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Development's Tortoise and Hare: Pubertal Timing, Pubertal Tempo, and Depressive Symptoms in Boys and Girls

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Abstract

Although the sequence of pubertal maturation remains consistent across most individuals, the timing and tempo of development fluctuate widely. While past research has largely focused on the sequelae of pubertal timing, a faster tempo of maturation might also present special challenges to children for acclimating to new biological and social milestones. Using latent growth curve modeling, the present study investigated how pubertal tempo and pubertal timing predicted depressive symptoms over a 4-year period in a sample of children recruited from New York City area public schools. Rate of intraindividual change in parent-reported Tanner stages was used as an index of pubertal tempo, and more advanced Tanner development at an earlier chronological age was used as an index of pubertal timing. For girls ($N = 138$, $M = 8.86$ years old at Time 1), pubertal timing emerged as the most salient factor, and the tempo at which girls progressed through puberty was not significant. In boys ($N = 128$, $M = 9.61$ years old at Time 1), both timing and tempo of development were significant; notably, however, the effects of pubertal tempo were stronger than those of timing. These findings highlight the need to consider multiple sources of individual variability in pubertal development and suggest different pubertal challenges for boys and girls.

Keywords

puberty; gender differences; depression; pubertal timing; pubertal tempo

The hare darted out of sight almost at once. ... The tortoise plodded on and plodded on.—Aesop, *The Hare and the Tortoise*

Nearly a half-century ago, Marshall and Tanner categorized secondary sexual changes occurring at puberty—specifically breast development in girls, genital development in boys, and pubic hair development in both genders—into five stages of development (Marshall & Tanner, 1969, 1970; Tanner, 1962). Stage 1 indexed no visible signs of maturation, and Stage 5 indexed a full level of maturation, consistent with adult physical stature. Marshall

and Tanner observed considerable fluctuations in children's progression through these stages. In particular, they recorded two separate sources of variation: (a) *pubertal timing*, which they described as "variation in the chronological age at which adolescence begins and different stages of physical maturity are reached" and (b) *pubertal tempo*, which they described as "variation in the time taken to pass through the various stages of development." (Marshall & Tanner, 1969, p. 291, and Marshall & Tanner, 1970, p. 14, respectively).

Since the publication of Marshall and Tanner's seminal work, psychologists have been intrigued by individual differences in the process of maturation and how these might relate to later health and emotional well-being (see Mendle, Turkheimer, & Emery, 2007, for a review). While the pubertal timing literature is expansive, less is known about the developmental impact of pubertal tempo. The present article aims to contribute to a broader knowledge of the salience of pubertal tempo in psychosocial adjustment, specifically investigating depressive symptoms in early adolescence. As children differ from each other both in their timing and in their tempo of maturation, we contrast how pubertal timing and pubertal tempo each predict trajectories of depressive symptoms in early adolescence.

Pubertal Timing

The timing of puberty, particularly in girls, stands out as one of the most well-researched and well-replicated antecedents of adolescent difficulties. Early pubertal timing in girls has been associated with risk in multiple functional domains including psychological (i.e., depression, anxiety, disordered eating, delinquency; Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997; Stice, Presnell, & Bearman, 2001; Wichstrom, 2000;); medical (i.e., human papillomavirus infection, obesity, teenage pregnancy; Fisher, Rosenfeld, & Burk, 1991; Udry, 1979; Wellens et al., 1992), and academic (i.e., truancy, academic difficulties, lack of pursuit of higher education; Stattin & Magnusson, 1990). Of particular relevance to the current study, early maturation, across a variety of measurement modalities and samples, has been associated with higher levels of depressive symptoms, poor self-esteem, suicide attempts, and *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., American Psychiatric Association, 1994) criteria for major depressive disorder (Ge, Conger, & Elder, 2001; Graber et al., 1997; Patton et al., 1996; Stice et al., 2001).

The predominant explanation for the association between early pubertal timing and subsequent psychological outcomes is the *maturation disparity hypothesis* (reviewed in Ge & Natsuaki, 2009), which references the mismatch between physical and emotional development. As children mature, they are faced with a startling array of new challenges. These include new parental expectations, burgeoning feelings of sexual attraction, hormone-related emotional lability, and shifting peer relationships (Brooks-Gunn & Warren, 1985; McClintock & Herdt, 1996; Paikoff & Brooks-Gunn, 1991; Steinberg, 1987). Because physical and emotional development do not necessarily occur in synchrony, earlier maturing children are forced to grapple with these new stressors with fewer resources than peers who achieve the same developmental milestones at a later chronological age (Ge, Conger, & Elder, 1996, 2001; Petersen & Crockett, 1985; Petersen, Sarigiani, & Kennedy, 1991; Stattin & Magnusson, 1990). The maturation disparity hypothesis has been used to explain higher rates of psychopathology and problem behavior among early maturing children of both genders (e.g., Haynie, 2003; Weichold, Silbereisen, & Schmitt-Rodermund, 2003) and is sometimes alternatively termed the *stage termination hypothesis* (e.g., Petersen & Taylor, 1980).

Whereas in the literature pubertal timing is consistently associated with a wide gamut of negative outcomes in girls, the literature on pubertal timing for boys lacks the breadth and replicable pattern of results obtained for girls (Ge et al., 2001). Findings are particularly

inconsistent with regard to internalizing disorders. Some studies show early maturing boys experience elevations in negative affect, anxiety, and depression relative to later developing peers (Ge et al., 2001; Susman, Dorn, and Chrousos, 1991), while others implicate both early and late development (e.g., “off-time” maturation) with high rates of internalizing problems (Alsaker, 1992). Still, other studies show no correlation between pubertal timing and internalizing symptoms (Kaltiala-Heino, Marttunen, Rantanen, & Rimpela, 2003). An additional cluster of work suggests more positive social sequelae for early maturers, due to the advantages of increased body size and mass for athletics (Jones, 1965; Jones & Bayley, 1950). Late maturers are conversely believed to be prone to poor self-esteem and frequent self-derogation although Ge et al. (2001) cautioned that this is a largely “historical” perspective in need of revision.

The inconsistency in the pubertal timing literature for boys is likely due, in part, to the absence of a “hallmark” milestone for puberty that is analogous to menarche in girls. Age at menarche is among the more common measures for determining pubertal timing in girls. (Later, we discuss age at menarche as an indicator of pubertal timing in more detail.) Spermatarche, or first ejaculation, could plausibly be considered a comparable milestone to menarche, but there is greater Tanner stage variability when boys reach spermatarche than is the case for girls reaching menarche (Dorn, Dahl, Woodward, & Biro, 2006), and boys, moreover, often do not feel comfortable reporting spermatarche (Downs & Fuller, 1991; Gaddis & Brooks-Gunn, 1985). The absence of an easily assessed, reliably reported indicator of pubertal timing in boys further eliminates the possibility of retrospective studies, which are relatively common in investigations of timing of menarche and psychological outcomes (e.g., Fairburn, Welch, Doll, Davies, & O'Connor, 1997). This contributes to less overall understanding of how individual differences in the timing of maturation might predict relevant psychosocial outcomes in boys.

Pubertal Tempo

While pubertal timing indexes a level of maturation relative to *others* of the same gender at the same chronological age, pubertal tempo is defined with reference to a child's *own* previous development. Some people mature comparatively rapidly (to use an analogy from Aesop, they are developmental hares), whereas others have a slower, more gradual progression through puberty (e.g., developmental tortoises). Pubertal tempo is a measure of this rate of intraindividual change over time. In Marshall and Tanner's investigation, the magnitude of individual variation in time taken to pass through the five stages of pubertal development was not insignificant. For breast development, the mean interval between Stage 2 and Stage 5 was 4 years, but the extremes ranged between 1.51 years (5th percentile) and nearly 9 years (95th percentile). For boys, the mean interval between Stage 2 and Stage 5 for genital development was approximately 3 years but ranged between 1.86 and 4.72 years (Marshall & Tanner, 1969, 1970). Marshall and Tanner, moreover, observed that timing and tempo appeared to be relatively independent of each other and that an earlier timing of pubertal onset was not systematically associated with either a more rapid or more dilatory maturational tempo.

