Published in final edited form as:

J Youth Adolesc. 2015 April ; 44(4): 922–939. doi:10.1007/s10964-015-0255-7.

Clarifying the Associations between Age at Menarche and Adolescent Emotional and Behavioral Problems

Erikka B. Vaughan,

Department of Psychological and Brain Sciences, Indiana University, Bloomington, IN 47405, USA

Carol A. Van Hulle,

Waisman Center, University of Wisconsin, Madison, WI 53706, USA

William H. Beasley,

Department of Pediatrics, University of Oklahoma Health Sciences Center, Oklahoma City, OK 73104, USA

Joseph L. Rodgers, and

Department of Psychology and Human Development, Vanderbilt University, Nashville, TN 37235, USA

Brian M. D'Onofrio

Department of Psychological and Brain Sciences, Indiana University, Bloomington, IN 47405, USA

Erikka B. Vaughan: ebvaugha@uemail.iu.edu

Abstract

Better understanding risk factors for the development of adolescent emotional and behavioral problems can help with intervention and prevention efforts. Previous studies have found that an early menarcheal age predicts several adolescent problems, including depressive symptoms, delinquency, and early age at first intercourse. Few studies, nevertheless, have explicitly tested (a) whether the associations with menarcheal age vary across racial/ethnic groups or (b) whether the sources of the associations are within-families (i.e., consistent with a direct, causal link) or only between-families (i.e., due to selection or confounding factors). The current study analyzed data from a nationally representative US Sample of females ($N = 5,637$). We examined whether race/ethnicity moderated the associations between early menarche and several adolescent problems by using multiple-group analyses and we examined the degree to which genetic and environmental

© Springer Science+Business Media New York 2015

Correspondence to: Erikka B. Vaughan, ebvaugha@uemail.iu.edu.

Author contributions: E. V. conceived of the study, participated in the theoretical framing, design, measurement and analytic plan, conducted analyses, and drafted the manuscript; C. V. H. participated in the operationalization and measurement of key variables and revisions of the manuscript, and provided analytical consultation; W. B. participated in the measurement of key variables and assisted in interpretation of the study and revisions of the manuscript; J. R. participated in the theoretical framing, measurement of key variables, interpretation of the study, and revisions of the manuscript; B. D. helped conceive of the study, participated in the theoretical framing, design, measurement, and analytic plan, and helped draft and revise the manuscript. All authors read and approved the manuscript.

Conflict of interest: The authors declare that they have no conflict of interest.

factors shared by family members account for the associations by comparing sisters and cousins with differing menarcheal ages. Menarcheal age predicted subsequent depressive symptoms, delinquency, and early age at first intercourse in the population. The magnitudes of the associations were similar across all racial/ethnic groups for all outcomes. The within-family associations (i.e., when comparing siblings and cousins with different menarcheal age) were large and statistically significant when predicting early intercourse, but not the other outcomes. The findings suggest that selection or confounding factors account for the associations between menarcheal age and subsequent depressive symptoms and delinquency, whereas the independent association between menarcheal age and early age at first intercourse is consistent with a direct, causal effect.

Keywords

Age at menarche; Internalizing; Externalizing; Mechanisms

Introduction

Research has demonstrated that emotional and behavioral problems during adolescence are associated with chronic and persistent problems during adulthood. Specifically, elevated symptoms of adolescent depression predict excessive adult drinking, smoking, criminal behavior, and relationship difficulties (Allen et al. 2014; Pine et al. 1999; Wickrama and Wickrama 2010); delinquency during adolescence predicts subsequent substance use and elevated externalizing symptoms and psychiatric disorder in adulthood (Reef et al. 2009, 2011; Reinke et al. 2012); and an early age at first intercourse predicts the contraction of sexually transmitted infection and engagement in later risky sexual behaviors (Kaestle et al. 2005; Sandfort et al. 2008). Understanding the etiology of adolescent adjustment problems may help efforts to prevent both elevated symptoms during adolescence and the continuation of related problems into adulthood.

Both normative (Bongers et al. 2003) and problematic changes in behavior during adolescence may be linked, in part, to pubertal development (Caspi and Moffitt 1991; Ge et al. 2003). Thus, it is important to distinguish between normative patterns of mild increases in risky behavior and emotional and behavioral patterns that are problematic and put adolescents at risk for later psychopathology. Among females, early pubertal *timing*—experiencing advanced pubertal status compared to peers—has been tied to elevated depressive symptoms, delinquency, and early initiation of sexual behavior during adolescence (Negri and Susman 2011). Whether or not pubertal timing per se would put adolescents at risk for both proximal and persistent psychopathology is less clear. Given the demonstrated associations with later outcomes, if we can further understand how and why pubertal timing is tied to these outcomes, we may be able to improve our intervention and prevention efforts for adolescent emotional and behavioral problems. Alternatively, we may prevent expensive research efforts aimed at linking pubertal timing and psychopathology that are exploring the wrong processes. As such, we sought to further examine the mechanisms through which pubertal timing influences these outcomes in the current study.

Several theories posit mediating mechanisms that account for the associations between the timing of pubertal development and adolescent emotional and behavioral problems. The *maturation disparity hypothesis* (Ge and Natsuaki 2009) infers that early pubertal timing poses risk because of the discrepancy between advanced physical changes and immature psychosocial capabilities that render early maturers less able to manage the biopsychosocial changes associated with puberty. Heightened social expectations (actual or perceived) from peers and adults due to the older appearance of individuals who reach menarche earlier (e.g., receipt of romantic interest from older individuals and rising responsibilities in school and at home) may act as stressors, increasing risk for depressive symptoms (Resnick et al. 1997) or may act as pathways into romantic relationships with older and/or deviant peer groups that increase risk for engagement in delinquent acts (Haynie 2003; Resnick et al. 1997) and early intercourse (Vanoss Marín et al. 2000). This stress and attention may be particularly difficult to navigate for early physiological maturers because they may not have the cognitive and emotional maturity to respond to parental and peer expectations. Consistent with this perspective, (Skoog and Stattin 2014) recently developed an integrated model, which posits that maturation-disparity will only predict problem behaviors in contexts that facilitate interaction with older and more deviant peers.

The *hormonal influence hypothesis* infers that early pubertal timing poses risks because the timing of hormonal changes that occur during puberty influence emotional and behavioral problems throughout adolescence.

Recent human research implicates neuroendocrine changes related to cortisol reactivity (Ellis et al. 2012) and pulmonary volume (Whittle et al. 2012) as mediating the link between pubertal timing and adolescent depressive symptoms. Additionally, some animal models provide insight into a possible connection between pubertal timing and subsequent impulsivity, which predicts both adolescent delinquent and sexual behavior. Findings from studies of hamsters suggest that the brains of newborns are highly sensitive to gonadal hormones, and that this sensitivity declines as offspring age (Schulz et al. 2009). If this is comparable for humans, girls who experience gonadarche (most prominently indicated by menarche and breast development) at an earlier age would be exposed to the influence of gonadal hormones when the brain is still more sensitive to gonadal influence. For instance, early-maturing girls would experience greater gonadal influence during the neural development of reward responsivity than during the neural development of executive control (Steinberg 2008), putatively resulting in longstanding influences on impulsive behavior. Although theoretically compelling, the hormonal influence hypothesis has received limited empirical support (Ge and Natsuaki 2009).

The *psychosocial acceleration theory* (Belsky et al. 1991) is a life history theory that posits experiencing psychosocial stressors during early childhood accelerates pubertal timing, and that accelerated pubertal timing influences subsequent patterns of behavior as part of a reproductive strategy (Ellis et al. 2012; Neberich et al. 2008). From this view, a reproductive strategy that maximizes time for reproduction and the number of offspring is strategic if environmental experiences indicate uncertainty in early childhood caretaking. The putative link between pubertal timing and adolescent depression and delinquency is therefore unclear from this theoretical lens because they are not directly tied to reproduction. One review from

this perspective posited that both accelerated and decelerated pubertal timing are linked to depressive outcomes (Del Giudice and Ellis 2014), and an empirical article indicated that early menarcheal age did not mediate a link between early psychosocial stressors and delinquency (Belsky et al. 2010). The same empirical article, however, indicated that menarcheal age mediates the link between psychosocial stressors during early childhood and adolescent sexual behavior (Belsky et al. 2010), which can be interpreted as more consistent with the acceleration of reproduction.

The maturation-disparity, hormonal influence, and psychosocial acceleration hypotheses posit nuanced mechanisms by which pubertal timing putatively causes emotional and behavioral problems during adolescence. Yet, there are two broad explanations for the associations between menarcheal age and adolescent emotional and behavioral problems (Harden 2014). One possibility, consistent with the aforementioned hypotheses, is that early menarcheal age is an independent risk factor for the development of adolescent emotional and behavioral outcomes (i.e., there is a causal relation). If so, the mechanisms responsible for the association between menarcheal age and adolescent outcomes would be specific to menarcheal age and independent of a female's family background. Alternatively, females from families with an earlier average menarcheal age may "select" into the adolescent outcomes. That is, certain factors that some families share may make members more likely to both have an earlier menarcheal age *and* to experience any associated outcomes (Mendle et al. 2007). In such a case, the observed association between pubertal timing and the development of emotional and behavioral problems would be spurious. Indeed, some family background characteristics that may be shared by sisters, such as family structure and inter-parental conflict, predict both timing of menarche (e.g., Ellis et al. 1999) and adolescent emotional and behavioral problems (e.g., Donahue et al. 2010). Furthermore, research indicates substantial contributions of *both* genetic factors (i.e., heritability estimates) and shared environmental factors to the variability in menarcheal age (Anderson et al. 2007; van den Berg et al. 2006), depressive symptoms (Boomsma et al. 2005; Silberg et al. 1999), delinquent acts (Tuvblad et al. 2006), and early age at first intercourse (Moore et al. 2014). These findings bring about the possibility that genetic factors or environmental factors that make siblings similar could confound the associations between menarcheal age and subsequent problems (Moore et al. 2014).

