statistical Approach

To examine the mediational models and developmental pathways, we used path analysis (AMOS 18.0; Arbuckle, 2007) and followed suggested steps to test mediation using the bootstrap procedure (Shrout & Bolger, 2002). This procedure is recommended as a better test of mediation (MacKinnon, Lockwood, & Williams, 2004; Shrout & Bolger, 2002) than the traditional Baron and Kenny (1986) test of mediation or the Sobel test (1982). The bootstrap procedure resamples the data to provide a distribution that allows for significance testing of mediation. Additionally, the bootstrap procedure has more power, does not assume multivariate normality (an assumption for conducting the Sobel test and produces more accurate and more conservative confidence intervals (Taylor, MacKinnon, & Tein, 2008).

The steps outlined above and the longitudinal design of the study provided a rigorous test of our mediational hypotheses. Cole and Maxwell (2003) suggest using a longitudinal multi-wave design to test for mediation, which allows for non-overlapping time points between

each of the observed variables when testing a mediation model (MacKinnon, 2008). Additionally, Cole and Maxwell (2003) emphasize the importance of controlling for preceding levels of predicted variables when testing mediation in order to assess if mediators account for prospective changes in the predicted variables. Likewise, the longitudinal design of this study allowed us to test the predictability of changes in factors, one of the main aims of the study. Last, we controlled for T1 depressive symptoms in all analyses to provide the most stringent test of our hypotheses, allowing us to partial out any shared variance that T1 depressive symptoms might have with our proposed mediators and predict prospective changes in depressive symptoms over time.

NE-Stressors Model: Stressors and Anxious Arousal Mediate the Prospective Association between NE and Depressive Symptoms

To examine the NE-stressors model, we first established that T1 NE predicted T4 depressive symptoms after controlling for T1 depressive symptoms (see above). Figure 2 summarizes the results of the tests for the model predicting that sequential T2 stressors and T3 anxious arousal mediate the relationship between T1 NE and T4 depressive symptoms. Next, we tested the mediation model using the bootstrap procedure with 2,000 resamples at the 95% confidence level. We report confidence intervals, and confidence intervals that do not include zero are considered significant. First, a simple model to test the NE-stressors model was tested. In this model all predictive paths were present, as well as correlation between variables assessed at the same time point. Additionally, we controlled for T1 stressors, T1 and T2 anxious arousal, and T1 depressive symptoms. This model did not fit the data well: $\chi^2(11) = 156.100$; $p < .001$; CFI = 0.890; RMSEA = 0.194. Following this we added theoretically meaningful correlations between variables across time points, as recommended by modification index analyses (e.g. T1 anxious symptoms to T2 stressors, T1 depressive symptoms to T3 anxious symptoms, and T1 stressors to T4 depressive symptoms). The model had an excellent fit: $\chi^2(4) = 3.511$; $p < .05$; CFI = 1.000; RMSEA = 0.000.

The bootstrap procedure supported mediation in the model. First, again the direct effect of T1 NE on T4 depressive symptoms, after controlling for initial depressive symptoms was significant. Second, all other direct effects modeled in Figure 2 were significant. The 95th percentile bias-corrected confidence interval for the full mediated effect of sequential T2 stressors and T3 anxious arousal mediating the association from T1 NE to T4 depressive symptoms ranged from .082 to .200. Since this confidence interval did not include zero, mediation was supported for the full model. Additionally, the direct effect of T1 NE on T4 depressive symptoms was reduced (from $\beta = .16$, $p < .01$, to $\beta = .10$, $p < .05$) after including the sequential mediators. To support the sequential mediation hypothesis we also tested mediation for each smaller part of the full model. T2 stressors mediated the association between T1 NE and T3 anxious arousal, controlling for T1 stressors. The 95th percentile bias-corrected confidence interval for this mediated effect ranged from .027 to .123, which did not include zero. T3 anxious arousal mediated the association between T2 stressors and T4 depressive symptoms, controlling for T2 anxious arousal. The 95th percentile bias-corrected confidence interval for this mediated effect ranged from .020 to .120, which did not include zero. Inclusion of T2 stressors and T3 anxious arousal explained 78% of the

association between T1 NE and T4 depressive symptoms after accounting for T1 depressive symptoms. This corresponds to nearly full mediation.

NE-Anxiety Model: Anxious Arousal and Stressors Mediate the Prospective Association between NE and Depressive Symptoms

The analyses used to test the NE-stressors model indicate a particular sequential mediation model, but it is possible that alternate temporal orderings of these variables may exist. The following analyses tested a particular conceptually plausible alternative, the NE-anxiety model. The same steps were taken to test if sequential T2 anxious arousal and T3 stressors mediated the relationship between T1 NE and T4 depressive symptoms (see Figure 3 for direct effects of each variable).

