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Pubertal Synchrony and Depressive Symptoms: Differences by Race and Sex

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Abstract

Individual differences in the timing and tempo of pubertal development have been shown to be related to depressive symptoms during adolescence, particularly among girls. Another measure of variability in pubertal development is pubertal synchrony, the degree to which the development of pubertal indicators (e.g., breast growth and ancillary hair growth) are synchronized within the individual. Pubertal synchrony also has been hypothesized to be related to depressive symptoms, but, to date, only one study has tested this hypothesis. However, it remains unclear whether pubertal synchrony confers risk for depressive symptoms more proximally in time or differentially among boys or non-White youth. The current study examined the relation between pubertal synchrony and depressive symptoms concurrently and six months later as a function of race and sex in a community sample of 215 youth (53% female, 44.7% African American; Mean age = 12.90 years ($SD = 0.86$)). Girls with asynchronous development at Time 1 reported significantly higher depressive symptoms at Time 2 than girls with synchronous development and boys with asynchronous development. In addition, boys with asynchronous development at Time 1 had lower depressive symptoms at Time 2 than boys with synchronous development. Race did not moderate pubertal synchrony – depression relations. These results suggest that pubertal asynchrony is a risk factor for girls, but a protective factor for boys, and lend support for pubertal synchrony as a potential contributor to the gender gap in depression that emerges during adolescence.

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Authors' Contributions

AS generated hypotheses, created the database, ran and interpreted data analysis, and drafted the manuscript; AAG helped generate hypotheses, and helped draft the manuscript; LYA provided feedback on the manuscript; LBA helped design the original study and write the grant that funded the study, participated in the design and coordination of this study, and provided feedback on all drafts of the manuscript. All authors read and approved the final manuscript.

Data Sharing Declaration

The datasets generated and/or analyzed during the current study are not publicly available but may be available from the corresponding author on reasonable request.

Conflicts of Interest

The authors report no conflict of interests.

Ethical approval

The Temple University Institutional Review Board approved the protocol (IRB protocol #6844).

Informed Consent

Written informed consent was collected from all study participants after explaining their role in the study and before starting data collection.

Introduction

Boys and girls have comparable levels of depressive symptoms during childhood, but girls become twice as likely to be depressed compared to boys during adolescence (Hankin & Abramson, 2001). Notably, the emergence of this gender gap coincides with the onset of puberty (Lee, Guo, & Kulin, 2001), leading researchers to investigate whether pubertal processes may be responsible for the preponderance of depression among women. Indeed, more advanced pubertal development has been shown to be associated with increases in depressive symptoms for girls, but not boys, above and beyond the effects of age (Conley & Rudolph, 2009). Deviations in the timing or rate of pubertal development, otherwise referred to as pubertal timing and pubertal tempo, also have been shown to predict depressive symptoms during adolescence (Alloy, Hamilton, Hamlat, & Abramson, 2016; Mendle, Harden, Brooks-Gunn, & Graber, 2010). Another type of individual difference in pubertal development is pubertal *synchrony*, or the degree to which pubertal indicators are synchronized within the individual. Pubertal synchrony also has been hypothesized to be related to depressive symptoms, but has been understudied. The current study aims to address this gap and to examine sex and racial differences in the relation between pubertal synchrony and depressive symptoms.

Broadly, puberty consists of two distinct, but related, processes: adrenarche and gonadarche. Adrenarche starts around ages 6-8 when the adrenal glands begin to release adrenal androgens, which are responsible for the growth of ancillary and pubic hair (Saenger & DiMartino-Nardi, 2001), occurring around the age of 10 in girls and 11 in boys, on average (Mouritsen et al., 2013). During gonadarche, a cascade of hormonal changes results in the production of estradiol and testosterone by the ovaries and testes, respectively. This increase in estradiol and testosterone induces morphological changes (e.g., growth of sex organs like ovaries and testes and external changes like breast development, voice change, and facial hair), and ultimately, menarche in girls (Grumbach, 2002). These morphological changes begin between the ages of 9-11 in girls and 10-12 in boys (Mouritsen et al, 2013). Thus, synchrony can be thought of as the degree to which the development of ancillary hair matches the development of other morphological changes (e.g., breast growth in girls, facial hair or voice change in boys). Some degree of asynchrony is typical, particularly during the earlier stages of development, but as puberty progresses, most girls achieve development of pubertal indicators (e.g., breast development and ancillary hair growth) simultaneously (Susman et al., 2010).

