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The Impact of Asynchronous Pubertal Development on Depressive Symptoms in Adolescence and Emerging Adulthood among Females

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Abstract

Puberty is accompanied by numerous psychological and interpersonal challenges, including a dramatic rise in the prevalence of depression among girls. Pubertal timing has been identified as a potent predictor of depressive symptoms among females, but less is known about other features of puberty. The present study sought to address this gap in the literature by examining the effect of pubertal synchrony, the degree to which morphological indicators of puberty develop concurrently, on depressive symptoms in adolescence and emerging adulthood in a longitudinal sample. Among 355 female participants, asynchronous development at age 13 was associated with increased depressive symptoms at age 20, but not age 15. Additional analyses indicated that pubertal timing moderated the association between synchrony and depressive symptoms at age 20, such that girls who exhibited asynchronous development had the highest levels of depressive symptoms when they matured later than peers. Results provide initial empirical support for the role of pubertal synchrony in the development of depression among females and are discussed with regard to the biopsychosocial processes that may connect features of puberty with the long-term development of psychopathology.

Keywords

puberty; depression; pubertal synchrony; pubertal timing; longitudinal

Introduction

In addition to the physiological changes that exemplify puberty, the pubertal experience is characterized by a variety of psychological and interpersonal challenges (Graber, 2013). Among girls, the transition to adolescence is marked by a dramatic increase in rates of depressive symptoms, which facilitates the emergence of a gender gap in depression prevalence that persists through much of adulthood (Kessler, 2003; Kuehner, 2003; Wade, Cairney, & Pevalin, 2002). Given the magnitude of the gender disparity in the prevalence of

depression, identifying factors that may contribute to the onset and persistence of heightened depressive symptoms among females is of critical importance. Research has suggested that variations in the experience of puberty can lead to increases in depression among adolescent females (Graber, 2013; Mendle, Turkheimer, & Emery, 2007) and that the effect of puberty on the development of depression continues across adolescence and into emerging adulthood (Copeland *et al.*, 2010; Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2004).

A number of biological, hormonal, and psychosocial hypotheses have attempted to explain the link between puberty and psychopathology (see reviews by Ge & Natsuaki, 2009 and Rudolph, 2014), with the most prominent highlighting the psychological and social costs of maturing in a manner that is “deviant” compared to peers (Petersen & Taylor, 1980). The *maturation disparity* hypothesis suggests that girls who mature off-time relative to peers may feel abnormal and alienated from others, resulting in increased stress and a sense of isolation that can contribute to the development of psychopathology, including depression (Brooks-Gunn, 1984; Caspi & Moffitt, 1991; Simmons & Blyth, 1987). Consequently, most studies have focused on the role of pubertal timing in the development of depressive symptoms among girls (see reviews by Graber, 2013 and Mendle *et al.*, 2007), with a particular emphasis on the detrimental effects of early timing. However, although the maturation disparity hypothesis was originally developed with regard to timing, it also highlights the risks associated with other pubertal characteristics in which adolescents can deviate from peers. Features of puberty other than timing have generally been overlooked in the literature to date (Mendle, 2014), although there is growing recognition that a variety of pubertal characteristics can impact psychological well-being (Mendle, Harden, Brooks-Gunn, and Graber, 2010).

One such characteristic is pubertal synchrony, the degree to which morphological indicators of puberty develop concurrently. In general, physical development does not occur with perfect simultaneity, with approximately half of girls exhibiting breast development prior to the onset of other morphological changes such as changes in body composition, acceleration in skeletal growth, genital development, and the growth of body hair (Susman & Dorn, 2013; Wheeler, 1991). As puberty progresses, development becomes more synchronous, such that girls tend to achieve maturation of secondary sexual characteristics (i.e., breast and pubic hair) concurrently (Susman *et al.*, 2010). Unfortunately, little is known about the specific mechanisms that promote synchronous or asynchronous development (Susman & Dorn, 2013).

