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Timing and tempo: Exploring the complex association between pubertal development and depression in African American and European American girls

Kate Keenan

Department of Psychiatry and Behavioral Neuroscience, University of Chicago

Kristen Culbert

Department of Psychology, Michigan State University

Kevin J. Grimm

Department of Psychology, Arizona State University

Alison Hipwell and Stephanie Stepp

Department of Psychiatry, University of Pittsburgh

Abstract

The relative contribution of pubertal timing and tempo to the development of depression has not been tested in a large, representative sample, nor has the interface among pubertal maturation, depression, and race. Participants were a community-based sample of 2,450 girls from the Pittsburgh Girls Study (PGS) who were interviewed annually from ages 9 to 17 years. Pubertal timing and tempo were characterized as a unitary construct and also separately for pubic hair and breast development using child and maternal report. Depression symptoms were assessed annually. African-American females had higher depression symptoms and progressed through puberty earlier, but at a slower tempo than European American girls. Girls with earlier timing had higher levels of depression symptoms at age 10 years. Slower tempo was associated with higher depression symptoms at age 10, and faster tempo was associated with increases in depression from ages 10 to 13. As well, race moderated the associations among timing, tempo, and depression symptoms, and the association between race and depression was partially mediated by pubertal timing and tempo. Pubertal timing and tempo and race contribute to the developmental course of depression from early to late adolescence. The pattern of association varies as a function of the developmental window within which depression is assessed. Thus, repeated measures of depression symptoms and puberty across the span of pubertal development are necessary for exploring the relative importance of dimensions of pubertal development to depression etiology.

Keywords

Puberty; timing; tempo; depression; race

¹Empirical bootstrap confidence bounds were calculated for each indirect effect with 200 bootstrapped samples.

Introduction

The prevalence of depressive disorders substantially increases from childhood to adolescence, largely due to increases in depression among girls during adolescence (see Keenan and Hipwell, 2005 and Kuehner, 2003 for reviews). This interaction of sex and age on depression prevalence has led to a focus on the role of puberty in the development of depression. Existing research provides evidence that pubertal development is linked to changes in mood, cognition, and behavior, and exposure to environmental stressors that are relevant to depression. At a biological level, pubertal stage (i.e., maturational stage of secondary sex characteristics) is associated with systems that appear to be part of the pathophysiology of depression including neural response to reward (Forbes et al., 2010) and functioning of the hypothalamic-pituitary-adrenal (HPA) and hypothalamic-pituitary-gonadal axes, especially among girls (Stroud et al., 2004). At a psychosocial level, pubertal development has been implicated in increased risk for depression in girls via heightened stress associated with physical maturation, transitions in social relationships, and greater frequency and vulnerability to negative life events (for review, see Cyranowski, Frank, Young, & Shear, 2000). Although these data support an association between pubertal maturation and risk for depression, a more nuanced understanding of the nature of the association is needed for testing etiological model testing and ultimately informing the development of targeted preventions.

After extensive research, however, the role of puberty in the *etiology and development* of depression remains unclear. Within nearly each dimension assessed — pubertal status, timing, and tempo, there are conflicting results. For example, in a subsample of African American and European American girls from the Pittsburgh Girls Study followed longitudinally, pubertal *status*, the stage of pubertal development achieved at the time of assessment, at age 9 did not influence the stability of depression symptoms or the predictive utility of depression symptoms to later depressive disorders from ages 9-11 years (Keenan et al., 2008). In contrast, using 4 annual waves of data for 9-16 year old European American and American Indian girls in the Great Smoky Mountain Study, Angold et al. (1998) reported a 3-fold increase in depressive disorders among girls who had reached Tanner stage 3 compared to girls in Tanner stage 2.

More empirical support is seen for an association between pubertal *timing*, the age at which a given pubertal milestone is reached, and depression (Ge et al., 2003; Mendle et al., 2007; Negri & Susman, 2011). Substantial individual variability is observed for age of pubertal timing. For example, the age at which Tanner stage 2 is reached can range from 7-13 years (Styne & Grumbach, 2002), and age at menarche, one of the most commonly used indices of pubertal timing, can range from 8 and 15 years (Chumlea et al., 2003). The timing of a specific pubertal milestone is typically evaluated in relation to same age peers. In a number of studies, higher risk for depression is observed among girls who experience earlier pubertal timing compared to on-time or late maturing girls (Joinson et al., 2011; Stice et al., 2001). In other studies, however, no direct effect is observed between pubertal timing and depressive disorders (Angold et al., 1998; Boden, Fergusson, & Horwood, 2011) or depression scale scores (Foster et al., 2008). Furthermore, the association between depression and early puberty varies at different developmental periods, but not always in a

consistent pattern. Crockett et al. (2013) reported associations between early pubertal timing and depression scores at age 11, but effects did not persist longitudinally (at age 15). Graber and colleagues (1997) tested the association between perceived timing and diagnoses of major depression among high school students in the Oregon Adolescent Depression Project: earlier perceived timing was not associated with current depressive disorders, but was with lifetime history of depression. Copeland et al. (2010) observed a statistical trend ($p = .05$) towards an effect of earlier pubertal timing on depressive disorders in adolescence and a significant timing-depression association in early adulthood. This effect between earlier pubertal timing and depression in early adulthood, however, was accounted for by adolescent conduct disorder.

Inconsistency in effects of pubertal timing may be partially the result of different definitions of timing. For example, menarche occurs relatively late in pubertal development and has a strong genetic component (Dvornyk & Wagar-ul-Haq, 2012). Moreover, age at menarche varies as a function of pubertal pathway. In the Avon Longitudinal Study of Parents and Children (ALSPAC), for example, girls whose pubertal development started with growth of pubic hair as opposed to breast development or synchronous development had an earlier age of reaching Tanner Stage 2, but a later age of menarche (Christensen et al., 2010). These findings point to the potential utility of assessing pubertal maturation across a range of secondary sex characteristics and exploring whether puberty-depression effects vary with different pubertal indicators (e.g., breast vs. pubic hair).

