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Affective, Biological, and Cognitive Predictors of Depressive Symptom Trajectories in Adolescence

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Abstract

Heterogeneity in the longitudinal course of depressive symptoms was examined using latent growth mixture modeling among a community sample of 382 U.S. youth from ages 11 to 18 (52.1% female). Three latent trajectory classes were identified: Stable Low (51%; displayed low depressive symptoms at all assessments), Increasing (37%; reported low depressive symptoms at age 11, but then significantly higher depressive symptoms than the Stable Low class at ages 13, 15, and 18), and Early High (12%; reported high early depressive symptoms at age 11, followed by symptoms that declined over time yet remained significantly higher than those of the Stable Low class at ages 13, 15, and 18). By age 15, rates of Major Depressive Disorder diagnoses among the Early High (25.0%) and Increasing (20.4%) classes were more than twice that observed among the Stable Low class (8.8%). Affective (negative affectivity), biological (pubertal timing, sex) and cognitive (cognitive style, rumination) factors were examined as predictors of class membership. Results indicated general risk factors for both high-risk trajectories as well as specific risk factors unique to each trajectory. Being female and high infant negative affectivity predicted membership in the Increasing class. Early puberty, high infant negative affectivity for boys, and high rumination for girls predicted membership in the Early High class. Results highlight the importance of examining heterogeneity in depression trajectories in adolescence as well as simultaneously considering risk factors across multiple domains.

Keywords

DEPRESSION; TRAJECTORIES; TEMPERAMENT; PUBERTY; COGNITIVE RISK FACTORS; ADOLESCENCE

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Cross-sectional and longitudinal studies have found that prevalence rates of depressive disorders rise from 2–4% in childhood to nearly 20% by age 18 (e.g., Cohen et al., 1993; Kessler, Avenevoli, & Merikangas, 2001). Adolescent-onset depression is associated with social impairment, recurrent depression in adulthood, and greater risk for comorbid mental health problems including substance use (e.g., Zisook et al., 2007). One important indicator of risk for depressive disorders is depressive symptoms. Depressive symptoms are both normative in adolescence and predictive of more severe symptoms and eventual depressive disorders over time (Fergusson, Horwood, Ridder, & Beautrais, 2005; Garber, Keiley, & Martin, 2002; Pine, Cohen, Cohen, & Brook, 1999). While sample-wide analyses have clearly identified that, on average, depressive symptoms increase across adolescence, such analyses may mask important heterogeneity in the course of depressive symptoms. In addition, identifying risk factors that place youth on a high-risk trajectory is critical for understanding the onset, course, and prevention of depression. In a recent review, Hankin (2012) noted “Despite considerable progress in distinct lines of vulnerability research, there is an explanatory gap in our ability to more comprehensively explain and predict who is likely to become depressed, when and why.” (p. 695).

The purpose of the current study was to fill this gap. Our first purpose was to examine whether there are subgroups of adolescents who follow high-risk trajectories of depressive symptoms and, if so, identify the age(s) at which these youth diverge from a normal or low-risk trajectory and describe their subsequent risk for depression diagnoses. In addition, we employed a multiple levels of analysis approach to identifying predictors of adolescent depressive symptom trajectories, testing affective, biological, and cognitive factors to predict membership in distinct depressive symptom trajectories in adolescence.

Heterogeneity of Depressive Symptom Trajectories Across Adolescence

Most studies find that depressive symptom levels are lowest in the late childhood/early adolescent period up to and including age 11, then display an increasing trend starting at around age 13, with a period of rapid increase occurring between ages 15 and 18 (e.g., Garber et al., 2002; Hankin et al., 1998). After age 18, rates of depressive symptoms in community samples tend to level off and remain relatively stable throughout most of adulthood (Hankin et al., 1998; Yaroslavsky, Pettit, Lewinsohn, Seeley, & Roberts, 2012). However, sample-wide analyses of average depressive symptoms mask important individual differences in depressive symptom trajectories, and there is evidence of both continuity and change in depressive symptoms in adolescence. The majority of adolescents in community samples display consistently low depressive symptoms across late childhood and adolescence (e.g. Costello, Swendsen, Rose, & Dierker, 2008; Frye & Liem, 2011; Reinke, Eddy, Dishion, & Reid, 2012; Sterba, Prinstein, & Cox, 2007). Most trajectory analyses also find evidence for a sizable minority who display a pattern of low depressive symptoms initially, which increase dramatically over time (Brendgen, Wanner, Morin, & Vitaro, 2005; Costello et al., 2008; Dekker et al., 2007; Frye & Liem, 2011; Reinke et al., 2012). At least three studies have also found that a sizable minority of youth display high depressive symptoms initially, which decline over time (Costello et al., 2008; Frye & Liem, 2011; Reinke et al., 2012). Finally, some studies find a small group of youth with consistently high depressive symptoms (Brendgen et al., 2005; Frye & Liem, 2011; Sterba et al., 2007; Reinke et al., 2012; Rodriguez, Moss, & Audrain-McGovern, 2005).

Prior studies have demonstrated specific and general risk factors in predicting adolescent depression trajectories. Brendgen et al. (2005) reported that girls with a highly reactive temperament who experienced rejection by same-sex peers were more likely to follow the increasing trajectory of depressive symptoms. Some risk factors exert general influences on high-risk depressive symptom profiles (e.g., increasing profile, stably high profile),

including being female (Brendgen et al., 2005; Costello et al., 2008; Frye & Liem, 2011), trauma history (Frye & Liem, 2011), and postpartum maternal depression (Sterba et al., 2007).