To date, pubertal synchrony primarily has been investigated in relation to physical health outcomes in girls. For example, girls with relatively more advanced breast development have been shown to have a higher body mass (Biro et al., 2003) and a greater percentage of body fat than girls with more advanced pubic hair development (Novotny, Daida, Morimoto, Shepherd, & Maskarinec, 2011). However, researchers have theorized that variations in pubertal synchrony, like pubertal timing and tempo, also may be related to psychological outcomes. Indeed, some have proposed that asynchronous development may lead to heightened anxiety (Eichorn, 1975) or the development of psychopathology more broadly (Brooks-Gunn & Warren, 1985). Mendle (2014) speculated that the effect of pubertal synchrony could operate in both directions: (1) youth who experience multiple pubertal

changes concurrently may be overwhelmed by numerous changes happening at once and, in turn, may be more vulnerable to psychopathology (making synchronous development a risk factor), or (2) youth who experience multiple changes concurrently may perceive puberty to be a more cohesive, predictable process and feel more psychologically secure (suggesting that asynchronous development would be a risk factor). However, there has been little empirical investigation into the relation between synchronous development and the experience of psychopathology.

To date, only one study has investigated the impact of asynchronous development on depressive symptoms. In this study, utilizing a community sample of 355 Australian adolescent girls assessed at ages 13, 15, and 20, Thompson, Hammen, and Brennan (2016) found that girls with asynchronous development at age 13 reported higher levels of depressive symptoms at age 20, but not at age 15. Further, both girls with more advanced ancillary hair development compared to breast development and girls with more advanced breast development compared to ancillary hair development reported higher levels of depressive symptoms than youth with synchronous development. This study was an important first step in understanding the role of pubertal synchrony in the development of depression during adolescence. However, little is known about how pubertal synchrony may be related to psychopathology among boys. Additionally, the sample used in this study was predominantly White. Although the authors ran sensitivity analyses and determined that their results did not change when non-White participants were excluded, little is known about how the relation between pubertal synchrony and depressive symptoms may differ by race and ethnicity.

Further, this study tested the effect of pubertal synchrony on depressive symptoms two and seven years later, but not cross-sectionally or over short time intervals. The effects of other variations in pubertal development, like pubertal timing and pubertal tempo, are evident during development (Mendle et al., 2010), as well as into young adulthood (Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2004). However, it remains unclear whether the effects of pubertal synchrony occur concurrently, or more proximally to when pubertal synchrony is assessed.

Examining the effect of pubertal synchrony on depression among boys and non-White youth is important for a number of reasons. First, as previously discussed, rates of depression in adolescence have been shown to vary by sex (Hankin & Abramson, 2001). Second, rates of depression appear to differ by race (Bailey, Mokonoogo & Kumar, 2019), although the relationship between race and depression often is unclear. Indeed, some studies have found that Black youth report more depressive symptoms than their White peers (Mrug, King, & Windle, 2016); whereas others have found no differences in the level of depressive symptoms reported by Black and White adolescents (Gaylord-Harden, Burrow, & Cunningham, 2012). However, other large-scale epidemiological studies have shown that, compared to their White peers, Black adolescents were less likely to experience a depressive episode in the past year (Mojtabai et al., 2016). Further, there is evidence to suggest that the synchronization of pubertal indicators differs by both sex and race (Susman et al., 2010). For example, breast development starts earlier than pubic hair development in girls, but girls complete breast and pubic hair development at approximately the same age. Further, Black

girls enter each stage of development earlier than White girls. Conversely, boys enter genital development before pubic hair development, but pubic hair development is completed in less time. Black boys enter pubic hair and genital development earlier than White boys; Black boys and White boys complete genital development in approximately the same amount of time, but Black boys take six months longer to complete pubic hair development. These differences may have important implications for the effect of pubertal synchrony on depressive symptoms. Consequently, understanding the relation between pubertal synchrony and depressive symptoms among all youth can help construct a more comprehensive picture of how race and sex differences in depression emerge during adolescence.

