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Anxiety and Depression During Childhood and Adolescence: Testing Theoretical Models of Continuity and Discontinuity

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Abstract
The present study sought to clarify the *trajectory* (i.e., continuous vs. discontinuous) and *expression* (i.e., homotypic vs. heterotypic) of anxiety and depressive symptoms across childhood and adolescence. We utilized a state-of-the-science analytic approach to simultaneously test theoretical models that describe the development of internalizing symptoms in youth. In a sample of 636 children (53% female; M_{age} = 7.04; SD_{age} = 0.35) self-report measures of anxiety and depression were completed annually by youth through their freshman year of high school. For both anxiety and depression, a piecewise growth curve model provided the best fit for the data, with symptoms decreasing until age 12 (the "developmental knot") and then increasing into early adolescence. The trajectory of anxiety symptoms was best described by a *discontinuous homotypic* pattern in which childhood anxiety predicted adolescent anxiety. For depression, two distinct pathways were discovered: A *discontinuous homotypic* pathway in which childhood depression predicted adolescent depression and a *discontinuous heterotypic* pathway in which childhood anxiety predicted adolescent depression. Analytical, methodological, and clinical implications of these findings are discussed.

Keywords Depression · Anxiety · Discontinuity · Heterotypic · Growth curve modeling

Childhood and adolescence are critical periods for the emergence of anxiety and depression. Symptoms during both developmental epochs impact social, emotional, and academic development (Costello et al. 2003; Garber and Horowitz 2002; Rapee et al. 2009; Rudolph 2014). Despite extensive research documenting the prevalence (e.g., Costello et al. 2003), impact (e.g., Garber and Horowitz 2002; Rapee et al. 2009), and comorbidity (Cummings et al. 2014) between anxiety and depression in youth, however, debates concerning prospective continuity (e.g., linear, quadratic, discontinuous growth) and sequential comorbidity (i.e., heterotypic models of psychopathology) remain unresolved. An incomplete understanding of the growth within and between symptoms of anxiety and depression undermines our ability to operationalize and target risk for internalizing distress in youth (Rutter et al. 2006). The present study aimed to address these fundamental developmental psychopathology questions by modeling competing explanations of symptom development to clarify the prospective relation between childhood and adolescent internalizing symptoms.

An Organizational Framework of Psychopathology

An organizational perspective posits that the inability to successfully navigate developmental challenges leads to a biological and psychological reorganization that sets the foundation for psychopathology (Cicchetti and Toth 1998). These reorganizations can impact the *expression* (i.e., the overt manifestation of clinical symptoms of distress) and *trajectory* (i.e., the longitudinal course of symptoms) of psychopathology over time. With regard to expression, symptoms may take a *homotypic* form in which the symptom manifestation remains the same following a developmental transition (i.e., depression predicts depression) or a *heterotypic* form in which one symptom

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expression begets an alternative symptom expression (i.e., anxiety predicts depression; Cicchetti et al. 1994).[1] As for the *trajectory*, symptoms may be continuous, as reflected in a linear or accelerated/decelerated path (i.e., faster increase/decrease in symptoms), or discontinuous, defined as non-dependent growth patterns across developmental epochs (Klimes-Dougan et al. 2010; Schulenberg et al. 2003). Overall, assessing the expression *and* trajectory of internalizing symptoms is critical for creating conceptual models that best describe patterns of growth during these vulnerable ages.

With regard to symptom expression, the majority of extant research posits *homotypic* expressions of *continuous* trajectories of psychopathology. For instance, research suggests that a depressotypic organization may lead to an accelerated, continuous, homotypic pattern of depression in adolescence (Hankin et al. 1998), before transitioning into a stable, continuous homotypic pattern in adulthood (Hankin et al. 1998; Rutter et al. 2006). In recent years, however, it has become increasingly common to simultaneously test both *homotypic* and *heterotypic* continuous expressions of internalizing symptoms (e.g., McLaughlin and King 2015). *Homotypic continuity* occurs when a transactional relation between the environment and impairment manifests as the same symptom expression across developmental epochs (e.g., stress-generation theories for depression; Rudolph et al. 2016). *Heterotypic continuity* occurs when one symptom manifestation lays the foundation for a new organization or when an existing underlying vulnerability interacts with a new biological or environmental context (Cicchetti and Toth 1998). For example, an inability to self-regulate may manifest as symptoms of separation anxiety in childhood but transform into social-evaluative fears (Weems 2008) or depression (Flannery-Schroeder 2006) as one biologically and socially matures.

As for symptom trajectories, continuous patterns of growth have received far more attention than discontinuous models. However, there are several theoretical explanations for discontinuous patterns of psychopathology (see Rutter et al. 2006; Schulenberg et al. 2003). For instance, certain transitions may lead to a temporary discontinuity of symptoms, but lay an organizational foundation for symptoms to return at a later developmental stage (i.e., *transition-linked turning points;* see Graber and Brooks-Gunn 1996). This foundation may be for the same (*homotypic discontinuity)* or for different (*heterotypic discontinuity*) prospective symptom expressions. Additionally, discontinuity may occur when the same symptom serves a different function across different developmental stages. For instance, the functional shift from marijuana use for experimentation in adolescence to self-medication in adulthood corresponds to non-dependent trajectories and can best be described within a *homotypic discontinuous* model (Schulenberg et al. 2003). Within this example, modeling the trajectory of marijuana use from adolescence through adulthood as continuous would be inappropriate, and lead to unstable risk predictions for chronic marijuana use in adulthood. Thus, clarifying if the transition between childhood and adolescence is characterized by homotypic or heterotypic and continuous or discontinuous patterns can influence our interpretation of early symptom expressions.

Methodological and Analytical Considerations

Mixed findings concerning the continuity of anxiety and depressive symptoms are rooted in the diverse methodological approaches utilized in the field. A majority of continuity research concerning anxiety and depression comes from family and epidemiological studies (e.g., Kessler et al. 2005; Weissman et al. 1999) which may be ill-equipped to capture clinically relevant symptom fluctuations during childhood and adolescence. Instead, multi-wave, longitudinal studies that include repeated (e.g., annual) follow-up assessments of symptoms may be better able to identify important turning points in symptom trajectories during rapid periods of development (Cohen et al. 2014; Rutter et al. 2006). To date, most multi-wave, longitudinal research assesses symptom continuity within childhood (e.g., Olatunji and Cole 2009; Sterba et al. 2007) or adolescence (e.g., Hale et al. 2008; McLaughlin and King 2015) but not both. A notable exception comes from Harrington and Rutter's pioneering work (see Harrington et al. 1996; Rutter et al. 2006) concerning depression continuity across the lifespan. In community and clinical samples, the authors found support for homotypic continuity between adolescent and adult depression but heterotypic discontinuity between child and adolescent presentations.

The present study sought to extend Harrington and Rutter's research by utilizing a state-of-the-science analytic approach. Traditionally, autoregressive cross-lagged panel models have been used to test the association within and between anxiety and depression over time (e.g., Cole et al. 1998). Although these approaches properly model the point-to-point associations, they are ill-equipped to detect larger developmental trajectories and cannot differentiate between within and between-person variance. Parallel process growth curve modeling (GCM), on the other hand, improves on cross-lagged approaches by examining linear and non-linear patterns of individual growth over time (Duncan et al. 2013); however, these models cannot account for the changes

[1] We note the terms heterotypic and homotypic continuity have been used to describe both manifest behaviors and latent internalizing processes over time (Cicchetti et al. 1994; Lahey et al. 2014). As a major aim of our study was the translational importance of distinguishing between continuous and discontinuous models of psychopathology for assessment purposes, we focus our discussion of heterotypic and homotypic continuity on symptom manifestations.

occurring between specific time points. A resolution to these collective limitations may be an autoregressive latent trajectory (ALT) approach, which maximizes the strengths of cross-lagged panel and parallel process GCM approaches by modeling the point-to-point associations within the context of larger developmental trajectories (Bollen and Curran 2004; McLaughlin and King 2015).

In addition to utilizing an ALT model, piecewise growth curve modelling (PGCM) can provide an analytical framework to examine theories of discontinuity. PGCM allows distinct growth periods to exist within a given model and can identify critical turning points in a pattern's trajectory (Kohli et al. 2015). PGCM is a recommended approach when assessing symptoms across developmental epochs (Chou et al. 2004) and for modeling the prospective relation between co-occurring symptoms (Mamey et al. 2015). To date, no study has used PGCM to test the association between anxiety and depressive symptoms in youth, and only one study utilized a parallel process GCM approach to test anxiety-depression comorbidity hypotheses across distinct epochs. Keenan et al. (2009) found support for a homotypic continuity model in females between the ages of 6–12 for both anxiety and depressive symptoms. These findings stand in contrast to findings by Harrington and Rutter (Harrington et al. 1996; Rutter et al. 2006), as well as more recent parallel process GCM/ALT research (Hale et al. 2009; Leadbeater et al. 2012; McLaughlin and King 2015) that supported heterotypic models for adolescent depression. We propose that using a PGCM approach, and explicitly testing if internalizing trajectories between childhood and adolescence are non-dependent, will better capture patterns of comorbidity and help clarify conflicting findings concerning homotypic and heterotypic models.