As with the above analyses, a simple model tested the NE-anxiety model. In this model all predictive paths were present, as well as correlation between variables assessed at the same time point. Additionally, we controlled for T1 anxious symptoms, T1 and T2 stressors and T3 depressive symptoms. This model did not fit the data well: $\chi^2(11) = 153.748$; $p < .001$; CFI = 0.876; RMSEA = 0.193. Following this we added theoretically meaningful correlations between variables across time points, as recommended by modification index analyses. The NE-anxiety model still did not fit the data well according to conventional standards $\chi^2(7) = 94.861$; $p < .001$; CFI = 0.924; RMSEA = 0.190. As this model did not fit as well as the prior model, we continued to test whether gender and/or age moderated pathways in the NE-stressors model.

Gender in the Developmental Pathway Models

We were interested to see if gender moderated the NE-stressors model. First, separate groups were created for girls and boys. Next, we tested for group invariance by conducting two analyses, one where all primary paths were allowed to freely vary and another where all primary paths were constrained to be equal between girls and boys. The two models did not significantly differ, $\Delta\chi^2(6) = 4.027$, $p = .67$, suggesting that the data fit this NE-stressors model similarly for girls and boys.

Age in the Developmental Pathway Models

We were also interested to see if age moderated the NE-stressors model. Separate groups were created for younger adolescents (age 11–14) and older adolescents (age 15–17). Next, we tested for group invariance by conducting two analyses, one where all primary paths were allowed to freely vary and another where all primary paths were constrained to be equal between younger adolescents and older adolescents. The two models did not significantly differ, $\Delta\chi^2(6) = 3.424$, $p = .75$, suggesting that the data fit this NE-stressors model similarly for younger adolescents and older adolescents.

Discussion

Adolescence is a period of marked change, including an increase in risk for elevated depressive symptoms. Many youth navigate through this period of storm and stress (Arnette, 1999) remarkably well without negative outcomes, still a substantial minority experience

maladaptive outcomes, including elevated levels of depressive symptoms (Petersen et al., 1993). As such, this study investigated the developmental pathways linking together risk factors to lead to a rise in depressive symptoms during this crucial developmental period. More specifically, the purpose of the current study was to examine particular plausible developmental trajectories linking baseline levels of the temperamental vulnerability of NE to prospective increases in depressive symptoms through the mediating influences of changes in anxious arousal and experienced stressors over time in adolescence and to explore whether gender or age moderated these meditational pathways. The NE-stressors model, in which T2 experienced stressors and subsequent T3 anxious arousal mediated the association between T1 NE and T4 depressive symptoms, fit the data well. However, the NE-anxiety model did not fit the data as well. Additionally, neither gender nor age moderated the NE-stressors model. In sum, results from this study suggest a specific developmental pathway that delineates how youth with greater levels of NE develop higher levels of depressive symptoms over time, and this process applies equally across gender and age.

One aim of the study was to investigate two specific plausible developmental pathways. NE, experienced stressors, anxious symptoms and depressive symptoms have been shown to relate to one another in various ways in adolescence, and this study showed that they link together through a specific pathway. More importantly, given the strength of the association between NE and a broad range of negative outcomes (Lahey, 2009), it is noteworthy that NE significantly led to increased depressive symptoms in this sample through this specific pathway, particularly the temporal ordering predicted by the NE-stressors model. Furthermore, the NE-stressors developmental pathway, in which NE led to increases in reported stressors, which was followed by elevated anxious arousal, explained most of the longitudinal association between NE and prospective elevations in depressive symptoms in this sample.

The first link of the NE-stressors causal chain, NE predicting increased experienced stressors, provides plausible support for NE playing a role in stress generation (Hammen, 2005). Hankin (2010) recently found that NE, but not PE, predicted generation of objectively rated daily stressors, suggesting that NE may lead to stress generation. A different, equally plausible interpretation of the NE to stressors link is that those with higher levels of NE interpret events more negatively or have a lower threshold for stressors, and thus report more stressors than those with lower levels of NE. Because of the nature of this study (subjective self-report), we cannot support one of these processes over the other. Despite these differing interpretations and difficulty distinguishing the exact process by which NE leads to greater reported stressors, this study provides further support for the NE to stressors link leading broadly to depressive symptoms in adolescence. Following an increase in experienced stressors from higher NE, as the second link in the NE-stressors model supports, is an elevation in initial anxious arousal. It may be that those with greater levels of NE not only experience more stressors but also are less well equipped to regulate their anxious symptoms when faced with stressors. Consequently their initial arousal may then increase over time, and as the third link in the model suggests may develop into depressive symptoms.

Findings from this study support and build upon previous research (Kercher, Rapee, & Schniering, 2009; Lakdawalla & Hankin, 2008) showing that NE serves as a preexisting vulnerability that leads to increased experienced stressors, which culminates in elevated depressive symptoms. We replicated and extended Kercher and colleagues' work, which was likewise conceptually testing the elaborated cognitive vulnerability-transactional model of depression (Hankin & Abramson, 2001), by demonstrating that increased anxious arousal symptoms follows temporally from greater stress exposure and precedes prospective increases in depressive symptoms.