Although the extant literature on latent classes of depressive symptoms and associated risk factors has contributed substantial knowledge, there are limitations to many prior depression trajectory analyses. Some are limited in age range, sampling youth either prior to the adolescent transition (e.g. Sterba et al., 2007) or after the adolescent transition (e.g. Frye & Liem, 2011). Others rely upon limited measures of depressive symptoms (e.g. Costello et al., 2008). Some examine only one gender (e.g. Stoolmiller, Kim, & Capaldi, 2005). Few have compared symptom trajectories with depression diagnoses, which is important for understanding the relation of symptoms to clinically significant psychopathology. The majority of studies have considered gender and stress as predictors of depression trajectories, with only a handful examining other risk factors. The current study followed youth from early to late adolescence (ages 11 to 18) using a well-validated measure of depressive symptoms, compared symptom trajectories with depression diagnoses, and examined a wide variety of theory-driven risk factors.

Potential Risk Factors for Distinct Depressive Symptom Trajectories

Numerous theories for adolescent depression have been proposed, covering a range of risk factors including genetics, pubertal hormones and timing, coping styles, emotional reactivity, negative cognitions, interpersonal relationships, and stress exposure. The ABC model of adolescent depression offers an integrated, developmentally sensitive model of how multiple factors (affective, biological, and cognitive) may confer risk for depression in adolescence (Hyde, Mezulis, & Abramson, 2008). Using the ABC model as a theoretical framework, the current study examined multiple potential risk factors across developmental domains that may explain individual differences in depressive symptom trajectories. Given the salience of the adolescent period for divergence of symptom trajectories, particular emphasis was given to identifying childhood risk factors premorbid to the onset of depressive problems.

Affective risk for depression

Affective models of depression suggest that individual differences in emotional reactivity represent an early temperamental risk factor for depressive disorders (Compas, Connor-Smith, & Jaser, 2004). A constellation composed of high negative affect, high reactivity, high intensity of emotional reactions, low adaptability, and low approach is typically labeled “negative affectivity”. Extensive research links high childhood negative affectivity with depressive symptoms and disorders in adolescence (Compas et al., 2004; Goodyer, Ashby, Altham, Vize, & Cooper, 1993). Given the relative stability of negative affectivity over time and conceptual overlap between negative affectivity and depressive symptoms, it is important to consider the extent to which childhood negative affectivity predicts change in depressive symptoms, particularly increases in symptoms, as opposed to simply being associated with continuity of depressive symptoms. Examining childhood negative affectivity as a predictor of divergent depressive symptom trajectories may clarify the role of negative affectivity as a premorbid risk factor for adolescent-onset depression.

Biological risk for depression

Pubertal timing (early, on time, or late) is crucial to understanding the emergence of depression in adolescence. Early puberty may interfere with the child’s ability to complete normative developmental tasks before being faced with the sociocultural demands of adulthood that accompany pubertal development, and may be accompanied by changes in

body image that make youth more susceptible to peer stressors (Ge & Natsuaki, 2009). Early puberty is associated with depressive symptoms among both boys and girls (Ge, Conger, & Elder, 2001; Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997; Kaltiala-Heino, Kosunen, & Rimpela, 2003).

Cognitive risk for depression

Cognitive models of depression suggest that individuals' characteristic cognitive responses to stress or depressed mood may confer vulnerability to depression. Two of the most empirically supported cognitive vulnerabilities to depression are negative cognitive style and rumination. The hopelessness theory of depression (Abramson, Metalsky, & Alloy, 1989) defines negative cognitive style as the trait tendency to make negative inferences about causes, consequences, and self-characteristics of stressful events, and hypothesizes that those who encounter stressful events and exhibit this negative cognitive style are at elevated risk for depression. Strong prospective support for negative cognitive style as a vulnerability factor for depression has been shown among adolescents and adults (e.g., Abela et al., 2011; Alloy et al., 2006). A second cognitive vulnerability factor for depression is a ruminative response style. Nolen-Hoeksema (1991) described rumination as "focusing on depressive symptoms and the possible causes and consequences of those symptoms" (p. 569). Prospective studies have shown that individuals who ruminate about their negative emotions are at increased risk for developing depressive disorders (e.g., Abela & Hankin, 2011; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008).

Several researchers have hypothesized that cognitive vulnerability to depression may be consolidating and stabilizing in early-to-middle adolescence (e.g., Cole et al., 2008; Mezulis, Hyde, & Abramson, 2006). The consolidation of cognitive vulnerability at this time is consistent with the timing of the rise in depressive symptoms. Thus, the transition from late childhood into adolescence may be an important developmental period in which cognitive vulnerability to depression exerts its influence on subsequent depression trajectories.

The Current Study

In the current study, we used data from the longitudinal Wisconsin Study of Families and Work (Hyde, Klein, Essex, & Clark, 1995). Our first aim was to identify heterogeneity in trajectories of depressive symptoms from age 11 to 18 using latent growth mixture modeling. The second aim was to associate distinct symptom trajectories with depression diagnoses. Our third aim was to examine theory-driven predictors of depression symptom trajectories. We examined affective (infant temperament), biological (pubertal timing), and cognitive (cognitive style, rumination) predictors of trajectory group membership. We further tested whether the effects of these risk factors varied as a function of biological sex.