The Present Study

The present 7-year study annually assessed anxiety and depressive symptoms in 636 youth beginning in childhood. The study replicated past parallel process GCM (e.g., Hale et al. 2009), and ALT-approaches (McLaughlin and King 2015) to determine if utilizing a PGCM framework could resolve past discrepancies. We examined PGCM models using a "developmental knot" (see Kohli et al. 2015) approach to pinpoint when trajectories became discontinuous. Developmental knots represent the point at which symptom trajectories alter course. In recent years, new analytic methods allow researchers to simultaneously compare different potential locations along the symptom trajectory when the exact placement of the knot is unknown (Kohli et al. 2015). Using these novel approaches can help determine at what age internalizing symptom trajectories may become heterotypic and/or discontinuous.

Overall, we hypothesized two etiologically distinct models. First, a *continuous homotypic* pattern in which childhood anxiety leads directly to adolescent anxiety. This hypothesis is informed by research demonstrating similar growth patterns in childhood and adolescent anxiety (Hale et al. 2008; Olatunji and Cole 2009; see Van Oort et al. 2009 for an exception), and null findings for depressive symptoms predicting general anxiety symptoms (Aune and Stiles 2009; Cohen et al. 2014). Second, we hypothesized a *discontinuous heterotypic* pattern, with childhood anxiety symptoms predicting adolescent depressive symptoms. This hypothesis is based on collective research showing distinct growth patterns between childhood anxiety (negative; Olatunji and Cole 2009) and adolescent depression (positive; Leadbeater et al. 2012) and that anxiety symptoms predict subsequent depressive symptoms (Aune and Stiles 2009; Cohen et al. 2014). Exploratory analyses tested when the "developmental knot" occurs and if our findings varied by gender.

Methods

Participants and Procedure

Participants included 636 youth (53% female; $M_{age} = 7.04$; $SD_{age} = 0.35$) who were recruited to participate in a multi-wave study. For the initial phase of recruitment, consent forms were distributed to families of 725 2nd graders in 11 schools across several small urban and rural Midwestern towns. All children who were able to complete the surveys were eligible for the study. Of the eligible children, 576 (80%) received parental consent. Parents provided written consent, and children provided oral assent. Participants and nonparticipants at Wave 1 (W_1) did not significantly differ in gender, $\chi^2(1) = 0.15$, *ns*, ethnicity (white vs. minority), $\chi^2(1) = 0.59$, *ns*, or school lunch status (full pay vs. subsidized), $\chi^2(1) = 0.35$, *ns*. In the 3rd grade, an additional 60 classmates of the participating children were recruited. Participating youth were from diverse ethnic backgrounds (67% White; 22% African American; 11% other) and varied in socioeconomic status (35% received subsidized school lunch). Participants completed questionnaires annually, first in small groups of four to five students (2nd to 5th grade) and then in classrooms (6th to 9th grade). Youth received a small prize for participating. The participation rate was 91% at baseline, and 90%, 90%, 88%, 84%, 75%, 75%, and 72% at subsequent grades.

Measures

Anxiety Symptoms From 2nd to 9th grade, youth completed the Revised Child Manifest Anxiety Scale (RCMAS; Reynolds and Richmond 1985), a 28-item measure assessing anxiety

symptoms during the past two weeks (e.g., "I worry about what is going to happen."). Youth answered either yes or no for each symptom. Scores represented the sum of the 28 items (αs = 0.88–0.92). The RCMAS shows strong internal consistency (Reynolds and Richmond 1978) and test-retest reliability (Wisniewski et al. 1987). Construct validity has been established through comparisons of youth with and without anxiety disorders (Seligman et al. 2004).

Depressive Symptoms From 2nd to 9th grade, youth completed the short form of the Mood and Feelings Questionnaire (SMFQ; Angold et al. 1995), a 13-item measure assessing depressive symptoms during the past two weeks (e.g., "I felt unhappy or miserable."). The response format was modified from a 3- to 4-point scale (*Not at All* to *Very Much*; see Lau and Eley 2008). Scores were computed as the average of the 13 items (αs = 0.87–0.93). The SMFQ shows strong internal consistency and reliability and construct validity has been established in clinical and community samples (Angold et al. 1995; Thapar and McGuffin 1998).

Data Analytic Plan

Assessments took place within each grade level across eight separate assessments. Given that participants differed in age at each of the assessments, two series of models were compared during hypothesis testing: (1) grade-based models that mirrored the assessment timeline and included age as a covariate, which resulted in 8 time points (i.e., 2nd to 9th grade); and (2) aged-based models in which time was reflected by participants' ages (without rounding in years) during the assessment periods, which resulted in 9 time points (ages 7 to 15). Analyses using these two approaches converged on an identical pattern of results[2]; however, age-based models provided a better fit. Thus, for reasons of parsimony, we present only age-based models.

To test the first hypothesis that anxiety followed a continuous growth trajectory whereas depression followed a discontinuous growth trajectory, within-construct growth patterns were examined in accordance with recommendations by Cheong et al. (2003). For each symptom, four models were tested: (1) an initial model examining a single linear trajectory across all nine ages; (2) a model examining quadratic trajectories across all nine ages; and (3) a series of piecewise latent growth curve models with "knots" at age 10, 11, 12, and 13 to determine the best placement. In each piecewise model, two intercept and slope terms were estimated to examine distinct childhood and adolescent periods of non-dependent growth.[3]

Next, we tested our hypotheses that anxiety would evidence continuous homotypic growth, whereas depression would evidence discontinuous heterotypic growth. Similar to the final growth curve models examined by McLaughlin and King (2015), two separate growth models were compared: (1) a parallel process growth model that utilized the optimal growth model for each symptom category (Model 1; Fig. 1a), and (2) an autoregressive latent trajectory model, which was identical to Model 3 except that cross-lagged associations between anxiety and depression residual variances were added (Model 2; Fig. 1b). To provide comparisons with McLaughlin and King (2015), auto-regressive paths were not added to the auto-regressive latent trajectory model and disturbances were allowed to covary for measures assessed during the same period (e.g., the disturbance of anxiety symptoms at age 8 was correlated with the disturbance of depressive symptoms at age 8). At each stage of model specification, gender-specific models were examined to determine if boys and girls differed in their knot placement or type of growth pattern (i.e., continuous or discontinuous). Across analyses, model fit did not vary as a function of gender. Once the best-fitting comorbidity model was established, the effects of childhood symptoms on adolescent symptoms were examined. For this final model, gender and race were included as covariates to control for any demographic influences on symptom trajectories.

Across all models, the following recommendations by Hu and Bentler (1998) the following cutoffs were used to examine whether our model adequately fit the data: CFI $\geq$ 0.95, RMSEA $\leq$ 0.06, SRMR $\leq$ 0.08. The measure of $\chi^2/df < 3.00$ was also used as an indicator of acceptable model fit. Models were then compared across each fit index to assess relative improvements in model fit. The Akaike Information Criterion (AIC) was also used to facilitate comparisons between different models. A lower AIC statistic suggests better model fit. For all GCMs, alternate methods for modeling time were examined. Prior to conducting analyses, data were examined for missingness and violations of analytic assumptions. Little's Missing Completely at Random test was non-significant ($\chi^2(2072) = 1722.42$, $p = 0.99$) suggesting that data were missing completely at random. Thus, data were estimated using full information maximum likelihood (FIML) estimates,

[2] Grade-based models for both anxiety and depression suggested that piecewise models provided the best fit, with the knot being placed at grades 6 or 7 (when the majority of participants would be age 12). Specifically, for anxiety models, the knot fit equally well in 6th and 7th grade (ΔCFI < 0.01, $\Delta\chi^2$/df = 0.20, ΔRMSEA < 0.01, ΔSRMR = 0.01). For depression models, the knot fit best when placed at grades 6 or 7, with slightly better fit for grade 7 (ΔCFI = 0.02, $\Delta\chi^2$/df = 1.12, ΔRMSEA = 0.01, ΔSRMR < 0.01).

[3] The secondary intercept can be removed in alternate arrangements for piecewise growth functions. We selected this arrangement with a second intercept because of our interest in predicting average symptoms at the beginning of the second growth period, adolescence. Alternate piecewise growth functions with a single intercept per symptom were also examined. Both single-intercept models also fit well and produced similar findings.