In the alternative theoretically plausible temporal ordering, the NE-anxiety model, NE did not predict elevations in anxious arousal symptoms in the first link and anxious arousal symptoms did not predict elevations in stressors in the second link. Although NE is theoretically related to anxiety (Clark & Watson, 1991), NE may not predict anxious arousal symptoms over this time frame. In addition, there are fewer empirical data to support that anxiety contributes to increased stressors than there is for NE contributing to this process. Therefore, it seems likely that anxious arousal is a consequence of increased experienced stressors and not a precursor to it. Furthermore, findings from this study cannot support stress generation directly from anxious symptoms, which is in contrast to research that supports this process with individuals with comorbid depression and anxiety (Harkness & Luther, 2001). Even though the NE-anxiety model was not supported, it cannot be disproved that this developmental pathway does not occur in other contexts. It may be that with a more general measure of anxious symptoms, or in a group with comorbid depression and anxiety, anxiety may follow NE and precede experienced stressors to lead to elevations in depressive symptoms. Likewise, other plausible alternative models were not explored in this study. For example, there is evidence that NE interacts with experienced stressors to predict depressive symptoms (e.g. Kendler, Kuhn, & Prescott, 2004), and it may be possible that anxious arousal might mediate this interaction. It is possible that a more parsimonious model, in which experienced stressors predicts later depressive symptoms through intermediate depressive symptoms, might fit the data better. In addition, there may be other time frames over which this model may be supported or other samples for which this pathway would exist. Future research should further examine other models testing the longitudinal association of NE and depressive symptoms in youth.

Another aim of the study was to investigate gender and age moderation in the pathway. Multigroup SEM analyses showed that neither gender nor age moderated the NE-stressors developmental pathway leading to depressive symptoms. These results suggest that this specific temporal unfolding of risk factors for depressive symptoms is the same for girls and boys, and for younger and older adolescents. Findings from this study are noteworthy because there are two established explanations for the gender and age differences in depressive symptoms in adolescence; there are gender specific models and general models. Hankin and Abramson (2001) purport that a general model of depression that is applicable to both boys and girls, in contrast to gender specific models (e.g. Keenan et al., 2009; Zahn-Waxler, 2000), may provide a better fit to understanding the development of depressive symptoms. Keenan, Feng, Hipwell, & Klosterman (2009) found that girl's level of depressive symptoms during early adolescence was most strongly predicted by their level of depressive symptoms during early childhood, suggesting a homotypic path of depressive

symptoms instead of a heterotypic path by which anxious arousal symptoms predict depressive symptoms. Such gender specific models inform our understanding of the ontogeny of depression in adolescence as it applies to specific groups. However, these models do not apply generally to all groups and leave unanswered what pathways may lead to greater depressive symptoms more generally for adolescents. Likewise, because being an older or younger adolescent did not moderate the pathway, this specific temporal ordering of NE-stressors model may be one that occurs throughout adolescence. In sum, the findings from this study apply equally well to boys and girls, and to younger and older adolescents. Additionally, as Hankin and Abramson (2001) propose in their elaborated cognitive vulnerability-transactional stress model for depression, the greater incidence of depressive symptoms in girls and older adolescents in this sample seems most likely attributable to a higher rate of specific preexisting vulnerabilities, such as NE, than to differential pathways. However, this study does not refute the notion that gender specific models, such as those hypothesized by Keenan & Hipwell (2005), may be supported for other risk factors and processes leading to increased depressive symptoms in adolescence.

This study advances the literature in several ways. First, to our knowledge this is the first study to examine NE, experienced stressors, anxious symptoms and depressive symptoms together, and it is the first to use a prospective longitudinal design to investigate the relationship among all four factors. Although other studies have examined relations among several of the factors, none has tested the temporal ordering of these risk factors linking together to increase depressive symptoms in adolescence. Second, the use of a multi-wave longitudinal design allowed us to rigorously test our mediation hypotheses. Cole and Maxwell (2003) assert that it is optimal to utilize such a design to test mediation and prospective change in variables over time, which was one of the goals of this study. Accordingly, sequential experienced stressors and anxious arousal accounted for 78% of the association between NE and depressive symptoms in the NE-stressors model after accounting for initial level of depressive symptoms, which is quite large and represents nearly full mediation. Moreover, by controlling for previous level of each predicted variable we were able to test for changes in variables longitudinally. This enabled a rigorous test of the *a priori* specified developmental pathways and the temporal unfolding of specific factors. When interpreting the association of NE and stress, however, one must take caution when interpreting results, as some research suggests that negative life events and neuroticism covary in young adults (Magnus, Diener, Fujita, & Pavot, 1993), and this association has been found to be mediated by familial factors (Kendler, Gardener and Prescott, 2003).