Most of what is known about pubertal tempo comes from medical studies, whose goals are simply to observe and report on secular trends in maturation (e.g., Biro et al., 2006; Pantiotiou et al., 2008). Collectively, these studies imply that pubertal tempo may be a better predictor of age at menarche than timing of pubertal onset (Papadimitriou, Fretzayas, & Nicolaidou, 2006). Within psychological research, a single study has linked intraindividual change to children's psychosocial outcomes. Ge et al. (2003) investigated puberty and symptoms of major depression in African American children. In this study, the authors measured pubertal timing and what they termed *pubertal change* at two time points

2 years apart. Children who progressed through more than 1 standard deviation of pubertal milestones at Time 2 relative to their Time 1 status were considered to have experienced *accelerated pubertal change*. For girls, early pubertal timing significantly predicted a more severe level of depressive symptoms, but there was no effect of pubertal change. For boys, early pubertal timing predicted increased symptoms of depression at Time 1 and accelerated pubertal change predicted increased symptoms at Time 2. Ge and colleagues never explicitly discussed pubertal tempo, interpreting their results by considering puberty a stressful life transition that all children will experience. This was consistent with their study hypotheses, and, more relevantly, two assessments of puberty cannot provide a true gauge of pubertal tempo. Nevertheless, this work provided preliminary evidence that timing and change both predict depressive symptoms and may do so differently for boys and girls.

There are naturally methodological complexities inherent in disentangling pubertal timing and pubertal tempo into separate processes. Because pubertal tempo is defined as the rate of intraindividual change, it requires detailed longitudinal data on participants in a specific age range. In addition, measures such as age at menarche necessarily reflect some degree of measurement error, as they actually conflate pubertal timing and pubertal tempo. Because time from pubertal onset to menarche varies across individuals (Biro et al., 2006), it would be possible for a girl with a rapid pubertal tempo to experience an average-to-late onset of maturation but comparatively early menarche. This would not be reflected in studies in which age at menarche is used as a proxy for pubertal timing. Such variability would also not be reflected in any study, of either boys or girls, in which pubertal status relative to peers at a single time point is selected as being indicative of the overall trajectory of pubertal timing.

The Maturation Compression Hypothesis

We posited that variation in pubertal tempo is a developmentally salient, but understudied, predictor of psychological well-being. In particular, we were interested in the psychological difficulties that might be associated with a more rapid pubertal tempo. A logic similar to that of the maturation disparity hypothesis might explain why progressing through puberty quickly could be problematic. A faster than average developmental tempo might demand an improbably swift assimilation of new biological and social milestones; evoke different and potentially stronger reactions from parents, peers, and teachers; and compress the time available for resolution of the developmental tasks of the pre-adolescent period. In contrast, a comparatively slower developmental progression might be less noticed or attended to both by others and by the maturing child. We refer to this consideration of the challenges of a shorter pubertal duration as the *maturation compression hypothesis*.

In the present article, we sought to provide a preliminary test of the maturation compression hypothesis. Specifically, we tested three main components: (a) that there are significant individual differences in both pubertal timing and pubertal tempo; (b) that pubertal timing and pubertal tempo each independently predict the development of depressive symptoms; and (c) that the effects of pubertal timing and tempo on the development of depressive symptoms differ across gender. Because it targets both interindividual differences and intraindividual change, our study represents a clear advance in understanding how dimensions of pubertal development are associated with psychological distress.

Method

Procedure and Participants

Participants were drawn from the Girls and Boys Health and Development (GBHD) Project, a longitudinal study designed to examine the social and emotional correlates of biological

development during late childhood and early puberty. The GBHD comprises a community sample of families from the New York City metropolitan area. As the project originally targeted girls' development and was later expanded to include boys, girls and boys participated separately in two sets of data collection, each collection spanning a 4-year period and including four separate waves of assessments. All assessments and procedures were approved by the institutional review board (IRB) at Columbia University. Girls and their families were recruited in 1995–1996 and boys and their families were recruited in 2001–2002 through public elementary schools. Schools were targeted for recruitment on the basis of a high level of ethnic diversity and location in working- and middle-class neighborhoods in Brooklyn, Queens, and Yonkers. A series of flyers describing the study were distributed to the children in school when girls were in third grade and boys were in fourth grade. This grade difference was intentional, to account for the later onset of pubertal development in boys.

Parents (typically mothers) returned cards with their contact information if they were interested in learning more about the project. During a follow-up call, research assistants extensively apprised parents of the study requirements. Parents who wanted to participate were scheduled for a home visit in the late afternoon after school. Because flyers were passed out in multiple waves to the children, a precise count of how many families received this information could not be established. Of those who participated in a follow-up call, 85% enrolled in the study. Only parents who indicated that they (and their children) would be able to complete the project in English were enrolled. Mothers of 100% of the girls and 99% of the boys reported that English was spoken at home. As schools were selected on the basis of demographic characteristics designed to increase sample diversity, approximately 14% of mothers reported that another language in addition to English was spoken at home; most often this language was Spanish.

At the start of the home visit, mothers signed informed consent forms on their own behalf and for their child's participation. Children additionally gave assent for their participation. Two data collectors conducted each home visit. Only female data collectors conducted home visits with girls and their mothers, while male and female data collectors conducted home visits with boys and their mothers. For boys, the male data collector was assigned to interactions with the child, while the other data collector interacted with the child's mother. At least one data collector was matched to family ethnicity whenever possible.

During the home visit, mothers completed a structured interview, providing information on demographic variables, family composition, family medical and psychological history, and children's temperament, behavior, and emotional states. Both mothers and children additionally completed a variety of standard, paper- and -pencil measures assessing psychological distress, problem behavior, and social functioning. Under the guidelines of the IRB, children who reported clinically significant levels on these measures were recontacted and offered referrals for services. In addition, referrals were given to any parent interested in this information, regardless of child or parent survey reports. Children received a gift (e.g., T-shirt, tote bag, miniature football) for their participation, and mothers were paid \$60 at baseline and \$75 in subsequent years for their family's participation in the home visit and other study protocols.

Following the initial home visit, participants were asked to complete three subsequent assessments, including repeated use of all paper- and -pencil questionnaire items. For girls, follow-up assessments were initially planned for 6-month intervals. As protocols were time intensive for families, this resulted in a reduced sample size at Wave 2. Subsequent assessments (Waves 3 and 4) were therefore conducted at approximately 1-year intervals. The number of girls who participated at each time point was 138 (Wave 1), 78 (Wave 2), 92

(Wave 3), and 116 (Wave 4). At Wave 4 assessment, 19.5% of the original sample had been lost.

As indicated, girls were recruited from third grade classrooms and were 8.86 years old on average at Wave 1 ($SD = 0.49$), with their age ranging from 7.6 years to 10.2 years. Of the 138 girls, 63 (46%) were European American, 51 (37%) were African American, and 24 (17%) were Hispanic/Latina. At the start of the study, 58.7% of girls lived in two-parent households. At successive assessments, 9%, 3%, and 6% of mothers (respectively) reported changes in their marital or significant partner status.

Boys, recruited from fourth grade classrooms, were 9.61 years old on average at Wave 1 ($SD = 0.70$), with their age ranging from 8.1 years to 12.0 years. Of the 128 boys, 40 (31%) were European American, 55 (43%) were African American, and 33 (26%) were Hispanic/Latino. At Wave 1, 53.1% of boys lived in two-parent households. At successive assessments, 10%, 7%, and 9% of mothers (respectively) reported changes in their marital or significant partner status.

For boys, follow-up assessments were conducted annually. Due to concerns about participant burnout, the home visit at Time 4 was replaced with a structured telephone interview, conducted separately for boys and their mothers, and with mailed questionnaires. The number of boys who participated at each time point was 128 (Wave 1), 103 (Wave 2), 91 (Wave 3), and 82 (Wave 4), corresponding to a loss of 35.9% of the sample.

With regard to sample attrition, girls who participated in all assessments did not significantly differ in their baseline pubertal status from girls who missed one or more assessments. Girls who participated at all time points reported significantly lower baseline levels of depression on the Children's Depression Inventory (CDI; Kovacs, 1985) at Time 1 ($M = 5.36$, $SD = 4.86$) than girls who missed one or more assessments ($M = 9.35$, $SD = 7.71$); were more likely to be European American (69.8% vs. 30.2%), and less likely to be African American (29.9% vs. 45.9%). Boys who participated in all assessments did not significantly differ from boys who missed one or more assessments with regards to baseline pubertal status or to baseline depressive symptoms. However, boys who missed one or more assessments were significantly different with regards to race/ethnicity (15.2% European American for boys who missed at least one assessment vs. 40.2% European American for boys who participated in all assessments). As will be described later in more detail, we used full information maximum likelihood our analytic plan to deal with missing data and response bias.