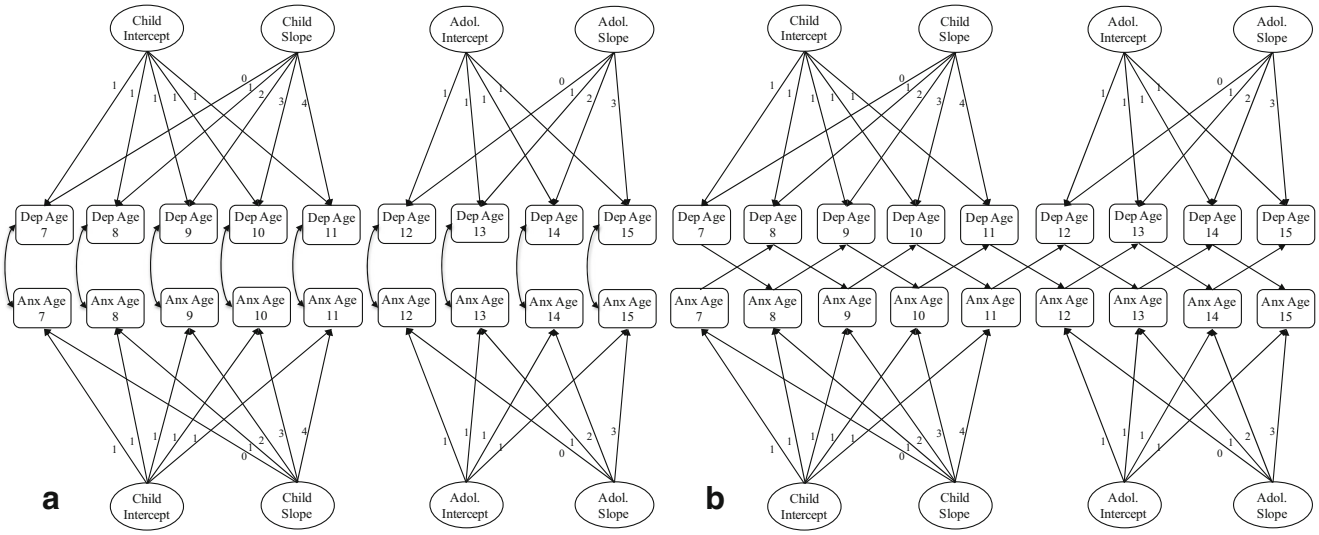

Fig. 1 Model A shows the combined piecewise model without cross-lagged paths between symptoms. Model B shows the combined piecewise model with cross-lagged paths. In all models, correlations were examined between symptoms or their residuals within each measurement period. Per modeling conventions, all latent variables were allowed to correlate, but are not presented here for display clarity. Intercept and slope terms are unique for anxiety and depression. Use of the term "child" and "adolescent" is based on age benchmarks as recommended by the National Institute of Child Health and Human Development (NICHD) Pediatric Terminology Harmonization Initiative

an optimal method for reducing bias in missing data relative to other estimation methods (Enders and Bandalos 2001). Depressive symptoms were significantly kurtotic and were thus transformed with a natural log transformation. Results did not differ between transformed and non-transformed data. As such, analyses based on non-transformed data are presented. Analyses were conducted in Mplus 7.0.

Results

Descriptive statistics are presented in Table 1. Of note, a relatively stable estimate of variance across time was observed for both anxiety and depression. Meanwhile, anxiety and depression mean levels decreased through 6th grade before increasing in the final three waves.

Table 1 Descriptive statistics of study variables

	Anxiety *M* (SD)	Depression *M* (SD)
2nd Grade	11.48 (6.72)	1.71 (0.68)
3rd Grade	10.08 (6.44)	1.60 (0.58)
4th Grade	9.24 (6.72)	1.53 (0.57)
5th Grade	8.68 (7.00)	1.51 (0.60)
6th Grade	8.40 (6.72)	1.45 (0.50)
7th Grade	7.56 (6.44)	1.39 (0.45)
8th Grade	7.84 (6.44)	1.44 (0.51)
9th Grade	8.68 (7.00)	1.53 (0.59)

Anxiety = Revised Child Manifest Anxiety Scale (RCMAS; Reynolds and Richmond 1985); Depression = short form of the Mood and Feelings Questionnaire (SMFQ; Angold et al. 1995). Please see the Methods section for descriptive statistics concerning gender, race, and poverty status

Determining the Trajectory of Anxiety and Depressive Symptoms

We first examined whether anxiety and depression followed a continuous or discontinuous trajectory and, if discontinuous, the placement of the developmental knot. The top panel of Table 2 presents a summary of the models for anxiety. An initial linear growth model evidenced poor fit across most indicators. Adding the quadratic term improved the model across all indicators and suggested good model fit; however, the best fitting model was the piecewise model with the "knot" at age 12, χ^2 (30) = 50.89, χ^2/df = 1.70, $p = 0.010$, CFI = 0.99, RMSEA = 0.03 (90% CI = 0.02–0.05), SRMR = 0.04, AIC = 26,041.19. The childhood slope suggested symptoms significantly decreased during this period ($M_{childhood} = -0.82$, $p < 0.001$), whereas the adolescent slope suggested a marginally significant increase ($M_{adolescence} = 0.21$, $p = 0.068$). Figure 2 displays the estimated and actual means based on this model's intercepts and slopes.

The middle panel in Table 2 presents a complete summary of all the models for depression. The model examining linear growth evidenced poor fit. Similar to anxiety models, adding the quadratic term improved model fit and resulted in acceptable model fit, but, the piecewise model with the "knot" at age 12 evidenced the best fit, χ^2 (30) = 43.29, χ^2/df = 1.44, p = 0.055, CFI = 0.98, RMSEA = 0.03 (90% CI = <0.01–0.04),

Table 2 Comparisons of model fit for within and between symptom growth models

	χ^2	df	*p*	χ^2/df	CFI	RMSEA (90% CI)	SRMR	AIC
Anxiety growth models								
Uniform linear	254.57	39	<0.001	6.54	0.87	0.09 (0.08–0.10)	0.12	26,251.55
Uniform quadratic	77.36	35	<0.001	2.21	0.97	0.04 (0.03–0.06)	0.04	26,061.78
Piecewise "knot" at age 10	92.90	30	<0.001	3.10	0.96	0.06 (0.04–0.07)	0.05	26,089.93
Piecewise "knot" at age 11	64.12	30	<0.001	2.14	0.98	0.04 (0.03–0.06)	0.04	26,055.60
Piecewise "knot" at age 12	50.89	30	0.010	1.70	0.99	0.03 (0.02–0.05)	0.04	26,041.19
Piecewise "knot" at age 13	75.75	30	<0.001	2.53	0.97	0.05 (0.04–0.06)	0.05	26,070.10
Depression growth models								
Uniform linear	158.90	39	<0.001	4.07	0.85	0.07 (0.06–0.08)	0.10	6105.38
Uniform quadratic	73.66	35	<0.001	2.10	0.95	0.04 (0.03–0.06)	0.05	5985.15
Piecewise "knot" at age 10	84.15	30	<0.001	2.81	0.93	0.05 (0.04–0.07)	0.06	6011.14
Piecewise "knot" at age 11	57.96	30	0.001	1.93	0.96	0.04 (0.02–0.05)	0.04	5971.11
Piecewise "knot" at age 12	43.29	30	0.055	1.44	0.98	0.03 (<0.01–0.04)	0.03	5948.25
Piecewise "knot" at age 13	61.24	30	0.001	2.04	0.96	0.04 (0.03–0.06)	0.05	5974.55
Anxiety and depression growth models								
Piecewise "knot" at 12	146.32	114	0.022	1.28	0.99	0.02 (0.01–0.03)	0.04	30,267.75
Piecewise and cross-lagged with "knot" at 12	134.21	98	0.001	1.37	0.99	0.02 (0.01–0.03)	0.04	30,283.28
Piecewise model with covariates	161.92	134	0.051	1.21	0.99	0.02 (<0.01–0.03)	0.04	30,219.82

CFI, Comparative Fit Index; *RMSEA*, Root Mean Square Error of Approximation; *SRMR*, Standardized Root Mean Residual; Fig. 1 depicts differences between piecewise models with and without cross-lagged paths. Anxiety = Revised Child Manifest Anxiety Scale (RCMAS; Reynolds and Richmond 1985); Depression = short form of the Mood and Feelings Questionnaire (SMFQ; Angold et al. 1995)

SRMR = 0.03, AIC = 5948.25. Within both childhood and adolescence, the slope factors had significant means, but in opposite directions, with symptoms decreasing across childhood ($M_{depression} = -0.06$, $p < 0.001$) and increasing in adolescence ($M_{depression} = 0.03$, $p = 0.001$). Figure 3 displays the estimated and actual means based on the model's intercepts and slopes. Thus, for both anxiety and depression, the trajectory is best described as discontinuous, with decreasing symptoms until age 12, followed by a non-dependent increasing symptom pattern in adolescence.