Although these theories refer to pubertal timing in a broad sense, pubertal timing is a multi-faceted construct, with several possible indicators. Most research exploring pubertal timing has used menarcheal age (i.e., age at first menstruation) to index pubertal timing (Dorn et al. 2006). Self or parent report of menarcheal age is a useful measure because it is a discrete event that indicates a female has likely experienced the maturation of the hypothalamic–pituitary–adrenal axis, and is in the midst of the maturation of the hypothalamic–pituitary–gonadal axis (Bordini and Rosenfield 2011; Marshall and Tanner 1969). Consistent with the above theoretical frameworks, many studies have identified that early menarcheal age is associated with adolescent emotional and behavioral problems (see Mendle et al. 2007; Negri and Susman 2011 for reviews). Few studies, however, have used design features that can test the generalizability of the associations and rule out confounding factors that may account for the identified associations (Harden 2014). The current study, therefore, used

statistical analyses and methodological design features to examine the mechanisms by which depressive symptoms, delinquency, and early intercourse are associated with menarcheal age during adolescence by testing (a) whether associations differ across racial and ethnic groups and (b) whether associations remain when controlling for genetic and environmental confounds (i.e., remain within-families) or whether they are attenuated (i.e., between-family processes account for the association).

Testing Race and Ethnicity as a Moderating Factor

In the US, many contextual factors (e.g., cultural distinctions, the experience of systemic discrimination) are systematically related to race and ethnicity (Williams 1999). Therefore, if race and/or ethnicity moderate the predicted associations between menarcheal age and adolescent emotional and behavioral problems in the US, the results would suggest that these associations are modifiable because they would vary by context. There may be several manifestations of different contexts, including cultural values, rites of passage, and perceptions of pubertal changes. The possibility that differences in developmental context influence associations between menarcheal age and adolescent adjustment is captured in the *contextual amplification hypothesis*, which posits the stressfulness and/or developmental “fit” of a context may exacerbate or attenuate the putative risk of an early menarcheal age (Ge and Natsuaki 2009). One posited context that may influence the association between menarcheal age and emotional and behavioral problems is the parent and child orientation toward the physical changes that accompany pubertal change. Research suggests that changes in BMI are related to pubertal development. Further, research indicates that Black girls have more body satisfaction than White girls, particularly during this developmental period, and that body satisfaction is less associated with mental health for Latina girls (Ge et al. 2001). If body dissatisfaction, for instance, is a mechanism linking menarcheal age to adolescent emotional and behavior problems, then the association between menarcheal age and such problems may vary across race and/or ethnicity.

The extant findings exploring whether race/ethnicity moderates associations between menarcheal age and adolescent behavior are inconsistent. Some studies suggest no moderation in the link between menarcheal age and adolescent internalizing (Benjet and Hernández-Guzmán 2002; Carter et al. 2012), but others suggest a weaker menarcheal age-internalizing link for Black and Latina females in the US (Carter et al. 2009, 2011; DeRose et al. 2011). Notably, few of these studies explicitly tested race as a moderator. One study found that menarcheal age did not predict externalizing problems among African American youth (DeRose et al. 2011), but a larger study recently found that the link between early menarche and peer deviance and delinquency were similar across racial and ethnic groups (Mrug et al. 2014). Finally, one large study using data from the National Longitudinal Study of Adolescent Health demonstrated that the menarcheal age-sexual initiation link was strongest for Latina females, weaker among Caucasian females, and weakest among African American females (Cavanagh 2004).

Findings regarding racial and ethnic differences may be inconsistent for several reasons. First, as some researchers have noted (e.g., DeRose et al. 2011), many studies have included suboptimal sample sizes of non-Caucasian participants, limiting the statistical power to

detect potentially small associations. Second, because few studies have explicitly examined race as a moderating factor, methodological differences among studies (e.g., whether menarcheal age was measured prospectively or by retrospective report, the age range during which the outcome was measured, etc.) may have led to inconsistencies in findings. It, therefore, will be important to compare studies that are more methodologically similar. To address these limitations in the literature, the current study tested whether race and ethnicity moderated these associations in a large, nationally representative US sample, as some previous work has examined.

Testing Within-Family Associations

In order to determine whether the associations are consistent with a causal effect or are due to confounding factors, researchers must use methods that can account for putative genetic and environmental confounds. To address this issue, some studies have included variables measuring the early childhood environment and family experiences as covariates in models of menarcheal age predicting adolescent adjustment (e.g., Joinson et al. 2011), but including measured covariates does not rule out unmeasured confounds (see Moore et al. 2014 for a discussion of this). Research designs that compare associations within families can help rule out unmeasured confounding factors (Rodgers et al. 2000; D'Onofrio et al. 2013).

Quasi-experimental, multi-level familial designs leverage the natural variations among individuals within a family to control for unmeasured genetic and environmental factors shared among related individuals (D'Onofrio et al. 2013; Lahey and D'Onofrio 2010; Rutter 2007). If there is a direct link between menarcheal age and adolescent emotional and behavioral problems, the association should persist in a within-family analysis that controls for unmeasured genetic and environmental factors. If, however, selection is occurring, the association would only be present in a between-family analysis, and would be diminished in a within-family analysis. In the current study we utilized two kinds of quasi-experimental family designs: sibling and cousin comparisons. Specifically, we compared the emotional and behavioral outcomes of sisters and female cousins who differed in their menarcheal age, thereby controlling for genetic and environmental factors shared among sisters or female cousins (D'Onofrio et al. 2013). Each method has benefits and limitations. Sister comparisons account for more confounding factors because sisters have more genetic and environmental similarity than cousins. Cousin comparisons, however, relax key assumptions of the sister comparison (e.g., you cannot compare singleton siblings, but you can compare singleton cousins to cousins with siblings) and can provide either converging or diverging evidence to evaluate the results of a sister comparison.

If the mechanisms underlying the associations between menarcheal age and the outcomes found in the population are independent of family background (i.e., all factors that make family members similar), then individuals within the same family who differ in menarcheal age should also differ in the outcomes. This finding would be consistent with the hypothesis that menarcheal age is a causal contributor to adolescent adjustment problems for females. For example, if different sisters reached menarche at different times, then they may have had different expectations from parents and peers at different ages, which would put them at differential risk for emotional and behavioral problems.

If the mechanisms are consistent with selection, then genetic and/or environmental factors that are shared by sisters and differ between families would account for the association between menarcheal age and adolescent emotional and behavioral problems. The key mechanisms would therefore be shared by sisters, and the process by which pubertal timing is associated with risk would be something that differs between families. In this case sisters would have the same rates of behavior and emotional problems regardless of their menarcheal ages.

Importantly, these designs do *not* control for unmeasured genetic and environmental factors that *differ* between sisters that may also explain identified links between menarcheal age and adolescent adjustment problems. Nevertheless, when associations persist even after controlling for unmeasured covariates shared among sisters they are *more likely* to be consistent with causal inferences. Putative risk factors that fit this description *and* that are of clinical significance (i.e., demonstrate a substantial effect size) should be more rigorously examined for prevention and intervention efforts (Kraemer et al. 1997).

Few studies have used within-family designs to test the association between menarcheal age and adolescent emotional and behavioral outcomes (Harden 2014). We are unaware of any such study of the link between menarcheal age and depressive symptoms. Some studies examining externalizing behaviors have been conducted. Burt et al. (2006) examined timing of menarche and conduct disorder using bivariate twin analyses and found that shared environmental factors that accounted for variation in menarcheal timing were highly correlated with shared environmental factors that accounted for variation in conduct problems at approximately the same age. Rodgers et al. (2015) replicated the results of Burt et al. (2006) using both childhood and adolescent conduct problems in the National Longitudinal Survey of Youth. These results suggest that menarcheal age does not independently predict conduct problems, but that environmental factors shared within families account for the association between the two. Because conduct problems and delinquency are closely related (Woolfenden et al. 2002), this same process may apply to delinquent behaviors during adolescence. Rowe (2002) and Moore et al. (2014) both examined menarcheal age and adolescent sexual behavior in the National Longitudinal Study of Adolescent Health and found that shared genetic factors largely accounted for the observed link between menarcheal age and early intercourse.

The existing quasi-experimental studies suggest that factors shared among sisters (genetic and/or environmental) explain the association between pubertal timing and subsequent emotional and behavioral outcomes. This pattern of findings suggests that menarcheal age may not independently contribute to risk for the development of emotional and behavioral patterns during adolescence, which is not consistent with a causal influence interpretation. Yet, these studies were limited in the breadth of adolescent outcomes explored (i.e., none for depressive symptoms or delinquent acts per se) and the study designs (i.e., mostly bivariate twin analyses). The findings from sibling comparison analyses may be more generalizable than those of twin analyses, because siblings are a much more typical type of family relationship than twins. The current study, therefore, extended previous work by examining multiple outcomes using multiple family designs to examine whether our findings would be consistent with previous work.

The Current Study

Although several studies have indicated associations between menarcheal age and depressive symptoms, delinquent acts, and sexual initiation, few studies have utilized nationally representative, prospective longitudinal designs with the ability to compare menarcheal age across racial/ ethnic groups and within families. The current study, therefore, rigorously tested whether menarcheal age independently predicted these outcomes across people of varying backgrounds. We had two main aims. First, we tested whether associations between menarcheal age and adolescent emotional and behavioral problems were similar across broad ethnic and racial categorizations in the US. If menarcheal age consistently predicts outcomes across racial and ethnic groups, findings would suggest that the association is generalizable across a range of sociocultural contexts. Second, we tested whether the association between menarcheal age independently predicted risk for the development of adolescent adjustment problems, including depressive symptoms, delinquency, and early age at first intercourse, when comparing across sisters and female cousins. If menarcheal age predicts the outcomes within-families, our findings would indicate that the association between menarcheal age and adolescent outcomes is *independent of* genetic and environmental factors shared among families, which would be consistent with a causal model. If menarcheal age is only associated with the outcomes between-families, our finding would be consistent with a selection model, suggesting that the association between menarcheal age and adolescent outcomes is *confounded by* genetic and/or environmental factors shared. That is, menarcheal age would be an indicator of factors that *both* increase the likelihood of early menarcheal age *and* increase the likelihood of experiencing adolescent behavioral problems.

Methods

Participants

The sample included reports from female participants of the 1979 National Longitudinal Survey of Youth (NLSY79) and the subsequent surveys of the daughters of NLSY79 females (CNLSY) (Bureau of Labor Statistics 2008a, b; Chase-Lansdale et al. 1991). The sample of mothers came from two merged subsamples within the NLSY79 cohort. The first subsample is a nationally representative, household sample of youths ($n = 6,111$) who were 14–21 years of age on December 31, 1978. The second subsample is an over-sample of Black and Hispanic/Latina youths, also age 14–21 ($n = 3,642$). All daughters of the women in the initial samples comprise the females in the CNLSY, who were the target individuals for the current study. These daughters have been assessed biennially since 1986 (with year of birth ranging from 1970 to 2008 in the sample used) on various domains including emotional and behavioral adjustment. Because of the wide age range of daughters in the CNLSY sample, participants were in the target study age range (with the measures described below ranging from being assessed at birth to being assessed at age 17) at different times, with some not being old enough to report on the key outcome variables at the most recent measurement wave ($n \approx 396$), and others having reported on the outcome variables as early as the 1970s ($n \approx 259$). Additionally, the age at which participants were first assessed varies from birth ($n \approx 3,415$) to age 12–14 ($n \approx 6$). Of a total of 5,627 females included in the full

data set, we used data from both mother-reports and self-reports for those daughters in the CNLSY who met our requirements for inclusion on the menarche variable and the critical outcome measures—depressive symptoms ($n = 3,069$), delinquent acts ($n = 3,279$), and early age at first intercourse ($n = 3,021$).