The Current Study

The current study examined race and sex differences in the relation between pubertal synchrony at age 12-13 and the development of depressive symptoms both concurrently and six months later. Specifically, sex or race were tested as moderators of the relation between pubertal synchrony and depressive symptoms. Hypotheses were tested in a community sample of adolescents that was roughly half male and half female and half White and half African American/Black, offering a unique opportunity to examine how the relation between pubertal synchrony and depressive symptoms differs by sex and between White and African American/Black youth. It was hypothesized that there would be a significant interaction between pubertal synchrony and sex in predicting depressive symptoms, such that girls with asynchronous development would have higher levels of depression than girls with synchronous development. Hypotheses about the effect of pubertal synchrony on depressive symptoms among boys and the interaction between pubertal synchrony and race were non-directional, as there was no prior literature on which to base these hypotheses. These hypotheses can be considered exploratory.

Methods

Participants and Procedure

The study sample was a subset of participants from the Adolescent Cognition and Emotion Project (Project ACE, Alloy et al., 2012), a longitudinal study examining the etiology of depression in adolescence. A community sample of 639 adolescents (aged 12 or 13 years old at baseline) and their mothers or primary female caregivers were recruited from Philadelphia-area public and private middle schools via mailings supported and approved by the Philadelphia School District (N=8,662) and advertisements placed in local print media likely to reach the population of interest (approximately N=134). Interested biological mothers or primary female caregivers, hereafter collectively referred to as mothers, completed phone screenings to determine study eligibility. To be included in Project ACE, the adolescent was 12 or 13 years old, identified as Caucasian/White, African American/Black, or Biracial, and the mother agreed to participate. Participants were excluded from the study if either the adolescent's or mother's English reading or speaking skills were below a level sufficient to understand and complete study assessments, or if either the adolescent or mother endorsed a history of severe psychiatric illness (e.g., a psychotic disorder), intellectual disability, pervasive developmental disorder, or learning disability. Participants

were not excluded if they or their mother had a history of Major Depressive Disorder. Mothers provided written informed consent and written informed assent was obtained from all adolescents. For additional information about recruitment methods and criteria, please see Alloy et al. (2012).

Project ACE aimed to assess youth at baseline and again every six months. Visits were split into 6-month visits (one session) and annual visits (split across two sessions). Participants frequently missed follow-ups but returned for later sessions. Data from the current study were drawn from baseline (Time 1) and the participants' first follow-up, approximately six months later (Time 2; $M = 7.27$ months, $SD = 1.27$). At Time 1, participants reported on their demographic characteristics (e.g., sex, race) and completed self-report measures assessing pubertal development and depressive symptoms. At Time 2, participants reported again on their depressive symptoms.

The subsample for the current study included adolescents who had complete data on the primary study variables at Time 1 and Time 2 and who identified as either African American/Black or Caucasian/White. If a participant was missing data on one of the primary study variables, they were dropped from the sample and not included in the present study. Attrition analyses examined whether there were significant differences between those included in the current study and those excluded. As the number of participants who identified as Biracial was insufficient to examine meaningful group differences, all Biracial participants were excluded from the current sample. Two hundred fifteen participants (53% female, 44.7% African American) were included in the final study sample. Participants were an average age of 12.90 years ($SD = 0.86$) at Time 1 and 13.52 years ($SD = 0.86$) at Time 2.

Participants in the current sample did not significantly differ from those not included in terms of sex ($\chi^2(1) = .008$, $p = .93$), pubertal synchrony ($\chi^2(1) = .91$, $p = .34$), depressive symptoms at Time 1 ($t(604) = -1.76$, $p = .08$), or school lunch eligibility ($\chi^2(1) = 2.84$, $p = .09$). Participants included in the sample were more likely to identify as White ($\chi^2(1) = 4.028$, $p = .05$), and were younger at Time 1 ($t(610) = -2.54$, $p = .01$) compared to those not included. Participants included in the study also had mothers with more education and higher household incomes than those excluded ($t = -2.36$, $p = .02$ and $t = -2.95$, $p = .003$, respectively).