Measures

Demographic data—Children reported their date of birth, and exact age was calculated from the date of home visit. During each follow-up assessment, mothers reported family characteristics, child ethnicity, their own education and occupational status, and the education and occupational status of any other parent/caregiver in the household. We used these responses to construct a measure of family socioeconomic status (SES) based on the Hollingshead Scale, with the standard scoring accounting for different household types (Hollingshead, 1975). The range of possible scores for girls' family SES was 7.5–65.0 at Wave 1 ($M = 37.50$, $SD = 14.55$). The mean Hollingshead score equated to a high school diploma and employment as clerical workers, sales workers, or owners of small businesses. In subsequent years, Hollingshead scores were comparable: $M = 35.99$ at Wave 2, $M = 36.76$ at Wave 3, and $M = 38.30$ at Wave 4. For boys, the range of family SES was 9.0–66.0 at Wave 1 ($M = 36.50$, $SD = 13.88$), which represents the same employment and education level as reported by girls' families. Hollingshead scores for boys' families remained

comparable across waves: $M = 38.24$ at Wave 2, $M = 36.59$ at Wave 3, and $M = 37.85$ at Wave 4.

Pubertal status—At each wave of data collection, mothers were provided with schematic drawings representing the five Tanner stages of pubertal maturation and asked to circle the figure that looked most like their child (Morris & Udry, 1980). Tanner Stage drawings provide a method for categorizing the status of physical development based on external primary and secondary sex characteristics, where Stage 1 represents no visible signs of pubertal development and Stage 5 indicates a level of maturation consistent with adult status. In the current study, mothers of children of both genders were asked to complete Tanner ratings for pubic hair development and either breast or genital development (depending on child gender). If a mother indicated that she did not have sufficient knowledge of the child's current pubertal status, the child completed the pubertal ratings using the same line drawings used by the mother. For boys at Wave 1, all ratings were completed by the mother. There were 13 boys who reported on their own pubertal status at Wave 2 (13%), 17 at Wave 3 (19.3%), and 8 at Wave 4 (17%). Boys' own ratings of level of pubertal development were strongly and positively correlated with their mothers' ratings at the previous assessment wave (polychoric $r = .63$ at Wave 3 and 0.59 at Wave 4). For girls, all ratings at Waves 1–3 were completed by the mother, and eight girls rated their own pubertal status at Wave 4 (7.4%). As with the boys' ratings, girls' own ratings of their pubertal status at Wave 4 were strongly and positively correlated with their mothers' ratings at Wave 3 (polychoric $r = .79$). The distribution of Tanner stages by gender at Waves 1–4 is shown in Tables 1 and 2.

For the current analyses, we used the Tanner stage assessments of pubic hair growth in girls and genital development in boys. These were chosen in accordance with the following considerations from Dorn et al. (2006): (a) averaging Tanner scores for different pubertal characteristics to create a single measure conflates divergent hormonal processes; (b) “testicular volume in boys is a particularly good choice for research if one is interested in knowing when pubertal processes begin” (p. 40); and (c) reports of pubic hair development in girls are generally more accurate than reports of breast development, due to difficulty distinguishing adipose tissue from breast tissue without palpation by a trained examiner (e.g., Bonat, Pathomvanich, Keil, Field, & Yanovski, 2002; Kaplowitz & Oberfield, 1999).

In general, maternal accuracy of Tanner ratings varies when compared with physician examination (Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987; Dorn, Susman, Nottelmann, Inoff-Germain, & Chrousos, 1990). In our sample, 41% of girls reported that they had reached menarche at Wave 4. Of these girls, 75.5% were rated by mothers as being at Tanner Stage 4 or 5 for pubic hair development. For girls who reported they had not reached menarche, 25% were rated as being at Tanner Stage 4 and none at Tanner Stage 5. Although individual indicators of puberty are not perfectly correlated within girls, these associations suggest that there was convergent validity in maternal ratings by Wave 4. Moreover, these maternal ratings are consistent with Marshall & Tanner's (1969) findings that 77% of participants achieved menarche at either Stage 4 or 5 of pubic hair development.

Pubertal timing and tempo—Pubertal timing was operationalized as a latent factor representing whether level of Tanner development of a child was more or less advanced than that of other participants at the beginning of the study period. As can be seen in Tables 1 and 2, most children were still at Tanner Stage 1 at Wave 1. Therefore, children with a more advanced Tanner stage at the initiation of the study were the earliest maturing children. Pubertal tempo was operationalized as a latent factor representing how quickly a child progressed through Tanner stages over the study period. The latent factors representing pubertal timing and tempo are described in more detail in the Analyses section.

Depressive symptoms—Participants completed the CDI at each wave. The CDI has been adapted from the Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) for use with children between the ages of 7 and 17 years. For each item on the CDI, three statements reflecting depressive symptoms are provided and participants endorse the statement that best describes their current mood. Each statement is on a 0–2 scale, and the 26 items are summed with higher scores indicating greater endorsement of depressive symptoms. The CDI demonstrated good reliability in the current sample (Cronbach's $\alpha = .81$ at Wave 1, $.86$ at Wave 2, $.84$ at Wave 3, and $.87$ at Wave 4). A score of 19 has typically been used to signify clinically relevant levels of depressions. Across the four waves, girls' depression scores ranged from 0 to 34 ($M = 6.58$, $SD = 6.38$). In boys, depression scores ranged from 0 to 39 ($M = 6.40$, $SD = 5.65$).

Analyses

All analyses were modeled with the software program Mplus (Muthén & Muthén, 1998–2009). Full information maximum likelihood (FIML) was used to account for missing data, as this has been recommended as the preferred method for analysis of missing data (Schafer & Graham, 2002). As noted in Wothke (1998, p. 224), the FIML method “uses all the information of the observed data, including information about the mean and variance for the missing portion of the variable, given the observed portion of other variables.” Nested models were compared with each other using the chi-square difference test. Differences in model chi squares are themselves distributed as chi squares, with degrees of freedom equal to the difference between the models' degrees of freedom. In all models, we controlled for potential racial and ethnic differences in pubertal timing, pubertal tempo, and depressive symptoms by including two dummy-coded variables for African American race and Hispanic ethnicity as statistical covariates.

Test 1: Latent Growth Models (LGM) of Pubertal Development in Boys and Girls to Estimate Pubertal Timing and Tempo

Our first step was to establish that participants in our sample exhibited significant individual differences in pubertal timing and pubertal tempo. We modeled timing and tempo of pubertal development, separately for boys and girls, using latent growth models (LGM; McArdle & Nesselroade, 2002; Meredith & Tisak, 1990). Analyzing longitudinal data with LGM allows one to characterize both *intraindividual* change and *interindividual* differences in change (McArdle & Nesselroade, 2002), in accordance with the major goals of longitudinal research (Baltes & Nesselroade, 1979). The basic latent growth curve model can be written as follows (Ferrer, Hamagami, & McArdle, 2004):

$$y_{it} = \eta_{1i} + \lambda_t * \eta_{2i} + e_{it}, \quad (1)$$

where η_{1i} is a latent factor representing the initial level (often referred to as the *latent intercept*) and η_{2i} is a latent factor representing the *slope* of change over time. In a LGM of puberty, the latent intercept factor represents *pubertal timing*. Children with a higher latent intercept have higher pubertal status at the earliest age of observation; that is, they are early maturing relative to their peers. The latent slope factor represents *pubertal tempo*: Children with a higher latent slope are predicted to increase in pubertal status more quickly over time.

In addition, λ_t is a vector of age-specific basis coefficients representing the shape of change over time. Constraining the values of λ_t constitutes a test of specific hypotheses regarding the shape of change. For example, if the values of λ_t were fixed to be zero at all time points, the implied shape would be a flat line (a “no-growth” model). A no-growth model, obviously, is a highly implausible account of puberty. Rather, we constrained the values of

λ_t to increase linearly with age (0, 1, 2, 3, and so on). At first glance, a linear model may seem inappropriate for pubertal development: Over an entire lifespan, the mean relation between pubertal status and age is, of course, S-shaped, with nearly everyone at Tanner Stage 1 in childhood and everyone reaching a plateau at Tanner Stage 5 in adulthood. Over the ages observed in the current study, however, the observed relation between mean pubertal status and age was indeed approximately linear, as shown in Figure 1.