Although some degree of asynchrony in morphological development is not uncommon, variations in synchrony have long been theorized to influence psychological well-being. Eichorn (1975) was the first to hypothesize that synchrony could impact mental health, arguing that asynchronous maturation could lead to heightened anxiety. Brooks-Gunn and Warren (1985) also suggested that synchronous versus asynchronous development could be associated with psychopathology, and although they did not predict the direction of the effect, Brooks-Gunn (1984) theorized that girls with more advanced breast development may experience particular interpersonal and psychological challenges due to the observable nature of the physical changes. Mendle (2014) offered theoretical explanations in both directions: (a) synchronous development may lead girls to feel more psychologically secure

because they are experiencing all aspects of puberty simultaneously, suggesting an association between asynchrony and psychopathology, or (b) undergoing multiple changes simultaneously may lead synchronously-developing adolescents to feel overwhelmed, suggesting an association between synchronous development and psychopathology. Although there is a body of literature indicating that girls have specific expectations and opinions around pubertal development in general (e.g., Brooks-Gunn, 1984; Brooks-Gunn, Newman, Holderness, & Warren, 1994; Brooks-Gunn & Ruble, 1982; O'Sullivan, Meyer-Balzburg, & Watkins, 2000), little is known about how girls perceive synchronous or asynchronous morphological maturation.

Furthermore, the impact of pubertal synchrony on psychopathology has never been studied empirically. Instead, the limited body of existing synchrony research has provided descriptive information regarding physical development, with a particular focus on secondary sexual characteristics. One recent study using a community sample determined that girls are more likely to demonstrate asynchronous breast and pubic hair development in the early stages of puberty (Susman *et al.*, 2010). Two additional studies on synchrony found that adolescent girls who exhibit more advanced breast development also have lower breast density than other girls (Novotny, Daida, Morimoto, Shepherd, & Maskarinec, 2011) and have higher body mass and greater percentage body fat than girls exhibiting more advanced pubic hair development (Biro *et al.*, 2003). Thus, despite theoretical reasons to suspect that pubertal synchrony may affect the development of psychopathology, empirical research is needed to test this phenomenon.

Additionally, features of puberty including synchrony, timing, and pubertal tempo (i.e., the speed of maturation) are necessarily “intertwined” (Mendle, 2014). The dynamic nature of pubertal development means that one adolescent can experience early maturation that progresses at a relatively slow tempo, while another initiates puberty on-time but exhibits a rapid tempo, such that their development eventually appears uniform. Similarly, individuals might display varying stages of morphological maturation at one point in development, but exhibit synchronous development at other points in adolescence, depending on the timing and tempo of maturation across each morphological feature. Thus, it has been hypothesized (Mendle, 2014) that pubertal characteristics may interact with one another to affect risk for psychopathology. For example, early timing may be perceived by girls as particularly deviant when development is also highly synchronous, such that multiple physical markers are maturing early. Alternatively, asynchronous development may be interpreted as especially deviant when it also occurs off-time relative to peers. The interactive nature of pubertal characteristics has been supported by research suggesting that the duration of puberty is affected by the timing of pubertal onset (Marti-Henneberg & Vizmanos, 1997). However, very little research has examined interactive effects of pubertal characteristics in the prediction of psychopathology, although one recent study found evidence for an interaction between timing and pubertal tempo in the prediction of externalizing behavior among boys (Marceau, Ram, Houts, Grimm, & Susman, 2011).

The present study seeks to address marked gaps in the literature by examining the effect of pubertal synchrony on the development of depressive symptoms among females in a longitudinal sample. Additional analyses will explore potential interactive effects of

synchrony and timing, due to the established association between early timing and the development of depressive symptoms among girls (Graber, 2013) as well as studies linking both early and late timing to depression among females (Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997; Natsuaki, Biehl, & Ge, 2009). Given that adolescent depression tends to persist or even worsen into emerging adulthood (Lewinsohn, Rohde, Klein, & Seeley, 1999; Rao, Hammen, & Daley, 1999; Schulenberg & Zarrett, 2006) as well as previous findings suggesting that pubertal timing is associated with psychopathology beyond adolescence (Copeland *et al.*, 2010; Graber *et al.*, 2004), the present analyses examine depressive symptoms at two distinct points in adolescence and emerging adulthood.

Hypotheses

Study hypotheses were generated in accordance with theory due to the lack of empirical research on pubertal synchrony and depressive symptoms. As summarized previously, explanations for an association between synchrony and psychopathology have been offered in both directions (Eichorn, 1975; Mendle, 2014): synchronous development may contribute to psychopathology due to the experience of multiple overwhelming changes simultaneously or asynchronous development may be associated with psychopathology due to a heightened sense of abnormality and isolation from peers. More advanced breast development has also been identified as particularly likely to contribute to psychopathology (Brooks-Gunn, 1984). In light of the presence of several competing theories in the literature, we hypothesize that girls will show varying levels of depressive symptoms in adolescence and emerging adulthood in response to synchronous development versus asynchrony, but we do not make a prediction about the direction of the hypothesized effect.