Primary Measures

Menarcheal Age—We constructed the menarcheal age variable using information from mother-reports ($n = 2,081$), self-reports ($n = 795$), and/or both mother and self-reports ($n = 459$). Until daughters were 14 or 15 years of age, mothers were asked whether or not her daughter had experienced her first menstrual period, and if so, both the age (e.g., 11) and month/year (e.g., June, 1998) at which her daughter experienced it. Beginning at age 14 or 15, daughters were asked the same questions about their own menstrual periods, usually if their mothers had not already responded. In cases in which both mothers and daughters responded, their responses were moderately correlated ($r = .43$ in the analyzed sample). Because individuals were asked both the age and the month/year, we prioritized the use of the month/year measure because it was more precise. Ideally, each participant would have a single report of the month/ year at which she reached menarche. However, there were several ways in which participants diverged from this ideal. Some participants had both mother and self-reports, some had multiple reports from the same rater ($n = 190$ for mothers and $n = 75$ for daughters), and some only had a report of age, without a report of month/year ($n = 486$). To address different patterns of reporting, we averaged mother and self-reports, averaged multiple reports by the same rater, used month/year reports when available, and if not, used the age report + .5 to estimate within-year variability as guided by previous research (Rodgers et al. 2008).

Mean menarcheal age for the entire sample (presented in Table 1) was similar to means found in previous research (American Academy of Pediatrics and American College of Obstetricians Gynecologists 2006; Ge et al. 2003; Wu et al. 2002). Though some past studies have analyzed menarcheal age by dividing the sample into timing categories, research suggests that categorization of pubertal timing may conceal relationships with depression and delinquency (Negri et al. 2008), so we analyzed menarcheal age as a quantitative/continuous variable for our primary analyses.

Adolescent Depressive Symptoms—Daughters completed a subset of seven items from the Center for Epidemiological Studies Depression Scale (CESD; Radloff 1977) beginning at 14 or 15 years of age. These items assessed how often in the past week individuals had problems with appetite, concentration, mood, motivation, and sleep (on a Likert scale from 0 to 2 where 0 = rarely, 1 = sometimes, and 2 = most of the time). Cronbach's alpha for the items, calculated from age 15–17 ranged from 0.68 to 0.71, consistent with what has been demonstrated in previous work (Orth et al. 2009). Participants who responded to at least five of seven items for at least one wave of data collection were included in the analyses. The mean of the 5–7 completed items was taken to create a summary score that was used for analyses. Because participants in the CNLSY were assessed biennially, most participants have a score for age 14 *or* age 15, but some have a score for both ages if the assessment fell at the beginning of 1 year and the end of another.

Due to this assessment schedule, we considered age 14–15 as one wave and age 16–17 as a second wave. For each individual we found the mean response score across the full age range from 14 to 17 years to maximize our power to detect an association. For most participants ($n = 2,095$) this was based on a single score. We chose to assess depressive symptoms beginning at age 14–15 because by this age there is a full differentiation between girls who have reached menarche early and on time, and those who will reach menarche late in relation to the average girl.

Adolescent Delinquency—Daughters completed seven items assessing delinquency based on the Self-Reported Delinquency Interview (SRD; Elliott and Huizinga 1983). The SRD has been used in studies examining adolescent delinquency (e.g., Moffitt & Caspi 2001) and predicts criminal convictions in the CNLSY (Lahey et al. 2006). The items were coded for whether each individual did (1) or did not (0) severely injure someone, lie to a parent about something significant, steal from a store, vandalize others' property, get in severe trouble at school, skip school, or run away overnight/stay out overnight without permission. The variable represents the number of times an individual engaged in one of these acts across two assessment waves. Thus, we took the mean number of endorsed SRD items across the ages of 14, 15, 16, and 17 and rounded this mean to the nearest whole number to obtain an approximate measure of the number of delinquent acts in which participants engaged across this age range. Participants with data for at least one assessment wave were included in analyses. The mean number of delinquent acts in which a participant engaged from age 14–17 was 1.4. Cronbach's alpha ranged from 0.55 to 0.67 across age 14–17.

Early Age at First Intercourse—At each assessment wave beginning at age 14–15, daughters were asked to self-report whether or not they had ever engaged in sexual intercourse, and if so, how old they were, in years, when they first did. Although we chose to focus on the entire range of the predictor, because we were focusing on problem behaviors as outcomes, we chose to analyze age at first intercourse as a binary variable of “early” or not. Consistent with previous research (Donahue et al. 2013; Zimmer-Gembeck and Helfand 2008), we used age 16 as a cutoff such that all females whose age at first intercourse was younger than 16 years or earlier was considered “early.” We therefore created a dichotomous measure for whether or not daughters had engaged in early intercourse. All participants who were at least 18 by the last assessment wave and had responded to this measure by age 17 were included in our analyses.

Race/Ethnicity—In the NLSY79 (the mother generation), race/ethnicity was coded as “Latina,” “Black,” or “Non-Latina, Non-Black” at the initial assessment. The Non-Latina, Non-Black group consisted of all individuals who did not fall within the other two groups, and was predominantly Caucasian. These broad categorical groupings were based on how participants in the mother sample self-identified from a list of over 20 more specific racial/ethnic categories. Daughters' race/ethnicity was subsequently categorized based on her mother's self-report.

Analytic Plan

Analyses were conducted in *Mplus* version 7 (Muthén and Muthén 1998–2010). We standardized the outcome measure for depressive symptoms in order to help with the interpretation of the results. We used full information maximum likelihood with robust standard errors (*mlr*) due to missingness and skewness of the data. We used sampling weights to account for the non-random sampling methods used to collect national data to assure that the parameter estimates were representative of the population. We ran separate multilevel models for each adolescent outcome—depressive symptoms (standardized for interpretation), delinquent acts (in mean number of acts), and early age at first intercourse (as early or not). The outcome for depressive symptoms and delinquent acts was the mean value across age 14 and 17. We chose to use the mean across this developmental period which typically falls within the high school years as the outcome in order to maximize power and include data from as many participants as possible. Because some participants either only completed the assessments at one age across the full age range, and others had not lived through the full risk period, comparing the two age ranges would, in some cases, not be comparing the same people. For all models, variance was estimated at three levels—individual girls, nested within her mother’s current household (all girls who lived with the same mother), nested within her mother’s original household (all mothers who lived in the same original NLSY79 household). Females who were the biological children of the same mother were considered sisters. Female offspring of mothers who lived in the same household in the original National Longitudinal Survey of Youth data collection were considered cousins. All models included birth order and age at the last assessment wave as covariates.

Our series of four separate analytical models answered four main research questions. In Model 1, we tested whether menarcheal age predicted the outcomes across the population. We tested both linear and curvilinear models because some research indicates that late pubertal timing may be associated with emotional and behavioral problems (e.g., Carter et al. 2012). Menarcheal age was centered at the sample mean for all girls who were not missing on the outcome of interest, and no predictors at the family level were included.

In Model 2, we tested whether the association attained in Model 1 was similar across broad racial and ethnic groups using multiple group analyses. We compared a model in which all parameters were constrained to be equal across Black, Latina, and Non-Black, Non-Latina groups to a model in which the estimate for menarcheal age predicting the outcome in question varied freely across groups. We carried out Chi square difference tests; a constrained model that fit the data significantly worse than the full model was evidence for moderation. We also examined the magnitude of the point estimates for each group.

In Model 3, we examined the independence of menarcheal age as a risk factor by testing whether the attained association remained when testing among cousins. Menarcheal age was centered at the mean for all female cousins in a family who were not missing on the outcome of interest. The centered menarcheal age score was a measure of how much each individual deviated from the average menarcheal age among cousins within a family. This analysis partially controlled for genetic (6.25–12.5 % for differentially related cousins) and

environmental factors shared among cousins. Additionally, the mean menarcheal age for the all cousins was included in the model as a predictor to control for the family-level effect.

In Model 4, we further examined the independence of menarcheal age as a risk factor by testing whether the attained association remained when comparing sisters who differed in their menarcheal age. This model did not include estimates for cousin comparisons. Instead, we separately explored comparisons of sisters. Menarcheal age was centered at the mean for all sisters who were not missing on the outcome of interest. The centered menarcheal age score was a measure of how much each individual deviated from the average menarcheal age among sisters within a family. This analysis partially controlled for genetic factors (of which maternal half-siblings share 25 % and maternal full siblings share 50 %) and controlled for all environmental factors shared among sisters (Lahey and D’Onofrio 2010). Therefore, a persistent association above and beyond these shared genetic and environmental factors among sisters provides support for an independent association, consistent with an inference that early menarcheal age is a causal risk factor. In contrast, an attenuated association provides evidence that menarcheal age is a selection factor. The mean menarcheal age for all sisters was included as a predictor in the model to control for the family-level effect. To preserve statistical power for the analyses, the current models did not distinguish between full and half-sisters and did include rare cases of identical twins (who share 100 % of their genes).

Finally, we tested a series of sensitivity analyses to examine some of our analytic assumptions. We did not include these within the main analyses because these analyses would be less than optimal for estimating the associations with as much data as possible. We ran four sets of sensitivity analyses. First, we tested whether we would have a similar pattern of findings when using only mother reports of menarche or only daughter reports of menarche in models. Second, due to possible concerns about collapsing reports of adolescent depressive symptoms and adolescent delinquency across a 4-year age range, we tested the associations separately at ages 14–15 and 16–17. Third, because many past studies examining pubertal timing have used categorical measures (e.g., early timing, average timing, and late timing) and because some research suggests that findings may differ when using categorical instead of continuous measures of menarcheal age (Negri et al. 2008) we included models with menarcheal age as a categorical predictor. Menarcheal ages at least one standard deviation below the mean (i.e., 11 years or younger) were considered “early” and menarcheal ages at least one standard deviation above the mean (i.e., 13.58 years or older) were considered “late”. Fourth, because our main analyses included siblings within the same household with different genetic relatedness, we sought to test the analyses in families that only had full siblings.