Based on prior trajectory analyses, we expected to find a distinct group of youth with stable low symptoms; a group of youth with increasing symptoms; and a group of youth with symptoms that are high at the start of the study which remain stable and/or decline. Since the risk factors examined here have all been associated with adolescent depression, we anticipated that negative affectivity, rumination, cognitive style, early pubertal timing, and being female would be associated with being on one or more of the high risk trajectories relative to a normative, stable low group. However, we had no a priori hypotheses regarding which risk factors, if any, would differentially predict one high risk trajectory over another.

Methods

Participants

Participants were 382 youth (52.1% female) who have participated in a longitudinal study of child development since birth. A total of 570 mothers were recruited during pregnancy for participation in the Wisconsin Study of Families and Work (formerly named the Wisconsin Maternity Leave and Health Project; Hyde et al., 1995). Data were collected at age 1 ($N = 480$) and during the summer following Grades 5 ($N = 306$; mean age = 11.5, $SD = .32$), 7 ($N = 372$; $M = 13.5$, $SD = .33$), 9 ($N = 337$; $M = 15.5$, $SD = .33$), and 12 ($N = 324$; $M = 18.5$, $SD = .33$). Every effort was made to retain all participants across the study from birth through the age 18 assessment. For the present study, 382 of the original 570 participants (67%) were still participating at the time of the adolescent assessments. Of these 382 participants, 219 youth (57%) participated in all four adolescent assessments, 105 (27%) participated in three adolescent assessments, and 58 (15%) participated in two adolescent assessments. Of these, 93% were White, 3% African American, 2% American Indian, 1% Asian/Pacific Islander, and 1% Hispanic. Independent samples t -tests were used to compare the 382 participants included in the analyses sample with the 188 participants from the original recruitment sample no longer participating by adolescence on the following variables assessed in infancy: family income; maternal and paternal education; and maternal and paternal depressive symptoms. The 188 participants from the original recruitment sample no longer participating by adolescence had significantly lower paternal education at the start of the study ($t = 2.35$, $p < .05$) than the 382 participants included in the current study; there were no significant differences on maternal education; family income; maternal depressive symptoms; or paternal depressive symptoms (all p values $> .10$).

Procedure

Mothers were enrolled in the study when pregnant with the participating child. The present study utilizes data from the 12-month assessment (mother report of infant temperament) but otherwise focuses on the pre-adolescent and adolescent assessments at ages 11, 13, 15, and 18. When participants were 12 months old, their mothers completed a questionnaire assessing infant temperament. At ages 11, 13, 15, and 18, participants completed a number of questionnaires administered on a laptop computer during in-home visits. Participants who had moved out of the area completed paper questionnaires by mail. Diagnostic interviews were conducted in person or by phone at the age 15 assessment. The study was approved by the University of Wisconsin Institutional Review Board. Parents provided consent and children provided assent for their participation until age 18, when participants provided consent. At each wave of data collection participants received monetary compensation.

Measures

Depressive symptoms—Depression symptoms were assessed at ages 11, 13, 15, and 18 with the Children's Depression Inventory (CDI; Kovacs, 1981), a 27-item self-report scale designed for use with children between ages 8 and 17. For each item, participants identified one of three statements that best described themselves in the previous 2 weeks (e.g., "I thought about bad things happening to me"). In the current study, given that assessments were conducted in summer, we omitted three items that referenced school. Participants' scores on the remaining 24 items were averaged and then multiplied by 27 to create a total score that is comparable to the complete 27-item CDI. The CDI has been widely used in depression research (Sitarenios & Stein, 2004) and has demonstrated good internal consistency and test-retest reliability (Saylor, Finich, Spirito, & Bennett, 1984; Smucker, Craighead, Craighead, & Green, 1986). Internal consistencies were .79 at age 11, .83 at age 13, and .86 at ages 15 and 18.

Depression diagnose—Trained graduate students conducted diagnostic interviews using the Kiddie-Schedule for Affective Disorders and Schizophrenia (K-SADS; Orvaschel, 1995) when participants were 15. The K-SADS is a semi-structured diagnostic interview administered to a child or adolescent (ages 6–18) and his or her primary caregiver. The interview provides DSM-IV diagnoses of a wide range of psychiatric disorders. Training in administration of the K-SADS was provided by Dr. Helen Orvaschel of Nova Southeastern University, FL, for the eight diagnostic interviewers. To ensure the reliability of the diagnostic interviews, the first 20 interviews were reviewed by Dr. Orvaschel and any discrepancies were resolved by consensus. After that point, all interviewers participated in a weekly group meeting with audiotaped interviews being periodically reviewed by Dr. Orvaschel for accuracy and validity. Any interviews that raised diagnostic issues or ambiguities were brought to the group for a consensus decision. These meetings were supervised by a senior faculty member.

Diagnostic interviewers first interviewed mothers about their child's symptoms, and then interviewed the adolescents. Interviews covered lifetime history of psychopathology, including unipolar depression diagnoses (major depressive disorder; dysthymia; depressive disorder NOS; and adjustment disorder with depressed mood). The diagnostic interviewers scored the interview according to the K-SADS manual, using both parent and child information.

Negative affectivity—Infant negative affectivity was assessed with the withdrawal negativity subscale of the Infant Behavior Questionnaire (IBQ; Rothbart, 1981). Mothers completed the parent-report questionnaire when the children were 12 months old. The withdrawal negativity subscale consists of 21 items measuring distress to novelty/fear and startle (e.g., "How often did your baby fuss, cry, or show distress while waiting for food?"). Mothers reported on each item across the prior 2-week time period using a 7-point Likert scale ranging from 1 (*never*) to 7 (*always*). Items were averaged across the respective subscale (distress to novelty and startle) and then these scale scores were averaged to compute overall withdrawal negativity. Internal consistency of the withdrawal negativity subscale was .76.