Identifying the Expression Pattern Across Development

We next examined whether anxiety and depressive symptoms showed a homotypic or heterotypic expression across development. The bottom panel in Table 2 presents a summary of simultaneous anxiety and depression models. Given our previous results, the "knot" was placed at age 12 for both anxiety and depression. The initial piecewise model (see Fig. 1a) evidenced good model fit across all indicators. The cross-lagged piecewise latent trajectory model (see Fig. 1b) also evidenced good model fit, but did not significantly improve model fit compared with the more parsimonious latent trajectory model (three of the fit indices were identical and two suggested a worse fit and, since these models are nested, a Satorra-Bentler scaled chi-square difference test was computed and was not significant, $\chi^2 = 12.84$, df = 16, $p = 0.685$). Additionally, of the 14 cross-lagged paths, only two were significant: (1) Age 9 anxiety predicted Age 10 depression ($\beta = 0.13$, $p = 0.036$), and (2) Age 10 depression predicted Age 11 anxiety ($\beta = 0.13$, $p = 0.021$). Gender and race were thus added as predictors to the latent trajectory model without cross-lagging to form our final model, χ^2 (134) = 161.92, χ^2/df = 1.21, $p = 0.051$, CFI = 0.99, RMSEA = 0.02 (90% CI, <0.01–0.03), SRMR = 0.04, AIC = 30, 219.82.

Table 3 summarizes our final model solution while Fig. 4 displays the developmental expression of symptoms. With regard to adolescent anxiety, the childhood anxiety intercept and slope significantly predicted the adolescent anxiety intercept (*p*-values < 0.01), but the childhood depression intercept and slope did not forecast adolescent anxiety (*p*-values > 0.10). With regard to adolescent depression, the childhood depression intercept and slope, and the childhood anxiety intercept significantly predicted the adolescent depression intercept (*p*-values < 0.05). Additionally, gender positively predicted both adolescent slopes (*p*-values < 0.01), such that girls reported a greater increase in anxiety and depressive symptoms across adolescence. No other paths or covariates significantly predicted symptom intercepts or slopes (*p*–values > 0.05). The covariance matrix for all study variables is presented in Table 4.

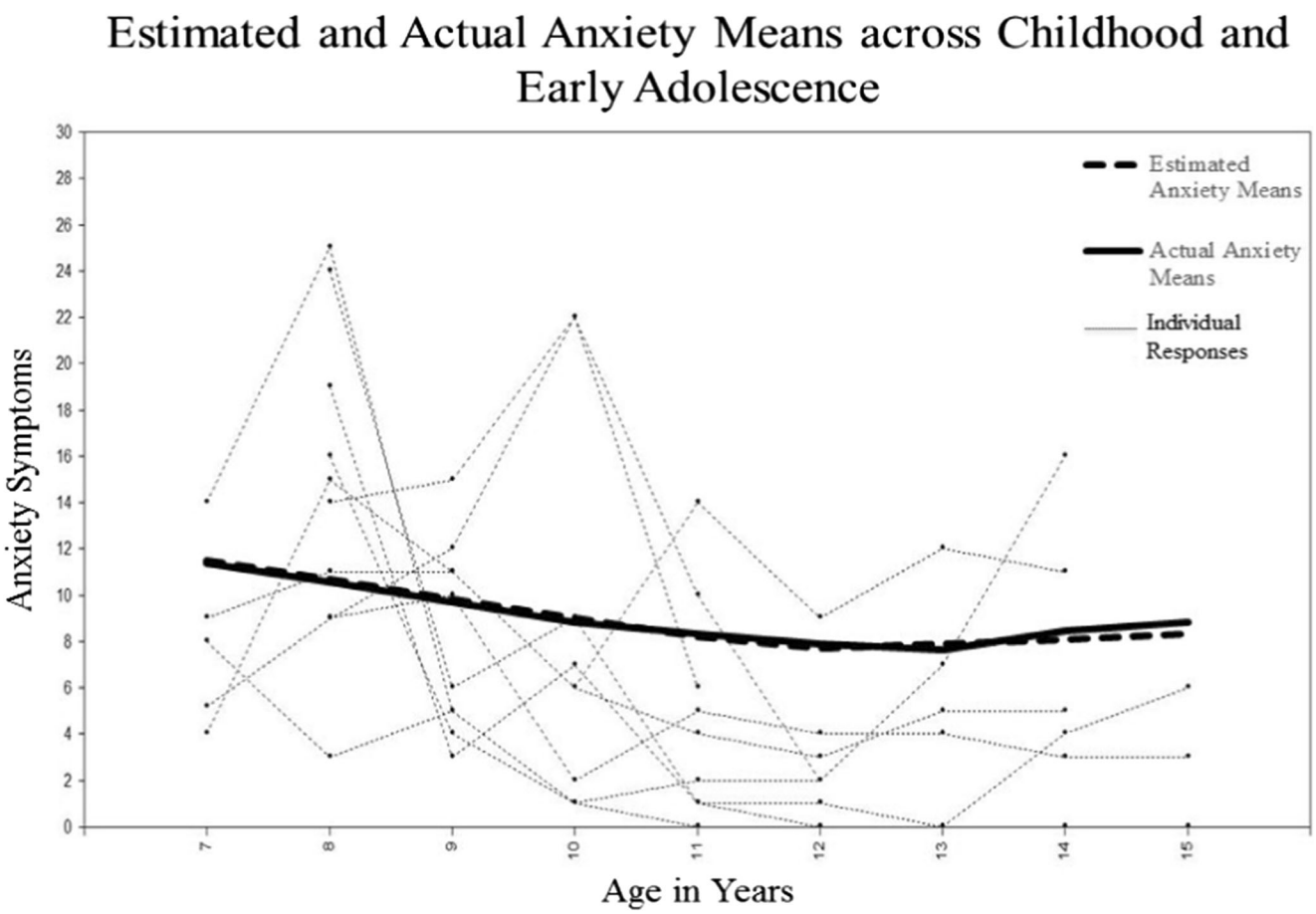

Fig. 2 Estimated and actual anxiety means from the Revised Child Manifest Anxiety Scale (RCMAS). Estimated means were derived from a piecewise longitudinal model with a "knot" placed at age 12. A second intercept and slope were estimated at the "knot". Individual data from a random selection of 10 cases is also displayed and was generated using Mplus version 7.2

Discussion

Contemporary methodological, analytical, and clinical approaches to childhood and adolescent internalizing symptoms typically assume continuous trajectories and homotypic symptom expression. Prospective examinations of anxiety *or* depression, mixed-level/growth curve modeling approaches that test linear or quadratic patterns, and preventative initiatives that screen for emerging symptoms in childhood to target adolescent psychological distress are examples of how these assumptions manifest. The present study offers a concerted challenge to these conventions by revealing discontinuous symptom trajectories for anxiety and depression and a heterotypic developmental pathway for depression. These findings, coupled with details on when symptom trajectories begin to differ (i.e., the developmental knot), provide a nuanced explanation for the development of adolescent internalizing symptoms. Below, we discuss how our findings advance the field's understanding of adolescent anxiety and depression as well as the methodological and clinical implications of our study.

Adolescent Anxiety Development

Our finding that anxiety exhibited a homotypic pattern of symptom expression was consistent with the larger literature (Bittner et al. 2007; Reinke and Ostrander 2008). As for symptom *trajectory*, support for a discontinuous model was inconsistent with our hypotheses. The majority of research to date demonstrates that within both childhood (e.g., Olatunji and Cole 2009) and adolescence (e.g., Hale et al. 2008), general anxiety symptoms continuously decrease over time.[4] However, the few studies that examined anxiety symptoms across childhood and adolescence have found trajectory patterns similar to the present study. Costello et al. (2003) found that anxiety symptoms decreased between ages 9 and 12 before slightly increasing. Van Oort et al. (2009) found a similar pattern across symptoms of generalized anxiety disorder, separation anxiety

[4] Specific manifestations of anxiety (e.g., panic disorder), however, may have different trajectories during adolescence (Nelemans et al. 2014). This issue is discussed further in the limitations section.