Third, initial level of depressive symptoms was controlled for, providing a stringent test of the hypothesized developmental pathways. Fourth, we retained a large proportion of participants from our baseline assessment, so the attrition rate was low across time in this study. Finally, because anxiety and depression share many commonalities (Clark & Watson, 1991; Watson, 2005), we assessed and analyzed anxious arousal symptoms from the tripartite model of depression using items from the anxious arousal scale of the MASQ (Watson et al., 1995). In doing so, we sought to minimize the overlap between anxious and depressive symptoms in our analyses, allowing us to predict depressive symptoms from

anxious arousal only and not general anxiety, which may overlap more broadly with depressive symptoms.

Despite these strengths, this study has several limitations. First, the time between assessment points was relatively short, with only 5 weeks between each time-point. The developmental pathway might differ across longer or shorter timeframes. It is possible that several of the constructs may be less stable over a longer time frame. Additionally, the exact time of the unfolding of these specific factors is unknown. Second, this study established that the developmental pathway existed similarly for older and younger adolescents, but younger children have also reliably exhibited depressive symptoms (Carlson & Kashani, 1988; Weiss & Garber, 2003) and older samples have provided support for the link among NE, stressors and depressive symptoms (e.g., Hankin, 2010; Lakdawalla & Hankin, 2008). Furthermore, with the emergence of adolescence comes many changes, and adolescence might be a distinct period in which the emergence of increased depressive symptoms occurs in a specific way. Because of the age of this sample, it is not known if this pathway would still be supported in a younger sample of children or older sample of adults; therefore this is an area for future research. Third, the data in this study were assessed by use of self-report questionnaires only, which introduces the possibility of a reporter- and single method-bias. In addition, this study only utilized data from a single informant, which also introduces a single-subject bias. Self-report measures of stressors can also be problematic when considering the accuracy of the dating of events, which could influence report of variables at other time-points. The use of other assessment procedures (e.g. interviews, multiple informants) in addition to self-report data would allow for a more thorough and statistically sound investigation of the present study's research questions. Last, although using the ANX sub-scale of the MASQ as our measure of anxious symptoms minimized the overlap in negative affect between anxious and depressive symptoms, the findings from this study are specific to anxious arousal symptoms only and may not generalize to different anxiety facets (e.g. general distress/GAD, social anxiety, etc.). Anxiety is hierarchical (Lahey et al., 2004, and it is possible to measure other aspects of anxious symptoms that may generalize more widely to anxiety (Silverman & Ollendick, 2005).

Summary and Clinical Implications

Our study highlights the importance of clinically assessing other factors when evaluating the role that temperament, more specifically NE, plays on increases in depressive symptoms in youth. In adolescents with heightened negative emotionality, it may be useful to assess the stressors an individual has recently experienced with the effort to better understand what type of initial reaction there may be to these stressors. Results from this study suggest that it is likely that stressors and the resulting initial anxious arousal from these stressors may be heightened beyond what may be expected in an individual without a more negative temperament. If, as our data from the NE-Stressors pathway illustrate, the presence of greater stressors is followed by elevations in anxious arousal, then targeting this initial arousal clinically via interventions (e.g., relaxation, mindfulness) may have an ameliorative effect on reducing later depressive symptoms (Ma & Teasdale, 2004; Teasedale et al., 2000). In addition, because this study did not support differences due to gender and age in the NE-

stressors pathway, interventions aimed to target these symptoms may be useful with boys and girls, as well as older and younger adolescents.

In summary, this study establishes support for the unfolding of specific factors that lead to the onset of depressive symptoms in adolescence. The data supported the NE-stressors model, in which initial NE led to increased experienced stressors, greater experienced stressors in turn led to elevated anxious arousal symptoms, and this culminated in greater depressive symptoms in adolescence using a multi-wave longitudinal design. This developmental pathway was the same for both girls and boys, and younger and older adolescents. In addition, this study points toward the importance of understanding how specific factors link together in developmental pathways toward increased depressive symptoms in adolescence.

Acknowledgments

This work was supported, in part, by NIMH grants R03-MH 066845 and R01-MH 077195 to Benjamin L. Hankin.

References

Abela, JR.; Hankin, BL. Depression in children and adolescents: Causes, treatment, and prevention. In: Abella, JR.; Hankin, BL., editors. Handbook of Depression and Children and Adolescents. New York: London Press; 2008. p. 6-32.

Angold A, Costello EJ, Erkanli A. Comorbidity. Journal of Child Psychology and Psychiatry. 1999; 40:57–87. [PubMed: 10102726]

Arbuckle, JL. Amos 16.0 Users Guide. Spring House, PA: Amos Development Corporation; 2007.

Arnette JJ. Adolescent storm and stress, reconsidered. American Psychologist. 1999; 54:317–326. [PubMed: 10354802]

Avenevoli, S.; Knight, E.; Kessler, RC.; Merikangas, KR. Epidemiology of depression in children and adolescents. In: Abella, JR.; Hankin, BL., editors. Handbook of Depression and Children and Adolescents. New York: London Press; 2008. p. 6-32.