Rumination—Depressive rumination was assessed at age 11 using a short form of the Ruminative Response Scale (RRS) of the Response Style Questionnaire (RSQ; Nolen-Hoeksema & Morrow, 1991). In the original 22-item RRS, participants indicate how frequently they engage in ruminative responses when they feel sad, down, or low, on a scale from 1 = *almost never* to 4 = *almost always*. Based upon consultation with Nolen-Hoeksema at the time of the study design (personal communication, 2001), our 5-item form of the RRS included rumination items that emphasized rumination about sad, depressed, or down affect (e.g., "When I feel sad or down, I think about how alone I feel"). The full RRS has been used with adolescents in several prior studies (e.g., Rood, Roelofs, Bögels, Nolen-Hoeksema, & Schouten, 2009). Internal consistency was .68.

Negative cognitive style—Negative cognitive style was assessed at age 11 with the Children's Cognitive Style Questionnaire (CCSQ; Mezulis et al., 2006). On a Likert scale ranging from 1 (*don't agree at all*) to 5 (*agree a lot*), participants indicate their agreement with statements regarding their attributions (1 item each for internality, stability, and globality), self-inferences (1 item), and anticipated consequences (1 item) for 4 hypothetical negative events. Responses to the 4 negative scenarios were averaged to compute three composite scores (negative attributional style, negative self-inference, and negative consequence), which were then averaged to create a negative cognitive style composite.

score. Higher scores on the CCSQ indicate more negative cognitive styles. Internal consistency was .86 at age 11.

Pubertal timing—Pubertal status was assessed at age 11 with line drawings of the 5 Tanner stages of pubertal status with instructions to identify the pictures that looked most like the participant's body (Marshall & Tanner, 1969, 1970). Tanner ratings based on line drawings are a widely used self-assessment of pubertal status and correlate adequately with physical examination (Dorn & Biro, 2011; physical examination was rejected as a method in this study because of its intrusiveness). Both boys and girls indicated their pubic hair development, while girls additionally indicated breast growth and boys indicated genital growth. The stages ranged from 1 (a picture of a pre-pubertal body) to 5 (a picture of a mature body). The two Tanner ratings were averaged to form a composite score for pubertal status (as is customary, Marshall & Tanner, 1969, 1970). Pubertal status was also assessed via age of menstruation, which we collected at age 15 via child self-report. Early Puberty was defined, for girls, as Tanner stage 3 or higher at age 11 and/or menstruation prior to age 11.5 years old. For boys, Early Puberty was defined as Tanner stage 3 or higher at age 11. We had 308 participants with data from which to assign a pubertal status (Tanner stage and/or age of menstruation data).¹ The variable was coded as 1 = Early Puberty (31.2%) and 0 = No Early Puberty (i.e., on-time or late; 68.8%).

Data-Analytic Plan

To identify heterogeneity in the patterns of depressive symptoms over time, we performed growth mixture modeling using Mplus 6.0 software (Muthén & Muthén, 1988–2009). All statistical analyses employed full information maximum likelihood (FIML) estimation with robust standard errors to account for the naturally skewed distribution of depressive symptoms. Mplus also offers state-of-the-art methods for handling missing values, which allowed all participants to be included in latent growth analyses regardless of whether they had completed all depressive symptom assessments. The number of latent trajectories was examined iteratively, starting with the null hypothesis of only one latent class and specifying an increasing number of classes. Evaluation of the output for each subsequent iteration included interpretability of the results, meaningfulness of the classes, and relevant model fit statistics (see Table 1). To examine depression diagnoses across latent growth trajectory classes, we employed chi-square tests of class by diagnosis frequency distributions using SPSS 19.0. Results are reported as likelihood ratios. Finally, we examined predictors of trajectory class membership using multinomial logistic regression in Mplus 6.0. Predictors were entered as centered, continuous variables for all variables except child sex and pubertal status, which were categorical predictors. Significant interactions were interpreted by examining each independent variable in the interaction at one standard deviation above and below the mean and then the distribution of class membership within each quadrant.

Results

Trajectories of Depressive Symptoms

Evaluation of the model statistics indicated that a three-class model provided the best fit to the data (see Table 1). Youth were placed into classes based upon most likely class membership statistics, and all subsequent analyses were based upon this class membership assignment. Average latent class probability for most likely class membership ranged from .83 to .98. We labeled the majority class (51% of the sample) the Stable Low class (see

¹Of the 382 participants, 224 participants completed the Tanner questions at age 11. Given a .78 correlation ($p < .001$) between child and mother Tanner report at age 11, we used the mothers' Tanner scores for 65 of the participants with missing data. We also used pubertal status data for 19 participants who did not have Tanner data, resulting in a total N of 308 for pubertal timing.

Figure 1). These adolescents had consistently very low depressive symptoms at all assessments. We labeled the next largest class (37% of the sample) the Increasing class. These youth displayed low depressive symptoms at the onset of adolescence (age 11), which consistently increased over time. Finally, we labeled the smallest class (12% of the sample) the Early High class. These youth started the study at age 11 with the highest depressive symptoms. Although their symptoms decreased over time, they remained significantly higher than the Stable Low class at every assessment. Correlations amongst study variables are shown in Table 2. Descriptive statistics and ANOVA comparisons are shown in Table 3.