Finally, e_t is a vector of time-specific (or age-specific) residual errors. For each individual i at each age t , a latent, continuous, normally distributed factor (y_{ti}) was assumed to underlie the observed ordinal (Tanner stage) data (Y_{ti}), such that $Y_t = c$ (representing the category of the ordinal data, i.e., Tanner stage), if $\tau_c < y_t \leq \tau_{c+1}$.

In the current analyses, four thresholds (the number of ordinal data categories minus one) were estimated, and the thresholds were constrained to be invariant across ages. In order to estimate the threshold factors freely, we had to fix the mean of the latent intercept factor to zero.

The full LGM for the pubertal development model is illustrated in Figure 2 (race/ethnicity regressions not depicted). For all LGM, we controlled for the race/ethnicity by regressing both the latent intercept and latent slope on two dummy-coded variables corresponding to African American or Hispanic race/ethnicity, with European American children as the control group. As can be seen in Figure 1, mean age trends in Tanner stages overlapped considerably across racial/ethnic groups, with a trend for African American children to have higher pubertal status and European American children to have lower pubertal status at each age in both boys and girls.

Test 2: Dual LGM of Puberty and Depressive Symptoms

We next fit dual LGMs to test the relation among pubertal timing, pubertal tempo, and symptoms of depression in early adolescence. Again, models were fit separately for boys and girls. The full dual LGM for boys is shown in Figure 3; the model for girls was identical, except the age range was 8–13 years. The left side of Figure 3 depicts the LGM for pubertal development, and it is identical to the model we have described. The right side of Figure 3 depicts the LGM for depressive symptoms. Because the observed depressive symptoms were distributed continuously, the lowest level of the model—linking the observed data to an underlying continuous distribution—was unnecessary for the LGM of depressive symptoms.

The key portions of the dual LGM analyses were the regressions from the latent growth factors of depressive symptoms to the latent growth factors of puberty. The first regression, labeled β_{X1-Y1} , tested whether children who had higher initial pubertal status (i.e., were early maturing) also showed more depressive symptoms at the start of the study. This can be thought of as an effect of pubertal *timing*. The second regression, labeled β_{X1-Y2} , tested whether children who were early maturing also showed greater changes in depressive symptoms from ages 8 to 14. This can also be thought of as an effect of pubertal timing. In contrast, the third and fourth regressions, labeled β_{X2-Y1} and β_{X2-Y2} , tested whether children who matured more rapidly than their peers demonstrated higher overall levels and greater increases in depressive symptoms, respectively. These effects can be thought of as effects of pubertal *tempo*.

In all dual-process LGMs, we also controlled for race/ethnicity by regressing the latent intercept and latent slope factors for both pubertal development and depressive symptoms onto two dummy-coded variables representing African American and Hispanic race/

ethnicity. The race/ethnicity regressions are not shown in Figure 2 for the sake of illustrative clarity.

Test 3: Multiple-Group LGM of Gender Differences

To test if the relations of pubertal timing and tempo with depressive symptoms differed across boys and girls, we fit a series of multiple-group LGMs to analyze data from boys and girls in a single model. The first and second set of models in this series tested which growth curve parameters for pubertal development and depressive symptoms, respectively, could be constrained to be equal for boys and girls. We then combined the most parsimonious models for gender differences in pubertal development and depressive symptoms in order to test the key parameters of interest: whether the effects of pubertal timing and tempo on depressive symptoms differed between boys and girls. This multiple-group LGM was compared with a reduced model in which the effects of pubertal timing and tempo were constrained to be equal across genders.

Results

Test 1: Are There Individual Differences in Both Pubertal Timing and Pubertal Tempo?

Results from LGM of timing and tempo of pubertal development are shown in Table 3. For both boys and girls, the full LGM (Figure 2) was compared with a more restricted model in which the variance in the linear slope (Ψ_{22}) was fixed to zero. In girls, the restricted model fit significantly worse ($\Delta\chi^2 = 8.41$, $\Delta df = 2$, $p = .02$) than the full model. In boys, the restricted model also fit significantly worse ($\Delta\chi^2 = 66.72$, $\Delta df = 2$, $p < .001$) than the full model. These results indicate that there are indeed significant individual differences in the tempo of pubertal development.

For boys, the mean slope equaled 2.12 ($SD = 2.14$). This can be interpreted in terms of the estimated thresholds for the categorical Tanner stage variables. For example, the threshold for Tanner Stage 4 was estimated to be 10.17; thus, a boy with an average pubertal tempo would achieve Tanner Stage 4 at around 13 years old, or almost 5 years from the beginning of the study ($10.17/2.12 = 4.80$ years). In contrast, a boy whose pubertal tempo was 1 standard deviation above the mean pubertal tempo would achieve Tanner Stage 4 in only 3 years ($10.17/3.58 = 2.84$ years), around age 11. For girls, the mean slope equaled 3.29 ($SD = 1.06$). Thus, a girl with average pubertal tempo would achieve Tanner Stage 4 in approximately 4 years, or around age 12 ($14.25/3.29 = 4.33$ years), whereas a girl whose pubertal tempo was 1 standard deviation above the mean would achieve Tanner Stage 4 in approximately 3 years ($14.24/4.35 = 3.28$ years), around age 11.

African American girls had significantly earlier pubertal timing than European American girls, $\beta = .62$, 95% confidence interval (CI) [0.15, 1.09], but did not have significantly faster pubertal tempo, $\beta = -.20$, $p = .59$. There were no significant differences between Hispanic girls and European American girls with respect to either timing, $\beta = .50$, $p = .15$, or tempo, $\beta = -.42$, $p = .39$. For boys, neither Hispanic boys nor African American boys were significantly different than European American boys in either pubertal timing $\beta_{\text{AfAmerican}} = .24$, $p = .33$; $\beta_{\text{Hispanic}} = .08$, $p = .79$, or pubertal tempo $\beta_{\text{AfAmerican}} = -.21$, $p = .41$; $\beta_{\text{Hispanic}} = -.39$, $p = .18$.

For both boys and girls, there was a moderate negative correlation ($r = -.40$) between the latent intercept and latent slope factors. This negative correlation may reflect a ceiling effect: early maturing children who are already at an advanced Tanner stage at the beginning of the study would have both a higher latent intercept and, because they have less pubertal development remaining, a lower latent slope. As can be seen from the R^2 estimates, which

range from .83 to .93 in boys and .86 to .91 in girls, the parameters of the LGM accounted very well for the observed Tanner stages at each age.

Test 2: Do Pubertal Timing and Tempo Independently Predict Depressive Symptoms?

Results from the dual LGM of puberty and depressive symptoms are shown in Table 4 (see Figure 3 for model). In boys, the (standardized) regression parameters indicate effects of both pubertal timing and pubertal tempo on change in depressive symptoms. The mean latent slope for depressive symptoms (μ_2) in boys was actually negative, indicating that boys' depressive symptoms, on average, decreased in their during pre-adolescence. The effects of pubertal timing and pubertal tempo on the latent slope were positive, indicating that boys who mature earlier and boys who mature more rapidly than average experience less negative (i.e., less rapid) decreases in depressive symptoms. That is, boys with early pubertal timing or rapid pubertal tempo did not experience the reduction in depressive problems that was usually enjoyed by boys their age. Hispanic boys had a significantly higher initial level of depressive symptoms than European American boys, $\beta = .98$, 95% CI [0.29, 1.67] but did not have significantly different change in depressive symptoms, $\beta = -.49$, $p = .27$. There were no differences between African American and European American boys with regards to initial level, $\beta = .41$, $p = .19$, or change in depressive symptoms, $\beta = -.48$, $p = .23$.