For all analyses, we used linear models when predicting depressive symptoms (measured quantitatively/continuously), negative binomial models when predicting delinquent acts (measured as a count variable), and logistic models when predicting early age at first intercourse (a binary outcome). In the multiple group analysis, we estimated the mean levels of depressive symptoms for each group, as well as thresholds for the count and binary outcome. When predicting delinquency and early age at first intercourse, thresholds at the family level were estimated separately for the three known classes of race/ethnicity.

Results

Descriptive statistics and correlations for menarcheal age, depressive symptoms, delinquent acts, and early age at first intercourse are presented in the Table 1. The average menarcheal age was 12.29, and ranged from age 7.58 to age 17.67. The mean and range of menarcheal age was almost identical across subsamples for specific outcomes ($M_{\text{dep}} = 12.29$, range = 7–17; $M_{\text{del}} = 12.29$, range = 7–17; $M_{\text{int}} = 12.30$, range = 7–17). We found significant, small correlations between menarcheal age and depressive symptoms, delinquent acts, and early age at first intercourse (Cohen 1992). Intraclass correlations indicated that, cousins in the same extended family share 24 % of the variance in menarcheal age, 8 % of the variance in depressive symptoms and 15 % of the variance in delinquency was within extended families. When examining the data as sisters nested within nuclear families, 28 % of the variance in menarcheal age, 10 % of the variance in depressive symptoms, and 15 % of the variance in delinquency was within groups of children that shared the same biological mother.

Depressive Symptoms

Unadjusted linear and nonlinear associations are depicted in Fig. 1. In the linear model, menarcheal age inversely predicted mean depressive symptoms between age 14 and 17 across the population ($b = -0.05$, $p < 0.05$) (see Table 2). For every 1 year decrease from the mean menarcheal age, there was a 0.05 standard deviation increase in depressive symptoms, indicating that an earlier menarcheal age was associated with more depressive symptoms. In the nonlinear model (Figure 1, Panel C), the quadratic term was significant ($b = 0.02$, $p < 0.01$). However, the non-linear association (depicted in Fig. 1, Panel A) does not indicate that late menarcheal age is predictive of more depressive symptoms. Further, the fit statistics for the linear (AIC = 8,690.052; BIC = 8,714.171) and nonlinear (AIC = 8,687.058; BIC = 8,717.207) models indicate that model fit of the more parsimonious linear model is comparable to that of the nonlinear model. For reference, we have depicted the model-derived estimates for the linear, quadratic, and categorical models. In the model in which menarcheal age was categorized into early, average, and late, early menarcheal age predicted more depressive symptoms compared to average menarcheal age, but late menarcheal age did not predict depressive symptoms compared to average age. Because we were interested in early menarcheal age, we chose to use the linear model for subsequent analyses.

The estimate for menarcheal age predicting adolescent depressive symptoms was -0.03 for Latina girls, -0.05 for Black girls, and -0.05 for Non-Black, Non-Latina girls. Modified Chi square difference tests [$\chi^2(2) = 3.15$, $p = 0.21$] indicated, however, that the constrained models did not fit significantly worse than the models in which the parameters for menarcheal age varied across groups. We, therefore, ran the subsequent models in the entire sample rather than fitting separate parameters in each racial/ethnic group. The association between menarcheal age and adolescent depressive symptoms was attenuated and not statistically significant when comparing cousins ($b = -0.02$, $p = 0.43$) and further attenuated when comparing sisters ($b = -0.01$, $p = 0.68$). Thus, menarcheal age did not predict depressive symptoms when comparing cousins and sisters, suggesting that confounding factors account for the association between menarcheal age and depressive symptoms found in the population.

Findings from sensitivity analyses (see Table 3 for population estimates and Table 4 for within-family comparisons) were generally consistent with the main findings, such that when (a) using mother-reported menarcheal age, (b) self-reported menarcheal age, (c) early versus other menarcheal age, (d) predicting age 14–15 depressive symptoms only, (d) and predicting among full siblings only, the population estimate was small and significant. The within-family analyses, were, as in the main analysis attenuated and no longer statistically significant.

Delinquency

Menarcheal age also predicted delinquent acts ($b = -0.04, p < 0.01$) in the population (see Table 2). Every 1-year increase from the mean menarcheal age was associated with a 0.04 decrease in mean delinquent acts over the age period. The nonlinear model yielded a non-significant quadratic term ($b = -0.01, 95\% \text{ CI } -0.02, 0.01, p = 0.49$). As depicted in Fig. 1, Panel B, the linear and nonlinear models provide a similar association except in the tails, which include very few people. The model fit statistics for the linear model (AIC = 10,289.261; BIC = 10,313.645) and the nonlinear model (AIC = 10,290.697; BIC = 10,321.178) provided further evidence that the linear model was superior and should be used for subsequent analyses. The estimate for menarcheal age predicting adolescent delinquency was -0.03 for Latina girls, 0.01 for Black girls, and -0.06 for Non-Black, Non-Latina girls. Modified Chi square difference tests [$\chi^2(2) = 5.4, p = 0.13$] indicated, however, the constrained model did not fit significantly worse than the full model, suggesting that race and ethnicity did not moderate the associations. In the subsequent models, which were fit to the entire sample, the association was attenuated and not significant when comparing cousins ($b = 0.01, p = 0.76$) and sisters ($b = 0.01, p = 0.75$). Thus, within extended and immediate families menarcheal age did not predict delinquent acts, which suggest confounding factors account for the population associations.

Sensitivity analyses were broadly consistent with these findings (see Table 3 for population estimates and Table 4 for within-family comparisons). Among the sensitivity analyses, only the population estimates yielded significant estimates for menarcheal age predicting engagement in delinquent acts. In some cases (i.e., for self-report only and categorical menarcheal age), nevertheless, the point estimates for the sister comparison analyses were larger than the population estimates.

Early Age at First Intercourse

Menarcheal age predicted early age at first intercourse in the population (see Table 2 and Fig. 1, Panel C). Every year menarcheal age was delayed was associated with a 31 % decrease in the odds of early intercourse (OR 0.69, 95 % CI 0.64, 0.75). In the nonlinear model, the quadratic term was not significant ($b = -0.03, 95\% \text{ CI } -0.06, 0.01, p = 0.19$), suggesting that the linear model was a better fit for the data. Moreover, fit statistics for the linear (AIC = 3,680.721; BIC = 3,698.763) and nonlinear (AIC = 3,681.175; BIC = 3,705.231) models also provide evidence that the linear model should be used because it provides similar fit with more parsimony.

When comparing across race and ethnicity, odds ratios for menarcheal age predicting early age at first intercourse were 0.78 for Latina girls, 0.74 for Black girls, and 0.62 for Non-Black, Non-Latina girls. Modified Chi square difference tests [$\chi^2(2) = 5.4, p = .07$] indicated that when predicting early age at first intercourse, the constrained model was not significantly different than the model in which the estimate for menarcheal age predicting early age at first intercourse varied across Latina, Black, and Non-Latina, Non-Black girls. Fit indices suggested that the constrained model fit the data equally to the full model ($BIC_{\text{constrained}} = 18,342.9, BIC_{\text{full}} = 18,353.5$) or that the models fit the data equally well ($AIC_{\text{constrained}} = 18,277.8, AIC_{\text{full}} = 18,276.6$). We, therefore, fit the subsequent models to the entire dataset without constraints. The association persisted at the same magnitude when comparing cousins (OR 0.69, 95 % CI 0.60, 0.80) and comparing sisters (OR 0.69, 95 % CI 0.57, 0.82), suggesting the association exists at the within-family level. The results, therefore, suggest the association between menarcheal age and early age at first intercourse was independent of confounding factors shared by siblings and cousins.

Sensitivity analyses were generally consistent with the primary findings (see Tables 3, 4). Analyses that included mother-report of menarcheal age only, self-report of menarcheal age only, or categorical menarcheal age yielded a pattern in which the link between menarcheal age and early age at first intercourse persisted in point estimates, but the estimates had large confidence intervals and were no longer statistically significant in the within-family comparisons. In contrast, the series of analyses that only included full siblings resulted in a pattern in which menarcheal age predicted early age at first intercourse both across the population and within families at the same magnitude, which were also statistically significant.

Discussion

Hypotheses from several theoretical orientations—including hypotheses of maturation-disparity, hormonal influence, and psychosocial acceleration—suggest that menarcheal age is part of an etiological pathway for the development of depressive symptoms, delinquent acts, and/or sexual behavior during adolescence. In contrast, selection or confounding factors that influence menarcheal age and outcomes may actually account for the associations between menarcheal age and emotional and behavioral problems during adolescence seen in the population. We found that menarcheal age predicted offspring depressive symptoms, delinquency, and early age at first intercourse across the population, consistent with previous studies. We also found that race/ethnicity did not moderate any of the associations. Finally, when testing the association within-families, we found that the link between menarcheal age and depressive symptoms and delinquent acts was attenuated and not statistically significant, but the association between menarcheal age and early sexual intercourse remained. Outcome-specific results are described below.

Depressive Symptoms

The association between menarcheal age and adolescent depressive symptoms in the population was quite small. Other studies and reviews have implicated a large role of pubertal timing in the prediction of depressive symptoms (Mendle et al. 2007). Some studies

specifically linking menarcheal age and depression nevertheless show small associations (e.g., Carter et al. 2012; Joinson et al. 2011), suggesting that menarcheal age alone may not be a strong predictor of these outcomes.

We found that race and ethnicity did not moderate the small association between menarcheal age and adolescent depressive symptoms across the population. The current study replicates a finding from another nationally representative longitudinal study (Carter et al. 2012) in which the association was similar across Black and Caucasian girls. This finding suggests that contexts systematically related to race or ethnicity may not have differential influences on the association between menarcheal age and depressive symptoms. Together, these findings suggest that large, nationally representative samples have greater power to detect small associations in racial and ethnic minority groups.

We found that the small association between menarcheal age and depressive symptoms did not persist when comparing female cousins and sisters. These findings are consistent with a selection model and suggest that factors shared among siblings and cousins in an extended family account for the association between early menarcheal age and depressive symptoms. As such, the within-family results (i.e., the comparison of siblings and cousins) are inconsistent with the interpretations associated with the aforementioned theories and previous studies (Negri and Susman 2011) linking menarcheal age to adolescent depressive symptoms. The current study, therefore, provides evidence for menarcheal age as a risk marker, but not an independent risk factor for the development of depressive symptoms.