Measures

Pubertal Synchrony.—Pubertal synchrony was computed at Time 1 using items from the Pubertal Development Scale (PDS, Petersen et al., 1988), a five-item self-report questionnaire assessing pubertal development. The questions ask about growth in height, body hair, skin change, breast change (females only) or voice change (males only), and menstruation (females only) or facial hair growth (males only). With the exception of menstruation, all questions were rated using a 4-point Likert scale (1 = no development, 2 = development has barely begun, 3 = development is definitely underway, 4 = development is complete). Menstruation was scored dichotomously as either a 1 = “I have not yet begun to menstruate” or 4 = “I have begun to menstruate.” Scores for each item were averaged to yield a total score ranging from 1-4, with higher scores indicating more pubertal development. The PDS has acceptable psychometric properties, with an average internal

consistency of 0.77 for the five item measure, and good convergent validity, with Pearson's correlations ranging from $r = 0.61$ - 0.67 with physician ratings of development (Petersen et al., 1988). The measure also has been shown to adequately capture basal hormone variability (Shirtcliff et al., 2009) and has been used successfully in samples of older adolescents (Kong et al., 2013). Internal consistency in this sample was $\alpha = 0.72$ for girls and $\alpha = 0.71$ for boys.

Consistent with prior studies (Novotny et al., 2011; Thompson et al., 2016), pubertal synchrony among female participants was determined by computing differences in self-reported breast growth and body hair growth. Among male participants, pubertal synchrony was determined by computing differences between self-reported voice change and body hair growth. Participants initially were categorized into one of three categories of development: *synchronous development* ($N = 93$, 43.3%) for youth who reported no difference between the two pubertal indicators, more advanced *gonadarche* ($N = 15$, 7.0%) for youth who reported more advanced breast development (for females) or voice change (for males) relative to their body hair development, or more advanced *adrenarche* ($N = 107$, 49.7%) for youth who reported more advanced body hair development relative to their breast development (for females) or voice change (for males). As the number of participants in the gonadarche group was too small to examine meaningful group differences, the pubertal synchrony variable then was re-categorized as a dichotomous variable with youth classified as either synchronous ($N = 93$, 43.3%) or asynchronous ($N = 122$, 56.7%) in their pubertal development.

Depressive Symptoms.—Depressive symptoms were assessed using the Children's Depression Inventory (CDI), a 27 item self-report measure that assesses cognitive, affective, and behavioral symptoms of depression among children and adolescents (Kovacs, 1985). Items are rated on a 0-2 scale, and participants are asked to choose the statement that best describes how they have been feeling over the last two weeks (e.g. "I am sad once in a while" / "I am sad many times" / "I am sad all the time"). Items scores are summed, with higher scores indicative of more depressive symptomatology. The CDI has demonstrated sound psychometric properties, with good reliability and validity evidenced in prior studies (Saylor, et al., 1984; Masip et al., 2010). Internal consistency for this measure was $\alpha = .84$ at Time 1, and $\alpha = .81$ at Time 2.

Demographic Information.—Self-reported sex, race, socioeconomic status (SES), and birth date, used for calculating participants' ages at Time 1 and Time 2 in the study, were collected at the baseline visit of the main study. Socioeconomic status was assessed by parent report of family income, parents' education, and eligibility for receipt of the National School Lunch program (NLSP) (0=no, 1=yes). Eligibility for the NLSP is determined based on an income to needs ratio, taking into account the income of the family and the size of the household. It was significantly correlated with both maternal education ($r = -.40$, $p < .001$) and household income ($r = .51$, $p < .001$). Eligibility for school lunch was used as a proxy for SES in the current study. Age is highly correlated with pubertal development, and there is evidence that SES is related to the timing of pubertal development (Obeidallah, Brennan,

Brooks-Gunn, Kindlon, & Earls, 2000). Thus, age and SES were included as covariates in the present study.