Pubertal development is also more complex than what is captured by measuring timing of milestones, which are essentially static measures of a dynamic process that occurs over several years. *Pubertal tempo*, defined as the rate of within-person progression through multiple stages of maturation, may also alter depression risk. Only a few studies have examined pubertal tempo as a risk factor for depression in girls, but like studies on pubertal timing, the findings are inconsistent. In an early study on pubertal progress assessed in a population based study of Dutch adolescents, the rate of progress from ages 10-12 years to ages 12-14 years was not significantly associated with either parent or youth report of changes in anxiety/depression scores (Laitinen-Krispijn, et al., 1999). Only recently have investigators assessed tempo using multiple, repeated assessments of pubertal stage of development. Mendle et al. (2010) reported that early pubertal timing, but not tempo predicted level of depression in a racially and ethnically diverse sample of 138 girls. In the largest study conducted to date, both early pubertal timing and faster tempo predicted internalizing symptoms broadly (i.e., withdrawn, somatic complaints, anxiety, and depression) at age 15 in a sample of over 350 European American girls (Marceau et al. 2011). These differences in results could be due in part to sample size and methodology. For example, the number and timing of assessments of pubertal stage could impact the measurement of tempo given that pubertal change is not linear (Ellis et al. 2011). Another factor that may contribute to the mixed findings on the association between tempo and depression risk is the racial composition of participants included in prior studies. In only one study was the sample racially diverse (Mendle et al., 2010), but the sample size was too small to test race differences.

Race differences in both depression and pubertal maturation are compelling and likely to be important for understanding the effect of puberty on depression. Data from our own research indicate that rates of depression symptom endorsement are higher among African American girls than among European American girls (Keenan et al., 2008) even after controlling for poverty (Keenan et al., 2009). This finding is consistent with other studies of youth differences in depression between African American and European American adolescents (Franko et al., 2005; Garrison et al., 1990; Kistner et al., 2003), although some studies, such as those based on recall of lifetime prevalence of depression, do not show race differences (e.g., Merikangas et al., 2010). Further, pubertal development occurs at a significantly younger age among African American girls compared to European American girls (Biro et al., 2006; Chumlea et al., 2003). Higher levels of depression symptoms among African American girls, therefore, may be partially due to an earlier onset of pubertal development, although no prior study has directly examined this hypothesis by comparing repeated assessments of pubertal stage and depression symptoms among African American and European American girls. There are unfortunately little to no data on race differences in pubertal tempo, and as noted above, representation of African American girls in prior studies examining pubertal tempo effects on mood symptoms have been insufficient for hypothesis testing (e.g., Mendle et al., 2010). Examining race effects on the association between patterns of pubertal development (i.e., timing and tempo) and depression would therefore be a significant contribution to our understanding of race differences in depression.

The continued interest in probing the associations among pubertal timing, tempo and depression, even in the context of fairly mixed results is in part due to the substantive support derived from controlled animal studies on the effect of pubertal hormones on brain development and the expression of sex differentiated phenotypes. Specifically, the developmental timing of exposure to gonadal hormones and the dose of exposure have organizational effects (i.e., permanent/long-term, persisting after initial hormone exposure) on the adolescent brain that are likely relevant to sex-linked forms of psychopathology, including depression (for reviews, see Schulz et al., 2009; Berenbaum & Beltz, 2011). Animal data indicate that the brain is more sensitive to the organizational effects of gonadal hormones on sex differentiated behavior at a younger adolescent age (e.g., earlier maturation; Schulz et al., 2009), and human data have demonstrated positive associations between levels of sex hormones and tempo of pubertal maturation, with a faster tempo associated with higher levels of circulating estrogen, androstenedione, dehydroepiandrosterone sulfate (DHEA-S), and follicular stimulating hormone (de Ridder et al., 1992). Such effects may have important implications for individual differences in risk for depression during/after puberty, particularly given the significant positive associations reported between these hormones (e.g., estradiol, DHEA-S, androstenedione) and depression (e.g., Angold et al., 1999; Brooks-Gunn & Warren, 1989; Goodyer et al., 2003; Paikoff et al., 1991; Weber et al., 2000). Thus, within this organizational hypothesis framework, higher hormone concentrations (and more rapid exposure) may enhance the magnitude of neural and phenotypic organization that contribute to depression risk, particularly if higher hormone exposure occurs during greater neural sensitivity, i.e., earlier pubertal maturation

Although pubertal hormone mechanisms are critical, they are unlikely to completely account for puberty-depression relationships. Several psychosocial mechanisms have been proposed

to explain etiologic links between pubertal timing and depression. Two leading psychosocial theories include the early timing or developmental readiness hypothesis (Peskin, 1973; Petersen & Taylor, 1980) and the off-time or maturational deviance hypothesis (Simmons & Blyth, 1987). The early timing/developmental readiness hypothesis predicts that girls who experience earlier pubertal maturation would be at increased risk for depression symptoms because they are not cognitively prepared for the emotional, social, and physiological changes of puberty (e.g., Ge et al., 2003; Marceau et al., 2011). The off-time or maturational deviance hypothesis posits that vulnerability to depression is highest among girls who experience either earlier or later pubertal maturation relative to peers because these girls would experience pubertal and perhaps other milestones or transitions at atypical developmental periods (e.g., Ge et al., 2003; Marceau et al., 2011). These psychosocial theories are, however, largely limited to providing an explanation for individual differences in psychopathology and timing of pubertal maturation, whereas an organizational hormone hypothesis may help to explicate associations between pubertal timing and tempo and risk for depression, as well as account for effects that persist even after the completion of puberty.