A pairwise parameter comparison of the regression coefficients for pubertal timing and pubertal tempo confirmed that the relation of pubertal tempo with depressive symptoms was significantly greater than that of pubertal timing, Wald test = 5.071, $p = .024$. In addition, we tested a nested model in which the regression effect of pubertal tempo was fixed to equal 0, and we compared the difference in R^2 for change in depressive symptoms. A model with only the effect of pubertal timing accounted for only 2.7% of the variance in change in depressive symptoms, whereas a model with both pubertal timing and pubertal tempo accounted for 42% of the variance in change in depressive symptoms.

In contrast, the regression parameters in girls indicated effects of only pubertal timing on depressive symptoms. There were no effects of pubertal tempo, nor were there any effects (of either timing or tempo) on change in depressive symptoms. In other words, early maturing girls experienced higher average levels of depressive symptoms during pre-adolescence, consistent with previous research findings. Independent of this effect of pubertal timing, rapidly maturing girls demonstrated no worse outcomes. Finally, there were no significant effects of race/ethnicity on either level or change in depressive symptoms in girls.

Test 3: Do the Effects of Pubertal Timing Versus Tempo Differ Across Genders?

Multiple-group LGM was used to test whether the growth curve parameters for pubertal development and depressive symptoms could be constrained to be equal across boys and girls without significant decrement in model fit. The first set of nested model comparisons indicated that boys and girls significantly differed in their mean pubertal timing (intercept) and mean pubertal tempo (slope), $\Delta\chi^2 = 30.9$, $\Delta df = 2$, $p < .001$. However, all other growth parameters for pubertal development, including the variances and covariance of the intercept and slope factors and regressions of intercept and slope on race/ethnicity could be constrained to be equal across genders, $\Delta\chi^2 = 6.86$, $\Delta df = 9$, $p = .68$. Similarly, the second set of nested model comparisons indicated that for growth curves of depressive symptoms, the intercept and slope variances and covariance, time-specific residual variances, and regressions of intercept and slope on race and ethnicity could be constrained to be equal across genders, $\Delta\chi^2 = 12.87$, $\Delta df = 10$, $p = .23$.

Consequently, the final multiple-group LGM constrained all parameters to be equal across genders, except for the means of the intercept and slope factors and the effects of pubertal timing and tempo on the development of depressive symptoms. Results from the final multiple-group LGM are shown in Table 5. A nested model that constrained the regression coefficients to be equal across gender fit significantly worse than the full model where coefficients were free to differ across gender, $\Delta\chi^2 = 11.14$, $\Delta df = 4$, $p = .03$. This indicates that the relative impact of pubertal timing and pubertal tempo significantly differed across boys and girls.

Discussion

Puberty represents a universal transition, yet seems to hold more resonance and present more of a stumbling block for some people than for others. Children certainly differ in their response, awareness, and capacity to navigate the challenges of puberty. But they also differ in their biological experiences, and the psychological significance of puberty is not unrelated to these individual maturational nuances. Early timing of puberty, particularly in girls, is one of the best-researched predictors of psychological distress during adolescence. This article highlights the importance of another individual difference in the process of maturation: pubertal tempo, a topic which has been understudied to date.

Our study concentrates on depressive symptoms as children progress from late childhood into puberty. Consistent with previous literature, our results replicated the well-established finding that girls who experience an earlier onset of maturation also report a greater level of depressive symptoms during puberty. While early pubertal timing also predicted depressive severity for boys, we notably obtained stronger effects for pubertal tempo than for pubertal timing: Boys who matured more quickly than peers reported more depressive symptoms, with those boys who matured early and quickly being at greatest risk. This was not the case for boys with more normative developmental timing and tempo.

To date, the process of puberty in boys has been more difficult to capture than that of girls and has yielded less consistent results than the girls' literature. This is particularly true for associations of pubertal timing with clinical outcomes for boys. Our results indicate that pubertal tempo may perhaps be a more salient augur in the trajectory of boys' puberty than timing alone. This is in support of the maturation compression hypothesis proposed in this article. Our findings additionally help to explain why the pubertal timing literature for boys has yielded relatively disparate results; to understand the psychological significance of boys' development, we may need to move beyond simple consideration of timing of development.

It is worth cautioning that the dearth of research on pubertal tempo makes interpretation of our findings results somewhat speculative. Yet it seems notable that timing was the strongest predictor of depressive symptoms in girls, and tempo was the strongest predictor in boys. This finding is similar to results obtained by Ge et al. (2003) in an investigation of pubertal timing versus pubertal change at two time points in African American children. On a more intuitive level, it seems apparent that puberty comprises markedly different biological processes, maturational challenges, social changes, benefits, and risks for boys and girls. One possible explanation of the gender difference is that timing and tempo may hold different importance for boys and girls because of inherent differences in the pubertal process. Specifically, girls mature, on average, 1 year earlier than boys. This means that when early maturing girls start to manifest visible signs of puberty, they are the first of all their classmates and peers to do so. When early maturing boys start to manifest signs of puberty, they may be the first *boys* but are not the first of their overall peer group to make this transition. In effect, timing in girls may supersede the effects of tempo for the simple reason that early maturation places girls in a uniquely vulnerable position that is not shared

either by an early maturing boy or by a later but more speedily developing child of either gender. For this reason, early onset of development may have something of a “trumping” effect over tempo in girls. In support of this, both boys and girls tend to report feeling embarrassment, excitement, fear, pride, and other mixed emotions about their development (Brooks-Gunn, Newman, Holderness, & Warren, 1994; Dubas, Graber, & Petersen, 1991; Gaddis & Brooks-Gunn, 1985), but the most consistently negative reactions to pubertal changes have been voiced by girls who experience early pubertal timing (Dubas et al., 1991).

Certainly, understanding normal developmental processes—and where they may go awry for some children—is a research endeavor that holds direct clinical relevance. There are a multitude of school-based health and intervention programs presently used to reduce the known correlates of early pubertal timing, including depression and low self-esteem, as well as externalizing behaviors and risky sexual activity (e.g., Aten, Siegal, Enaharo, & Auinger, 2000; Botvin, Griffin, & Nichols, 2006; Eddy, Reif, & Fetrow, 2000; O’Dea and Abraham, 2000). Our results offer a number of implications for clinicians and researchers interested in further calibrating existing interventions to be both effective and meaningful for children. Although some of these programs discuss normative pubertal changes, they tend not to be gender specific nor do they include a psychoeducational component that addresses individual variations in development. Yet the importance of “normalizing,” and placing stressors in context has repeatedly been shown to be clinically effective (see Gabbard, Beck, & Holmes, 2007). Although children’s biological processes may not be malleable, their responses to such processes are. By suggesting that relations of individual differences at puberty with outcomes may differ across gender, we offer in this study specific areas to target for reframing the impact of the pubertal transition within an intervention context.

Limitations

There has been little research on pubertal tempo coupled with inconsistent findings on associations of psychological outcomes with pubertal timing in boys. As the present study is one of the first, if not only, studies on pubertal tempo to date, we consider this a preliminary exploration. This is particularly important given the limitations in our study. First and foremost, our initial sample size was not large and experienced attrition over time. Some children, moreover, went more than 1 year between assessments. This question of missing data is perhaps a more troubling scenario for our study than for other longitudinal research, given the nature of pubertal tempo and the need to measure subtle changes occurring during a relatively compact span. Although we used growth curve modeling with FIML to account for missing data, our statistical power may have been compromised to detect all effects. Of particular note, we obtained a null effect of pubertal tempo for girls. Replications in other samples with a greater number of participants and less attrition will be critical to determine whether this is a “true” null effect or simply an artifact of sample size concerns. For this reason, we feel it worth emphasizing that our interpretations regarding the gender difference are particularly provisional, in accordance with the confines of the data.

In addition to attrition and missing data, our findings are further limited by concerns about generalizability. Our sample was drawn from families who responded to recruitment in public schools. Parents managing high levels of life stress, unusually tumultuous puberty in their children, and depression or other forms of psychopathology (for themselves, their partners, or their children) may have been less likely to participate in a time-intensive psychological study. As we simply do not know the reasons some families responded to the recruitment flyer and others did not, we are unable to ascertain generalizability of results.