Depression Diagnoses by Depression Trajectory

We examined prevalence rates of major depressive disorder (MDD) as well as other depression diagnoses (dysthymia; depressive disorder NOS; and adjustment disorder with depressed mood) by class, and statistically compared the Early High and Increasing classes to the Stable Low class. Given the high early depressive symptoms among the Early High class, we also considered whether rates of depression diagnoses varied if they were childhood-onset versus adolescent-onset. Results are shown in Table 4. Lifetime prevalence of MDD among youth in the Increasing class was more than twice that observed in the Stable Low class; prevalence of MDD among youth in the Early High class was nearly triple that of the Stable Low class. These descriptive analyses also suggest that, across groups, most youth first met criteria for MDD at age 12 or later. Thus, the depressive symptoms displayed at age 11 by youth in the Early High class do not appear to be simply a proxy for childhood-onset depression but rather a potential early indicator of risk for adolescent-onset depression.

Affective, Biological, and Cognitive Predictors of Depression Trajectories

Means, standard deviations, and frequencies of all predictors by class are shown in Table 5. We conducted a multinomial logistic regression to examine predictors of class membership. We modeled child sex, negative affectivity, pubertal timing, cognitive style, and rumination, as well as two-way interactions with sex. Main effects and interactions were interpreted at a significance level of $p < .05$. The Stable Low group served as the reference category. Regression results are presented in Table 6.

Membership in the Increasing class relative to the Stable Low class was predicted by sex and infant negative affectivity. There was a trend ($p = .06$) for cognitive style at age 11 to predict membership in the Increasing class as well. None of these main effects were moderated by sex.

Membership in the Early High class relative to the Stable Low class was predicted by early puberty and rumination at age 11. The main effect of rumination was moderated by sex. Examination of this interaction indicated that the effect of rumination on membership in the Early High class was strongest for girls. There was also evidence for a significant interaction between infant negative affectivity and sex. Examination of this interaction indicated that the effect of infant negative affectivity on membership in the Early High class was strongest for boys.

To better specify distinct predictors of high risk trajectories, we conducted a final multinomial logistic regression examining membership in the Early High class relative to membership in the Increasing class. This analysis confirmed that sex ($\beta/SE = -2.55, p = .01$), rumination ($\beta/SE = 3.25, p < .00$); and negative affectivity ($\beta/SE = -1.96, p = .05$) differentially predicted membership in the Early High class relative to the Increasing class, such that being female and being high in negative affectivity were associated with greater likelihood of being in the Increasing Class relative to the Early High class while being high

in rumination was associated with greater likelihood of being in the Early High class relative to the Increasing class. Although early puberty differentiated membership in the Early High class relative to the Stable Low class in the prior analysis, it did not significantly differentiate membership in the Early High class relative to the Increasing class ($\beta/SE = 1.23, p = .22$).

Discussion

The current study examined the development of depression in adolescence by examining heterogeneity in symptom trajectories across adolescence using a multi-wave design and self-report measures, parental-report measures, and diagnostic interviews.

Trajectories of Depressive Symptoms in Adolescence

We were particularly interested in identifying when and how high-risk youth diverge from their low-risk peers. Prior depression trajectory analyses have typically identified a large group of youth with stable low depressive symptoms, as well as smaller groups with increasing, decreasing, and/or stable high depressive symptoms (Brendgen et al., 2005; Costello et al., 2008; Dekker et al., 2007; Frye & Liem, 2011; Reinke et al., 2012; Sterba et al., 2007). Results from the current study were largely consistent with prior depression trajectory analyses in adolescence. The majority of youth (51%) displayed consistently low depressive symptoms at all assessments. We also identified two high-risk trajectories. First, just over one-third of youth (36%) displayed a pattern of increasing depressive symptoms. At age 11, youth in this Increasing class were indistinguishable from youth in the Stable Low class based on depressive symptoms, but by age 13 they had diverged significantly and their symptoms increased steadily at each subsequent assessment. Second, we identified a small group of youth (13%) who displayed a pattern of high depressive symptoms at age 11 – markedly higher than those observed in either the Increasing or Stable Low classes. Youth on this Early High trajectory reported declining symptoms over time, though it would not be accurate to characterize this pattern as a reduction in overall depression risk. Despite net declines in depressive symptoms over time, Early High trajectory youth reported significantly higher depressive symptoms than their peers in the Stable Low trajectory at each assessment.

Although other studies have identified a group of youth with steady high depressive symptoms across adolescence (e.g., Reinke et al., 2012), our study did not identify such a group. This likely is due to our community sample and the relatively small number of youth ($N=52$) with high depressive symptoms at age 11 who were classified as being in the Early High trajectory. It is possible that some of the youth in the Early High trajectory actually displayed a stable high symptom trajectory but we lacked the statistical power to distinguish them. In a larger sample we would have had greater power to detect a small group of youth with stable high depressive symptoms.

While describing these three depressive symptom trajectories is interesting in and of itself and provides replication of key findings from prior trajectory analyses, the proximal goals of this study were to both identify and predict depression trajectories in adolescence as necessary steps toward a more distal goal of informing preventive and early intervention efforts by depression researchers and clinicians. Below we examine the Increasing and Early High classes in terms of depression diagnoses and significant predictors.