Data Analysis Plan

Univariate analyses of covariance (ANCOVA) were employed to test the hypothesized interaction between pubertal synchrony and sex and the exploratory interaction between pubertal synchrony and race predicting differences in levels of depressive symptoms concurrently and six months later. Age and SES were entered as covariates in both models, and baseline depressive symptoms were entered as a covariate when predicting depressive symptoms six months later. Significant interactions were followed up with tests of simple main effects.

Results

Descriptive Statistics

Descriptive statistics for all study variables, stratified by race and sex, are presented in Table 1, and bivariate correlations among primary study variables are presented in Table 2. Pubertal synchrony was not significantly correlated with depressive symptoms at Time 1 or Time 2, and the strength of the correlation decreased from Time 1 to Time 2. When the sample was stratified by sex, pubertal synchrony was not significantly correlated with depressive symptoms at Time 1 or Time 2 in boys or girls. However, the strength of the correlation increases in girls from Time 1 to Time 2, but decreases in boys (Boys: Time 1: $r = .10$, Time 2: $r = -.17$; Girls: Time 1: $r = .03$, Time 2: $r = .12$). When the sample was stratified by race, pubertal synchrony was not significantly correlated with depressive symptoms at Time 1 or Time 2 in White youth or Black youth. The strength of the correlation was comparable at Time 1 and Time 2 among White youth, but decreases among Black youth (White youth: Time 1: $r = .09$, Time 2: $r = .08$; Black youth: Time 1: $r = .09$, Time 2: $r = -.08$).

Differences by Sex

Time 1.—There was not a significant main effect of pubertal synchrony predicting depressive symptoms at Time 1, $F(1, 200) = 1.13$, $p = .29$. There also was not a significant interaction between pubertal synchrony and sex in predicting depressive symptoms at Time 1, $F(1, 200) = 0.38$, $p = .54$.

Time 2.—There was not a significant main effect of pubertal synchrony predicting depressive symptoms at Time 2, $F(1, 199) = .06$, $p = .80$. However, there was a significant interaction between pubertal synchrony at Time 1 and sex predicting depressive symptoms at Time 2, $F(1, 199) = 11.05$, $p = .001$ (Figure 1). Tests of simple main effects revealed that among boys, those with asynchronous development at Time 1 had significantly lower depressive symptoms at Time 2 than those with synchronous development (Mean Difference = 1.81, $p = .02$). Among girls, those with asynchronous development at Time 1 had significantly higher depressive symptoms than those with synchronous development at Time 1 (Mean Difference = 1.55, $p = .02$). Moreover, girls with asynchronous development at Time 1 had significantly higher depressive symptoms at Time 2 than boys with

asynchronous development at Time 1 (Mean Difference = 2.19, $p = .001$). Boys and girls with synchronous development at Time 1 did not significantly differ in depressive symptoms at Time 2 ($p = .13$).

Differences by Race

Time 1.—There was not a significant main effect of pubertal synchrony predicting depressive symptoms at Time 1, $F(1, 200) = .75$, $p = .39$. There also was not a significant interaction between pubertal synchrony and race in predicting depressive symptoms at Time 1, $F(1, 200) = 0.31$, $p = .58$.

Time 2.—There was not a significant main effect of pubertal synchrony predicting depressive symptoms at Time 2, $F(1, 199) = .003$, $p = .96$. There also was not a significant interaction between pubertal synchrony and race in predicting depressive symptoms at Time 2, $F(1, 199) = 0.25$, $p = .62$.