There are several possible empirical explanations for why the current results are not consistent with previous findings. First, most previous research has not tested the within-family association between menarcheal age and adolescent depressive symptoms. The result may mean that factors shared among families (i.e., that differ between families) account for the link, and most previous research has not been able to rule out this possibility. One caveat to these findings for this and other outcomes is that the imprecision with which cousin and sister comparisons were measured (i.e., based on individuals living in the same household rather than biological relatedness alone) may result in more similar estimates for cousin and sister comparisons. This would especially be the case if genetic confounds account for the association. Second, the current analysis does not preclude the possibility that more precise measurement of menarcheal age would have yielded stronger estimates. The magnitude of the estimates, nevertheless, were still very small, indicating that if menarcheal age is an independent risk factor, its contribution to risk for depressive symptoms is likely quite limited. The results from sensitivity analyses were largely consistent with these findings.

Delinquent Acts

The association between menarcheal age and the development of delinquency was also small. We also did not find evidence for race or ethnicity moderating the small association between menarcheal age and delinquent acts, which replicates results from Rodgers et al. (2015). The current finding also replicates the findings of another recent large-scale study that directly tested race and ethnicity as a moderator in finding no statistically significant difference by race and ethnicity for menarcheal age predicting delinquency (Mrug et al.

2014). The association between menarcheal age and delinquency did not, however, persist within families. This finding is consistent with a selection model, and suggests that genetic and environmental factors shared by siblings and cousins account for the observed association between menarcheal age and delinquent acts in the population. For example, (Harden and Mendle 2012) posited that the androgen receptor gene may be part of an array of genes that are similar within families and may influence menarcheal age and engagement in delinquent behavior. Further, our findings may also be consistent with research using bivariate twin analyses that implicate shared environmental factors as explaining the association between menarcheal age and conduct problems (Burt et al. 2006), a construct associated with delinquency.

The link between menarcheal age and adolescent delinquent acts was generally consistent across sensitivity analyses. In two sensitivity analyses, the within-family point-estimate for menarcheal age predicting delinquent acts was larger than the population point-estimate. Because the within-family model requires greater statistical power, the larger point-estimate suggests that in a larger sample the link between menarcheal age and delinquent acts may persist within families. The current study, nevertheless, does not provide evidence for a within-family link.

Early Age at First Intercourse

We found a moderate association between menarcheal age and early age at first intercourse across the population; for every 1 year increase from the mean menarcheal age, there was a .31 decrease in the odds of having engaged in sexual intercourse by age 17. Consistent with findings for depressive symptoms and delinquent acts, we did not find evidence for race or ethnicity as a moderator. Nevertheless, another large-scale longitudinal study found that Caucasian females showed the strongest association between menarcheal age and early age at first intercourse, then Latina females, and then Black females (Cavanagh 2004). Given the conflicting findings between large scale studies, future research should continue to examine race and ethnicity as putative moderators of this association, particularly when compared to peers rather than national averages.

Menarcheal age continued to predict early age at first intercourse in within-family analyses, both when comparing siblings and comparing cousins. These findings are consistent with an inference that menarcheal age has a direct effect on intercourse before the age of 16.

Although very few studies have utilized genetically informed designs to test the link between menarcheal age and early intercourse, the current findings are counter to those of a recent study conducted in the National Survey of Adolescent Health, which showed that shared genetic factors accounted for the link (Moore et al. 2014). Our estimates from the cousin-comparisons, which account for approximately 25 % of genetic factors, and sibling-comparisons, which account for approximately 50 % of genetic factors, were similar. As such, the findings are not consistent with genetic confounding, although additional studies that explicitly test genetic and environmental confounds are needed.

The current findings may be consistent with both the psychosocial acceleration theory and mechanistic hypotheses linking menarcheal age to early age at first intercourse. First, the maturation disparity hypothesis suggests that early maturers are more likely to engage

socially with older adolescents, particularly in romantic contexts (Graber and Sontag 2006). This may be one pathway to early intercourse for females who reach menarche early. Second, the hormonal influence hypothesis suggests that the increase in androgen hormones may influence engagement in sexual behavior. Future work should continue to investigate these and other putative mechanisms to determine which processes mediate the association.

Limitations and Strengths

The current findings should be considered in light of some limitations. First, the associations we identified within the population may be interpreted as smaller than those identified in some previous work. This small association may be related to the measurement of menarcheal age in this study which included varying informants (mother, daughter, or both) and varying formats (e.g., age vs. month and year) of reporting menarcheal age. The average mother-reported menarcheal age was earlier than the average self-reported menarcheal age. This was likely because mothers were asked whether their daughters had reached menarche until age 14 or 15, and daughters responded thereafter. Although ideally both reporters would have been asked across all time points, we sought to address this by running sensitivity analyses in which only mother or only self-reports were used. The small association may also be difficult to compare to other studies, and may, in fact, be in the confidence interval of findings from previous studies. A meta-analysis examining associations between pubertal timing more broadly and depression in adolescence found that the overall estimates across studies had large confidence intervals, indicating a wide range of estimates in which the true association lies (Galvao et al. 2014).

Second, sibling comparisons make a few key assumptions about how risk factors operate. The experiences of one sibling are assumed to not influence the other siblings (Lahey and D'Onofrio 2010). Further, it is assumed that females with sisters are representative of singletons and females with brothers only, when all of these factors may make home environments distinct for any individual girl. Also, the sister comparison controls for fewer genetic factors than twin comparisons, so analyses that compare twins, siblings, and unrelated individuals would yield stronger internal validity. The current study included a range of genetic relatedness within families (e.g., full and half-sisters), but did not explicitly test differences by genetic relatedness. It is important to note, nevertheless, that we found consistent results when comparing both siblings and cousins, which suggests the results are not due solely to the assumptions/limitations inherent in each design.

Third, the study was unable to specify menarche compared to same-age peers, rather to national norms. Some researchers posit the importance of specifically comparing adolescent girls to the individuals within their social networks, which is more salient and meaningful. Fourth, the study was unable to elucidate processes related to pubertal timing and outcomes in boys because our measure of pubertal timing was limited to the female participants in the sample. Finally, we were limited in our measurement of race and ethnicity which was based on the mother's report, precluding the examination of more specific moderation (e.g., no specific ethnicities for Black participants, no knowledge of whether participants were biracial).

The current study also had several strengths. The sample used was larger and more diverse than samples used in much of the extant literature. Notably, our findings regarding race and ethnicity for the depressive and delinquent outcomes were consistent with recent large-scale studies with diverse samples in the US. Additionally, we utilized several quasi-experimental family designs that allowed us to control for unmeasured genetic and environmental confounds shared within families. Although most previous research has controlled for measured covariates, our analytical design dramatically increased the genetic and environmental factors for which we controlled when testing whether the association between menarcheal age and adolescent outcomes was causal. Finally, we tested multiple adolescent outcomes within the same study, thereby extending the reach of research using quasi-experimental methods in this area of research.

Future Directions

We see at least two directions for future research. First, a majority of previous work examining the link between pubertal timing and adolescent behavioral patterns has focused on menarcheal age as an index of pubertal timing. The current study suggests that menarcheal age is not a causal risk factor for depressive symptoms or delinquency. Recent work suggests that other indicators, such as the development of secondary sex characteristics, perception of pubertal timing or physical maturation compared to peers, and pubertal tempo, are more proximally related to adolescent emotional and behavior experiences than menarcheal age, particularly because they more explicitly engage social comparisons and expectations (Carter et al. 2009; Halpern et al. 2007; Joinson et al. 2012; Mendle 2014). If other indicators of pubertal timing, perceptions of pubertal timing and tempo are considered, there may again be contextual differences by race/ethnicity. For example, pubertal timing according to the Pubertal Development Scale was linked to depressive symptoms among Caucasian females, but not African American females (Hamlat et al. 2014). Future studies, when possible, should test these different measures in large, longitudinal studies and continue to examine both ethnic moderation and use rigorous methods to rule out alternative explanations.

Second, the integrative model of maturation-disparity within a context, proposed by Skoog and Stattin (2014) suggests that problems associated with pubertal timing are short-term and do not endure into adulthood. The proposed mechanisms may differ in whether or not the putatively associated difficulties would be proximal to pubertal change, or whether pubertal timing would predict more longstanding difficulties. From the maturation-disparity framework, for example, challenges (e.g., risky sexual behavior) for early maturing girls may dissipate as they achieve cognitive and emotional maturation. Recent studies that have followed large birth cohorts have indicated that emotional and behavioral problems associated with pubertal timing, including menarcheal age, may diminish as girls age beyond the period of pubertal transition (Copeland et al. 2010; Joinson et al. 2011; Boden et al. 2011). Future work should continue to examine these developmental questions.

Conclusion

Consistent with previous research, we found associations between menarcheal age and three adolescent outcomes—depressive symptoms, delinquency, and age at first intercourse. Adding to the equivocal literature about racial and ethnic differences in these associations, we found that all associations were similar across Latina, Black, and Non-Latina, Non-Black individuals, indicating similar mechanisms explaining the associations across these groups. When further examining these mechanisms, we found that the association between menarcheal age and depressive symptoms and delinquency did not persist within families. This and other recent findings suggest that interventions for the development of depressive symptoms and delinquency during the adolescent period would not benefit from further examination of proposed causal mechanisms linking them to menarcheal age. Instead, intervention and prevention efforts should identify the factors that differ between extended families that are correlated with both menarcheal age and these adolescent problems. In contrast, we found that the association between menarcheal age and early age at first intercourse *did* persist within families, although other research has suggested genetic confounding may account for the association. This finding suggests that further exploration of putative causal mechanisms linking menarcheal age to early age at first intercourse may provide insight into improvements for intervention efforts, but that further evidence of this possibility is necessary.

Acknowledgments

This research was supported by the Indiana University Adam W. Herbert Graduate Fellowship, and Grants F31-HD079266-01A1 and R01-HD061384 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development. The research was approved by the Institutional Review Board at Indiana University. The authors would like to thank Dr. Martin Rickert for preparing the three-panel figure within the manuscript.