The Increasing Trajectory: Emergent risk for adolescent depression from a convergence of risk factors—If it is reassuring to depression researchers and clinicians that the majority of youth (51%) are on a relatively low-risk depression trajectory in adolescence, it should be alarming that the next largest group of youth (37%) is on a

trajectory of increasing symptoms and steadily accumulating risk for depression diagnoses. By age 15, youth in the Increasing class had the highest level of depressive symptoms in the entire sample and nearly a quarter of them had already met criteria for a major depressive episode. If we consider all clinically relevant Axis 1 depressive disorders, the lifetime prevalence for depression among this group of youth was nearly 40% by age 15. What is notable about these high-risk youth, however, is that at age 11 they were indistinguishable from their Stable Low peers in terms of early depressive symptoms. What, then, differentiates youth on the high-risk Increasing trajectory from youth on the low-risk Stable Low trajectory even in the absence of identifiable differences in depressive symptoms? We identified three risk factors that contributed to emergent risk for adolescent depression: being female; having high negative affectivity in infancy; and having more negative cognitive style in pre-adolescence.

The emergent sex difference in depression in adolescence is a well-established and robust finding (Hankin et al., 1998). What is novel about this finding is the specificity of the risk associated with being female – girls were more likely to be on the Increasing high-risk trajectory, but not the Early High high-risk trajectory. This is consistent with the depressive symptom trajectories identified among adolescent girls by Dekker and colleagues (2007), who found that among adolescent girls, depression symptom trajectories were characterized by either stability of symptoms or increases in symptoms. These concordant results indicate that future research should identify the mechanisms that propel girls onto an Increasing trajectory. These might be factors that themselves increase over the same period, such as stress or sexual victimization.

While extensive research has associated childhood negative affectivity with depressive symptoms and disorders in adolescence (e.g., Compas et al., 2004), there has been debate over the extent to which childhood negative affectivity predicts emergence of depressive symptoms and disorders as opposed to simply being an early form of depression. The current study clearly identifies infant negative affectivity as a premorbid risk factor for adolescent-onset depression. Youth who were described by their mothers as being highly emotionally reactive and displaying more negative affect than the typical child even as young as 12 months old were significantly more likely to be on the Increasing trajectory. However, these youth did not display greater depressive symptoms or diagnoses at age 11 than youth on the Stable Low trajectory, suggesting that infancy negative affectivity is not simply a proxy for early depressive symptoms. Thus, negative affectivity prior to adolescence may be an easily identifiable and powerful indicator of risk for depression in adolescence.

Finally, there has been considerable debate as to when cognitive risk factors begin to confer risk for depression, as these rely upon normative cognitive development in the transition to adolescence to emerge and consolidate (Mezulis, Funasaki, & Hyde, 2011). Researchers have also debated the direction of effects between cognitive style and depressive symptoms, with some researchers suggesting that negative cognitive style may actually emerge as a result of early depressive symptoms and functions instead as a cognitive “scar” of early depression, which in turn predicts depression recurrences (McCarty, Vander Stoep, & McCauley, 2007). In the current study, we found a trend for negative cognitive style at age 11 to predict being on the Increasing trajectory in adolescence. While this trend-level effect should be interpreted cautiously, this result supports other studies suggesting a prospective effect of negative cognitive style on the emergence of adolescent depression (e.g. Abela et al., 2011).

The Early High Trajectory: Entering adolescence on a high-risk developmental trajectory created by early risk factors—Although common wisdom suggests that

most youth enter adolescence with low depressive symptoms that then increase over time, youth on the Early High trajectory displayed a very different pattern. They entered adolescence with high depressive symptoms that declined somewhat over time. This class might be interpreted in one of two ways: either as a group of youth with childhood-onset depression rather than adolescent-onset depression, or as a group of youth with decreasing risk over time. Closer examination revealed that both interpretations were inaccurate. Total risk for depression did not appear to decrease over time for these youth. Their symptoms, while declining somewhat from age 11 to 18, remained significantly higher than those observed in the Stable Low group at all assessments. Similarly, they accumulated depression diagnoses at a rate exceeding that of both the low-risk Stable Low and high-risk Increasing groups; nearly 30% of youth in this class had experienced a major depressive episode by age 15. However, this high rate of depression diagnoses does not appear to be explained by a high rate of childhood-onset episodes. The high level of symptoms observed among these youth at age 11 appear to precede and indicate risk for future depressive episodes rather than simply being a marker of concurrent or prior depressive episodes. However, it will be critical to follow these youth across the transition to adulthood, as at least one other study has found that the Early High group may become indistinguishable from the Stable Low group by age 25, at least in terms of current symptoms (Costello et al., 2008).

Given the high depressive symptoms already being displayed by these youth at age 11, it is difficult to interpret statistically significant risk factors as “premorbid” and differentiate causal risk factors from correlate symptoms. However, we identified both affective and biological risk factors for being on the Early High trajectory that are likely premorbid to even the depressive symptoms at age 11. Infant negative affectivity was a risk factor for being on the Early High trajectory, particularly for boys. This finding has clear clinical relevance, as research has demonstrated several etiological pathways to adolescent depression among girls, but identifying high-risk boys has been more difficult.

The other notable biological risk factor for being on the Early High trajectory was early puberty. These are youth who, by age 11.5, had already attained Tanner Stage 3 and/or started menstruation, either of which would indicate a pubertal developmental trajectory a year or more advanced than typically developing youth. The concordance between early puberty and early depressive symptoms is consistent with prior studies suggesting that early puberty puts youth at elevated risk for mental health problems. While beyond the scope of this paper, other studies have found that the effects of early puberty on depression outcomes may be mediated by changes in peer sexual harassment and body image (e.g. Lindberg, Grabe, & Hyde, 2007).