With regard to potential interactive effects of synchrony and timing, we predict that timing will moderate the hypothesized association between synchrony and depression, such that the effect of synchrony on depressive symptoms in adolescence and emerging adulthood will be most deleterious among early-maturing girls.

Method

Participants

Participants were drawn from a large, community sample of women and their children who took part in the Mater Misericordiae Mothers' Hospital-University of Queensland Study of Pregnancy (MUSP; Keeping *et al.*, 1989), a longitudinal study of children born in Brisbane, Queensland, Australia between 1981 and 1984. All women with publicly-funded insurance seeking obstetric care at the Mater Misericordiae Mothers' Hospital were invited to participate in MUSP at their first prenatal visit. Over 7000 women were recruited, and a subsample of 815 mother-child pairs ($N = 403$ girls, 49.4%) were selected from the original sample in order to explore the effects of maternal depression on youth. This subsample was identified by oversampling for maternal depression based on scores on the seven depression items of the Delusions-Symptoms-States Inventory (DSSI; Bedford & Foulds, 1978), which was administered to mothers on four occasions between pregnancy and youth age 5. Maternal depression status was subsequently confirmed through diagnostic interviews administered at youth age 15 using the Structured Clinical Interview for DSM-IV (SCID;

First, Spitzer, Gibbon, & Williams, 1995). Mean maternal DSSI scores prior to youth age 5 were significantly associated with maternal depression diagnoses based on the SCID ($B = 0.03$, $SE = 0.01$, $p < .001$).

Youth and their mothers completed follow-up measures at youth ages 13, 15, and 20. Of the 403 girls within the subsample, a total of 355 females had complete data on pubertal status at age 13 and were included in analyses examining outcomes at age 15. Participants included in the age 15 analyses showed no differences in race/ethnicity ($\chi^2(1,394) = 0.19$, $p = .72$), maternal education ($\chi^2(1,399) = 0.01$, $p = .99$), maternal depression history by age 5 ($\chi^2(1,399) = 0.48$, $p = .50$), or maternal depression history by age 15 ($\chi^2(1,401) = 0.31$, $p = .64$) compared to the full sample of 403 girls.

A total of 326 female participants were retained at the follow-up assessment and included in analyses examining outcomes at age 20. The 29 participants lost to follow-up by the age 20 analyses were no different from those retained in the study at age 20 in race/ethnicity ($\chi^2(1,347) = 0.09$, $p = .99$), maternal education ($\chi^2(1,352) = 0.79$, $p = .60$), maternal depression history by age 5 ($\chi^2(1,352) = 1.49$, $p = .25$), maternal depression history by age 15 ($\chi^2(1,354) = 0.84$, $p = .44$), pubertal synchrony ($\chi^2(2,354) = 3.14$, $p = .21$), or pubertal timing ($\chi^2(2,354) = 1.28$, $p = .53$).

The sample was primarily lower middle class, and the majority of participants' mothers had completed some high school ($N = 341$, 96.1%). Most participants identified as Caucasian ($N = 331$, 93.2%), while a minority of participants identified as Asian ($N = 12$, 3.4%), Maori/Pacific Islander ($N = 2$, 0.6%), Aboriginal ($N = 2$, 0.6%), and other ($N = 1$, 0.3%). Data on race/ethnicity were not available for seven participants. Race (Caucasian vs. minority) was not associated with synchrony ($\chi^2(2,346) = 2.24$, $p = .33$) or timing ($\chi^2(2,346) = 1.88$, $p = .39$). Given previous research indicating that Asian females are more likely than Caucasian females to exhibit more advanced breast development (Novotny *et al.*, 2011), all analyses were repeated excluding non-Caucasian participants. There were no changes in the direction, significance, or effect sizes of the obtained results when excluding non-Caucasian participants.

Among the 355 participants, 47 had a history of diagnosable maternal depression by age 5 (13.3%). A total of 48 mothers met SCID criteria for current major depressive episode or dysthymia at youth age 15 (13.5%), while 151 mothers (42.5%) met diagnostic criteria for lifetime depression or dysthymia at age 15.