Avenevoli S, Stolar M, Li J, Dierker L, Merikangas KR. Comorbidity of depression in children and adolescents: Models and evidence from a prospective high-risk family study. Biological Psychiatry. 2001; 49:1071–1801. [PubMed: 11430849]

Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. Journal of Personality and Social Psychology. 1986; 51:1173–1182. [PubMed: 3806354]

Beesdo K, Bittner A, Pine DS, Stein MB, Dipl-Stat MH, Lieb R, Wittchen H. Incidence of social anxiety disorder and the consistent risk for secondary depression in the first three decades of life. Archives of General Psychiatry. 2007; 64:903–912. [PubMed: 17679635]

Bolger N, Zuckerman A. A framework for studying personality in the stress process. Journal of Personality and Social Psychology. 1995; 69:890–902. [PubMed: 7473036]

Carlson GA, Kashani JH. Phenomenology of major depression from childhood through adulthood: Analysis of three studies. The American Journal of Psychiatry. 1988; 145:1222–1225. [PubMed: 3421342]

Clark LA. Temperament as a unifying basis for personality and psychopathology. Journal of Abnormal Psychology. 2005; 114:505–521. [PubMed: 16351374]

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

Cole DA, Maxwell SE. Testing mediational models with longitudinal data: Questions and tips in the use of structural equation modeling. Journal of Abnormal Psychology. 2003; 112:558–577. [PubMed: 14674869]

Cole DA, Peeke LG, Martin JM, Truglio R, Seroczynski AD. A longitudinal look at the relation between depression and anxiety in children and adolescents. Journal of Consulting and Clinical Psychology. 1998; 66:451–460. [PubMed: 9642883]

Compas BE, Connor-Smith J, Jaser SS. Temperament, stress reactivity, and coping: Implications for depression in childhood and adolescence. Journal of Clinical Child and Adolescent Psychology. 2004; 33:21–31. [PubMed: 15028538]

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

Dougherty LR, Klien DN, Durbin E, Hayden EP, Olino TM. Temperamental positive and negative emotionality and children's depressive symptoms: A longitudinal prospective study from age three to age ten. Journal of Social and Clinical Psychology. 2010; 29:462–488.

Eley TC, Stevenson J. Specific life events and chronic experiences differentially associated with depression and anxiety in young twins. Journal of Abnormal Child Psychology. 2000; 28:383–394. [PubMed: 10949962]

Espejo EP, Hammen CL, Connolly NP, Brennan PA, Najman JM, Bor W. Stress sensitization and adolescent depressive severity as a function of childhood adversity: A link to anxiety disorders. Journal of Abnormal Child Psycology. 2006; 35:287–299.

Farmer A, Redman K, Harris T, Mahmood A, Sadler S, Pickering A, McGiffin P. Neuroticism, extraversion, life events and depression. British Journal of Psychiatry. 2002; 181:118–122. [PubMed: 12151281]

Farmer RF, Seeley JR. Temperament and character predictors of depressed mood over a 4-year interval. Depression and Anxiety. 2009; 26:371–381. [PubMed: 19123455]

Grant KE, Compas BE, Stuhlmacher AF, Thurm AE, McMahon SD, Halpert JA. Stressors and child and adolescent psychopathology: Moving from markers to mechanisms of risk. Psychological Bulletin. 2003; 129:447–466. [PubMed: 12784938]

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

Hankin BL. Cognitive vulnerability-stress model of depression during adolescence: Investigating depressive symptom specificity in a multi-wave prospective study. Journal of Abnormal Child Psychology. 2008a; 36:999–1014. [PubMed: 18437551]

Hankin BL. Rumination and depression in adolescence: Investigating symptom specificity in a multiwave prospective study. Journal of Child and Adolescent Clinical Psychology. 2008b; 37:701–713.

Hankin BL. Personality and depressive symptoms: Stress generation and cognitive vulnerabilities to depression in a prospective daily diary study. Journal of Social and Clinical Psychology. 2010; 29:369–401.

Hankin BL, Abramson LY. Development of gender differences in depression: Description and possible explanations. Annals of Medicine. 1999; 31:372–379. [PubMed: 10680851]

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

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

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

Hankin, BL.; Wetter, E.; Cheeley, C. Sex differences in child and adolescent depression: A developmental psychopathological approach. In: Abella, JR.; Hankin, BL., editors. Handbook of Depression and Children and Adolescents. New York: London Press; 2008. p. 6-32.

Harkness KL, Luther J. Clinical risk factors for the generation of life events in major depression. Journal of Abnormal Psychology. 2001; 110:564–572. [PubMed: 11727946]

Hettema JM, Kuhn JW, Prescott CA, Kendler KS. The impact of generalized anxiety disorder and stressful life events on risk for major depressive episodes. Psychological Medicine. 2006; 36:789–795. [PubMed: 16515735]

Joiner TE Jr, Wingate LR, Gencoz T, Gencoz F. Stress generation in depression: Three studies on its resilience, possible mechanism, and symptom specificity. Journal of Social and Clinical Psychology. 2005; 24:236–253.