In the present study, we aim to extend the emerging human research on the role of puberty in depression using nine waves of data from the Pittsburgh Girls Study (PGS, Keenan et al., 2010), a community-based sample of 2,450 girls who were interviewed annually since childhood. In the PGS, changes from pre to complete pubertal maturation were captured for most individuals via repeated parent and child reports of pubertal development between ages 9-17 years. Pubertal timing and tempo effects were tested using separate measures for pubic hair and breast development, as well as an overall pubertal development construct that included breast development and growth of pubic hair. Beginning at age 10 years, depression symptoms were assessed repeatedly by child and maternal report. Importantly, approximately half of the PGS sample is African American, and although poverty and race are significantly correlated, the distribution of participants across race and poverty is adequate to test for possible differences in puberty's effects on depression between African American and European American girls.

We hypothesize that pubertal timing and tempo will be associated with depression symptoms: girls with an earlier onset and faster tempo will have higher levels of depression at age 10 and will be less likely to show attenuation of symptoms over time compared to girls with later onsets and slower tempo. In addition, we expect that the previously observed race differences in depression symptoms in this sample will be partially accounted for by race differences in pubertal timing and tempo. These hypotheses are tested using non-linear growth models.

Methods

Participants

In the PGS, a stratified, random household sampling method, with over-sampling of households in low-income neighborhoods, was used to identify girls between the ages of 5 and 8 years. Neighborhoods in which at least 25% of the families were living at or below the poverty level were fully enumerated (i.e., all homes were contacted to determine if the

household contained an eligible girl), and a random selection of 50% of the households in non-risk neighborhoods were enumerated during 1998 and 1999. The enumeration identified 3,118 separate households in which an eligible girl resided. From these households, families who moved out of state and families in which the girl would be age ineligible by the start of the study were excluded. Of the 2,992 eligible families, 2,875 (96%) were successfully re-contacted to determine their willingness to participate in the longitudinal study. Of those families, 85% agreed to participate resulting in a total sample size of 2,450. The 2,450 girls were relatively evenly distributed across the four age groups (5-8 years). Approximately half of the girls were African American (52%), 41% were European American, and the remaining girls were described as multiracial or representing another race. Nearly all the primary caregivers were biological mothers (92%). More than half of the caregivers were cohabiting with a husband or partner and 25% of the families had yearly incomes of less than \$15,000.

Participants and their caregivers are interviewed annually. Data for the present analyses are based on the first 10 years of assessment. Retention of the sample has been very high, ranging from a high of 97.5% at wave 2 to 86.3% at wave 10. Compared to retained participants, those lost to attrition were more likely to be families not receiving public assistance and European American girls.

Measures

Information on girls' age, race, and whether the household was in receipt of public assistance (e.g. Women, Infants and Children program, food stamps, aid to families with dependent children) was collected by parental report. For these analyses, Asian American girls and girls with unknown race ($n = 26$) were excluded, leaving comparisons between African American and European American girls.

Symptoms of depression were measured using the Childhood/Adolescent Symptom Inventory-4th Edition (*CSI-4/ASI-4*; Gadow & Sprafkin, 1997; 2008) based on combined child and caregiver report beginning at age 10 years. This family of measures includes all nine symptoms of DSM-IV major depression disorder and has demonstrated reliability, validity and clinical utility (Gadow & Sprafkin, 1997; 2002; 2008). In the present study, items that were endorsed as 'a lot', 'all the time' or 'yes' by either informant were summed to generate symptoms counts. Correlations between parent and child total symptom count were low and consistent across development (average correlation = .21), a result that is comparable to other studies (e.g., van der Ende & Verhulst, 2005). In general, girls were more likely to report changes in sleep, appetite, concentration, and activity than caregivers, with the overlap in endorsement of these symptoms increasing with age. Given that parents and children appear to provide unique and meaningful reports of symptoms (Jensen et al., 1999), the standard and clinically justified approach to coding symptom presence has been to combine informants (Cantwell et al., 1999), which is the approach used in the present study.

Descriptive statistics for depression symptoms are presented in Table 1: on average girls were reported to have 1-2 symptoms at each age. Although the level of symptom endorsement is low, the morbidity associated with even 1-2 symptoms of depression is

significant. For example, minor depression (defined in the DSM as depressed mood or anhedonia plus one additional symptom) appears to be equivalent to major depression in term of prognosis regarding impairment, use of mental health services, future major depression episodes and suicidality (Fergusson et al., 2005; Gonzalez-Tejera et al., 2005). We also have provided evidence for the clinical significance of having 1-2 symptoms of depression during childhood in a sub-study to the PGS; the level of impairment among girls who met criteria for minor depression was significantly different from girls without a depression diagnosis and did not differ from girls with a diagnosis of major depression (Keenan et al., 2008). Thus, identifying etiologic effects on changes in relatively low levels of depression symptoms has clinical utility.

Pubertal development was assessed in two ways. First, the child completed ratings with the aid of schematic drawings of breast and pubic hair (Morris & Udry, 1980). Ratings of development from the drawings were used to estimate Tanner staging. Second, the child and caregiver were administered the *Petersen Physical Development Scale* (PPDS; Petersen et al., 1988). The PPDS includes questions about growth spurt, body hair, breast development and changes in skin scored on 4-point scales and one yes/no question about onset of menstruation. Girls were classified into five groups: pre-pubertal, beginning pubertal, mid-pubertal, advanced, and post-pubertal based on categories developed by Crockett (unpublished data). This approach has been used in other studies (e.g., Ge, Conger, & Elder, 2001), is consistent with the normal sequence of pubertal development identified by Tanner (1962) and has shown good reliability and validity (Robertson et al., 1992) and convergent validity with Tanner staging by physical exam (Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987). The main difference is that the Crockett staging includes menarche in the algorithm and the Tanner staging does not. These three measures were combined (as described below) to estimate pubertal development, an approach that is most likely to capture different dimensions of puberty (Shirtcliff, Dahl & Pollak, 2009). Child and parent reports of pubertal development were collected annually beginning at 9 years of age and continuing until both informants reported that the girl had reached Tanner stage 5. The percent of participants at each state of pubertal development at each age of assessment is presented in Table 2. Note that approximately two-thirds of participants had reached Tanner stages 2 or 3 by age 9, the age at which pubertal development was first assessed.