Procedure

At youth age 13, the original sample of MUSP participants completed a battery of self-report questionnaires and follow-up interviews, including an assessment of current pubertal status. At youth age 15, the subsample of 815 mother-child pairs completed additional self-report and interview measures. Youth and their mothers completed a second follow-up at youth age 20. Separate interviewers assessed youth and their mothers, and all interviewers were blind to maternal depression status. All participants gave consent/assent at each data collection point, and all procedures were approved by the Institutional Review Boards of the University of Queensland, Emory University, and the University of California, Los Angeles.

Measures

Pubertal status—Pubertal status was assessed at age 13 (Mean = 13.88 years, SD = 0.31). Youth were presented with a series of drawings illustrating stages of physical development as identified by Tanner (1969) and adapted by Morris and Udry (1980). They were then asked to rate their currently level of morphological development by identifying which of five drawings most closely resembled their own bodies. Female participants rated the secondary sexual characteristics that have been most commonly studied in the literature: breast and pubic hair development. In the present sample, the mean self-rated Tanner breast stage at age 13 was 3.59 (SD = 0.71) with a range of 1 to 5. The mean self-rated Tanner pubic hair stage was 3.81 (SD = 0.67) with scores ranging from 2 to 5. Table 1 summarizes the distribution of pubertal status at age 13. Previous research has established that self-ratings of the Tanner stages are positively correlated with physician and parent ratings of physical development and generally demonstrate moderate to strong kappa coefficients (Brooks-Gunn, Warren, Rosso, & Garguilo, 1987; Duke, Litt, & Gross, 1980; Morris & Udry, 1980).

Pubertal synchrony—Consistent with prior research (Novotny *et al.*, 2011), pubertal synchrony was ascertained by calculating the difference between participants' breast and pubic hair Tanner stage ratings at the age 13 assessment. Participants were assigned to one of three categories of synchrony. Those with a difference score of zero were classified as exhibiting synchronous development ($N = 227$, 63.9%). Girls whose pubic hair Tanner stage rating was greater than their breast Tanner stage rating were classified as exhibiting signs of *pubarche* ($N = 97$, 27.3%), while those whose breast development was more advanced than their pubic hair development were classified as exhibiting evidence of *thelarche* ($N = 31$, 8.7%). This method of classifying synchrony is commensurate with prior studies (Novotny *et al.*, 2011). Participants identified as exhibiting signs of pubarche had a range of difference scores from 1 to 3, with most girls exhibiting a difference score of 1 ($N = 86$, 88.7%). All girls classified as exhibiting signs of thelarche had a difference score of 1.

Pubertal timing—Pubertal status at age 13 was used to calculate timing relative to same-sex peers. Self-rated pubertal status was regressed on chronological age in order to create a metric of timing (Dorn, Dahl, Woodward, & Biro, 2006). Separate linear regressions were conducted for breast and pubic hair development. The resulting residual scores reflect the timing of each participant's pubertal development relative to peers. Higher scores (e.g., more positive residuals) indicate earlier maturation relative to peers.

Residuals for breast and pubic hair development were moderately positively correlated ($r = 0.54$, $p < .001$), so the residual scores were averaged in order to create a single timing score for each participant (e.g., Conley & Rudolph, 2009).

Participants were then assigned to one of three categories according to their averaged residual score. A categorical timing variable was used in order to compare early-maturing, on-time, and late-maturing participants, since a continuous variable would only allow for the evaluation of early- vs. late-maturing girls. Commensurate with prior research (Dorn *et al.*, 2006; Stroud & Davila, 2008), participants were assigned to categories using a cutoff of ± 0.67 standard deviations from the mean in order to assign approximately 20% of the

sample to each of the outlying groups. Girls above 0.67 standard deviations from the mean were classified as early-maturing ($N = 71$, 20.0%), while girls below -0.67 standard deviations from the mean were classified as late-maturing ($N = 84$, 23.7%). All other participants were classified as maturing on-time relative to peers ($N = 200$, 56.3%).

Depressive symptoms—Depressive symptoms were assessed using the Beck Depression Inventory-II (BDI-II; Beck *et al.*, 1996) administered at ages 15 and 20. The BDI-II has shown high internal consistency and convergent validity in both clinical and community samples of adults (Beck *et al.*, 1996). The psychometric properties of the BDI-II have also been replicated in community samples of adolescents (Osman, Barrios, Gutierrez, Williams, & Bailey, 2008).