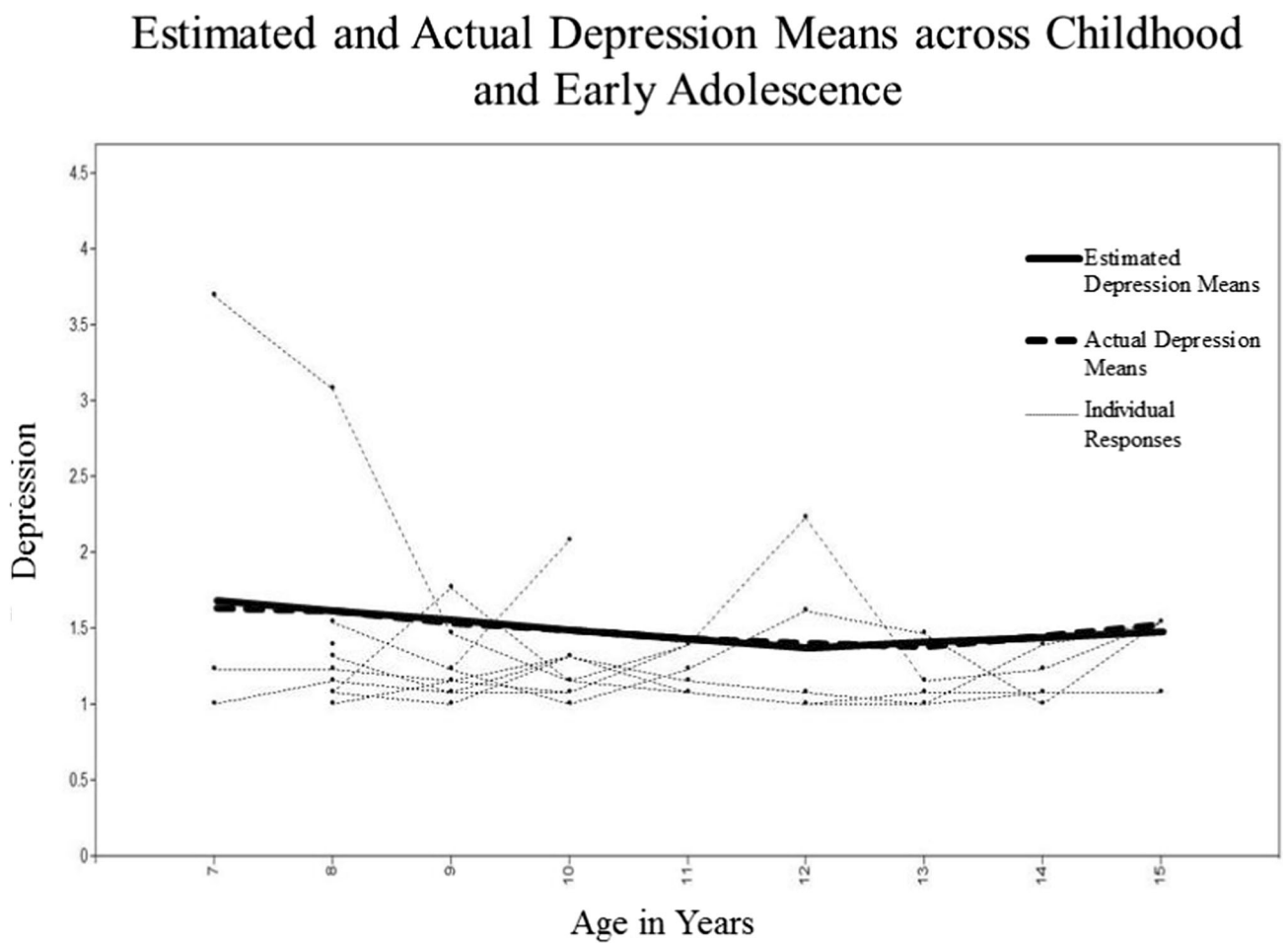

Fig. 3 Estimated and actual depression means from the Short Mood and Feelings Questionnaire (SMFQ) Depression scale. Estimated means were derived from a piecewise longitudinal model with a "knot" placed at age 12. A second intercept and slope were estimated at the "knot". Individual data from a random selection of 10 cases is also displayed and was generated using Mplus version 7.2

disorder, and social phobia. The authors suggested, and later demonstrated (Van Oort et al. 2011), that although intrapersonal risk factors for anxiety (e.g., global self-worth) may be stable across development, interpersonal risk factors for anxiety may uniquely relate to certain developmental stages.

A model of shared intrapersonal risk factors along with disparate interpersonal risk factors across development stages may explain the emergence of our discontinuous, homotypic anxiety profile. A homotypic *expression* is likely maintained by a combination of genetics, temperament, and early environmental experiences (Rapee et al. 2009) that are influential in forming an anxiotypic organization that can forecast both proximal (i.e., childhood) or distal (adolescent) anxiety symptoms. However, a discontinuous trajectory for anxiety suggests that this organizational framework alone is not sufficient for a developmentally-contiguous trajectory of symptoms. Instead, anxiety symptom trajectories may be dependent on variable, stage-specific developmental challenges, such as navigating new peer and family relationship issues (Laursen and Collins 2009; Rudolph et al. 2016). This hypothesis is consistent with a transition-linked turning point model, which postulates that latent vulnerabilities for symptoms are activated during sensitive periods of development (Graber and Brooks-Gunn 1996). Thus, childhood anxiety symptoms may best be seen as a *signal* for an anxiotypic organization as opposed to being part of a continuum of symptoms over time.

Adolescent Depression Development

Compared to anxiety, a larger body of research has focused on symptom expression and trajectory with regard to depression. Our findings are consistent with Harrington and Rutter's (Harrington et al. 1996; Rutter et al. 2006) research indicating that depression follows a discontinuous symptom trajectory. As with anxiety, these findings suggest that childhood and adolescent depression may be related to different psychosocial correlates (Garber and Horowitz 2002) and that the trajectory of adolescent depression is not dependent on the pattern of pre-existing

Table 3 Summary of piecewise growth curve model with "knot" at age 12

	β or γ	*p*
Predictors of adolescent anxiety intercept		
Childhood anxiety intercept	0.73**	<0.001
Childhood anxiety slope	0.86**	<0.001
Childhood depression intercept	−0.09	0.538
Childhood depression slope	0.03	0.826
Racial Minority	−0.06	0.166
Female	<0.01	0.988
Predictors of adolescent anxiety slope		
Childhood anxiety intercept	−0.30	0.288
Childhood anxiety slope	−0.66*	0.030
Childhood depression intercept	−0.29	0.428
Childhood depression slope	−0.67	0.238
Adolescent depression intercept	0.64	0.241
Racial Minority	0.08	0.365
Female	0.34**	<0.001
Predictors of adolescent depression intercept		
Childhood anxiety intercept	0.35*	0.014
Childhood anxiety slope	0.23	0.151
Childhood depression intercept	0.45*	0.020
Childhood depression slope	0.75**	0.001
Racial Minority	−0.03	0.594
Female	0.05	0.312
Predictors of adolescent depression slope		
Childhood anxiety intercept	0.15	0.782
Childhood anxiety slope	0.02	0.972
Childhood depression intercept	−0.34	0.254
Childhood depression slope	−0.67	0.064
Adolescent anxiety intercept	0.14	0.782
Racial Minority	0.07	0.468
Female	0.35**	<0.001

Racial Minority was dichotomously coded with white as the referent group and racial minority youth as the comparison group. Female was also dichotomously coded with males as the referent group. Anxiety = Revised Child Manifest Anxiety Scale (RCMAS; Reynolds and Richmond 1985); Depression = short form of the Mood and Feelings Questionnaire (SMFQ; Angold et al. 1995)

**$p < 0.01$ and *$p < 0.05$

symptoms (Rutter et al. 2006). Meanwhile, our hypothesis that a heterotypic model of depression would emerge was only partially supported as both heterotypic and homotypic pathways for depression were identified.

These results reflect the principle of equifinality (Cicchetti et al. 1994) and suggest that both a homotypic (e.g., Keenan et al. 2009) and heterotypic (e.g., Rutter et al. 2006) explanation exist for adolescent depression. For the past two decades, research has yielded discrepant findings concerning the etiological role of childhood anxiety and depression in the emergence of adolescent depression (Keenan et al. 2009; McLaughlin and King 2015; Reinke and Ostrander 2008). Although we proposed that our methodological and analytical approach may resolve these discrepant findings, the truth may be that the developmental processes leading to adolescent depression are too diverse for the answer to reside within a singular childhood-adolescent depression pathway. Instead, our findings suggest that various pathways may lead to a single outcome. Typically, equifinality applies to why a certain constellation of risk factors may exist for some depressed patients but not others (e.g., Cicchetti and Toth 1998; Hyde et al. 2008). However, our findings show that equifinality is also reflected within prospective symptom expressions, with some individuals evidencing homotypic and others heterotypic symptom expressions over time. Disentangling these different pathways may lead to the identification of unique mediating processes that can lay the foundation for more targeted, personalized approaches to preventing and treating adolescent depression.