Sensitivity Analyses

To test the robustness of these findings, the same models were re-run using a variable for pubertal synchrony that was computed using the facial hair item for boys instead of the voice change item. The pattern of results was similar to that presented above. There was not a significant interaction between pubertal synchrony and sex predicting depressive symptoms at Time 1, and there was not a significant interaction between pubertal synchrony and race predicting depressive symptoms at Time 1 or Time 2. However, there was a marginally significant interaction between pubertal synchrony and sex predicting depressive symptoms at Time 2, $F(1, 199) = 3.75$, $p = .054$. Tests of simple main effects revealed that among boys, there was no difference in depressive symptoms between those with synchronous and asynchronous development ($p = .50$). Among girls, those with asynchronous development had significantly higher depressive symptoms than those with synchronous development (Mean Difference = 1.56, $p = .03$). Moreover, girls with asynchronous development at Time 1 had significantly higher depressive symptoms at Time 2 than boys with asynchronous development at Time 1 (Mean Difference = 1.64, $p = .01$). Boys and girls with synchronous development at Time 1 did not significantly differ in depressive symptoms at Time 2 ($p = .59$).

Discussion

There is evidence that variations in the timing and tempo of pubertal development confer risk for depression during adolescence, and it has been hypothesized that asynchronous pubertal development also may be a risk factor. Although there is some evidence to suggest that asynchronous development is associated with increases in depressive symptoms prospectively among girls, questions remain about how asynchronous development is related to depression among boys or non-White youth. The current study investigated sex and racial differences in the association of asynchronous development with depressive symptoms both concurrently and six months later among adolescents who identified as either Caucasian/White or African American/Black. It was hypothesized that there would be a significant interaction between pubertal synchrony and sex predicting depressive symptoms at Time 1

and Time 2, such that girls with asynchronous development would report significantly higher depressive symptoms than girls with synchronous development. Tests of racial differences in the relation between pubertal synchrony and depressive symptoms were exploratory, as this question had not previously been addressed in the literature. Somewhat consistent with hypotheses, girls with asynchronous development reported higher depressive symptoms than girls with synchronous development at Time 2, but not at Time 1. Further, boys with asynchronous development reported significantly lower depressive symptoms than boys with synchronous development, and girls with asynchronous development reported significantly more depressive symptoms than boys with asynchronous development. Interestingly, there were no racial differences in the association between pubertal synchrony and depressive symptoms.

The finding that girls with asynchronous development reported more depressive symptoms six months later than girls with synchronous development is inconsistent with the findings of Thompson and colleagues (2016) and does not support their hypothesis that the deleterious psychological effect of asynchronous development may not reach the threshold of psychological significance until emerging adulthood. The authors found that asynchrony measured at age 13 was related to higher depressive symptoms at age 20, but not at age 15. However, the current study's findings suggest that these effects may be evident during adolescence six months later. One potential explanation for this discrepancy is the generational gap in the samples used in these studies. Youth in Thompson et al. (2016) were born between the years of 1981-1984, and youth in the current study were born between 1996-2000. Age and cohort differences in the onset of puberty, and particularly breast development, have been documented in the literature (Aksglaede, Sørensen, Petersen, Skakkebaek, & Juul, 2009). However, it is also possible that the effects of pubertal synchrony are evident at different ages across development. Future work should explore this possibility.

Moreover, asynchronous development conferred differential risk for depressive symptoms by sex, such that girls with asynchronous development reported significantly higher depressive symptoms six months later than girls with synchronous development and boys with asynchronous development. Additionally, boys with asynchronous development reported significantly lower depressive symptoms six months later than boys with synchronous development. These findings offer some insight to the speculation about the effects of pubertal synchrony described in Mendle (2014). Mendle suggested that asynchrony may operate as a risk factor, because youth view pubertal development as unpredictable, or it may operate as a protective factor, because youth are not experiencing multiple changes all at once. The current findings suggest that the effect of asynchronous development is sex specific. For boys, asynchronous development may be a protective factor, but for girls, pubertal asynchrony may be a risk factor.

Furthermore, a substantial portion (56.7%) of the study sample was classified as having asynchronous development, findings consistent with a previous study that found that youth in the earlier stages of puberty are more likely to have asynchronous development (Susman et al., 2010). In the sample used by Thompson and colleagues (2016), 36.1% of the sample was classified as asynchronous at age 13. Based on these data, asynchrony appears to reflect normative pubertal development. If asynchronous development is common and operates as a

protective factor for boys and a risk factor for girls, it may contribute to the gender gap in depression that emerges during adolescence (Hankin & Abramson, 2001).