Lastly, it is worth commenting on the limitations in our measure of puberty. Pubertal status at each wave was primarily drawn from parental reports of Tanner stages. In general, the

accuracy of parent report of pubertal development varies when compared with physical examination. Some studies show that parent reports of Tanner stage are reasonably well correlated with assessments of Tanner stage obtained by a trained examiner conducting a physical examination (e.g., .57–.87; Brooks-Gunn et al., 1987). Yet kappa coefficients, in which the likelihood of chance agreement is taken into account, are considerably lower (.33–.50; Dorn et al., 1990). Moreover, it would be reasonable to infer that some parents may not have had sufficient or recent opportunity to observe their children's bodies in enough detail for Tanner ratings to be accurate; indeed, a minority of parents voiced that they felt unable to complete these rating, and children therefore completed the ratings themselves. The inclusion of multiple reporters of development is certainly not optimal, particularly not when compared with the "gold standard" of a physical examination (Dorn et al., 2006).

In addition, our study was an investigation of a 4-year span of pubertal development, with assessments ending in early adolescence. Yet puberty was already underway for some participants at the start of the study, and conversely, a relatively small percentage of participants had completed development by the end of the study. This highlights a more general methodological problem inherent in the study of puberty: Capturing the complete spectrum of pubertal development in all individuals in a sample would necessitate assessing participants between the ages 6 and 16 with reasonable frequency. This is obviously an expensive and difficult proposition and leads to pubertal data that are often truncated. With regard to the present study, it is plausible that some of the less-developed participants at Wave 4 might still go on to experience a rapid period of developmental change. In fact, we can make no predictions about long-term effects and can specify the relations of pubertal timing and tempo and depressive symptoms in early adolescence only.

Conclusion

Above all, the present study reinforces the benefits to investigators of moving beyond pubertal timing to examine additional sources of individual variability at puberty. While a study in which pubertal timing is disentangled from pubertal tempo is a clear advance over studies with no measures of tempo, our findings concentrate on one outcome during early adolescence. It is not clear whether similar effects would be replicated for other outcomes or for pubertal changes occurring later in adolescence. Additional research into pubertal tempo will help both researchers and clinicians build a more comprehensive understanding of this critical developmental window.

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References

- Alsaker FD. Pubertal timing, overweight, and psychological adjustment. *Journal of Early Adolescence*. 1992; 12:396–419.
- American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*. 4. Washington, DC: Author; 1994.
- Aten MJ, Siegel DM, Enaharo M, Auinger P. Keeping middle school students abstinent: Outcomes of a primary prevention intervention. *Journal of Adolescent Health*. 2002; 31:70–78. [PubMed: 12090967]
- Baltes, PB.; Nesselroade, JR. History and rationale of longitudinal research. In: Nesselroade, JR.; Baltes, editors. *Longitudinal research in the study of behavior and development*. New York, NY: Academic Press; 1979. p. 21-27.

- Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. *Archives of General Psychiatry*. 1961; 4:561–571. [PubMed: 13688369]
- Biro FM, Huang B, Crawford PB, Lucky AW, Striegel-Moore R, Barton BA, Daniels S. Pubertal correlates in black and white girls. *Journal of Pediatrics*. 2006; 148:234–240. [PubMed: 16492435]
- Bonat S, Pathomvanich A, Keil MF, Field AE, Yanovski JA. Self-assessment of pubertal stage in overweight children. *Pediatrics*. 2002; 110:743–747. [PubMed: 12359788]
- Botvin GJ, Griffin KW, Nichols TR. Preventing youth violence and delinquency through a universal school-based prevention approach. *Prevention Science*. 2006; 7:403–408. [PubMed: 17136462]
- Brooks-Gunn J, Newman D, Holderness C, Warren MP. The experience of breast development and girls: Stories about the purchase of a bra. *Journal of Youth and Adolescence*. 1994; 23:539–565.
- Brooks-Gunn J, Warren MP. Measuring physical status and timing in early adolescence: A developmental perspective. *Journal of Youth and Adolescence*. 1985; 14:163–189.
- Brooks-Gunn J, Warren MP, Rosso J, Gargiulo J. Validity of self-report measures of girls' pubertal status. *Child Development*. 1987; 58:829–841. [PubMed: 3608653]
- Dorn LD, Dahl RE, Woodward HR, Biro F. Defining the boundaries of early adolescence: A user's guide to assessing pubertal status and pubertal timing in research with adolescents. *Applied Developmental Science*. 2006; 10:30–56.
- Dorn LD, Susman EJ, Nottelmann ED, Inoff-Germain G, Chrousos GP. Perceptions of puberty: Adolescent, parent, and health care personnel. *Developmental Psychology*. 1990; 26:322–329.
- Downs AC, Fuller MJ. Recollections of spermarche: An exploratory investigation. *Current Psychology: Research & Reviews*. 1991; 10:93–102.
- Dubas JS, Graber JA, Petersen AC. A longitudinal investigation of adolescents' changing perceptions of pubertal timing. *Developmental Psychology*. 1991; 27:580–586.
- Eddy JM, Reid JB, Fetrow RA. An elementary school-based prevention program targeting modifiable antecedents of youth delinquency and violence: Linking the Interests of Families and Teachers (LIFT). *Journal of Emotional & Behavioral Disorders*. 2000; 8(3):165–176.
- Fairburn C, Welch S, Doll H, Davies BA, O'Connor ME. Risk factors for bulimia nervosa: A community-based case control study. *Archives of General Psychiatry*. 1997; 54:509–517. [PubMed: 9193191]
- Ferrer E, Hamagami F, McArdle JJ. Modeling latent growth curves with incomplete data using different types of structural equation modeling and multilevel software. *Structural Equation Modeling*. 2004; 11:452–483.
- Fisher M, Rosenfeld WD, Burk RD. Cervicovaginal human papillomavirus infection in suburban adolescents and young adults. *Journal of Pediatrics*. 1991; 119:821–825. [PubMed: 1658285]
- Gabbard, GO.; Beck, JS.; Holmes, J. *Oxford textbook of psychotherapy*. New York, NY: Oxford University Press; 2007.
- Gaddis A, Brooks-Gunn J. The male experience of pubertal change. *Journal of Youth and Adolescence*. 1985; 14:61–69.
- Ge X, Conger RD, Elder GH. Coming of age too early: Pubertal influences on girls' vulnerability to psychological distress. *Child Development*. 1996; 67:3386–3400. [PubMed: 9071784]
- Ge X, Conger RD, Elder GH. Pubertal transition, stressful life events, and the emergence of gender differences in adolescent depressive symptoms. *Developmental Psychology*. 2001; 37:404–417. [PubMed: 11370915]
- Ge X, Kim IJ, Brody GH, Conger RD, Simon RL, Gibbons FX, Cutrona CE. It's about timing and change: Pubertal timing effects on symptoms of depression among African American youths. *Developmental Psychology*. 2003; 39:430–439. [PubMed: 12760513]
- Ge X, Natsuaki MN. In search of explanations for early pubertal timing effects on developmental psychopathology. *Current Directions in Psychological Science*. 2009; 18:327–331.
- Graber JA, Lewinsohn PM, Seeley JR, Brooks-Gunn J. Is psychopathology associated with the timing of pubertal development? *Journal of the American Academy of Child and Adolescent Psychiatry*. 1997; 36:1768–1776. [PubMed: 9401339]
- Haynie D. Context of risk? Explaining the link between girls' pubertal development and their delinquency involvement. *Social Forces*. 2003; 82:355–397.