References

- Allen JP, Chango J, Szewedo D, Schad M. Long-term sequelae of subclinical depressive symptoms in early adolescence. *Development and Psychopathology*. 2014; 26(01):171–180.10.1017/S095457941300093X [PubMed: 24444175]
- American Academy of Pediatrics, Committee on Adolescence, American College of Obstetricians Gynecologists, Committee on Adolescent Health Care. Menstruation in girls and adolescents: Using the menstrual cycle as a vital sign. *Pediatrics*. 2006; 118(5):2245–2250.10.1542/peds.2006-2481 [PubMed: 17079600]
- Anderson CA, Duffy DL, Martin NG, Visscher PM. Estimation of variance components for age at menarche in twin families. *Behavior Genetics*. 2007; 37:668–677.10.1007/s10519-007-9163-2 [PubMed: 17680356]
- Belsky J, Steinberg L, Draper P. Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development*. 1991; 62(4):647–670. [PubMed: 1935336]
- Belsky J, Steinberg L, Houts RM, Halpern-Felsher BL. The development of reproductive strategy in females: Early maternal harshness → earlier menarche → increased sexual risk taking. *Developmental Psychology*. 2010; 46(1):120–128.10.1037/a0015549 [PubMed: 20053011]
- Benjet C, Hernández-Guzmán L. A short-term longitudinal study of pubertal change, gender, and psychological well-being of Mexican early adolescents. *Journal of Youth and Adolescence*. 2002; 31(6):429–442.10.1023/a:1020259019866

- Boden JM, Fergusson DM, Horwood LJ. Age of menarche and psychosocial outcomes in a New Zealand birth cohort. *Journal of the American Academy of Child and Adolescent Psychiatry*. 2011; 50(2):132–140. [PubMed: 21241950]
- Bongers IL, Koot HM, van der Ende J, Verhulst FC. The normative development of child and adolescent problem behavior. *Journal of Abnormal Psychology*. 2003; 112(2):179–192.10.1037/0021-843x.112.2.179 [PubMed: 12784827]
- Boomsma DI, Van Beijsterveldt CEM, Hudziak JJ. Genetic and environmental influences on anxious/depression during childhood: A study from the Netherlands Twin Register. *Genes, Brain and Behavior*. 2005; 4(8):466–481.10.1111/j.1601-183X.2005.00141.x
- Bordini B, Rosenfield RL. Normal pubertal development: Part I: The endocrine basis of puberty. *Pediatrics in Review*. 2011; 32(6):223–229.10.1542/pir.32-6-223 [PubMed: 21632873]
- Bureau of Labor Statistics, U. S. Children of the NLSY79, 1979–2008. 2008a. [computer file]
- Bureau of Labor Statistics, U. S. National Longitudinal Survey of Youth 1979 cohort, 1979–2008 (rounds 1–23). 2008b. [computer file]
- Burt SA, McGue M, DeMarte JA, Krueger RF, Iacono WG. Timing of menarche and the origins of conduct disorder. *Archives of General Psychiatry*. 2006; 63(8):890–896.10.1001/archpsyc.63.8.890 [PubMed: 16894065]
- Carter R, Caldwell C, Matusko N, Antonucci T, Jackson J. Ethnicity, perceived pubertal timing, externalizing behaviors, and depressive symptoms among Black adolescent girls. *Journal of Youth and Adolescence*. 2011; 40(10):1394–1406.10.1007/s10964-010-9611-9 [PubMed: 21088874]
- Carter R, Jaccard J, Silverman WK, Pina AA. Pubertal timing and its link to behavioral and emotional problems among at-risk African American adolescent girls. *Journal of Adolescence*. 2009; 32(3):467–481.10.1016/j.adolescence.2008.07.005 [PubMed: 18801563]
- Carter R, Silverman WK, Jaccard J. Race and perceived pubertal transition effects on girls' depressive symptoms and delinquent behaviors. *Journal of Youth and Adolescence*. 2012.10.1007/s10964-012-9885-1
- Caspi A, Moffitt TE. Individual differences are accentuated during periods of social change: The sample case of girls at puberty. *Journal of Personality and Social Psychology*. 1991; 61(1):157–168.10.1037/0022-3514.61.1.157 [PubMed: 1890586]
- Cavanagh SE. The sexual debut of girls in early adolescence: The intersection of race, pubertal timing, and friendship group characteristics. *Journal of Research on Adolescence*. 2004; 14(3):285–312.10.1111/j.1532-7795.2004.00076.x
- Chase-Lansdale PL, Mott FL, Brooks-Gunn J, Phillips DA. Children of the National Longitudinal Survey of Youth: A unique research opportunity. *Developmental Psychology*. 1991; 27(6):918–931.10.1037/0012-1649.27.6.918
- Cohen J. A power primer. *Psychological Bulletin*. 1992; 112(1):155–159.10.1037/0033-2909.112.1.155 [PubMed: 19565683]
- Copeland W, Shanahan L, Miller S, Costello EJ, Angold A, Maughan B. Outcomes of early pubertal timing in young women: A prospective population-based study. *American Journal of Psychiatry*. 2010; 167(10):1218–1225. [PubMed: 20478880]
- Del Giudice, M.; Ellis, BJ. *Evolutionary foundations of developmental psychopathology*. 3. Vol. 1. New York: Wiley; 2014.
- DeRose L, Shiyko M, Foster H, Brooks-Gunn J. Associations between menarcheal timing and behavioral developmental trajectories for girls from age 6 to age 15. *Journal of Youth and Adolescence*. 2011; 40(10):1329–1342.10.1007/s10964-010-9625-3 [PubMed: 21203809]
- Donahue KL, D'Onofrio BM, Bates JE, Lansford JE, Dodge KA, Pettit GS. Early exposure to parents' relationship instability: Implications for sexual behavior and depression in adolescence. *Journal of Adolescent Health*. 2010; 47(6):547–554.10.1016/j.jadohealth.2010.04.004 [PubMed: 21094431]
- Donahue K, D'Onofrio B, Lichtenstein P, La ngström N. Testing putative causal associations of risk factors for early intercourse in the Study of Twin Adults: Genes and Environment (STAGE). *Archives of Sexual Behavior*. 2013; 42(1):35–44.10.1007/s10508-012-9947-1 [PubMed: 22441771]

- D'Onofrio BM, Lahey BB, Turkheimer E, Lichtenstein P. Critical need for family-based, quasi-experimental designs in integrating genetic and social science research. *American Journal of Public Health*. 2013; 103(S1):S46–S55.10.2105/ajph.2013.301252 [PubMed: 23927516]
- Dorn LD, Dahl RE, Woodward HR, Biro F. Defining the boundaries of early adolescence: A user's guide to assessing pubertal status and pubertal timing in research with adolescents. *Applied Developmental Science*. 2006; 10:30–56.10.1207/s1532480xads1001_3
- Elliott DS, Huizinga D. Social class and delinquent behavior in a national youth panel: 1976–1980. *Criminology*. 1983; 21(2):149–177.10.1111/j.1745-9125.1983.tb00256.x
- Ellis BJ, Del Giudice M, Dishion TJ, Figueredo AJ, Gray P, Griskevicius V, Wilson DS. The evolutionary basis of risky adolescent behavior: Implications for science, policy, and practice. *Developmental Psychology*. 2012; 48(3):598–623.10.1037/a0026220 [PubMed: 22122473]
- Ellis BJ, McFadyen-Ketchum S, Dodge KA, Pettit GS, Bates JE. Quality of early family relationships and individual differences in the timing of pubertal maturation in girls: A longitudinal test of an evolutionary model. *Journal of Personality and Social Psychology*. 1999; 77(2):387–401.10.1037/0022-3514.77.2.387 [PubMed: 10474213]
- Galvao TF, Silva MT, Zimmermann IR, Souza KM, Martins SS, Pereira MG. Pubertal timing in girls and depression: A systematic review. *Journal of Affective Disorders*. 2014; 155:13–19. [PubMed: 24274962]
- Ge X, Conger RD, Elder GH Jr. Pubertal transition, stressful life events, and the emergence of gender differences in adolescent depressive symptoms. *Developmental Psychology*. 2001; 37(3):404–417.10.1037/0012-1649.37.3.404 [PubMed: 11370915]
- Ge X, Kim IJ, Brody GH, Conger RD, Simons RL, Gibbons FX, Cutrona CE. It's about timing and change: Pubertal transition effects on symptoms of major depression among African American youths. *Developmental Psychology*. 2003; 39(3):430–439. [PubMed: 12760513]
- Ge X, Natsuaki MN. In search of explanations for early pubertal timing effects on developmental psychopathology. *Current Directions in Psychological Science (Wiley-Blackwell)*. 2009; 18(6):327–331.10.1111/j.1467-8721.2009.01661.x
- Graber JA, Sontag LM. Puberty and girls' sexuality: Why hormones are not the complete answer. *New Directions for Child and Adolescent Development*. 2006; 2006(112):23–38.10.1002/cd.160 [PubMed: 16869171]
- Halpern C, Kaestle C, Hallfors D. Perceived physical maturity, age of romantic partner, and adolescent risk behavior. *Prevention Science*. 2007; 8(1):1–10.10.1007/s11121-006-0046-1 [PubMed: 16972191]
- Hamlat EJ, Stange JP, Abramson LY, Alloy LB. Early pubertal timing as a vulnerability to depression symptoms: Differential effects of race and sex. *Journal of Abnormal Child Psychology*. 2014; 42(4):527–538.10.1007/s10802-013-9798-9 [PubMed: 24014162]
- Harden KP. Genetic influences on adolescent sexual behavior: Why genes matter for environmentally oriented researchers. *Psychological Bulletin*. 2014; 140(2):434–465.10.1037/a0033564 [PubMed: 23855958]
- Harden KP, Mendle J. Gene-environment interplay in the association between pubertal timing and delinquency in adolescent girls. *Journal of Abnormal Psychology*. 2012; 121(1):73–87.10.1037/a0024160 [PubMed: 21668078]
- Haynie DL. Contexts of risk? Explaining the link between girls' pubertal development and their delinquency involvement. *Social Forces*. 2003; 82(1):355–397.
- Joinson C, Heron J, Araya R, Paus T, Croudace T, Rubin C, et al. Association between pubertal development and depressive symptoms in girls from a UK cohort. *Psychological Medicine, FirstView*. 2012; 10.1017/S003329171200061X
- Joinson C, Heron J, Lewis G, Croudace T, Araya R. Timing of menarche and depressive symptoms in adolescent girls from a UK cohort. *The British Journal of Psychiatry*. 2011; 198(1):17–23.10.1192/bjp.bp.110.080861 [PubMed: 21200072]
- Kaestle CE, Halpern CT, Miller WC, Ford CA. Young age at first sexual intercourse and sexually transmitted infections in adolescents and young adults. *American Journal of Epidemiology*. 2005; 161(8):774–780.10.1093/aje/kwi095 [PubMed: 15800270]