At age 15, the mean BDI-II score in the present sample was 6.97 ($SD = 7.05$) with a range of 0 to 44. A total of 55 participants (15.5%) met criteria for at least mild depression at age 15, with BDI-II scores of 14 or above. At age 20, the mean BDI-II score was 8.05 ($SD = 9.18$) with scores ranging from 0 to 52. A total of 55 participants (16.9%) met criteria for at least mild depression at age 20. BDI-II scores in the present sample demonstrated high internal consistency at age 15 (Cronbach's $\alpha = .87$) and acceptable internal consistency at age 20 (Cronbach's $\alpha = .70$).

Maternal depressive symptoms—Maternal depressive symptoms at youth age 5 were included as a covariate in all analyses and were assessed using the 7-item depression subscale of the DSSI, as discussed above. This subscale demonstrates high internal consistency and convergent validity with other validated measures of depression (Henry, Crawford, Bedford, Crombie, & Taylor, 2002). Scores on this subscale reflect the frequency of recent depressive symptoms. A total of 47 mothers in the present sample (13.2%) received a score of 7, indicating no evidence of depressive symptoms at youth age 5. The mean DSSI score was 12.00 ($SD = 4.49$) with a range of 7 to 31.

Statistical Analyses

Univariate analyses of covariance (ANCOVA) were employed to examine hypothesized differences between categories of pubertal synchrony in the prevalence of youth depressive symptoms at ages 15 and 20 as well as the hypothesized interaction between synchrony and timing. Synchrony was a categorical variable comprised of three categories: synchronous development, pubarche, and thelarche. Timing was also comprised of three categories: early-maturing, on-time, and late-maturing.

Given that the present sample was selected from the larger MUSP study by oversampling for maternal depression, all analyses included maternal depressive symptoms at youth age 5 as a covariate. All analyses also incorporated maternal education status as a covariate, due to the association between social disadvantage and pubertal timing (Ellis & Essex, 2007; Obeidallah, Brennan, Brooks-Gunn, Kindlon, & Earls, 2000). In order to control for prior youth depression, analyses examining age 15 depressive symptoms also included participants' scores on the Anxious/Depressed subscale of the Youth Self-Report (YSR; Achenbach, 1991), which was completed at the age 13 assessment. Analyses examining age

20 depressive symptoms covaried depressive symptoms at age 15 based on the BDI-II. All analyses were adjusted for the observed mean of all covariates.

Results

Descriptive Statistics

Descriptive statistics for all study variables are presented in Table 2, stratified by pubertal synchrony and pubertal timing.

Pubertal Synchrony

Synchrony was not significantly associated with depressive symptoms at youth age 15 ($F(2,342) = 0.67, p = .51$, partial $\eta^2 = 0.00$). However, synchrony significantly predicted depressive symptoms at youth age 20 ($F(2,290) = 3.90, p = .02$, partial $\eta^2 = .03$). Girls with synchronous development at age 13 exhibited significantly fewer depressive symptoms at age 20 than girls demonstrating signs of either thelarche ($p = .03$) or pubarche ($p = .03$). The association between synchrony and age 20 depressive symptoms is presented in Figure 1.

Interactive Effects of Pubertal Synchrony and Pubertal Timing

The next set of analyses examined the interaction between synchrony and timing in the prediction of youth depressive symptoms. At age 15, the main effect of synchrony on depressive symptoms was not significant ($F(2,336) = 1.94, p = .15$, partial $\eta^2 = .01$). However, there was a main effect of timing on age 15 depressive symptoms ($F(2,336) = 4.29, p = .02$, partial $\eta^2 = .03$), such that late-maturing girls exhibited significantly higher levels of symptoms than on-time girls ($p = .01$). There were no significant differences between girls who matured on-time and those who developed earlier than peers. The interaction of synchrony and timing did not significantly predict depressive symptoms at age 15 ($F(4,336) = 1.31, p = .27$, partial $\eta^2 = .02$).