Developmental and Gender Differences

Comparisons between age and grade-based models suggested that chronological age best describes the year-to-year as well as the epoch-to-epoch changes in anxiety and depressive symptoms. Although there was no assessment of pubertal or other psychobiological processes relevant to development, the superiority of an age-based compared to a grade-based model suggests that the direct (e.g., neurobiological development; Andersen and Teicher 2008) or moderating (Hyde et al. 2008; Rudolph 2014) influence of intrapersonal development should not be discounted in studies of anxiety or depression. Despite anxiety typically having an earlier onset compared to depression (Kessler et al. 2005), our findings suggest that a shift in anxiety and depressive trajectories occur at a similar developmental point, age 12. Future research should investigate the presence of other "developmental knots" as both depression (Hankin et al. 1998; Lewinsohn and Essau 2002) and anxiety (Nelemans et al. 2014) symptoms may increase during mid to late adolescence (i.e., ages 15–18).

Finally, a few gender differences are worth noting. We found that girls experienced elevated anxiety symptoms in childhood and a sharper increase in adolescent anxiety symptoms. These findings are consistent with past research that identified gender differences emerging for general anxiety symptoms beginning by age 6 (Lewinsohn et al. 1998). Although our study did not reveal mean level gender differences in general anxiety symptoms in adolescence, girls' increasing trajectory of symptoms during this period is consistent with findings that adolescent females continue to be more at risk for anxiety (Rapee et al. 2009). Meanwhile, null gender differences in childhood depression and an increasing trajectory of adolescent depressive symptoms in girls is consistent with past research (Garber and Horowitz

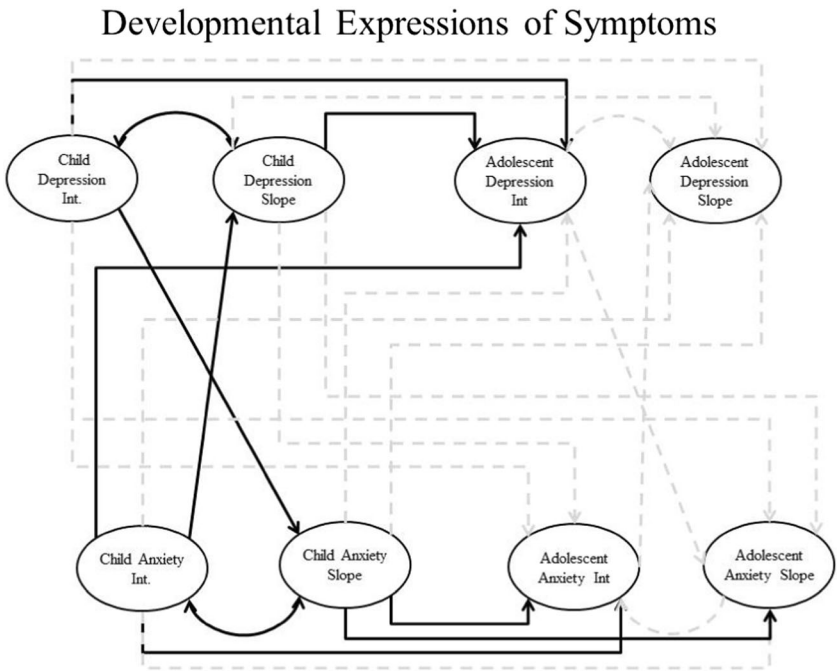

Fig. 4 Developmental model of the expression of anxiety and depressive symptoms. Significant paths ($p < 0.05$) are represented by solid black lines and nonsignificant paths ($p > 0.05$) are represented by dashed gray lines. Paths predicting anxiety and depressive symptoms are presented. In this model, specific paths were examined between all child symptom variables and all adolescent symptom variables. Additionally, correlations were examined between variables representing the same growth function (e.g., child intercepts) or, for endogenous variables, their disturbances (e.g., adolescent slopes). These correlations are not shown in order to enhance figure clarity. Correlations between disturbances of the same symptom type and period of growth (e.g., childhood depression intercept and slope) were also examined. Covariates of gender and race are not displayed in order to enhance figure clarity. Use of the term "child" and "adolescent" is based on age benchmarks as recommended by the National Institute of Child Health and Human Development (NICHD) Pediatric Terminology Harmonization Initiative

2002; Hankin and Abramson 2001). Interestingly, we did not find any evidence that the developmental knot varied for females versus males, suggesting that the growth of internalizing symptoms, as opposed to age of onset, more broadly begets mean level gender differences. Our findings reinforce past postulations (Cole et al. 2002) that static anxiety or depressive symptom scores may not be the best benchmark for predicting future risk.

Limitations, Implication, and Future Directions

Our findings should be considered within the context of notable limitations. First, only self-report measures were utilized. Importantly, past research shows that the use of multi-method and multi-informant approaches can add incremental validity to the assessment of psychosocial processes in youth (De Los Reyes et al. 2015; Ingram and Siegle 2009). It is important that future research replicate these findings using clinical interviews to better distinguish between pediatric depression and anxiety presentations. Second, we only investigated anxiety symptoms as a unitary construct. Anxiety is a heterogeneous class of internalizing symptoms that differentially relate to demographic characteristics (e.g., gender, age) and depressive symptoms (Cummings et al. 2014). Third, we did not assess pubertal development. Any assertion concerning symptom discontinuity being linked to puberty is speculative on our part. Fourth, our investigation of the developmental knot was exploratory in nature and future research is needed to confirm its placement at age 12. Last, we were unable to clarify exactly why childhood and adolescent symptoms were discontinuous. We discussed our findings within the context of a transition-linked turning point model (Graber and Brooks-Gunn 1996) however, it is also possible that internalizing symptoms serve a different function in childhood versus adolescence.

In 2016, the United States Preventative Services Task Force (USPSTF) made a dramatic shift in their recommendations for depression screening. Starting at age 12, universal depression screens were recommended in pediatric settings acknowledging with "high certainty" that there was a "net benefit" to monitoring depression at this age. Our findings support this assertion, as we found a continuous increase in depressive symptoms starting at age 12. As past research shows adolescent and adult depression to be continuous (Hankin et al. 1998; Rutter et al. 2006), the promise of simultaneously targeting current distress and prospective risk is empirically supported. However, the

Table 4 Covariance matrix for all observed variables

	Anx age 7	Anx age 8	Anx age 9	Anx age 10	Anx age 11	Anx age 12	Anx age 13	Anx age 14	Anx age 15	Dep age 7	Dep age 8	Dep age 9	Dep age 10	Dep age 11	Dep age 12	Dep age 13	Dep age 14	Dep age 15	Racial minority	Female
Anx age 7	46.27																			
Anx age 8	20.28	44.69																		
Anx age 9	16.50	22.18	43.04																	
Anx age 10	13.60	22.72	27.33	48.08																
Anx age 11	11.58	19.09	22.02	30.58	45.09															
Anx age 12	7.41	15.37	19.88	27.24	28.79	43.26														
Anx age 13	8.91	14.30	19.21	22.52	24.54	29.90	41.94													
Anx age 14	12.10	15.14	20.75	19.75	22.43	25.98	30.98	4458												
Anx age 15	9.76	16.62	19.21	16.35	17.91	21.97	27.78	34.02	47.46											
Dep age 7	2.05	1.64	1.20	1.62	1.11	0.51	0.88	0.88	0.72	0.45										
Dep age 8	1.24	2.61	1.40	1.63	1.29	1.05	1.03	0.83	123	0.17	0.39									
Dep age 9	0.73	1.64	2.43	1.97	1.41	1.34	1.17	1.20	1.02	0.13	0.15	0.33								
Dep age 10	0.75	1.42	1.63	2.84	1.97	1.63	1.29	1.12	0.98	0.13	0.14	0.17	0.35							
Dep age 11	0.24	1.23	1.36	1.93	2.52	1.88	1.57	1.17	1.06	0.06	0.10	0.13	0.18	0.29						
Dep age 12	0.38	1.00	1.30	1.50	1.55	2.15	1.82	1.62	1.60	0.01	0.08	0.09	0.11	0.15	0.24					
Dep age 13	0.24	0.77	1.08	1.33	1.32	1.64	2.29	1.81	1.90	0.06	0.07	0.07	0.09	0.11	0.13	0.24				
Dep age 14	0.67	0.97	1.43	1.37	1.35	1.64	2.02	2.73	2.37	0.05	0.06	0.09	0.09	0.09	0.12	0.16	0.30			
Dep age 15	0.60	1.19	1.35	1.34	1.38	1.65	2.03	2.45	3.32	0.05	0.09	0.07	0.09	0.10	0.15	0.10	0.23	0.39		
Racial minority	−0.01	0.07	−0.10	−0.17	0.13	−0.33	−0.20	−0.33	−0.13	−0.05	0.01	−0.01	0.03	0.02	−0.01	0.01	0.01	−0.02	0.97	
Female	0.26	0.40	0.46	0.32	0.46	0.36	0.76	0.97	1.26	−0.01	0.01	0.01	0.01	0.02	0.03	0.05	0.07	0.09	−0.02	0.25

Anx = Anxiety as measured by the Revised Child Manifest Anxiety Scale (RCMAS; Reynolds and Richmond 1985); Dep = Depression as measured by the short form of the Mood and Feelings Questionnaire (SMFQ; Angold et al. 1995). Racial Minority was dichotomously coded with white as the referent group and racial minority youth as the comparison group. Female was also dichotomously coded with males as the referent group

emergence of a discontinuous pattern of depression starting at age 12 suggests that adopting a similar approach for children may be limited. Instead, future research should aim to identify continuous indicators of risk and/or impairment that are independent of symptom expressions. These indicators will probably differ for adolescent depression stemming from an anxiotypic versus depressogenic organization. The identification of these multiple markers can lead to clinical and research protocols that better identify how childhood anxiety and childhood depression uniquely lead to depression outcomes in adolescence.