Kazdin AE, French NH, Unis AS. Child, mother, and father evaluation of depression in psychiatric inpatient children. Journal of Abnormal Child Psychology. 1983; 11:167–180. [PubMed: 6619431]

Keenan K, Feng X, Hipwell A, Klostermann S. Depression begets depression: Comparing the predictive utility of depression and anxiety symptoms to later depression. Journal of Child Psychology and Psychiatry. 2009; 50:1167–1175. [PubMed: 19344385]

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

Kendler KS, Gardner CO, Prescott CA. Personality and the experience of environmental adversity. Psychological Medicine. 2003; 33:1193–1202. [PubMed: 14580074]

Kendler KS, Kuhn J, Prescott CA. The interrelationship of neuroticism, sex and stressful life events in the prediction of episodes of major depression. The American Journal of Psychiatry. 2004; 161:631–636. [PubMed: 15056508]

Kercher AJ, Rapee RM, Schniering CA. Neuroticism, life events and negative thoughts in the development of depression in adolescent girls. Journal of Abnormal Child Psychology. 2009; 37:903–915. [PubMed: 19437113]

Kessler RC, Gruber M, Hettema JM, Hwang I, Sampson N, Yonkers KA. Co-morbid major depression and generalized anxiety disorders in the National Comorbidity Survey follow-up. Psychological Medicine. 2008; 38:365–374. [PubMed: 18047766]

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

Kovacs, M. Children's Depression Inventory (CDI). New York: Multi-health Systems, Inc; 1992.

Krueger RF. Personality traits in late adolescence predict mental disorders in early adulthood: A prospective-epidemiological study. Journal of Personality. 1999; 67:39–65. [PubMed: 10030020]

Krueger RF, Tackett JL. Personality and psychopathology: Working toward the bigger picture. Journal of Personality Disorders. 2003; 17:109–128. [PubMed: 12755325]

Lahey BB. Public health significance of neuroticism. American Psychologist. 2009; 64:241–256. [PubMed: 19449983]

Lahey BB, Applegate B, Waldman ID, Loft JD, Hankin BL, Rick J. The structure of child and adolescent psychopathology: Generating new hypothses. Journal of Abnormal Psychology. 2004; 113:358–385. [PubMed: 15311983]

Lakdawalla Z, Hankin BL. Personality as a prospective vulnerability to dysphoric symptoms among college students: Proposed mechanisms. Journal of Psychopathology and Behavioral Assessment. 2008; 30:121–131.

Landis D, Gaylord-Hayden NK, Malinowski SL, Grant KE, Carleton RA, Ford RE. Urban adolescent stress and hopelessness. Journal of Adolescence. 2007; 30:1051–1070. [PubMed: 17467052]

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

Lonigan CJ, Vasey MW, Phillips BM, Hazen RA. Temperament, anxiety, and the processing of threat-relevant stimuli. Journal of Clinical Child and Adolescent Psychology. 2004; 33:8–20. [PubMed: 15028537]

Ma SH, Teasdale JD. Mindfulness-based cognitive therapy for depression: Replication and exploration of differential relapse prevention effects. Journal of Consulting and Clinicial Psychology. 2004; 72:31–40.

MacKinnon, DP. Introduction to statistical mediation analysis. Mahwah, NJ: Erlbaum; 2008.

MacKinnon DP, Lockwood CM, Williams J. Confidence limits for the indirect effect: Distribution of the product and resampling methods. Multivariate Behavioral Research. 2004; 39:99–128. [PubMed: 20157642]

Magnus K, Diener E, Fujita F, Pavot W. Extraversion and neuroticism as predictors of objective life events: A longitudinal analysis. Journal of Personality and Social Psychology. 1993; 65:1046–1053. [PubMed: 8246112]

Mak AS, Blewitt K, Heaven PCL. Gender and personality influences in adolescent threat and challenge appraisals and depressive symptoms. Personality and Individual Differences. 2004; 36:1483–1496.

Nolen-Hoeksema S, Girgus JS. The emergence of gender differences in depression during adolescence. Psychological Bulletin. 1994; 115:424–443. [PubMed: 8016286]

Ormel J, Wohlfarth T. How neuroticism, long-term difficulties, and life situation change influence psychological distress: A longitudinal model. Journal of Personality and Social Psychology. 1991; 60:744–755. [PubMed: 2072254]

Petersen AC, Compas BE, Brooks-Gunn J, Stemmler M, Ey S, Grant KE. Depression in adolescence. American Psychologist. 1993; 48:155–168. [PubMed: 8442570]

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

Riskind JH, Black D, Shahar G. Cognitive vulnerability to anxiety in the stress generation process: Interaction between the looming cognitive style and anxiety sensitivity. Journal of Anxiety Disorders. 2010; 24:124–128. [PubMed: 19854612]