- Hollingshead, AB. Four-factor index of social status. New Haven, CT: Yale University Press; 1975.
- Jones MC. Psychological correlates of somatic development. *Child Development*. 1965; 36:899–911. [PubMed: 5892336]
- Jones MC, Bayley N. Physical maturing among boys as related to behavior. *Journal of Educational Psychology*. 1950; 41:129–148.
- Kaltiala-Heino R, Marttunen M, Rantanen P, Rimpela M. Early puberty is associated with mental health problems in adolescence. *Social Science and Medicine*. 2003; 57:1055–1064. [PubMed: 12878105]
- Kaplowitz PB, Oberfield SE. Reexamination of the age limit for defining when puberty is precocious in girls in the United States: Implications for evaluation and treatment. *Pediatrics*. 1999; 104:936–941. [PubMed: 10506238]
- Kovacs M. The Children's Depression Inventory (CDI). *Psychopharmacology Bulletin*. 1985; 21:995–1124. [PubMed: 4089116]
- Marshall WA, Tanner JM. Variations in the pattern of pubertal changes in girls. *Archives of the Diseases in Childhood*. 1969; 44:291–303.
- Marshall WA, Tanner JM. Variations in the pattern of pubertal changes in boys. *Archives of the Diseases in Childhood*. 1970; 45:13–23.
- McArdle, JJ.; Nesselroade, JR. Growth curve analysis in contemporary psychological research. In: Schinka, J.; Velicer, W., editors. *Comprehensive handbook of psychology: Vol. 2. Research methods in psychology*. New York, NY: Wiley; 2002.
- McClintock M, Herdt G. Rethinking puberty: The development of sexual attraction. *Current Directions in Psychological Science*. 1996; 5:178–183.
- Mendle J, Turkheimer E, Emery RE. Detrimental psychological outcomes associated with early pubertal timing in adolescent girls. *Developmental Review*. 2007; 27:151–171. [PubMed: 20740062]
- Meredith W, Tisak J. Latent curve analysis. *Psychometrika*. 1990; 55:107–122.
- Morris NM, Udry JR. Validation of a self-administered instrument to assess stage of adolescent development. *Journal of Youth and Adolescence*. 1980; 9:271–280.
- Muthén, LK.; Muthén, BO. *Mplus user's guide*. Los Angeles, CA: Authors; 1998–2009.
- O'Dea JA, Abraham S. Improving the body image, eating attitudes, and behaviors of young male and female adolescents: A new educational approach that focuses on self-esteem. *International Journal of Eating Disorders*. 2000; 28:43–57. [PubMed: 10800013]
- Paikoff R, Brooks-Gunn J. Do parent–child relationships change during puberty? *Psychological Bulletin*. 1991; 110:47–66. [PubMed: 1891518]
- Pantsiotou S, Papadimitriou A, Douros K, Priftis K, Nicolaidou P, Fretzayas A. Maturation tempo differences in relation to the timing of the onset of puberty in girls. *Acta Paediatrica*. 2008; 97:217–220.
- Papadimitriou A, Fretzayas A, Nicolaidou P. The tempo of puberty. *Journal of Pediatrics*. 2006; 149:732–733. [PubMed: 17095363]
- Patton GC, Hibbert ME, Carlin J, Shao Q, Rosier M, Caust J, Bowes G. Menarche and the onset of depression and anxiety in Victoria, Australia. *Journal of Epidemiology and Community Health*. 1996; 50:661–666. [PubMed: 9039386]
- Petersen AC, Crockett L. Pubertal timing and grade effects on adjustment. *Journal of Youth and Adolescence*. 1985; 14:191–206.
- Petersen AC, Sarigiani PA, Kennedy RE. Adolescent depression: Why more girls than boys? *Journal of Youth and Adolescence*. 1991; 20:191–215.
- Petersen, AC.; Taylor, B. The biological approach to adolescence: Biological change and psychological adaptation. In: Adelson, J., editor. *Handbook of adolescent psychology*. New York, NY: Wiley; 1980.
- Schafer JL, Graham JW. Missing data: Our view of the state of the art. *Psychological Methods*. 2002; 7:147–177. [PubMed: 12090408]
- Stattin, H.; Magnusson, D. Pubertal maturation in female development: Volume 2. *Paths through life*. Hillsdale, NJ: Erlbaum; 1990.

- Steinberg L. Impact of puberty on family relations: Effects of pubertal status and pubertal timing. *Developmental Psychology*. 1987; 23:451–460.
- Stice E, Presnell K, Bearman SK. Relation of early menarche to depression, eating disorders, substance abuse, and comorbid psychopathology among adolescent girls. *Developmental Psychology*. 2001; 37:608–619. [PubMed: 11552757]
- Susman EJ, Dorn LD, Chrousos GP. Negative affect and hormone levels in young adolescents: Concurrent and predictive perspectives. *Journal of Youth and Adolescence*. 1991; 20:167–190.
- Tanner, JM. Growth at adolescence. 2. Oxford, England: Blackwell; 1962.
- Udry JR. Age at menarche, at first intercourse, and at first pregnancy. *Journal of Biosocial Science*. 1979; 11:433–441. [PubMed: 511871]
- Weichold, K.; Silbereisen, RK.; Schmitt-Rodermund, E. Short- and long-term consequences of early versus late physical maturation in adolescents. In: Hayward, C., editor. *Gender differences at puberty*. Cambridge, England: Cambridge University Press; 2003. p. 241-276.
- Wellens R, Malina RM, Roche AF, Chumlea WC, Guo S, Siervogel RM. Body size and fatness in young adults in relation to age at menarche. *American Journal of Human Biology*. 1992; 4:783–787.
- Wichstrom L. Predictors of adolescent suicide attempts: A nationally representative longitudinal study of Norwegian adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*. 2000; 39:603–610. [PubMed: 10802978]
- Wothke, W. Longitudinal and multi-group modeling with missing data. In: Little, TD.; Schnabel, KU.; Baumert, J., editors. *Modeling longitudinal and multiple group data: Practical issues, applied approaches, and specific examples*. Mahwah, NJ: Erlbaum; 1998.

Figure 1.
Mean growth in Tanner stage for European American, African American, and Hispanic boys and girls.

Figure 2.

Latent growth model of pubertal development in boys from age 8 to age 14. Observed ordinal Tanner stage variables (Y_{8i} – Y_{14i}) are depicted as “circles within squares” to indicate that children were not measured at every age; that is, some data are missing by design at each age. Threshold structure for ordinal Tanner Stage variables is not depicted. Residual variances for the continuous latent variables (y_{8i} – y_{14i}) underlying observed ordinal variables were constrained to be equal across ages. Mean of the latent intercept factor (μ_1) was fixed to 0.0. Regressions of latent intercept and latent slope factors on dummy-coded race/ethnicity variables not depicted. Model for girls was identical, except age range was from 8 to 13 years. i = individual; η_{1i} = latent factor representing the initial level (often referred to as the *latent intercept*); η_{2i} = latent factor representing the *slope* of change over time; Ψ = variance in linear slope.

Figure 3.

Latent growth model of pubertal development and depression in boys from ages 8 to 14 years. Observed variables are depicted as “circles within squares” to indicate that children were not measured at every age; that is, some data are missing by design at each age. Residual variances for the continuous latent variables ($x_{8i}-x_{14i}$) underlying observed ordinal variables were constrained to be equal across ages. Residual variances for observed depression scores ($Y_{8i}-Y_{14i}$) were also constrained to be equal across ages. Mean of the latent intercept factor for puberty (μ_{x1}) was fixed to 0.0. Regressions of latent intercept and latent slope factors on dummy-coded race/ethnicity variables not depicted. Model for girls was identical, except age range was from 8 to 13 years. i = individual; η_{1i} = latent factor representing the initial level (often referred to as the *latent intercept*); η_{2i} = latent factor representing the *slope* of change over time; Ψ = variance in linear slope; β_{X1-Y1} = regression of depression intercept on puberty intercept; β_{X1-Y2} = regression of depression slope on puberty intercept; β_{X2-Y1} = regression of depression intercept on puberty slope; β_{X2-Y2} = regression of depression slope on puberty slope.

Table 1

Number of Boys at Each Tanner Stage at Waves 1 Through 4

Stage	Wave 1 (Mean age = 9.61 years)				Wave 2 (Mean age = 11.13 years)				Wave 3 (Mean age = 12.27 years)				Wave 4 (Mean age = 13.14 years)			
	Genital		Hair		Genital		Hair		Genital		Hair		Genital		Hair	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
1	59	50	95	79	24	24	44	44	2	2	12	14	0	0	0	0
2	38	32	25	21	39	39	39	39	21	24	25	28	8	17	8	17
3	17	14	0	0	23	23	12	12	35	40	29	33	13	27	15	32
4	5	4	0	0	13	13	3	3	26	30	19	22	18	38	19	40
5	0	0	0	0	1	1	1	1	4	5	3	3	8	17	5	11
Total	119		120		100		99		88		88		47		47	

Note. Genital = genital development; hair = pubic hair growth.