Finally, we found that one cognitive risk factor at age 11, rumination, predicted membership in the Early High class. This result should also be interpreted very cautiously. Rumination about depressed affect has been criticized as having too much conceptual overlap with depressive symptoms (Treynor, Gonzalez, & Nolen-Hoeksema, 2003) and in the current study it was measured at age 11 when youth in the Early High trajectory were already displaying elevated depressive symptoms, so that it cannot clearly be interpreted as a premorbid risk factor. However, several studies have shown that rumination exacerbates and prolongs depressed mood and as such is an important cognitive mechanism in depression maintenance and recurrence (Nolen-Hoeksema et al., 2008). In the current study, we also found a significant rumination by sex interaction, suggesting that rumination may be a cognitive vulnerability factor that disproportionally makes girls vulnerable to depressive symptoms.

Strengths, Limitations, and Future Directions

The latent class approach accounts for the heterogeneity in the longitudinal course of depressive symptoms from early through late adolescence, and sheds light on a general risk factor for both high-risk trajectories (negative affect) and specific risk factors unique to each high-risk trajectory (early puberty, rumination, cognitive style). Importantly, our findings of Early High, Increasing, and Stable Low classes replicate those of other latent class studies on depressive symptoms in adolescence (Brendgen et al., 2005; Costello et al., 2008; Frye & Liem, 2011; Reinke et al., 2012). While a strength of the current study is the community sample of adolescents (who were experiencing stressors normative to the adolescent period), this study is limited in its generalizability of the findings beyond non-Hispanic Whites. We also lacked data on depression intervention within our sample that may have impacted upon symptom trajectories; specifically, it is possible that one explanation for declining symptoms among the Early High group was early depression treatment which was not assessed in the study. Finally, we only collected depressive symptom data once every two years. Symptom measures such as the CDI only assess current symptoms, and thus fluctuations in symptoms between assessments are not well-characterized by these analyses. Thus, we encourage future research on the following: 1) latent class analysis of depressive symptoms every 3–6 months across adolescence; 2) risk factors assessed in childhood that predict membership in the Early High group; 3) other risk factors that may predict class membership, including HPA markers, pubertal hormones, immune markers, and life stressors; 4) latent classes of comorbid anxiety and depressive symptoms; and 5) research in more vulnerable populations (e.g., Ge, Natsuaki, & Conger, 2006; Repetto, Caldwell, & Zimmerman, 2004).

Clinical Implications for the Development of Depression Prevention Programs

This study has several important implications for the effective development and implementation of targeted depression prevention programs. Studies of adolescent depression demonstrating a mean rise in depressive symptoms and diagnoses suggest a clinical rationale for universal programs (Garber, Korelitz, & Samanez-Larkin, 2012). However, our results clearly indicate that the majority of youth do not need depression prevention programs. They enter adolescence on a low-risk trajectory characterized by consistently low depressive symptoms and relatively low depression diagnoses. Their resilience may come in the form of lower levels of risk factors for adolescent-onset depression – less negative affectivity, less negative cognitive style and rumination, and less likely to have early puberty – and/or in the form of higher levels of protective factors not measured in the current study. It is likely that this sizable group of youth contribute to the generally weak findings for the effectiveness of universal prevention programs for reducing depression risk.

Consistent with these findings, Horowitz and Garber (2006) stated that selective and indicated depression prevention programs have the most promise for effectively altering the depression trajectories of at-risk youth. Most non-universal depression prevention programs are indicated prevention programs, meaning they target youth already displaying signs of depression, e.g. high-symptom youth. These are differentiated from selective depression prevention programs, which target youth who are high in one or more empirically supported risk factors but who do not already display signs of depression – i.e. high-risk but low-symptom youth. It is important to observe that indicated prevention programs prior to age 13 would overlook youth on the Increasing trajectory in our study. These youth do not display pre-adolescent high depressive symptoms yet clearly they are on a high-risk trajectory. Youth on this Increasing trajectory would benefit most from selective prevention programs that target youth who are high in one or more empirically supported risk factors but who do not already display signs of depression.

If indicated depression prevention programs may inadvertently overlook youth on the Increasing trajectory, it is likely that they disproportionately target youth on the Early High trajectory. It is an open question as to whether indicated depression prevention programs adequately link the preventive intervention with the empirically supported risk factor within that sample. Our findings suggest that youth with early high depressive symptoms may have a markedly different pathway to adolescent depression than youth whose depressive symptoms emerge several years later. Here we see that early pubertal development may play a particularly salient role in conferring risk for early depressive symptoms, suggesting that preventive interventions that target the social and psychological sequelae of early puberty may be particularly beneficial to these youth. Similarly, rumination appears to confer risk at least for the maintenance of depressive symptoms among youth with early symptom trajectories.

Taken together, our findings highlight the importance of nuanced approaches to identifying heterogeneity in risk for depression in adolescence across multiple levels of analysis (affective, biological, and cognitive). Our results guide future research by providing preliminary suggestions for tailoring interventions to the empirically supported risk trajectory.

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Figure 1.
Depressive Symptom Trajectory Classes

Table 1

Latent Growth Mixture Model Statistics

Number of Classes	AIC	BIC	Entropy	LMR Adjusted LRT
1	7964.33	7984.48	.96	--
2	7816.50	7848.75	.94	$p = .013$
3	7775.76	7820.10	.88	$p = .041$
4	7766.15	7822.58	.83	$p = .110$
5	7727	7796.01	.81	$p = .240$

Notes: For the Bayesian Information Criterion (BIC) and the Akaike Information Criterion (AIC), lower values typically indicate better fitting models. Model entropy is a measure of classification accuracy with values closer to 1 (range: 0–1) indicating greater precision of classification accuracy. The Lo-Mendell-Rubin adjusted likelihood ratio test (LMR Adjusted LRT) of model fit compares the estimated model with a model with one fewer class (Lo, Mendell, & Rubin, 2001). The Lo-Mendell-Rubin Adjusted LRT yields a p -value that reflects whether the current model fits the data significantly better than a model with one less class. The three-class model displayed lower AIC and BIC compared to the two-class model while maintaining adequate entropy; the LMR Adjusted LRT indicated that the three-class model was a significantly better fit to the data than the two-class model. Although the AIC decreased slightly from the three-class to the four-class model, the BIC increased, entropy decreased, and the LMR Adjusted LRT indicated that the four-class model was not a significantly better fit than the three-class model.