At age 20, there were significant main effects of synchrony ($F(2,284) = 8.02, p < .001$, partial $\eta^2 = .05$) and timing ($F(2,284) = 4.52, p = .01$, partial $\eta^2 = .03$) on depressive symptoms. Consistent with the findings reported above, girls with synchronous development exhibited significantly lower depressive symptoms at age 20 than girls showing signs of thelarche ($p = .01$) or pubarche ($p = .001$). Additionally, girls who developed on-time had significantly lower age 20 depressive symptoms than late-maturing girls ($p = .004$).

Examining the interaction between synchrony and timing revealed that timing moderated the association between synchrony and age 20 depressive symptoms ($F(4,284) = 2.66, p = .03$, partial $\eta^2 = .04$), such that girls who exhibited asynchronous development had the highest levels of depressive symptoms when they matured later than peers. Late-maturing girls showing signs of either pubarche ($p = .01$) or thelarche ($p = .01$) exhibited significantly higher levels of depressive symptoms than those with synchronous development (Figure 2).

Of note, all analyses were repeated using a dichotomous synchrony variable in which girls exhibiting signs of thelarche and pubarche were collapsed into a single category in order to examine the general impact of asynchronous development on depression. The direction, significance, and effect sizes of all results at ages 15 and 20 were essentially unchanged.

Discussion

The present study sought to expand the literature on puberty and psychopathology by examining the role of pubertal synchrony in the development of depressive symptoms among females. Results suggest that asynchronous morphological development is associated with increased levels of depressive symptoms in emerging adulthood, but this association is not yet apparent in adolescence. Additional analyses found that pubertal timing moderated the association between synchrony and depressive symptoms in emerging adulthood, such that asynchronous development resulted in the highest levels of depressive symptoms among late-maturing girls. These findings represent the first empirical evidence of an association between asynchronous morphological development and psychopathology and offer preliminary support for an interactive, dynamic relationship between synchrony and timing in the prediction of depression among females.

Contrary to expectations, the effect of asynchronous development on depressive symptoms was not apparent until emerging adulthood. This suggests that the psychological effects of asynchronous development may emerge over time, so that by age 20 there is statistically significant evidence of heightened depression. This finding certainly does not exclude the likelihood that asynchronous development has deleterious psychological effects on adolescents, but simply suggests that these effects become evident statistically by age 20. While emerging adulthood is known as a challenging transitional period (Arnett, 2014), many concerns associated with this stage are similar to those of adolescence, including body image, self-esteem, and peer and romantic relationships. Thus, it may be that the impact of asynchronous development on these concerns has reached a threshold of psychological significance by age 20, particularly as emerging adults separate from their families of origin (Markiewicz, Lawford, Boyle, & Haggart, 2006). Future studies examining the effect of asynchronous pubertal development on psychopathology in adolescence and emerging adulthood are greatly needed, both to replicate the current results and to provide additional insight into the emergence of clinically significant depressive symptoms by age 20.

In the present sample, asynchronous development resulted in the highest levels of depressive symptoms among late-maturing girls, contrary to the majority of research supporting an association between early timing and depression (Graber, 2013; Mendle *et al.*, 2007). This suggests that off-time development may be pernicious in either direction, a conclusion supported by previous research that has identified curvilinear associations between timing and depression (Conley & Rudolph, 2009; Graber *et al.*, 1997; Natsuaki *et al.*, 2009). Particularly when combined with asynchronous morphological development, late maturation may provoke concerns about abnormality and “deviance” from peers that could ultimately lead to heightened risk for depression.

While the present findings corroborate the extensive literature documenting the effect of timing on depressive symptoms and provide initial evidence that synchrony also influences mental health, additional research is needed to elucidate the psychological, interpersonal, and biological mechanisms through which puberty affects psychopathology (Rudolph, 2014). In line with cognitive theories of depression (Beck, 1987), girls who mature asynchronously may develop the belief that puberty is an inconsistent, unpredictable