Analytic Approach

Analyses were conducted in a sequential manner, such that main effects were first examined followed by the inclusion of interaction terms to test our hypotheses regarding associations among pubertal development, depression symptoms, and race. This approach is appropriate given our goal of generating results on main effects that would be comparable to previous research and then extending this work to moderating effects within a large racially diverse sample.

Following Marceau et al (2011) estimates of the timing and tempo of pubertal development were calculated based on fitting nonlinear growth curve models to repeatedly measured indicators of pubertal development including the Tanner Stages and PPDS ratings by the participant and parent. To take into account the various indices of pubertal development, a

first-order pubertal development factor was specified with the various pubertal development measures as indicators and change in the first-order pubertal development factor was modeled with a second-order sigmoid growth model.

The longitudinal first-order factor model for pubertal development indicators can be specified as

$$y_{ijt} = \tau_j + \lambda_j \cdot f_{it} + u_{ijt} \quad (1)$$

where y_{ijt} is the j^{th} pubertal development score for individual i measured at time t , τ_j is the intercept for pubertal development score j , λ_j is the factor loading for pubertal development score j , f_{it} is the factor score (true pubertal development score) for individual i at time t , and u_{ijt} is the unique factor score for pubertal development score j for individual i measured at time t . We note that the intercepts, τ_j , and the factor loadings, λ_j , are invariant with respect to time, which was evaluated by traditional approaches to examine factor invariance (Meredith & Horn, 2001). The longitudinal factor model was fit with (A) all pubertal development measures, (B) just the breast development measures, and (C) just the pubic hair development measures. We also note that the factor analytic model takes into account the different scalings present in certain measures of pubertal development.

At the second-order level, changes in the first-order pubertal development factors, f_{it} , were modeled with a Gompertz growth model (Campbell et al., 2012). The Gompertz growth model can be written as:

$$f_{it} = g_1 \times \exp(-\exp(-g_{i2} \times (age_{it} - g_{i3}))) + d_{it} \quad (2)$$

where g_1 is the universal upper asymptote representing full pubertal maturity, g_{i2} is the individually-varying pubertal tempo parameter, g_i is the individually-varying pubertal timing parameter, and d_{it} is the time-dependent disturbance score for individual i at time t . Factor scores of g_{i2} and g_{i3} were estimated and then treated as observed variables in subsequent analyses studying changes in depression symptoms and the impacts of pubertal tempo and timing. We note that time was modeled in terms of age of the participant at each measurement occasion and ranged from 8 through 17. The model of Equations 1 and 2 was fit in Mplus using a first-order Taylor series expansion of the Gompertz growth model following Browne and du Toit (1991; see also Grimm et al., 2010; Grimm et al., 2011).

Longitudinal changes in depression symptoms were modeled via a multiphase or spline growth model (McArdle & Epstein, 1987; Meredith & Tisak, 1990; Cudeck & Klebe, 2001) with a fixed change or knot point at age 13 years based on the observation that race differences in depression symptoms and pubertal development narrowed from 13 to 17 years of age. The spline growth model with the intercept centered at age 10 can be expressed as:

$$x_{it} = b_{i1} + b_{i2} \cdot (\min(age_{it} - 10, 3)) + b_{i3} \cdot (\max(age_{it} - 13, 0)) + e_{it} \quad (3)$$

where x_{it} is the depression symptom score measured for individual i at time t , b_{i1} is the intercept or predicted depression symptom score for individual i at age 10, the first measurement occasion when depression symptoms were measured, b_{i2} is the linear rate of

change in depression symptoms prior to age 13 for individual i scaled in years, b_{i3} is the linear rate of change in depression symptoms after age 13 for individual i scaled in years, age_{it} is participant i 's age at time t , and e_{it} is the time-dependent residual for individual i measured at time t .

Variability in the intercept (b_{i1}), pre (b_{i2}), and post (b_{i3}) slopes from the growth model was then modeled based on pubertal timing and tempo based on data collected from ages 9 to 17 years, and race (African American vs. European American). The model including pubertal timing and tempo can be written as

$$\begin{aligned} b_{i1} &= \gamma_{10} + \gamma_{11} \cdot g_{i2} + \gamma_{12} \cdot g_{i3} + \gamma_{13} \cdot AA_i + \gamma_{14} \cdot AA_i \cdot g_{i2} + \gamma_{15} \cdot AA_i \cdot g_{i3} + \gamma_{16} \cdot g_{i2} \cdot g_{i3} + \gamma_{17} \cdot AA_i \cdot g_{i2} \cdot g_{i3} + h_{i1} \\ b_{i2} &= \gamma_{20} + \gamma_{21} \cdot g_{i2} + \gamma_{22} \cdot g_{i3} + \gamma_{23} \cdot AA_i + \gamma_{24} \cdot AA_i \cdot g_{i2} + \gamma_{25} \cdot AA_i \cdot g_{i3} + \gamma_{26} \cdot g_{i2} \cdot g_{i3} + \gamma_{27} \cdot AA_i \cdot g_{i2} \cdot g_{i3} + h_{i2} \\ b_{i3} &= \gamma_{30} + \gamma_{31} \cdot g_{i2} + \gamma_{32} \cdot g_{i3} + \gamma_{33} \cdot AA_i + \gamma_{34} \cdot AA_i \cdot g_{i2} + \gamma_{35} \cdot AA_i \cdot g_{i3} + \gamma_{36} \cdot g_{i2} \cdot g_{i3} + \gamma_{37} \cdot AA_i \cdot g_{i2} \cdot g_{i3} + h_{i3} \end{aligned} \quad (4)$$