- Kraemer H, Kazdin AE, Offord DR, Kessler RC, Jensen PS, Kupfer DJ. Coming to terms with the terms of risk. *Archives of General Psychiatry*. 1997; 54(4):337–343.10.1001/archpsyc.1997.01830160065009 [PubMed: 9107150]
- Lahey BB, D'Onofrio BM. All in the family: Comparing siblings to test causal hypotheses regarding environmental influences on behavior. *Current Directions in Psychological Science*. 2010; 19(5): 319–323.10.1177/0963721410383977 [PubMed: 23645975]
- Lahey BB, Van Hulle CA, Waldman ID, Rodgers JL, D'Onofrio BM, Pedlow S, et al. Testing descriptive hypotheses regarding sex differences in the development of conduct problems and delinquency. *Journal of Abnormal Child Psychology*. 2006; 34(5):737–755.10.1007/s10802-006-9064-5 [PubMed: 17033935]
- Marshall WA, Tanner JM. Variation in patterns of pubertal changes in girls. *Archives of Disease in Childhood*. 1969; 44:291–303. [PubMed: 5785179]
- Mendle J. Beyond pubertal timing: New directions for studying individual differences in development. *Current Directions in Psychological Science*. 2014; 23(3):215–219.10.1177/0963721414530144
- Mendle J, Turkheimer E, Emery RE. Detrimental psychological outcomes associated with early pubertal timing in adolescent girls. *Developmental Review*. 2007; 27(2):151–171.10.1016/j.dr.2006.11.001 [PubMed: 20740062]
- Moffitt TE, Caspi A. Childhood predictors differentiate life-course persistent and adolescence-limited antisocial pathways among males and females. *Development and Psychopathology*. 2001; 13(2): 355–375. [PubMed: 11393651]
- Moore SR, Harden KP, Mendle J. Pubertal timing and adolescent sexual behavior in girls. *Developmental Psychology*. 2014.10.1037/a0036027
- Mrug S, Elliott MN, Davies S, Tortolero SR, Cuccaro P, Schuster MA. Early puberty, negative peer influence, and problem behaviors in adolescent girls. *Pediatrics*. 2014; 133(1):7–14.10.1542/peds.2013-0628 [PubMed: 24324002]
- Muthén, LK.; Muthén, BO. *Mplus user's guide: Sixth edition*. Los Angeles, CA: Muthén and Muthén; 1998–2010.
- Neberich W, Penke L, Lehnart J, Asendorpf JB. Family of origin, age at menarche, and reproductive strategies: A test of four evolutionary-developmental models. *European Journal of Developmental Psychology*. 2008; 7(2):153–177.10.1080/17405620801928029
- Negriff S, Fung M, Trickett P. Self-rated pubertal development, depressive symptoms and delinquency: Measurement issues and moderation by gender and maltreatment. *Journal of Youth and Adolescence*. 2008; 37(6):736–746.10.1007/s10964-008-9274-y [PubMed: 25152546]
- Negriff S, Susman EJ. Pubertal timing, depression, and externalizing problems: A framework, review, and examination of gender differences. *Journal of Research on Adolescence*. 2011; 21(3):717–746.10.1111/j.1532-7795.2010.00708.x
- Orth U, Robins RW, Meier LL. Disentangling the effects of low self-esteem and stressful events on depression: findings from three longitudinal studies. *Journal of Personality and Social Psychology*. 2009; 97(2):307.
- Pine DS, Cohen E, Cohen P, Brook J. Adolescent depressive symptoms as predictors of adult depression: Moodiness or mood disorder? *American Journal of Psychiatry*. 1999; 156(1):133–135. [PubMed: 9892310]
- Marshall WA, Tanner JM. Variation in patterns of pubertal changes in girls. *Archives of Disease in Childhood*. 1969; 44:291–303. [PubMed: 5785179]
- Mendle J. Beyond pubertal timing: New directions for studying individual differences in development. *Current Directions in Psychological Science*. 2014; 23(3):215–219.10.1177/0963721414530144
- Mendle J, Turkheimer E, Emery RE. Detrimental psychological outcomes associated with early pubertal timing in adolescent girls. *Developmental Review*. 2007; 27(2):151–171.10.1016/j.dr.2006.11.001 [PubMed: 20740062]
- Moffitt TE, Caspi A. Childhood predictors differentiate life-course persistent and adolescence-limited antisocial pathways among males and females. *Development and Psychopathology*. 2001; 13(2): 355–375. [PubMed: 11393651]
- Moore SR, Harden KP, Mendle J. Pubertal timing and adolescent sexual behavior in girls. *Developmental Psychology*. 2014.10.1037/a0036027

- Mrug S, Elliott MN, Davies S, Tortolero SR, Cuccaro P, Schuster MA. Early puberty, negative peer influence, and problem behaviors in adolescent girls. *Pediatrics*. 2014; 133(1):7–14.10.1542/peds.2013-0628 [PubMed: 24324002]
- Muthén, LK.; Muthén, BO. *Mplus user's guide*. 6. Los Angeles, CA: Muthén and Muthén; 1998–2010.
- Neberich W, Penke L, Lehnart J, Asendorpf JB. Family of origin, age at menarche, and reproductive strategies: A test of four evolutionary-developmental models. *European Journal of Developmental Psychology*. 2008; 7(2):153–177.10.1080/17405620801928029
- Negriff S, Fung M, Trickett P. Self-rated pubertal development, depressive symptoms and delinquency: Measurement issues and moderation by gender and maltreatment. *Journal of Youth and Adolescence*. 2008; 37(6):736–746.10.1007/s10964-008-9274-y [PubMed: 25152546]
- Negriff S, Susman EJ. Pubertal timing, depression, and externalizing problems: A framework, review, and examination of gender differences. *Journal of Research on Adolescence*. 2011; 21(3):717–746.10.1111/j.1532-7795.2010.00708.x
- Orth U, Robins RW, Meier LL. Disentangling the effects of low self-esteem and stressful events on depression: findings from three longitudinal studies. *Journal of Personality and Social Psychology*. 2009; 97(2):307.
- Pine DS, Cohen E, Cohen P, Brook J. Adolescent depressive symptoms as predictors of adult depression: Moodiness or mood disorder? *American Journal of Psychiatry*. 1999; 156(1):133–135. [PubMed: 9892310]
- Radloff LS. The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*. 1977; 1(3):385–401.10.1177/014662167700100306
- Reef J, Diamantopoulou S, Meurs I, Verhulst F, Ende J. Developmental trajectories of child to adolescent externalizing behavior and adult DSM-IV disorder: Results of a 24-year longitudinal study. *Social Psychiatry and Psychiatric Epidemiology*. 2011; 46(12):1233–1241.10.1007/s00127-010-0297-9 [PubMed: 20936464]
- Reef J, Diamantopoulou S, Van Meurs I, Verhulst F, Van Der Ende J. Child to adult continuities of psychopathology: A 24-year follow-up. *Acta Psychiatrica Scandinavica*. 2009; 120(3):230–238.10.1111/j.1600-0447.2009.01422.x [PubMed: 19522882]
- Reinke WM, Eddy JM, Dishion TJ, Reid JB. Joint trajectories of symptoms of disruptive behavior problems and depressive symptoms during early adolescence and adjustment problems during emerging adulthood. *Journal of Abnormal Child Psychology*. 2012; 40(7):1123–1136.10.1007/s10802-012-9630-y [PubMed: 22492211]
- Resnick MD, Bearman PS, Blum RW, Bauman KE, Harris KM, Jones J, Udry JR. Protecting adolescents from harm: Findings from the National Longitudinal Study on Adolescent Health. *JAMA: The Journal of the American Medical Association*. 1997; 277(10):1001/jama.1997.03550100049038
- Rodgers J, Bard D, Johnson A, D'Onofrio B, Miller W. The cross-generational mother–daughter–aunt–niece design: Establishing validity of the MDAN design with NLSY fertility variables. *Behavior Genetics*. 2008; 38(6):567–578.10.1007/s10519-008-9225-0 [PubMed: 18825497]
- Rodgers JL, Cleveland HH, van den Oord E, Rowe DC. Resolving the debate over birth order, family size, and intelligence. *American Psychologist*. 2000; 55:599–612. [PubMed: 10892201]
- Rodgers JL, Van Hulle C, D'Onofrio B, Rathouz P, Beasley W, Johnson A, et al. Behavior problems and timing of menarche: A developmental longitudinal biometrical analysis using the NLSY-children data. *Behavior Genetics*. 2015; 45:51–70. [PubMed: 25246040]
- Rowe DC. On genetic variation in menarche and age at first sexual intercourse: A critique of the Belsky–Draper hypothesis. *Evolution and Human Behavior*. 2002; 23(5):365–372.10.1016/S1090-5138(02)00102-2
- Rutter M. Proceeding from observed correlation to causal inference: The use of natural experiments. *Perspectives on Psychological Science*. 2007; 2(4):377–395.10.1111/j.1745-6916.2007.00050.x
- Sandfort TGM, Orr M, Hirsch JS, Santelli J. Long-term health correlates of timing of sexual debut: results from a national US study. *American Journal of Public Health*. 2008; 98(1):155–161.10.2105/ajph.2006.097444 [PubMed: 18048793]