process, while synchronous maturation may reinforce the belief that one is progressing appropriately toward developmental milestones. Consistent with the maturation disparity hypothesis, the detrimental effects of asynchronous development may be attributable to perceptions of deviance from peers or the belief that one is developing abnormally. Additionally, given the strong relevance of interpersonal stress to depression in females (Hammen, 2003), the interpersonal significance of asynchronous development should not be overlooked. Signs of thelarche may be associated with particular interpersonal challenges (Brooks-Gunn, 1984), such that girls may receive romantic attention before they have attained the psychological and interpersonal resources to cope with this experience (e.g., Petersen & Taylor, 1980; Stattin & Magnusson, 1990). Furthermore, consistent with the *contextual amplification* and *personal accentuation* hypotheses (Caspi, Lynam, Moffitt, & Silva, 1993; Caspi & Moffitt, 1991; Ge & Natsuaki, 2009), which have been identified as highly relevant to the link between pubertal timing and depression (Benoit, Lacourse, & Claes, 2013; Rudolph & Troop-Gordon, 2010), difficult environmental contexts and pre-existing personal vulnerabilities may aggravate the stresses of asynchronous maturation, leading to increased depressive symptoms. For example, girls with problematic peer relationships or high levels of neuroticism may cope with the stresses of asynchronous development in a particularly ineffective manner, resulting in higher levels of depression.

Biological processes, including hormonal shifts, may also influence the association between synchrony and depressive symptoms (Angold, Costello, Erkanli, & Worthman, 1999; Rudolph, 2014). Asynchrony in the development of brain regions that regulate emotional processing (e.g., amygdala) and those responsible for executive functioning (e.g., prefrontal cortex) may enhance risk for psychopathology due to deficits in emotion regulation skills (Dahl, & Gunnar, 2009; Rudolph, 2014). The gonadal hormones responsible for variability in morphological maturation may also contribute to shifts in mood: previous research indicates that circulating levels of androgens and estrogens are related to negative emotionality and mood in females across the lifespan (Steiner, Dunn, & Born, 2003). Studies utilizing a biopsychosocial perspective are critically needed in order to understand the numerous contributing factors that link pubertal characteristics, including synchrony, with depression. Additionally, by elucidating the mechanisms linking synchrony and timing to psychopathology, parents, educators, and medical professionals will be outfitted with the knowledge to develop preventative interventions designed to limit the deleterious psychological impact of asynchronous and off-time development on girls' emotional and social functioning.

Strengths of the present study include the use of a moderately large sample assessed at two points in adolescence and emerging adulthood as well as the ability to statistically control for prior depressive symptoms when predicting later symptoms. Retrospective reporting bias was avoided by measuring constructs concurrently, including pubertal status. Additionally, to the authors' knowledge, this is the first study to explore associations between synchrony and psychopathology, addressing a notable gap in the literature on the psychological effects of puberty among young women and extending the limited prior research on features of puberty other than timing (Mendle *et al.*, 2010; Mendle, Harden, Brooks-Gunn, & Graber, 2012).

The primary limitation of the present study is the use of a single, relatively late assessment of pubertal status. While the use of a single measurement of pubertal development is consistent with much of the research on puberty and psychopathology (e.g., Rudolph & Troop-Gordon, 2010; Stroud & Davila, 2008; Teunissen *et al.*, 2011), puberty is a highly dynamic process and the degree of synchrony among different markers of development may shift over time due to variations in timing and tempo (Susman *et al.*, 2010). The present study provides a snapshot of the psychological effects of synchrony at one point in development, but may fail to capture the broader effects of synchronous versus asynchronous maturation across the pubertal transition. Assessing pubertal status at age 13 is not ideal given the normative age of onset and duration of puberty among girls. Furthermore, later assessments prevent comprehensive understanding of pubertal timing by failing to capture the interplay between timing and tempo (i.e., girls identified as late-maturing based on pubertal status at age 13 may have matured later than peers or may simply have progressed through puberty more slowly than others). In the future, the field of puberty research should move toward employing repeated measurements of pubertal status from late childhood through adolescence in order to capture the dynamic nature of pubertal development.

In addition, although self-ratings of the Tanner stages have generally shown high correlations with physician and parent ratings (Brooks-Gunn *et al.*, 1987; Morris & Udry, 1980) and many studies have utilized this method as part of the assessment of pubertal status (e.g., Angold, Costello, & Worthman, 1998; Conley & Rudolph, 2009; Rudolph, 2008), some have questioned whether self-ratings are an accurate and reliable method of assessing morphological development (Dorn *et al.*, 2006; Dorn, Susman, & Ponirakis, 2003). Furthermore, the use of a sample that was primarily Caucasian, limited to females, and oversampled for maternal depression is not optimal, and future studies should aim to explore associations between synchrony and psychopathology in more inclusive and representative samples. Finally, given the association between adiposity and pubertal development (Biro *et al.*, 2003; Kaplowitz *et al.*, 2001), the inability to control for body mass index or adipose tissue in the present analyses is not ideal, and future studies of puberty should certainly aim to measure and control for adiposity.