Rothbart MK. Commentary: Differentiated measures of temperament and multiple pathways to childhood disorders. Journal of Clinical Child and Adolescent Psychology. 2004; 33:82–87. [PubMed: 15028543]

Rothbart MK, Ahadi SA, Hershey K, Fisher P. Investigations of temperament at 3 to 7 years: The children's behavior questionnaire. Child Development. 2001; 72:1394–1408. [PubMed: 11699677]

Rudolph KD. Gender differences in emotional responses to interpersonal stress during adolescence. Journal of Adolescent Health. 2002; 30S:3–13. [PubMed: 11943569]

Shrout PE, Bolger N. Mediation in experimental and nonexperimental studies: New procedures and recommendations. Psychological Methods. 2002; 7:422–445. [PubMed: 12530702]

Silverman WK, Ollendick TH. Evidence-based assessment of anxiety and its disorders in children and adolescents. Journal of Clinical Child and Adolescent Psychology. 2005; 34:380–411. [PubMed: 16026211]

Sobel, ME. Asymptotic confidence intervals for indirect effects in structural equation models. In: Leinhardt, S., editor. Sociological methodology. San Francisco: Jossey-Bass; 1982. p. 290-312.

Steer RA, Clark DA, Kumar G, Beck A. Common and specific dimensions of self-reported anxiety and depression in adolescent outpatients. Journal of Psychopathology and Behavioral Assessment. 2008; 30:163–170.

Taylor AB, MacKinnon DP, Tein J. Tests of the three-path mediated effect. Organizational Research Methods. 2008; 11:241–269.

Teasdale JD, Segal ZV, Williams JMG, Ridgeway VA, Soulsby JM, Lau MA. Prevention of relapse/recurrence in major depression by mindfulness-based cognitive therapy. Journal of Consulting and Clinical Psychology. 2000; 64:615–623. [PubMed: 10965637]

Uhrlass DJ, Gibb BE. Childhood emotional maltreatment and the stress generation model of depression. Journal of Social & Clinical Psychology. 2007; 26:119–130.

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

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

Weiss B, Garber J. Developmental differences in the phenomenology of depression. Development and Psychopathology. 2003; 15:403–430. [PubMed: 12931835]

Wetter EK, Hankin BL. Mediational pathways through which positive and negative emotionality contribute to anhedonic symptoms of depression: A prospective study of adolescents. Journal of Abnormal Child Psychology. 2009; 37:507–520. [PubMed: 19184402]

Wills TA, Windle M, Cleary SD. Temperament and novelty seeking in adolescent substance use: Convergence of dimensions of temperament with constructs from Cloninger's theory. Journal of Personality and Social Psychology. 1998; 74:387–406. [PubMed: 9491584]

Yang H, Chiu Y, Soong W, Chen WJ. The roles of personality traits and negative life events on the episodes of depressive symptoms in nonreferred adolescents: A 1-year follow-up study. Journal of Adolescent Health. 2008; 42:378–385. [PubMed: 18346663]

Zahn-Waxler, C. The development of empathy, guild and internalization of distress: Implications for gender differences in internalizing and externalizing problems. In: Davidson, R., editor. Anxiety, depression, and emotion. New York: Oxford University Press; 2000. p. 222-265.

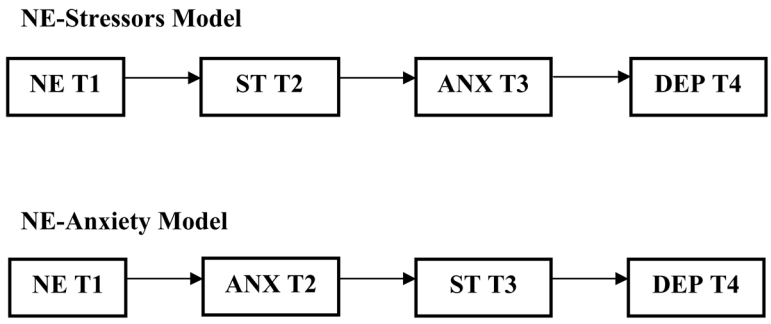

Figure 1. Hypothesized Models

NE-stressors model: Primary model that higher level of NE predicts greater amounts of stressors, which then generates increases anxious symptoms, and this increase in anxious symptoms leads to increases in depressive symptoms. NE-anxiety model: Alternate model that NE leads to increases in anxious symptoms which leads to higher levels of reported stressors, and this increase in stressors leads to increases in depressive symptoms.