Table 2

Correlation Matrix for All Study Variables.

	CDI 11	CDI 13	CDI 15	CDI 18	RUM	CS	NA	Early Pub
CDI 13	.49**							
CDI 15	.35**	.56**						
CDI 18	.27**	.33**	.35**					
RUM	.44**	.18**	.16**	.17**				
CS	.24**	.15**	.15**	.17**	.32**			
NA	.03	.03	.11 ⁺	.14**	.01	.02		
Early Pub	.21**	.27**	.23**	.11	.05	-.05	.03	
Sex	.00	.14**	.17**	.06	.01	.13*	.14*	.31**

Notes: The number following CDI indicates the age of assessment. Early Puberty was coded 0 = No Early Puberty and 1 = Early Puberty. Sex was coded -1 = male and 1 = female. NA = Negative affectivity.

Table 3

Depressive Symptoms by Trajectory Class

Age	Total	Depressive Symptoms M (SD)			ANOVA	
		Stable Low (SL)	Increasing (I)	Early High (EH)	F	Comparison
11	3.25 (4.44)	1.68 (1.80)	2.48 (3.02)	11.32 (6.27)	152.05**	(SL = I) < EH
13	4.25 (4.76)	2.39 (2.47)	5.17 (4.86)	8.62 (6.81)	45.95**	SL < I < EH
15	4.79 (5.44)	2.04 (2.10)	7.63 (6.33)	7.12 (6.18)	56.58**	SL < (I=EH)
18	5.44 (5.78)	1.85 (1.96)	9.85 (5.93)	5.43 (5.66)	112.03**	SL < EH < I

Table 4

Depression Diagnoses by Trajectory Class

Depression Diagnosis	Entire Sample	Stable Low	Increasing	Early High
<i>Major Depressive Disorder</i>				
Lifetime	15.2%	8.8%	20.4%, LR=8.66**	25.0%, LR=9.18*
Childhood Onset ⁺	2.1%	2.1%	2.2%, LR=.01	1.9%, LR=.01
Adolescent Onset ⁺⁺	13.1%	6.7%	18.2%, LR=10.12**	23.1%, LR=10.78**
<i>Other Depressive Disorder</i>				
Lifetime	10.2%	6.7%	14.6%, LR=4.48*	11.5%, LR=1.10
Childhood Onset ⁺	2.1%	1.6%	2.9%, LR=.55	0.0%, LR=1.4
Adolescent Onset ⁺⁺	8.4%	5.2%	11.7%, LR=3.82*	11.5%, LR=2.24

Notes:

⁺ less than 11 years, 11 months;

⁺⁺ greater than or equal to 12 years, 0 months;

Other Depressive Disorder = Dysthymia, Depressive Disorder NOS, and Adjustment Disorder with Depressed Mood. LR = Likelihood Ratio compared to Stable Low class;

* indicates LR significant at $p < .05$;

** indicates LR significant at $p < .01$.

Table 5

Means, Standard Deviations, Frequencies, Percentages of Predictor Variables by Class

Variable	Increasing (I)	Early High (EH)	Stable Low (SL)	Total	Increasing vs. SL	Early High vs. SL	
Frequency							
Sex (Female)	91 (66.4%)	24 (46.2%)	84 (43.5%)	199 (52.1%)	15.28**	.013	
Early Puberty	41 (33.6%)	18 (42.9%)	37 (25.7%)	96 (31.2%)	1.99	4.39*	
	Increasing (I)	Early High (EH)	Stable Low (SL)	Total	F	Increasing vs. SL	Early High vs. SL
Negative Affectivity	3.08 (.65)	2.86 (.54)	2.75 (.68)	2.89 (.67)	8.88**	.33 (.08)**	.11 (.11)
Mean (SD)							
Rumination	1.84 (.51)	2.20 (.58)	1.74 (.50)	1.84 (.53)	11.76**	.10 (.06)	.46 (.09)**
Cognitive Style	1.91 (.50)	2.06 (.58)	1.82 (.43)	1.88 (.48)	3.93*	.09 (.06)	.24 (.09)**

Note:

* < .05;

** < .01.

Class comparisons reported for Frequency (%) variables are Likelihood Ratios. Class comparisons reported for Mean (SD) variables are Mean Differences (Standard Error).

Table 6

Multinomial Logistic Regressions Predicting Membership in Increasing and Early High Classes

Predictor	Increasing Class		Early High Class	
	OR	p-value	OR	p-value
Sex	.41	.00	1.06	.60
Negative affectivity	1.93	.00	4.12	.25
Early Puberty	1.21	.50	2.27	.03
Rumination	1.45	.14	4.07	.00
Cognitive Style	1.68	.06	2.90	.28
Negative affectivity x Sex	.84	.34	2.63	.04
Early Puberty x Sex	1.57	.18	1.44	.16
Rumination x Sex	.88	.65	.51	.02
Cognitive Style x Sex	1.40	.34	1.60	.31

Note: Comparison group is Stable Low class for all analyses.