where g_{i2} is the estimated pubertal tempo variable, g_{i3} is the estimated pubertal timing variable, AA_i is a dummy-coded African-American indicator variable (0=European American, 1= African American), γ_{11} , γ_{12} , γ_{13} , γ_{14} , γ_{15} , γ_{16} , and γ_{17} are the level-2 regression coefficients for the effects of pubertal tempo, pubertal timing, race, and the interactions between race and pubertal timing and tempo on girls' depression symptoms measured at age 10; γ_{21} , γ_{22} , γ_{23} , γ_{24} , γ_{25} , γ_{26} , and γ_{27} are the level-2 regression coefficients for the effects of pubertal tempo, pubertal timing, race, and the interactions between race and pubertal timing and tempo on individual changes in depression symptoms from age 10 through 13; γ_{31} , γ_{32} , γ_{33} , γ_{34} , γ_{35} , γ_{36} , and γ_{37} are the level-2 regression coefficients for the effects of pubertal tempo, pubertal timing, race, and the interactions between race and pubertal timing and tempo on individual changes in depression symptoms from age 13 through 17, and h_{i1} , h_{i2} and h_{i3} are level-2 disturbance terms representing unexplained variability of the level-1 intercept and linear slopes. The model of Equations 3 and 4 was fit using SAS PROC MIXED (Littell et al, 2006).

Results

Change in depression symptoms

Longitudinal changes in depression symptoms were modeled with a multiphase growth curve model. The mean intercept was 1.89 representing the predicted sample-level depression symptom score at 10 years of age, the mean of the pre-slope was $-.05$ ($p < .05$), and the mean of the post-slope was also $-.05$ ($p < .05$), indicating that depression symptom scores very slightly decreased, on average, from ages 10 through 17 at approximately the same rate. Significant variability in the intercept, pre-, and post slopes of depression symptoms highlights between-person differences in the level of depression symptoms at age 10 as well as between-person differences in the rate of change from ages 10 through 13 and from ages 13 through 17. Standard deviations of the pre- ($SD = .31$) and post-slopes ($SD = .29$) in conjunction with the means of the pre- and post-slopes indicate that 95% of the sample had pre-slope values between $-.66$ and $.56$ and post-slope values between $-.62$ and $.52$, highlighting that some participants' depression symptom scores increased whereas others' scores were decreased during the same time period within each developmental phase.

These results are consistent with findings from other large, community-based studies over the same developmental period (e.g., Dekker et al., 2007).

Regarding the covariance among the intercept, pre-, and post-slopes, the intercept/pre-slope covariance was -0.09 ($r = -.25$) and the intercept/post-slope was -0.08 ($r = -.26$) indicating that girls with higher depression symptom scores at age 10 tended to show greater reduction in depression symptoms over time. Lastly, the pre-/post-slope covariance was -0.00 ($r = -.02$), suggesting that individual changes from age 10 to 13 were unrelated to individual changes from age 13 to 17.

Race effects on depression symptoms

Differences in depression symptoms at age 10 and changes in depression symptoms were first evaluated in relation to race. Compared to European American females, African American females had higher levels of depression symptoms at age 10 by 0.82 points ($SE = 0.06$, $p < .01$) and greater reductions in depression symptoms from age 10 through 13; 0.10 points ($SE = 0.03$) per year, $p < .01$. Race was unrelated to individual changes in depression symptoms from age 13 to 17. European American girls' levels of depression symptoms did not significantly change from age 10 through 17, on average. Overall, African American girls had higher levels of depression than European American girls across development.

Race effects on pubertal development

The timing of pubertal development was earlier and the tempo slower for African compared to European American females. Specifically, African American girls' breast development occurred 10.9 months earlier ($\beta = -.33$, $p < .001$) and pubic hair development occurred 8.9 months earlier ($\beta = -.30$, $p < .001$). African American girls' breast ($\beta = -.05$, $p < .05$) and pubic hair ($\beta = -.30$, $p < .001$) development occurred at a slightly slower rate than European American girls.

Association of pubertal timing and tempo, with depression symptoms

In an initial step of evaluating the impacts of pubertal timing and tempo on changes in depression symptoms, the pubertal timing and tempo variables and their interaction were the sole variables included as predictors of the intercept, pre-, and post-slopes of depression symptoms (i.e., Model 1 — no race effect included). Timing and tempo variables were standardized (mean=0, standard deviation=1) in order to compare the magnitude of their effects. Parameter estimates of the associations between pubertal development and depression symptoms, and their standard errors, are provided in Tables 3A, 3B, and 3C for overall pubertal development, breast development, and pubic hair development, respectively.

For all models of pubertal development ratings (i.e., overall development, breast development, and pubic hair development), timing (i.e., the age of reaching Tanner stage 2) and tempo (i.e., the rate of pubertal maturation from 9-17 years) were significantly associated with depression symptoms at age 10, tempo was associated with the change in depression symptoms from age 10 through 13, and timing with the rate of change in depression symptoms from age 13 through 17 (Table 3A).

More specifically, earlier pubertal timing was associated with higher depression symptoms at age 10 and greater reductions in depression symptoms from age 13 through 17 (Table 3A). Participants with earlier pubertal timing therefore had higher levels of depression symptoms at age 10, the age at which the highest level of depression symptoms was reported. Further, although a greater decrease in depression symptoms was observed from ages 13 through 17 for girls with earlier compared to later timing, in general earlier pubertal timing continued to be associated with higher levels of depression symptoms into late adolescence (see Figure 1).

Tempo was also inversely associated with depression symptoms at age 10: participants who evidenced a slower pace in reaching full pubertal maturation tended to have higher levels of depression symptoms and vice versa (i.e., those with a faster pace had lower depression symptoms) at age 10 years (see Table 3A, 3B, 3C & Figure 2). However, from ages 10 through 13 years, depression symptoms tended to decrease among participants with slower pubertal tempo and increase among those with faster pubertal tempo (see Table 3A, 3B, 3C). Similar results were found for breast development and pubic hair development. In addition, for breast development, the interaction between pubertal timing and tempo on age 10 depression symptoms also was significant (Table 3B), suggesting that the association between slower tempo and higher levels of depression symptoms at age 10 was stronger when timing was later. No other significant interactive effects of timing and tempo on within-individual level and changes in depression symptoms were observed.