Table 2

Number of Girls at Each Tanner Stage at Waves 1 Through 4

Stage	Wave 1 (Mean age = 8.86 years)				Wave 2 (Mean age = 9.69 years)				Wave 3 (Mean age = 10.56 years)				Wave 4 (Mean age = 11.84 years)			
	Breast		Hair		Breast		Hair		Breast		Hair		Breast		Hair	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
1	63	51	95	75	19	25	45	61	18	20	29	33	7	7	17	17
2	47	38	26	20	42	56	20	27	36	40	26	30	23	21	22	21
3	19	10	4	3	12	16	7	9	26	29	17	20	46	43	27	26
4	1	1	2	2	2	3	1	1	7	8	11	13	25	23	27	26
5	0	0	0	0	0	0	1	1	2	2	4	5	7	6	10	10
Total	130		127		75		74		89		87		108		103	

Note. Breast = breast development; hair = pubic hair growth.

Table 3

Parameters From Latent Growth Models of Pubertal Development in Boys and Girls

Model parameters	Boys		Girls	
	Estimate	95% CI	Estimate	95% CI
Growth factors				
Mean intercept (μ_1)	0.00	[0.00, 0.00]	0.00	[0.00, 0.00]
Mean slope (μ_2)	2.12	[1.48, 2.77]	3.29	[2.23, 4.34]
Variance of intercept (Ψ_{11})	28.55	[12.38, 44.73]	22.25	[4.57, 39.93]
Variance of slope (Ψ_{22})	2.14	[0.88, 3.40]	1.13	[0.00, 2.55]
Covariance of intercept–slope (Ψ_{12})	−5.47	[−9.09, −1.85]	−2.00	[−5.32, 1.31]
Tanner stage thresholds				
Stage 1–2	2.31	[0.30, 4.32]	6.78	[4.12, 9.41]
Stage 2–3	6.30	[4.06, 8.54]	11.01	[7.57, 14.45]
Stage 3–4	10.17	[7.61, 12.72]	14.25	[10.22, 18.27]
Stage 4–5	15.00	[11.86, 18.13]	18.59	[13.65, 23.54]
Age-Specific R^2				
Age 8	0.90		0.88	
Age 9	0.86		0.87	
Age 10	0.83		0.86	
Age 11	0.82		0.87	
Age 12	0.86		0.89	
Age 13	0.90		0.91	
Age 14	0.93		NA	
Model fit indices				
Log likelihood	−547.56		−348.55	
Bayesian information criterion	1115.02		718.00	

Note. CI = confidence interval.

Table 4

Parameters From Dual Latent Growth Models of Pubertal Development and Depressive Symptoms (Estimated Separately in Boys and Girls)

Model parameters	Boys		Girls	
	Estimate	95% CI	Estimate	95% CI
Growth factors: Puberty				
Mean intercept (μ_{X1})	0.00	[0.00, 0.00]	0.00	[0.00, 0.00]
Mean slope (μ_{X2})	2.19	[1.52, 2.86]	3.18	[2.39, 3.97]
Variance of intercept (Ψ_{X11})	29.57	[12.78, 46.37]	20.27	[8.10, 32.44]
Variance of slope (Ψ_{X22})	2.28	[0.93, 3.62]	1.10	[0.04, 2.16]
Covariance of intercept–slope (Ψ_{X12})	−5.74	[−9.55, −1.94]	−1.79	[−3.99, 0.42]
Growth factors: Depression				
Mean intercept (μ_{Y1})	6.94	[3.52, 10.37]	8.74	[1.44, 16.05]
Mean slope (μ_{Y2})	−0.91	[−2.03, 0.22]	0.55	[−1.63, 2.73]
Variance of intercept (Ψ_{Y11})	13.46	[1.71, 25.21]	10.40	[0.00, 32.70]
Variance of slope (Ψ_{Y22})	0.60	[0.00, 1.77]	0.43	[0.00, 3.26]
Covariance of intercept–slope (Ψ_{Y12})	−0.82	[−4.08, 2.45]	0.26	[−6.38, 6.90]
Regression effects (standardized)				
Puberty intercept–depression intercept (β_{X1-Y1})	−0.34	[−0.80, 0.12]	0.45	[0.06, 0.84]
Puberty intercept–depression slope (β_{X1-Y2})	0.67	[0.08, 1.27]	−0.60	[−1.91, 0.71]
Puberty slope–depression intercept (β_{X2-Y1})	−0.35	[−0.83, 0.13]	−0.25	[−0.79, 0.29]
Puberty slope–depression slope (β_{X2-Y2})	0.82	[0.12, 0.73]	−0.004	[−0.88, 0.87]
Tanner stage thresholds				
Stage 1–2	2.55	[0.48, 4.72]	6.45	[4.38, 8.51]
Stage 2–3	6.59	[4.26, 8.91]	10.63	[8.05, 13.21]
Stage 3–4	10.51	[7.85, 13.17]	13.85	[10.84, 16.86]
Stage 4–5	15.43	[12.16, 18.70]	18.09	[14.09, 22.09]
R^2 : Depression				
Latent intercept	0.24		0.42	
Latent slope	0.43		0.33	
Age 8	0.54		0.45	
Age 9	0.50		0.44	
Age 10	0.48		0.45	
Age 11	0.51		0.47	
Age 12	0.56		0.50	
Age 13	0.62		0.55	
Age 14	0.68		NA	

Note. Lower bound for confidence intervals (CI) of variance components bounded at zero. Log likelihood for model = −1347.27 in girls and −1602.038 in boys. Bold typeface indicates regression estimates where the confidence interval does not include 0 (i.e., significance).

Table 5

parameters From Multiple-Group LGM of Gender Differences (Boys and Girls Estimated Together)

Model parameters	Boys		Girls	
	Estimate	95% CI	Estimate	95% CI
Growth factors: Puberty				
Mean intercept (μ_{X1})	3.69	[1.94, 5.43]	0.00	[0.00, 0.00]
Mean slope (μ_{X2})	1.73	[1.15, 2.31]	3.15	[2.38, 3.91]
Variance of intercept (Ψ_{X11})	27.79 ^a	[15.41, 40.18]	27.79 ^a	[15.41, 40.18]
Variance of slope (Ψ_{X22})	1.97 ^a	[0.95, 2.99]	1.97 ^a	[0.95, 2.99]
Covariance of intercept–slope (Ψ_{X12})	−4.53 ^a	[−7.19, 1.86]	−4.53 ^a	[−7.19, 1.86]
Growth factors: Depression				
Mean intercept (μ_{Y1})	9.54	[5.30, 13.78]	8.91	[3.78, 14.04]
Mean slope (μ_{Y2})	−1.31	[−2.80, 0.20]	0.30	[−1.63, 2.23]
Variance of intercept (Ψ_{Y11})	14.21 ^a	[4.85, 23.58]	14.21 ^a	[4.85, 23.58]
Variance of slope (Ψ_{Y22})	0.71 ^a	[0.00, 1.77]	0.71 ^a	[0.00, 1.77]
Covariance of intercept–slope (Ψ_{Y12})	−0.94 ^a	[−3.69, 1.81]	−0.94 ^a	[−3.69, 1.81]
Regression effects (standardized)				
Puberty intercept–depression intercept (β_{X1-Y1})	−0.32	[−0.78, 0.14]	0.43	[0.12, 0.73]
Puberty intercept–depression slope (β_{X1-Y2})	0.58	[0.03, 1.13]	−0.50	[−1.05, 0.05]
Puberty slope–depression intercept (β_{X2-Y1})	−0.36	[−0.83, 0.11]	−0.16	[−0.58, 0.26]
Puberty slope–depression slope (β_{X2-Y2})	0.71	[0.15, 1.27]	−0.12	[−0.95, 0.71]
Tanner stage thresholds				
Stage 1–2	5.46 ^a	[3.26, 7.66]	5.46 ^a	[3.26, 7.66]
Stage 2–3	9.63 ^a	[7.18, 12.08]	9.63 ^a	[7.18, 12.08]
Stage 3–4	13.35 ^a	[10.64, 16.05]	13.35 ^a	[10.64, 16.05]
Stage 4–5	18.02 ^a	[14.90, 21.15]	18.02 ^a	[14.90, 21.15]
R^2 : Depression				
Latent intercept	0.15		0.35	
Latent slope	0.35		0.20	

Note. Lower bound for confidence intervals (CI) of variance components bounded at zero. Log-likelihood for model = −3142.19, and sample-size adjusted Bayesian information criterion = 6501.69. Bold typeface indicates regression estimates where the confidence interval does not include 0 (i.e., significance).

^aParameter constrained to be equal across boys and girls, based on results of previous nested model comparisons.