The mechanisms of the relation between asynchrony and depressive symptoms among girls remain unclear. Previous work on the effects of early pubertal timing on depressive symptoms in girls has speculated that these girls are at greater risk for depression because of society's reaction to their development and their vulnerability to becoming involved with older, more deviant peers (Ge, Brody, Conger, Simons, & Murry, 2002). It would follow then that asynchronous girls with more advanced gonadarche (e.g., more advanced breast development) are at the highest risk for depressive symptoms. The asynchrony variable used in the current study was dichotomized (synchronous/asynchronous) because of small cell sizes when using a trichotomized variable, so the current findings do not speak to this hypothesis. However, among only girls with asynchronous development, a variable coded such that 0=more advanced adrenarche and 1=more advanced gonadarche was computed to offer some preliminary data for this hypothesis ($N = 61$, 7 with more advanced gonadarche). The correlation between synchrony and depressive symptoms at Time 1 was $r = .04$, and at Time 2 was $r = -.05$ (the same pattern held when the same correlations were conducted using the entire sample of asynchronous girls at baseline, $N = 131$). This can be interpreted to mean that girls with more advanced gonadarche reported higher depressive symptoms at Time 1, but girls with more advanced adrenarche reported higher depressive symptoms at Time 2. It should be noted that this is consistent with Thompson and colleagues (2016), who found that girls with more advanced adrenarche reported the highest levels of depressive symptoms at age 20. Future work should investigate this with a larger sample, as well as the mechanisms of this association, which is counter to expectations.

Interestingly, race did not moderate the association of pubertal synchrony with depressive symptoms at Time 1 or Time 2. The examination of racial differences in the effect of pubertal synchrony on depressive symptoms was exploratory, as there was no prior work on which to base hypotheses. However, these findings are consistent with past work that has demonstrated that the effects of pubertal timing operate similarly in Black and White youth. For instance, one study found that pubertal timing was associated with higher depressive symptoms among both African American and European American girls (Keenan et al., 2004). In another study, there were no significant differences in the effects of pubertal timing on depressive symptoms between Black boys and White boys or between Black girls or White girls (Mendle, Harden, Brooks-Gunn, & Graber, 2010). However, there also is evidence that the pubertal transition confers more risk for depressive symptoms among White girls than White boys or their minority peers (Hayward, et al., 1999). Examining a three-way interaction could help clarify racial and sex differences in the effects of pubertal synchrony on depressive symptoms in this sample. The current sample was underpowered to examine a three-way interaction, but future work should investigate this possibility in a larger sample.

The current study had a number of strengths, including a diverse community sample with roughly equal proportions of males and females and White and Black youth and good representation of low SES youth, and the implementation of both cross-sectional and prospective measures of depressive symptoms. These strengths allowed the current study to

expand on past work on the relation between pubertal synchrony and depressive symptoms in girls and also investigate how these relations may differ among boys and Black youth. However, the current study should be interpreted in light of its limitations. First, pubertal synchrony was assessed at just one point in time. In light of evidence that the degree of synchronization between pubertal indicators actually changes over the course of development (Susman et al., 2010), it is possible that results could differ if pubertal synchrony was assessed at a different age. Indeed, puberty is a dynamic process, and future work should consider the effect of age at which pubertal synchrony is assessed and seek to explore whether these associations change over the course of development. Second, there is likely to be great heterogeneity in the ethnicities of youth collapsed into groups labeled “White” and “Black.” It is plausible that the effects of pubertal processes differ depending on the ethnic and cultural context in which youth are raised. Unfortunately, these data were not available for the current sample, but future work should investigate this possibility. Relatedly, due to the nature of the sample and exclusion criteria for participation, results may not generalize to youth who are not attending public or private schools, youth with learning disabilities, intellectual disabilities, or severe psychiatric illness, or youth who do not identify as either Black or White. Finally, there was a large difference in size between the larger parent sample and the current study sample. Although participants in Project ACE were contacted frequently (every six months) to complete follow-ups, participants frequently missed some follow-ups, but resumed participation later in the study. Youth with complete data at baseline and the six-month follow-up were prioritized for inclusion in the current study, as it was an aim of this study to examine the association of pubertal synchrony with depressive symptoms concurrently and at their first follow-up. The current study sample did not differ from those excluded on the basis of sex, pubertal synchrony, or depressive symptoms. However, they were more likely to be White and younger at Time 1 and were more likely to have mothers with more education and higher household income. Consequently, it is possible that this attrition could have biased the results.