Associations among race, pubertal timing and tempo and depression symptoms

Race, product terms between race and timing, race and tempo, and the three-way product term between race, timing, and tempo were then added as predictors of the intercept and slopes of depression symptoms. The effect of race on level of depression symptoms at age 10 (reported above) remained significant, but the magnitude of the effect was smaller when pubertal timing and tempo estimates were included (see main effects of Race in Table 3A, 3B, 3C). For example, the difference in level of depression symptoms between African American and European American girls (at age 10) was .82, whereas models including pubertal maturation resulted in a difference between the two races of .56-.62, a reduction of approximately 24-32% (Table 3A, 3B, 3C). Indirect effects were then tested to assess whether race differences in depression were partially mediated by pubertal timing and tempo. Empirical bootstrap confidence bounds were calculated for each indirect effect with 200 bootstrapped samples. Indirect effects from race to pubertal development to level of depression at age 10 were significant for overall puberty, breast development, and pubic hair development when examining both timing and tempo of development. Indirect effects through timing variables ranged from 0.15 to 0.25, whereas indirect effects through tempo variables ranged from 0.02 to 0.08, suggesting that the timing of pubertal development accounted for a larger portion of the association between race and depression.

Effects of race on changes in depression symptoms over time were maintained and similar in magnitude when measures of overall puberty (Table 3A), breast development (Table 3B), and pubic hair development (Table 3C) were included in the model, as African American girls exhibited greater decreases in depression symptoms than European American girls

from ages 10 to 13 only (i.e., race was unrelated to changes in depression symptoms from ages 13 to 17). Indirect effects from race to an index of pubertal development to changes in depression were either non-significant or negligible. The indirect effect with the largest magnitude was -0.04 from race to overall puberty tempo to changes in depression from age 10 to 13.

Effects of timing, tempo and their interaction largely remained the same when race was included in the model (Table 3A, 3B, 3C), with moderate changes in the magnitude of some associations. For example, the effect of slower tempo of pubic hair development on depression symptoms at age 10 remained significant but was reduced by nearly 50% when race was included in the model. Pubertal timing, which was significantly associated with changes in depression symptoms from ages 13 to 17 years without race in the model, was no longer significant when race was included (Tables 3A, 3B, 3C). One interesting change was emergence of a significant association between tempo of overall pubertal development and change in depression symptoms from ages 13-17 (Table 3A); although girls with faster pubertal tempo had greater increases in depression symptoms from 10-13 years, slower pubertal tempo predicted higher levels of depression symptoms at age 10 and greater increases in depression symptoms from ages 13 to 17.

Discussion

The present study is the first to test the effects of pubertal timing and tempo in a racially diverse sample, using prospective, repeated assessments of pubertal maturation and depression symptoms beginning in childhood and continuing into late adolescence. Results from the present study confirm some, but not all, prior findings on pubertal development and risk for depression in girls. Consistent with our hypothesis, regarding pubertal timing, *earlier* timing (defined as the age of reaching Tanner stage 2) was associated with higher levels of depression symptoms. This effect was observed for breast and pubic hair development as well as overall pubertal development, and the magnitude of the association was similar for African and European American girls. There was a small, but significant association between timing and changes in depression symptoms from ages 13-17 years, but no significant effects of timing on changes in depression symptoms from 10 — 13 years. In general the effect of pubertal timing on depression symptom is best characterized as an intercept effect that persisted over time despite year-to-year changes in depression symptoms, and was maintained even following the completion of puberty (e.g., age 17).

This pattern of results is fairly consistent with the few studies designed to test the effects of pubertal timing across development. Joinson et al. (2013) used data from the ALSPAC study to show an association between early menarche and depression symptoms at 13 years that was maintained through age 16 ½, but was attenuated at ages 18 and 19, the same point at which depression scores tended to level off. Adolescent females who reported early pubertal timing relative to same-age peers were more likely to meet criteria for major depression throughout adolescence and into early adulthood than their on-time peers (Graber et al., 2004). These maintenance effects are also consistent with an organizational hormonal hypothesis wherein the timing of gonadal hormone exposure would be expected to exert long-lasting changes in the brain and behavior (Zehr, Culbert, Sisk & Klump, 2007).

The effect of tempo of pubertal development from ages 10-17 years on depression was complex. First, timing and tempo were inversely associated, a pattern that has been reported in other studies (Biro et al., 2006; Llop-Viñlas et al., 2004). Our hypothesis that faster tempo would be associated with higher levels of and increases in depression symptoms was based on the assumption that faster tempo may indicate exposure to higher levels of sex hormones and/or psychosocial vulnerabilities, which would confer risk for depression. Links between faster tempo and depression risk were, however, only partially supported in our data. Faster tempo predicted greater increases in depression symptoms, but only from ages 10-13 years. In contrast to our hypothesis, *slower* tempo was associated with *higher* depression symptoms at age 10 (in all models of pubertal constructs: overall, breast development, and pubic hair) and increases in depression from ages 13 to 17 (when the overall pubertal development construct was used).

Although the finding that slower tempo was associated with childhood and later depression symptoms was unanticipated, our pattern of tempo effects is in-line with the off-time/maturational deviance hypothesis: girls who mature at a different time or rate (e.g., faster or slower tempo) from their peers would be expected to be at highest risk for depression as these girls would experience milestones or transitions at different developmental times than their on-time/average peers. There are also examples in the broader literature in which a slower maturation confers risk. For example, boys' greater vulnerability to mild genetic anomalies has been hypothesized to be due to their slower rate of physical development early in life (Gualtieri & Hicks, 1985; Taylor, 1985). The rate of maturation of the cerebral cortex as measured by cortical thinning in childhood is positively associated with intelligence (Shaw et al., 2006). In the present study, the nature of the association between pubertal tempo and depression symptoms was typically an inverse association and is therefore consistent with the psychosocial off-time/maturational deviance hypothesis for depression risk and with this broader research on maturation. This result requires a re-thinking of how exposure to sex steroids may confer risk for psychopathology. Consistent with hypotheses in other areas of development, an extended period of exposure to increasing sex steroids during development may provide more opportunities for disruption in neural circuitries involved in the regulation of emotion, cognitions, and behavior.