- Schulz KM, Molenda-Figueira HA, Sisk CL. Back to the future: The organizational–activational hypothesis adapted to puberty and adolescence. *Hormones and Behavior*. 2009; 55(5):597–604.10.1016/j.yhbeh.2009.03.010 [PubMed: 19446076]
- Silberg J, Pickles A, Rutter M, Hewitt J, Simonoff E, Maes H, Eaves L. The influence of genetic factors and life stress on depression among adolescent girls. *Archives of General Psychiatry*. 1999; 56(3):225–232.10.1001/archpsyc.56.3.225 [PubMed: 10078499]
- Skoog T, Stattin H. Why and under what contextual conditions do early-maturing girls develop problem behaviors? *Child Development Perspectives*. 2014; 8(3):158–162.10.1111/cdep.12076
- Steinberg L. A social neuroscience perspective on adolescent risk-taking. *Developmental Review*. 2008; 28(1):78–106.10.1016/j.dr.2007.08.002 [PubMed: 18509515]
- Tuvblad C, Grann M, Lichtenstein P. Heritability for adolescent antisocial behavior differs with socioeconomic status: gene–environment interaction. *Journal of Child Psychology and Psychiatry*. 2006; 47(7):734–743.10.1111/j.1469-7610.2005.01552.x [PubMed: 16790008]
- van den Berg S, Setiawan A, Bartels M, Polderman T, van der Vaart AW, Boomsma DI. Individual differences in puberty onset in girls: Bayesian estimation of heritabilities and genetic correlations. *Behavior Genetics*. 2006; 36(2):261–270.10.1007/s10519-005-9022-y [PubMed: 16408250]
- Vanoss Marín B, Coyle KK, Gómez CA, Carvajal SC, Kirby DB. Older boyfriends and girlfriends increase risk of sexual initiation in young adolescents. *Journal of Adolescent Health*. 2000; 27(6):409–418.10.1016/S1054-139X(00)00097-5 [PubMed: 11090743]
- Whittle S, Yücel M, Lorenzetti V, Byrne ML, Simmons JG, Wood SJ, et al. Pituitary volume mediates the relationship between pubertal timing and depressive symptoms during adolescence. *Psychoneuroendocrinology*. 2012.10.1016/j.psyneuen.2011.10.004
- Wickrama T, Wickrama KAS. Heterogeneity in adolescent depressive symptom trajectories: Implications for young adults' risky lifestyle. *Journal of Adolescent Health*. 2010; 47(4):407–413.10.1016/j.jadohealth.2010.02.013 [PubMed: 20864011]
- Williams DR. Race, socioeconomic status, and health: The added effects of racism and discrimination. *Annals of the New York Academy of Sciences*. 1999; 896(1):173–188.10.1111/j.1749-6632.1999.tb08114.x [PubMed: 10681897]
- Woolfenden SR, Williams K, Peat JK. Family and parenting interventions for conduct disorder and delinquency: A meta-analysis of randomised controlled trials. *Archives of Disease in Childhood*. 2002; 86(4):251–256.10.1136/adc.86.4.251 [PubMed: 11919097]
- Wu T, Mendola P, Buck GM. Ethnic differences in the presence of secondary sex characteristics and menarche among US girls: The Third National Health and Nutrition Examination Survey, 1988–1994. *Pediatrics*. 2002; 110:752.10.1542/peds.110.4.752 [PubMed: 12359790]
- Zimmer-Gembeck MJ, Helfand M. Ten years of longitudinal research on US adolescent sexual behavior: Developmental correlates of sexual intercourse, and the importance of age, gender and ethnic background. *Developmental Review*. 2008; 28(2):153–224.

Biographies

Erikka B. Vaughan is a doctoral student in the Department of Psychological and Brain Sciences at Indiana University. Her research uses quasi-experimental designs and longitudinal analyses to explore associations among individual differences, family factors, and the development of emotional and behavioral problems during childhood and adolescence.

Carol A. Van Hulle is an Assistant Research Scientist at the University of Wisconsin-Madison. She received her Ph.D. in Psychology from the University of Colorado. Her research has been wide-ranging within the field of child development, integrating quantitative genetic and behavioral approaches to temperament, language development, conduct problems, prenatal effects, and sensory modulation disorders.

William H. Beasley is an Assistant Professor of Research in the Department of Pediatrics at the University of Oklahoma Health Sciences Center, and President of Howard Live Oak, a small statistical consulting company. He graduated from the Quantitative Psychology program of the University of Oklahoma in 2010. In addition to Behavior Genetic methods, he is interested in statistical simulation and CQI (continuous quality improvement) techniques.

Joseph L. Rodgers is the Lois Autrey Betts Professor of Psychology and Human Development at Vanderbilt University. He earned his Ph.D. in Quantitative Psychology, with a minor in Biostatistics, from the University of North Carolina at Chapel Hill in 1981. His primary research focus involves building mathematical models of human behavior, with substantive interest in adolescent transition behaviors including smoking, drinking, delinquency, and sexual behavior. He also has substantive interest in human reproduction and fertility, including applications of epidemiological models, behavior genetic models, and nonlinear dynamic models.

Brian M. D’Onofrio is a Professor in the Department of Psychological and Brain Sciences at Indiana University. He received his Ph.D. in Clinical Psychology from the University of Virginia in 2005. His research, rooted in the field of developmental psychopathology, explores the causes and treatments of child and adolescent psychopathology through three main approaches: quasi-experimental designs, longitudinal analyses, and intervention studies.

Fig. 1.

Linear and nonlinear model-derived estimates of menarcheal age predicting adolescent outcomes. *Note:* Depicted models did not include covariates. Confidence intervals for the categorical estimates are represented in the figure by *lines* extending from each categorical point on the plots

Table 1

Correlations and descriptive statistics for main variables

	1	2	3	4	N	M	SD	Freq.	Range
1. Menarcheal age	–				3,335	12.29	1.25		7.58–17.66
2. Depressive symptoms	–0.06	–			3,148	4.92	3.65		0–21
3. Delinquent acts	–0.05	0.22	–		3,446	1.35	1.54		0–14
4. Early age at first intercourse	–0.23	0.20	0.39	–	2,741	–	–	935	1 = early

All correlations shown are significant at $p < 0.05$. Correlations for age at first intercourse are polychoric

Table 2

Unstandardized estimates of AAM predicting adolescent outcomes

Outcome	Population				Cousin comparison				Sister comparison			
	Linear		Nonlinear		Linear		95 % CI		Linear		95 % CI	
	b	95 % CI	b	95 % CI	b	95 % CI	b	95 % CI	b	95 % CI	b	95 % CI
Depressive symptoms												
Population estimate	-0.05**	[-0.08, -0.02]	-0.05**	[-0.08, -0.02]								
Population quadratic			0.02**	[0.001, 0.030]								
Within-family					-0.02**	[-0.07, 0.03]			-0.01	[-0.07, 0.05]		
Delinquent acts												
Population estimate	-0.04**	[-0.07, -0.01]	-0.04**	[-0.07, -0.01]								
Population quadratic			-0.01	[-0.02, 0.01]								
Within-family					0.01	[-0.04, 0.06]			0.01	[-0.05, 0.07]		
Early AFI												
Population estimate	0.69**	[0.64, 0.75]	0.68**	[0.63, 0.74]								
Population quadratic			0.98	[0.94, 1.01]								
Within-family					0.69**	[0.60, 0.80]			0.69**	[0.57, 0.82]		

*
 $p < .05$;**
 $p < .01$

Table 3

Sensitivity analyses: menarcheal age predicting adolescent outcomes

Outcome	Population		
	<i>N</i>	<i>b</i>	95 % CI
Depressive symptoms	3,071	−0.05**	[−0.08, −0.02]
Mother reported MA	2,314	−0.05*	[−0.09, −0.01]
Self reported MA	1,147	−0.05*	[−0.09, −0.00]
Outcome at 14–15	2,024	−0.05**	[−0.09, −0.02]
Outcome at 16–17	1,972	−0.02	[−0.05, 0.02]
Dichotomous MA	3,148	−0.07	[−0.02, 0.16]
Categorical MA	3,069		
Early compared to “on time”		0.21**	[0.10, 0.32]
Late compared to “on time”		0.02	[−0.09, 0.12]
Families with full siblings	1,706	−0.04**	[−0.08, −0.00]
Delinquent acts	3,282	−0.04**	[−0.07, −0.01]
Mother reported MA	2,515	−0.02	[−0.06, 0.02]
Self reported MA	2,222	0.07**	[−0.10, −0.02]
Outcome at 14–15	2,778	−0.07**	[−0.11, −0.03]
Outcome at 16–17	2,496	−0.01	[−0.05, 0.02]
Dichotomous MA	3,446	0.04	[−0.05, 0.13]
Categorical MA	3,279		
Early compared to “on time”		−0.03	[−0.14, 0.07]
Late compared to “on time”		−0.14**	[−0.26, −0.02]
Families with full siblings	1,815	−0.07**	[−0.11, −0.02]
		OR	95 % CI
Early intercourse	3,023	0.69**	[0.63, 0.75]
Mother report	2,255	0.80**	[0.72, 0.89]
Self report	2,741	0.65**	[0.58, 0.72]
Dichotomous MA	2,741	2.10**	[1.64, 2.70]
Categorical MA	3,021		
Early compared to “on time”		0.73**	[0.46, 1.01]
Late compared to “on time”		−0.67**	[−0.98, −0.35]
Families with full siblings	1,373	0.60**	[0.53, 0.69]

MA menarcheal age

*
 $p < 0.05$;**
 $p < 0.01$

Table 4

Sensitivity analyses: menarcheal age predicting adolescent outcomes

Outcome	Sisters			Cousins		
	N	b	95 % CI	N	b	95 % CI
Depressive symptoms	3,148	-0.01	[-0.07, 0.05]	3,148	-0.02	[-0.07, 0.03]
Mother reported MA	2,314	-0.03	[-0.11, 0.06]	2,314	-0.02	[-0.09, 0.05]
Self reported MA	1,147	0.03	[-0.07, 0.13]	1,147	0.01	[-0.09, 0.11]
Outcome at 14-15	2,024	-0.04	[-0.12, 0.05]	2,024	-0.04	[-0.10, 0.03]
Outcome at 16-17	3,119	0.04	[-0.05, 0.12]	3,119	0.04	[-0.05, 0.12]
Dichotomous MA	3,148	-0.03	[-0.20, 0.13]	3,148	0.02	[-0.13, 0.17]
Families with full siblings	1,706	-0.07	[-0.16, 0.02]	1,706	-0.06	[-0.13, 0.02]
Delinquent acts	3,446	0.01	[-0.05, 0.07]	3,446	0.01	[-0.04, 0.06]
Mother reported MA	2,515	-0.02	[-0.10, 0.05]	2,515	-0.02	[-0.09, 0.05]
Self reported MA	1,224	0.10	[-0.02, 0.23]	1,224	0.07	[-0.03, 0.16]
Outcome at 14-15	2,778	0.00	[-0.08, 0.08]	2,778	-0.02	[-0.20, 0.04]
Outcome at 16-17	2,496	0.02	[-0.06, 0.09]	2,496	0.02	[-0.05, 0.08]
Dichotomous MA	3,446	-0.09	[-0.25, 0.07]	3,446	-0.05	[-0.19, 0.10]
Families with full siblings	1,815	-0.02	[-0.10, 0.05]	1,815	-0.01	[-0.08, 0.06]

	OR	95 % CI	OR	95 % CI
Early intercourse	2,741	0.69**	2,741	0.69**
Mother reported MA	2,255	0.83	2,045	0.87
Self reported MA	1,223	0.86	1,131	0.77
Dichotomous MA	2,741	1.47	2,741	1.51
Families with full siblings	1,373	0.63**	1,706	0.56**

MA menarcheal age

*
 $p < 0.05$;**
 $p < 0.01$