Note: NE = Negative Emotionality; ST = Stressors; ANX = Anxious Symptoms; DEP = Depressive Symptoms

Note: NE = Negative Emotionality; ST = Stressors; ANX = Anxious Symptoms; DEP = Depressive Symptoms; T1 = Time 1; T2 = Time 2; T3 = Time 3; T4 = Time 4

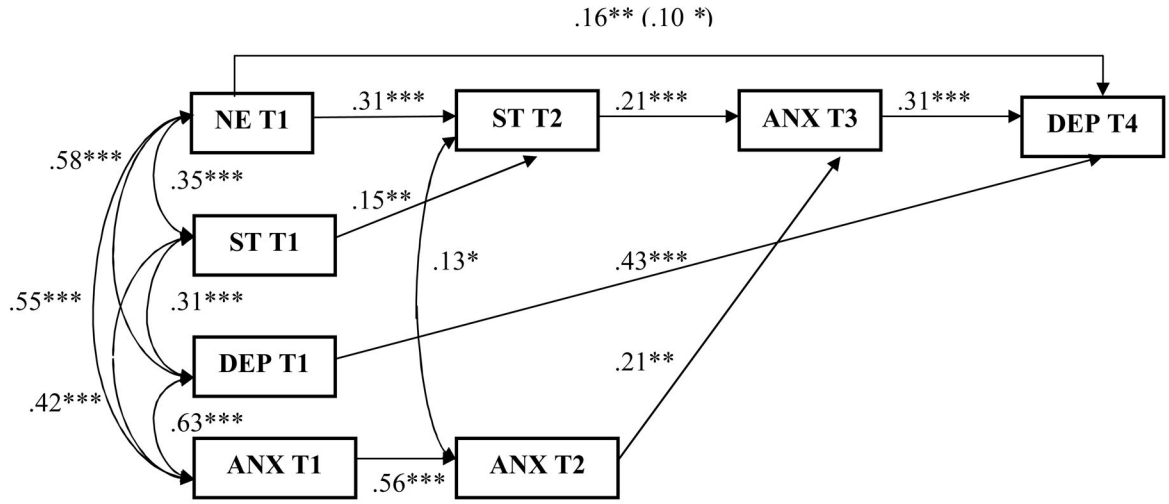

Figure 2. NE-Stressors Model

NE-stressors sequential mediation: Sequential mediation model of stressors and anxious symptoms mediating the association between NE and depressive symptoms.

* $p < .01$, *** $p < .001$

Note: Paths from NE T1 to ANX T3 ($\beta = .28$, $p < .001$) and from ST T2 to DEP T4 ($\beta = .08$, $p < .05$) were included in the model, but are not shown for clarity of presentation. NE = Negative Emotionality; ST = Stressors; ANX = Anxious Symptoms; DEP = Depressive Symptoms; T1 = Time 1; T2 = Time 2; T3 = Time 3; T4 = Time 4

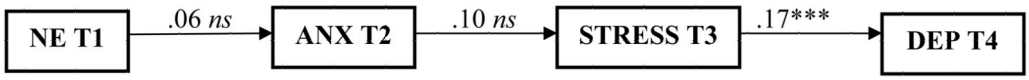

Figure 3. NE-Anxiety Model

Direct effects in the NE-anxiety model.

*** p <.001

Note: NE = Negative Emotionality; ST = Stressors; ANX = Anxious Symptoms; DEP = Depressive Symptoms; T1 = Time 1; T2 = Time 2; T3 = Time 3; T4 = Time 4

Table 1

Means, Standard Deviations and Correlations

	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*
1. NE T1									
2. ST T1	.36***								
3. ST T2	.36***	.28***							
4. ST T3	.25***	.33***	.31***						
5. ANX T1	.56***	.43***	.42***	.35***					
6. ANX T2	.45***	.28***	.60***	.34***	.56***				
7. ANX T3	.45***	.27***	.44***	.56***	.58***	.57***			
8. DEP T1	.59***	.34***	.44***	.39***	.63***	.62***	.58***		
9. DEP T4	.52***	.40***	.46***	.45***	.56***	.53***	.64***	.70***	
Overall									
M	2.26	1.86	1.76	1.72	2.20	2.20	2.21	12.81	15.22
SD	0.52	0.81	0.62	0.60	.75	0.73	0.72	8.64	12.77
Girls									
M	2.34	1.93	1.90	1.78	2.29	2.25	2.31	13.03	18.34
SD	0.54	0.84	0.59	0.60	0.82	0.78	0.81	8.82	15.00
Boys									
M	2.17	1.77	1.58	1.17	2.10	2.15	2.09	12.54	11.47
SD	0.49	0.75	0.67	0.59	0.64	0.65	0.58	8.43	7.97
Age 11–14									
M	2.22	1.83	1.61	1.63	2.07	1.91	1.93	11.22	12.68
SD	0.57	0.62	0.69	0.51	0.79	0.73	0.70	8.38	12.37
Age 15–17									
M	2.29	1.87	1.84	1.78	2.28	2.37	1.78	13.70	16.65
SD	0.50	0.89	0.57	0.63	0.72	0.67	0.63	8.68	12.80

Note. NE = Negative Emotionality; ST = Stressors; ANX = Anxious Symptoms; DEP = Depressive Symptoms; T1 = Time 1; T2 = Time 2; T3 = Time 3; T4 = Time 4

*** $p < .001$

J Abnorm Child Psychol. Author manuscript; available in PMC 2014 May 22.