Conclusion

The present study offers the first empirical evidence of the contribution of pubertal synchrony to the development of psychopathology, demonstrating that asynchronous pubertal development is associated with increased depressive symptoms in emerging adulthood, but not adolescence. This effect was particularly pronounced among girls who matured later than peers. Much additional research is needed, both to corroborate the present findings as well as to elucidate the complex interplay of psychological, interpersonal, and biological processes that contribute to the higher prevalence of depression among females beginning in adolescence. Precise, repeated measurements of pubertal development encompassing both morphological and hormonal indicators will further advance the field and allow for greater understanding of the dynamic, interactive processes that comprise puberty. The present study represents a valuable step toward the expansion of research beyond the current focus on timing (Mendle, 2014). Additional studies exploring the role of

synchrony in the development of psychopathology as well as the interactive effects of various characteristics of puberty will continue to further our understanding of the psychological correlates and consequences of the pubertal transition.

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Figure 1. Group differences in depressive symptoms at age 20 by pubertal synchrony, adjusting for maternal depression history, maternal education status, and age 15 depressive symptoms. Error bars represent the standard error of the mean.

Figure 2. Group differences in age 20 depressive symptoms by pubertal synchrony and pubertal timing, adjusting for maternal depression history, maternal education status, and age 15 depressive symptoms. Error bars represent the standard error of the mean.

Table 1

Participants' pubertal status at age 13 assessment based on self-rated Tanner stages.

Tanner stage	Breast	Pubic hair
Stage 1	1 (0.3%)	0 (0%)
Stage 2	18 (5.1%)	14 (3.9%)
Stage 3	130 (36.6%)	78 (22.0%)
Stage 4	182 (51.3%)	224 (63.1%)
Stage 5	24 (6.8%)	39 (11.0%)

Table 2

Descriptive statistics of study variables by pubertal synchrony.

Variable	Synchronous pubertal development						Pubarche						Thelarche					
	Early-maturing	On-time	Late-maturing	Total	Early-maturing	On-time	Late-maturing	Total	Early-maturing	On-time	Late-maturing	Total	Early-maturing	On-time	Late-maturing	Total	Early-maturing	On-time
<i>N</i>	38	124	65	227	22	63	12	97	11	13	7	31						
Caucasian race (%) ^a	36 (94.7%)	118 (95.9%)	60 (93.8%)	214 (94.3%)	18 (90%)	60 (98.4%)	12 (100%)	90 (92.8%)	10 (100%)	12 (92.3%)	5 (71.4%)	27 (87.1%)						
Mean (SD) Tanner breast stage at age 13 (Range: 1–5)	4.34 (0.48)	4.00 (0)	2.91 (0.29)	3.74 (0.60)	3.91 (0.29)	2.98 (0.13)	1.92 (0.29)	3.06 (0.61)	5.00 (0)	4.00 (0)	3.00 (0)	4.13 (0.76)						
Mean (SD) Tanner pubic hair stage at age 13 (Range: 2–5)	4.34 (0.48)	4.00 (0)	2.91 (0.29)	3.74 (0.60)	5.00 (0)	4.06 (0.25)	3.33 (0.65)	4.19 (0.58)	4.00 (0)	3.00 (0)	2.00 (0)	3.13 (0.76)						
Mean (SD) BDI-II score at age 15 (Range: 0–44)	8.26 (5.93)	5.70 (5.97)	7.17 (6.84)	6.59 (6.27)	10.18 (8.62)	5.92 (6.10)	10.92 (13.06)	7.51 (8.03)	7.09 (5.80)	7.08 (6.22)	12.43 (15.25)	8.29 (8.86)						
Mean (SD) BDI-II score at age 20 (Range: 0–52)	7.03 (7.32)	6.79 (8.40)	6.88 (7.33)	6.86 (7.88)	14.00 (14.24)	7.23 (7.50)	15.30 (13.34)	9.64 (10.49)	11.86 (11.11)	8.15 (8.44)	19.00 (17.13)	11.65 (11.91)						

Note.

^aValues do not match the total number of participants due to missing race/ethnicity data (N = 7).