Conclusion

It has been hypothesized that pubertal synchrony may be related to depression, but only one study to date has tested this question. It remains unclear whether pubertal synchrony is related to depressive symptoms cross-sectionally or more proximally in time, and whether these effects differ by sex or race. The current study found that girls with asynchronous development reported higher depressive symptoms six months later than girls with synchronous development or boys with asynchronous development. Further, boys with asynchronous development reported significantly lower depressive symptoms six months later than boys with synchronous development. There were no racial differences in the relation between pubertal synchrony and depressive symptoms. These findings demonstrate that the deleterious psychological effects of pubertal asynchrony at age 13 may emerge as early as six months later and add to the understanding of pubertal asynchrony as a risk vs. a protective factor. Specifically, the current study offers evidence to suggest that asynchrony may be risk factor for girls and a protective factor for boys. These results also suggest that asynchrony may be a normative aspect of pubertal development. Future work should

consider how the effects of pubertal synchrony may change over the course of development and study this effect through a developmental lens.

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Biography

Allison Stumper is a doctoral student in Lauren B. Alloy's Mood and Cognition Lab at Temple University. Her research interests include how biological and environmental factors interact to confer risk for adolescent depression, with an emphasis on the role of biological changes as a result of the pubertal transition.

Amber A. Graham is a doctoral student in the clinical psychology program at Temple University working in Dr. Lauren B. Alloy's Mood and Cognition Lab. Her research interests include how cognitive processing, community and family processes, and cultural identity, independently and in interaction, influence the development, course, and treatment of mood disorders in youth of color.

Lyn Y. Abramson is the Sigmund Freud Professor of Psychology at the University of Wisconsin-Madison. She received her doctorate in Clinical Psychology from the University of Pennsylvania. Her major research interests include the developmental, cognitive, motivational, and cultural determinants of information processing about the self and the effects of early psychological, physical, and sexual maltreatment on the development of cognitive styles and vulnerability to depression in adulthood.

Lauren B. Alloy is Laura H. Carnell Professor and Joseph Wolpe Distinguished Faculty in Psychology at Temple University. She received her doctorate in Experimental and Clinical Psychology from the University of Pennsylvania. Her major research interests include cognitive, psychosocial, developmental, and, neurobiological processes in the onset and course of depression and bipolar disorder.

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Figure 1.

The interaction between pubertal synchrony at Time 1 and sex predicting depressive symptoms at Time 2.

Note: CDI = Children's Depression Inventory; Error bars are ± 1 SD.

Table 1.

Descriptive statistics of primary study variables by race and sex.

	White Girls (N=65)			White Boys (N=54)			Black/AA Girls (N=49)			Black/AA Boys (N=47)		
	N/Mean	%/SD		N/Mean	%/SD		N/Mean	%/SD		N/Mean	%/SD	
Age T1	12.73	0.68		12.80	0.82		13.08	0.98		13.08	0.93	
Pubertal Synchrony												
Synchronous	30	46.2%		23	42.6%		23	46.9%		17	36.2%	
Asynchronous	35	53.8%		31	57.4%		26	53.1%		30	63.8%	
Depressive Symptoms T2	5.83	5.47		4.49	3.43		5.15	5.45		4.34	3.71	

Note. AA = African-American; T1 = Time 1; T2 = Time 2.

Bivariate correlations among primary study variables.

Note: Pubertal Synchrony coded as 0=synchronous, 1=asynchronous; CDI = Children's Depression Inventory; SES = socioeconomic status.