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Compliance with Ethical Standards

Conflict of Interest All authors declare that they have no conflict of interest.

Ethical Approval Institutional Review Board approval.

Informed Consent Informed consent was obtained for all study procedures.

References

Andersen, S. L., & Teicher, M. H. (2008). Stress, sensitive periods and maturational events in adolescent depression. *Trends in Neurosciences, 31*(4), 183–191.

Angold, A., Costello, E. J., Messer, S. C., & Pickles, A. (1995). Development of a short questionnaire for use in epidemiological studies of depression in children and adolescents. *International Journal of Methods in Psychiatric Research, 5*(4), 237–249.

Aune, T., & Stiles, T. C. (2009). The effects of depression and stressful life events on the development and maintenance of syndromal social anxiety: Sex and age differences. *Journal of Clinical Child & Adolescent Psychology, 38*(4), 501–512. https://doi.org/10.1080/15374410902976304.

Bittner, A., Egger, H. L., Erkanli, A., Costello, E. J., Foley, D. L., & Angold, A. (2007). What do childhood anxiety disorders predict? *Journal of Child Psychology and Psychiatry, 48*(12), 1174–1183. https://doi.org/10.1111/j.1469-7610.2007.01812.x.

Bollen, K. A., & Curran, P. J. (2004). Autoregressive latent trajectory (ALT) models a synthesis of two traditions. *Sociological Methods & Research, 32*(3), 336–383. https://doi.org/10.1177/0049124103260222.

Cheong, J., MacKinnon, D. P., & Khoo, S. T. (2003). Investigation of mediational processes using parallel process latent growth curve modeling. *Structural Equation Modeling, 10*(2), 238–262. https://doi.org/10.1207/S15328007SEM1002_5.

Chou, C. P., Yang, D., Pentz, M. A., & Hser, Y. I. (2004). Piecewise growth curve modeling approach for longitudinal prevention study. *Computational Statistics and Data Analysis, 46*(2), 213–225. https://doi.org/10.1016/S0167-9473(03)00149-X.

Cicchetti, D., & Toth, S. L. (1998). The development of depression in children and adolescents. *American Psychologist, 53*(2), 221–241. https://doi.org/10.1037/0003-066X.53.2.221.

Cicchetti, D., Rogosch, F. A., & Toth, S. L. (1994). A developmental psychopathology perspective on depression in children and adolescents. In W. M. Reynolds & H. F. Johnston (Eds.), *Handbook of depression in children and adolescents* (pp. 123–141). USA: Springer. https://doi.org/10.1007/978-1-4899-1510-8_7.

Cohen, J. R., Young, J. F., Gibb, B. E., Hankin, B. L., & Abela, J. R. (2014). Why are anxiety and depressive symptoms comorbid in youth? A multi-wave, longitudinal examination of competing etiological models. *Journal of Affective Disorders, 161*, 21–29. https://doi.org/10.1016/j.jad.2014.02.042.

Cole, D. A., Peeke, L. G., Martin, J. M., Truglio, R., & Seroczynski, A. D. (1998). A longitudinal look at the relation between depression and anxiety in children and adolescents. *Journal of Consulting and Clinical Psychology, 66*(3), 451–460.

Cole, D. A., Tram, J. M., Martin, J. M., Hoffman, K. B., Ruiz, M. D., Jacquez, F. M., & Maschman, T. L. (2002). Individual differences in the emergence of depressive symptoms in children and adolescents: a longitudinal investigation of parent and child reports. *Journal of Abnormal Psychology, 111*(1), 156–165. https://doi.org/10.1037/0021-843X.111.1.156.

Costello, E. J., Mustillo, S., Erkanli, A., Keeler, G., & Angold, A. (2003). Prevalence and development of psychiatric disorders in childhood and adolescence. *Archives of General Psychiatry, 60*(8), 837–844. https://doi.org/10.1001/archpsyc.60.8.837.

Cummings, C. M., Caporino, N. E., & Kendall, P. C. (2014). Comorbidity of anxiety and depression in children and adolescents: 20 years after. *Psychological Bulletin, 140*(3), 816–845. https://doi.org/10.1037/a0034733.

De Los Reyes, A., Augenstein, T. M., Wang, M., Thomas, S. A., Drabick, D. A., Burgers, D. E., & Rabinowitz, J. (2015). The validity of the multi-informant approach to assessing child and adolescent mental health. *Psychological Bulletin, 141*(4), 858–900. https://doi.org/10.1037/a0038498.

Duncan, T. E., Duncan, S. C., & Strycker, L. A. (2013). *An introduction to latent variable growth curve modeling: concepts, issues, and application*. New York: Routledge.

Enders, C. K., & Bandalos, D. L. (2001). The relative performance of full information maximum likelihood estimation for missing data in structural equation models. *Structural Equation Modeling, 8*(3), 430–457. https://doi.org/10.1207/S15328007SEM0803_5.

Flannery-Schroeder, E. C. (2006). Reducing anxiety to prevent depression. *American Journal of Preventive Medicine, 31*(6), 136–142. https://doi.org/10.1016/j.amepre.2006.07.006.

Garber, J., & Horowitz, J. L. (2002). Depression in Children. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression* (pp. 510–540). New York: Guilford.

Graber, J. A., & Brooks-Gunn, J. (1996). Transitions and turning points: navigating the passage from childhood through adolescence. *Developmental Psychology, 32*(4), 768–776. https://doi.org/10.1037/0012-1649.32.4.768.

Hale III, W. W., Raaijmakers, Q., Muris, P., & Meeus, W. (2008). Developmental trajectories of adolescent anxiety disorder symptoms: a 5-year prospective community study. *Journal of the American Academy of Child & Adolescent Psychiatry, 47*(5), 556–564. https://doi.org/10.1097/CHI.0b013e3181676583.

Hale III, W. W., Raaijmakers, Q. A., Muris, P., Van Hoof, A., & Meeus, W. H. (2009). One factor or two parallel processes? Comorbidity and development of adolescent anxiety and depressive disorder symptoms. *Journal of Child Psychology and Psychiatry, 50*(10), 1218–1226. https://doi.org/10.1111/j.1469-7610.2009.02115.x.

Hankin, B. L., & Abramson, L. Y. (2001). Development of gender differences in depression: An elaborated cognitive vulnerability–transactional stress theory. *Psychological Bulletin, 127*(6), 773–796. https://doi.org/10.1037/0033-2909.127.6.773.

Hankin, B. L., Abramson, L. Y., Moffitt, T. E., Silva, P. A., McGee, R., & Angell, K. E. (1998). Development of depression from preadolescence to young adulthood: emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology, 107*(1), 128–140. https://doi.org/10.1037/0021-843X.107.1.128.

Hankin, B. L., Snyder, H. R., Gulley, L. D., Schweizer, T. H., Bijttebier, P., et al. (2016). Understanding comorbidity among internalizing problems: integrating latent structural of models of psychopathology and risk mechanisms. *Development and Psychopathology, 28*, 987–1012. https://doi.org/10.1017/S0954579416000663.

Harrington, R., Rutter, M., & Fombonne, E. (1996). Developmental pathways in depression: multiple meanings, antecedents, and endpoints. *Development and Psychopathology, 8*(04), 601–616.

Hu, L. T., & Bentler, P. M. (1998). Fit indices in covariance structure modeling: sensitivity to underparameterized model misspecification. *Psychological Methods, 3*(4), 424–453. https://doi.org/10.1037/1082-989X.3.4.424.

Hyde, J. S., Mezulis, A. H., & Abramson, L. Y. (2008). The ABCs of depression: integrating affective, biological, and cognitive models to explain the emergence of the gender difference in depression. *Psychological Review, 115*(2), 291–313. https://doi.org/10.1037/0033-295X.115.2.291.