The most parsimonious explanation is that an atypical pace, either fast or slow, of pubertal development is suboptimal with regard to risk for depression (e.g., consistent with the off-time/maturational deviance hypothesis). It is possible, however that the pace of pubertal development is more or less problematic at different points of inflection in the pubertal growth curve. There are data to indicate that the duration of transition through the middle stages of puberty, stages 2 to 4, is shorter than the duration of the transitions at the beginning or end of pubertal development (Ellis et al. 2011). Rapid progression through the stages during which development is already relatively accelerated may yield temporary or more transient problems in mood regulation that are associated with acute increases in levels of sex steroids, but wane as the system adjusts. The complicated pattern of results between tempo and depression symptoms observed in the present study underscore the importance of testing models using repeated measures of depression over extended developmental periods. Had we only measured depression symptoms through early adolescence, we may have

concluded girls with faster pubertal tempo were on trajectory towards higher levels of depression symptoms. The inconsistent results regarding tempo among the few existing studies also may be explained by the inclusion of different developmental periods of assessment.

As hypothesized, the previously reported race differences in depression symptoms in girls in this sample and in other studies were attenuated after accounting for variance in pubertal development. African American girls reached Tanner stage 2 earlier than European American girls, their tempo of pubertal development was slower, and they had higher levels of depression symptoms. The magnitude of difference in level of depression symptoms at age 10 was reduced by nearly 33%, once differences in pubertal timing were controlled. This is evidence that at least some of the variance in race differences in depression is due to the consistently reported differences in pubertal maturation. A reasonable next step would be to explore whether earlier pubertal timing and higher levels of depression symptoms in African American girls, compared to European American girls, could be attributed to a third variable, such as exposure to environmental stressors and/or experiences of discrimination. Associations between race and environmental factors on pubertal maturation have been reported. For example, Deardorff et al. (2012) found that availability of neighborhood recreation facilities was inversely associated with pubertal development, but only among African American girls. Given that pubertal timing appears to be largely genetically influenced, there may be race differences in the gene systems or epigenetic processes involved in triggering pubertal development. This type of investigation is still developing (e.g., Gajdos et al., 2009; Demerath et al. 2013), but may be highly impactful in contributing to the understanding of genetic influences, including the environments that affect gene expression, on pubertal timing and tempo and risk for psychopathology.

A number of limitations to the present study should be considered. The first is the reliance on maternal and self-report of pubertal development and the inference of hormonal effects rather than on direct measurement of changes in exposure to gonadal hormones. Our hypotheses regarding the impact of puberty on risk for depression are largely centered on the possibility of a developmentally based diathesis whereby the timing and frequency of release of pubertal hormones interacts with other hormonal and neurotransmitter systems to create vulnerability. Timing and tempo of breast development and growth in pubic hair are only proximal measures of that diathesis. This is especially true for girls at earlier stages of development, when Tanner staging, even when completed by a physical exam is not highly correlated with estrogen levels (Huang et al., 2012). We therefore cannot exclude the possibility of alternative mechanisms, such as the morphological changes of puberty and/or psychosocial factors, underlying pubertal timing-depression links. For example, those who mature earlier than their peers could also be at greatest risk for depression symptoms due to a lack of emotional or cognitive readiness for the physical, physiological, and psychosocial changes of puberty (i.e., the early maturing or developmental readiness hypothesis; see Ge & Natsuaki, 2009). It is also possible that biological and psychological vulnerability intersect, such that hormonal effects associated with early pubertal timing may alter or enhance sensitivity to social and emotional processes (Zehr et al., 2007; Mendle & Ferrero, 2012).

A second limitation is the left hand censoring of pubertal development. By age 9 years, over 75% of African American girls and over 25% of European American girls had reached Tanner stage 2 for overall pubertal development. Although this was the earliest age of assessment of pubertal development in the PGS, it appears that individual differences in timing and tempo would be better captured by including earlier ages. The association between depression symptoms at age 10 years and slower pubertal tempo is based on assessments of pubertal development from ages 10-17, thus we can only infer that the overall pace, beginning with the earliest stages of adrenarche, was slow. Earlier assessments of both pubertal development and depression would also allow for a test of direction of effect. Our assessments of pubertal development predated that of child-reported depression symptoms. Given the relatively stable level of depression symptoms from age 10-17 years in the present study and the fact that the largest observed effects of timing and tempo were on depression symptoms at age 10, it would be important to explore the possibility that depression symptoms experienced during early childhood hastens the onset of pubertal development.

Finally, annual assessments may not be optimal for testing associations between the timing and tempo of pubertal development and depression. More frequent assessments would likely increase the temporal specificity. A recently fielded study will assess mood and estradiol metabolites in urine every three months for three years providing a much more rigorous assessment of the effects of timing and tempo on mood (Steinbeck et al., 2012).

In summary, the present study extends the existing literature by testing associations between pubertal timing and tempo using multiple informants, over an extended period of time, in a large, racially diverse community sample. Timing and tempo both explain individual differences in depression symptoms at age 10 and changes in depression over time. Whereas only *earlier* pubertal timing conferred risk for depression, *slower* and *faster* tempo were associated with depression symptoms, the effects of which varied across developmental periods. Race moderated the association between pubertal development and depression. Race differences in depression were in part attributable to earlier pubertal timing and slower tempo among African American girls. Identifying the factors that influence pubertal timing and progression may help reveal causal mechanisms related to the development of depression in childhood and adolescence.

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Figure 1. Effects of overall pubertal timing on depression symptoms from ages 10 to 17 years
 Depression symptoms from ages 10 to 17 years for individuals grouped by age of pubertal timing from earliest to latest as defined by -2 , -1 , 0 , $+1$, and $+2$ standard deviations from the mean for pubertal timing controlling for tempo.