Ingram, R. E., & Siegle, G. J. (2009). Methodological issues in the study of depression. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression* (pp. 69–92). New York: Guilford.

Keenan, K., Feng, X., Hipwell, A., & Klostermann, S. (2009). Depression begets depression: Comparing the predictive utility of depression and anxiety symptoms to later depression. *Journal of Child Psychology and Psychiatry, 50*(9), 1167–1175. https://doi.org/10.1111/j.1469-7610.2009.02080.x.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62*(6), 593–602. https://doi.org/10.1001/archpsyc.62.6.593.

Klimes-Dougan, B., Long, J. D., Lee, C.-Y. S., Ronsaville, D. S., Gold, P. W., & Martinez, P. E. (2010). Continuity and cascade in offspring of bipolar parents: a longitudinal study of externalizing, internalizing, and thought problems. *Development and Psychopathology, 22*(4), 849–866. https://doi.org/10.1017/S0954579410000507.

Kohli, N., Hughes, J., Wang, C., Zopluoglu, C., & Davison, M. L. (2015). Fitting a linear–linear piecewise growth mixture model with unknown knots: a comparison of two common approaches to inference. *Psychological Methods, 20*(2), 259–275. https://doi.org/10.1037/met0000034.

Lahey, B. B., Zald, D. H., Hakes, J. K., Krueger, R. F., & Rathouz, P. J. (2014). Patterns of heterotypic continuity associated with the cross-sectional correlational structure of prevalent mental disorders in adults. *JAMA Psychiatry, 71*(9), 989–996.

Lau, J. Y., & Eley, T. C. (2008). Attributional style as a risk marker of genetic effects for adolescent depressive symptoms. *Journal of Abnormal Psychology, 117*(4), 849–859. https://doi.org/10.1037/a0013943.

Laursen, B., & Collins, W. A. (2009). Parent-child relationships during adolescence. In R. M. Lerner & L. Steinberg (Eds.), *Handbook of adolescent psychology* (pp. 3–42). New York: Wiley.

Leadbeater, B., Thompson, K., & Gruppuso, V. (2012). Co-occurring trajectories of symptoms of anxiety, depression, and oppositional defiance from adolescence to young adulthood. *Journal of Clinical Child & Adolescent Psychology, 41*(6), 719–730. https://doi.org/10.1080/15374416.2012.694608.

Lewinsohn, P. M., & Essau, C. A. (2002). Depression in adolescents. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression* (pp. 510–540). New York: Guilford.

Lewinsohn, P. M., Gotlib, I. H., Lewinsohn, M., Seeley, J. R., & Allen, N. B. (1998). Gender differences in anxiety disorders and anxiety symptoms in adolescents. *Journal of Abnormal Psychology, 107*(1), 109–117. https://doi.org/10.1037/0021-843X.107.1.109.

Mamey, M. R., Barbosa-Leiker, C., McPherson, S., Burns, G. L., Parks, C., & Roll, J. (2015). An application of analyzing the trajectories of two disorders: a parallel piecewise growth model of substance use and attention-deficit/hyperactivity disorder. *Experimental and Clinical Psychopharmacology, 23*(6), 422–427. https://doi.org/10.1037/pha0000047.

McLaughlin, K. A., & King, K. (2015). Developmental trajectories of anxiety and depression in early adolescence. *Journal of Abnormal Child Psychology, 43*(2), 311–323. https://doi.org/10.1007/s10802-014-9898-1.

Nelemans, S. A., Hale, W. W., Branje, S. J., Raaijmakers, Q. A., Frijns, T., van Lier, P. A., & Meeus, W. H. (2014). Heterogeneity in development of adolescent anxiety disorder symptoms in an 8-year longitudinal community study. *Development and Psychopathology, 26*(01), 181–202. https://doi.org/10.1017/S0954579413000503.

Olatunji, B. O., & Cole, D. A. (2009). The longitudinal structure of general and specific anxiety dimensions in children: testing a latent trait–state–occasion model. *Psychological Assessment, 21*(3), 412–424. https://doi.org/10.1037/a0016206.

Rapee, R. M., Schniering, C. A., & Hudson, J. L. (2009). Anxiety disorders during childhood and adolescence: origins and treatment. *Annual Review of Clinical Psychology, 5*, 311–341. https://doi.org/10.1146/annurev.clinpsy.032408.153628.

Reinke, W. M., & Ostrander, R. (2008). Heterotypic and homotypic continuity: the moderating effects of age and gender. *Journal of Abnormal Child Psychology, 36*(7), 1109–1121. https://doi.org/10.1007/s10802-008-9236-6.

Reynolds, C. R., & Richmond, B. O. (1978). What I think and feel: a revised measure of children's manifest anxiety. *Journal of Abnormal Child Psychology, 6*(2), 271–280. https://doi.org/10.1007/BF00919131.

Reynolds, C. R., & Richmond, B. O. (1985). *Revised children's manifest anxiety scale*. Los Angeles: Western Psychological Services.

Rudolph, K. D. (2014). Puberty as a developmental context of risk for psychopathology. In M. Lewis & K. D. Rudolph (Eds.), *Handbook of developmental psychopathology* (pp. 331–354). New York: Springer. https://doi.org/10.1007/978-1-4614-9608-3_17.

Rudolph, K. D., Lansford, J. L., & Rodkin, P. C. (2016). Interpersonal theories of developmental psychopathology. In D. Cichetti (Ed.), *Developmental psychopathology*. New York: Wiley.

Rutter, M., Kim-Cohen, J., & Maughan, B. (2006). Continuities and discontinuities in psychopathology between childhood and adult life. *Journal of Child Psychology and Psychiatry, 47*(3–4), 276–295. https://doi.org/10.1111/j.1469-7610.2006.01614.x.

Schulenberg, J. E., Maggs, J. L., & O'Malley, P. M. (2003). How and why the understanding of developmental continuity and discontinuity is important. In J. T. Mortimer & M. J. Shanahan (Eds.), *Handbook of the life course* (pp. 413–436). New York: Springer. https://doi.org/10.1007/978-0-306-48247-2_19.

Seligman, L. D., Ollendick, T. H., Langley, A. K., & Baldacci, H. B. (2004). The utility of measures of child and adolescent anxiety: a meta-analytic review of the Revised Children's Manifest Anxiety Scale, the State–Trait Anxiety Inventory for Children, and the Child Behavior Checklist. *Journal of Clinical Child and Adolescent Psychology, 33*(3), 557–565. https://doi.org/10.1207/s15374424jccp3303_13.

Sterba, S. K., Prinstein, M. J., & Cox, M. J. (2007). Trajectories of internalizing problems across childhood: heterogeneity, external validity,

and gender differences. *Development and Psychopathology, 19*(02), 345–366. https://doi.org/10.1017/S0954579407070174.

Thapar, A., & McGuffin, P. (1998). Validity of the shortened Mood and Feelings Questionnaire in a community sample of children and adolescents: a preliminary research note. *Psychiatry Research, 81*(2), 259–268. https://doi.org/10.1016/S0165-1781(98)00073-0.

United States Preventative Task Force (USPTF) (2016) Depression in adults: screening, depression in children and adolescents: screening. Retrieved from www.uspreventiveservicestaskforce.org. Accessed 24 Feb 2016.

Van Oort, F. V. A., Greaves-Lord, K., Verhulst, F. C., Ormel, J., & Huizink, A. C. (2009). The developmental course of anxiety symptoms during adolescence: the TRAILS study. *Journal of Child Psychology and Psychiatry, 50*(10), 1209–1217. https://doi.org/10.1111/j.1469-7610.2009.02092.x.

Van Oort, F. V. A., Greaves-Lord, K., Ormel, J., Verhulst, F. C., & Huizink, A. C. (2011). Risk indicators of anxiety throughout adolescence: the TRAILS study. *Depression and Anxiety, 28*(6), 485–494. https://doi.org/10.1002/da.20818.

Weems, C. F. (2008). Developmental trajectories of childhood anxiety: identifying continuity and change in anxious emotion. *Developmental Review, 28*(4), 488–502. https://doi.org/10.1016/j.dr.2008.01.001.

Weissman, M. M., Wolk, S., Goldstein, R. B., Moreau, D., Adams, P., Greenwald, S., et al. (1999). Depressed adolescents grown up. *JAMA, 281*(18), 1707–1713.

Wisniewski, J. J., Genshaft, J. L., Mulick, J. A., & Coury, D. L. (1987). Test-retest reliability of the Revised Children's Manifest Anxiety Scale. *Perceptual and Motor Skills, 65*(1), 67–70. https://doi.org/10.2466/pms.1987.65.1.67.