Figure 2. Effects of overall pubertal tempo on depression symptoms from ages 10 to 17 years
 Depression symptoms from ages 10 to 17 years for individuals grouped by rate of pubertal tempo from fastest to slowest as defined by -2 , -1 , 0 , $+1$, and $+2$ standard deviations from the mean for pubertal timing controlling for timing.

Table 1

Descriptive statistics for depression symptoms for total sample and by race

Age	N	Total Sample		African American		European American	
		Mean	(SD)	Mean	(SD)	Mean	(SD)
10	2303	1.94	(1.66)	2.27	(1.65)	1.51	(1.56)
11	2272	1.82	(1.66)	2.18	(1.66)	1.35	(1.54)
12	2250	1.68	(1.66)	1.95	(1.67)	1.30	(1.56)
13	2216	1.73	(1.75)	1.98	(1.79)	1.40	(1.65)
14	2178	1.81	(1.83)	1.97	(1.84)	1.64	(1.84)
15	1631	1.76	(1.87)	2.01	(1.93)	1.38	(1.70)
16	1067	1.73	(1.93)	1.85	(1.83)	1.50	(1.97)
17	526	1.52	(1.92)	1.69	(1.87)	1.25	(1.94)

Note: Depression symptom counts assessed by combining positive endorsements by either caregiver or child report.

Table 2

Percent of girls at each Tanner stage at each age for total sample and within African American (AA) and European American (EA) girls

Age	N	Stage 1			Stage 2			Stage 3			Stage 4			Stage 5		
		Total	AA	EA	Total	AA	EA	Total	AA	EA	Total	AA	EA	Total	AA	EA
9	2327	30.3	19.3	43.7	40.6	40.7	41.0	28.1	38.5	14.7	1.0	1.5	0.6	0.0	0.0	0.0
10	2302	14.4	6.3	24.8	35.3	28.6	43.5	43.4	54.7	28.8	6.9	10.3	2.9	0.1	0.2	0.0
11	2271	4.4	1.5	8.4	20.2	13.2	29.2	50.3	52.4	47.1	24.4	31.8	15.1	0.7	1.2	0.2
12	2250	0.7	0.1	1.7	7.0	3.0	12.1	34.9	29.7	41.5	55.0	63.8	43.6	2.4	3.5	1.1
13	2218	0.0	0.0	0.1	1.6	0.3	3.4	17.5	11.4	25.1	71.9	76.7	65.5	8.9	11.6	5.9
14	2176	0.0	0.0	0.0	0.2	0.0	0.6	6.2	3.1	9.9	74.6	75.0	73.7	19.0	21.9	15.8
15	1632	0.0	0.0	0.0	0.1	0.0	0.3	3.1	1.5	5.4	65.4	64.8	66.3	31.3	33.7	28.0
16	1050	0.0	0.0	0.0	0.7	0.7	0.7	7.1	5.1	9.9	44.4	43.3	45.8	47.8	51.0	43.6
17	516	0.0	0.0	0.0	0.0	0.0	0.0	5.2	6.9	3.4	28.9	32.3	26.5	65.9	60.8	70.1

Note: Tanner stage based on average scores of caregivers' reports on the Petersen Pubertal Development Scale (PPDS), and by the girls' reports on the PPDS and the Udry.

Table 3

Effects of timing, tempo, and race on depression symptoms at age 10 and changes in depression symptoms

	Model 1			Model 2		
	Depression sxss at age 10	depression sxss ages 10-13	depression sxss ages 13-17	depression sxss at age 10	depression sxss ages 10-13	depression sxss ages 13-17
	β (SE)	β (SE)	β (SE)	β (SE)	β (SE)	β (SE)
A. Overall Development						
Race						
Tempo (γ_{21})	-0.17** (0.03)	0.06* (0.01)	-0.02 (0.01)	0.56** (0.07)	-0.07* (0.03)	-0.03 (0.03)
Timing (γ_{32})	-0.37** (0.03)	-0.00 (0.01)	0.05* (0.01)	-0.16** (0.05)	0.09** (0.02)	-0.07** (0.02)
Timing*Tempo (γ_{16})	-0.05 (0.03)	0.00 (0.01)	0.02 (0.01)	-0.33** (0.05)	0.01 (0.02)	0.02 (0.02)
				-0.03 (0.03)	-0.02 (0.01)	0.02 (0.01)
B. Breast Development						
Race						
Tempo(γ_{21})	-0.12** (0.03)	0.06** (0.01)	-0.01 (0.01)	0.62** (0.07)	-0.09** (0.03)	-0.01 (0.03)
Timing (γ_{32})	-0.40** (0.03)	-0.00 (0.01)	0.05** (0.02)	-0.18** (0.06)	0.10** (0.02)	-0.05 (0.03)
Timing*Tempo (γ_{16})	-0.07** (0.03)	-0.01 (0.01)	-0.01 (0.01)	-0.37** (0.05)	0.01 (0.02)	0.03 (0.02)
				-0.06 (0.04)	-0.03 (0.02)	0.02 (0.02)
C. Pubic Hair Development						
Race						
Tempo (γ_{21})	-0.19** (0.03)	0.05** (0.01)	-0.02 (0.01)	0.62** (0.07)	-0.09** (0.03)	-0.02 (0.03)
Timing (γ_{32})	-0.32** (0.03)	-0.01 (0.01)	0.05** (0.01)	-0.10* (0.05)	0.05* (0.02)	-0.04 (0.02)
Timing*Tempo (γ_{16})	-0.05 (0.03)	-0.00 (0.01)	0.01 (0.01)	-0.25** (0.05)	-0.01 (0.02)	0.03 (0.02)
				-0.04 (0.03)	-0.01 (0.02)	0.01 (0.02)

Model 1 = no race effect included; model 2 = race effect included; depression sxss = depression symptoms; bolded parameter estimates are statistically significant;

*
p < .05,

**
p < .01; interactive effects among race, timing, and tempo were non-significant.