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Girls' Sleep Trajectories Across the Pubertal Transition: Emerging Racial/Ethnic Differences

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A B S T R A C T

Purpose: This study aims to examine the longitudinal association between puberty and sleep in a diverse sample of girls and explore racial/ethnic differences in this association.
Methods: Using latent growth curve modeling, the present study measured pubertal development (timing and rate) and sleep (wake time and bedtime) in 1,239 socioeconomically and ethnically diverse girls starting when they were 6–8 years old and followed longitudinally for up to 8 years. Pubertal assessment was conducted annually in clinic by physical examination, classified by sexual maturation stage for breast and pubic hair development by trained raters.
Results: In line with previous research, black girls had the earliest pubertal development, followed by Hispanic, white, and Asian girls. Black girls, on average, reported significantly shorter sleep duration than Hispanic ($\beta = -.20$, $p < .001$), Asian ($\beta = -.29$, $p = .002$), and white ($\beta = -.35$, $p < .001$) girls. In a series of dual-process models, we found that early pubertal timing predicted shorter sleep duration for early-maturing black girls (breast development: $\beta = .13$, $p = .005$; pubic hair development: $\beta = .14$, $p = .012$). There was no evidence of any association between pubertal rate and sleep. All models controlled for family socioeconomic status and body mass index.

IMPLICATIONS AND CONTRIBUTION

Earlier pubertal timing in girls was associated with later bedtimes across the transition from middle childhood to adolescence. Therefore, clinicians should consider using pubertal onset as a "teachable moment" to promote sleep health. Early intervention may be especially significant for black youth, who experience puberty-related changes in sleep before their peers.

Conflict of Interest: The findings and conclusions of this report are solely the responsibility of the authors and do not necessarily represent the official views of the NIH. The NIH played no role in the study design, data collection, writing, or decision to submit the manuscript for publication. All authors declare they have no actual or potential conflicts of interest. The first author wrote the first draft of this paper and received no compensation.

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Conclusion: Sleep is essential for many aspects of youth development, including emotional, cognitive, and physical functioning. Developmental changes associated with puberty may put some early maturing girls at risk of shorter sleep duration in adolescence and exacerbate racial/ethnic disparities in health and well-being.

Early pubertal timing in girls is associated with increased risk of social, emotional, behavioral, and physical problems during adolescence and beyond [1,2]. Interestingly, many of the same risk-taking behaviors and poor mental and physical health outcomes are also associated with insufficient sleep [3–5], yet the links between sleep and pubertal onset are not well understood. Furthermore, given well-established racial/ethnic differences in both pubertal timing and sleep, some youth may be at greater risk than others. In particular, black or African-American girls typically enter puberty earlier [6,7] and have shorter sleep duration [8] than girls from other racial/ethnic groups, which may exacerbate health disparities in the United States.

These early racial/ethnic sleep inequalities foreshadow well-documented differences in sleep among black and white adults in the United States [9,10]. There is also evidence for reduced sleep duration among Hispanic adults, compared with white adults, over the past several decades [11]. Psychosocial stressors are the main hypothesized pathway to racial/ethnic disparities in sleep quantity and quality. For instance, poor sleep has been linked to stressors such as negative life events, neighborhood disorder, and financial strain, which are significantly more common among black and Hispanic families compared with white families [12,13]. Additionally, a new body of research suggests that everyday discrimination and perceived racism also negatively impact sleep, independent of socioeconomic status and other psychosocial stressors [14,15].

Another plausible, yet understudied, risk factor of sleep disparities is early pubertal timing. Research from laboratory studies suggests that pubertal youth experience delayed sleep phase (i.e., the natural tendency for later bedtime, as measured by later melatonin secretion), longer sleep latency (i.e., time taken to fall asleep), less delta slow-wave sleep (i.e., deep sleep), and a greater tolerance to stay up late (regardless of daytime sleepiness) compared with prepubertal youth [16–21]. Therefore, girls who experience puberty before their peers (including a higher proportion of black and Hispanic youth in the United States) likely undergo neurological reorganization of their sleep-wake cycle, leading to later bedtimes and shorter sleep duration than their same-aged peers. Early pubertal timing may also influence sleep via increased psychosocial stress. Youth who develop relatively early (i.e., gain weight or develop secondary sex characteristics when their peers still have childlike appearances) may be treated differently by adults and other youth, or encounter more adult-like expectations, leading to heightened risk of psychosocial problems [22,23]. Indeed, studies suggest that stressful social experiences contribute to longer sleep latency and shorter sleep duration [24,25]. Links have been established between short sleep duration and key developmental outcomes, including poor cognitive performance [26], depressive symptoms [5], obesity [27], inflammation [28], and risky behaviors [3] in adolescence, which underscores the need to better understand the early biological precursors to sleep behavior.

Importantly, puberty is not a discrete transition, but rather a developmental process that begins in late childhood through a cascade of neuroendocrine changes that manifest in physical growth and sexual maturation that can be measured by phenotypic indicators (e.g., secondary sex characteristics). The typical progression from pubertal initiation to full, physical maturation spans approximately 4 years, but there is vast individual variation, with a range of 1 year to 7 or more years [29]. Although less studied, pubertal rate might also cause increased stress due to the maturation compression hypothesis: faster rate requires more rapid adaption to new biological and social milestones relative to slower rate [30]. Girls who transition through puberty quickly (i.e., faster rate) may also be at risk of worse sleep, both because they experience relevant neurological changes sooner, and because they may be under higher stress related to the rapid transition. Overall, there is a lack of relevant, longitudinal data to explore possible racial/ethnic disparities in pubertal rate.

To our knowledge, no studies have examined whether pubertal timing and rate may explain different sleep trajectories across adolescence. We address this gap, and also consider whether associations between puberty and sleep trajectories may differ across racial/ethnic groups. Although pubertal development and behavioral research to date has largely focused on white women, this study included clinic-based sexual maturation ratings in a large, diverse sample of girls, which provides a rare opportunity to explore whether puberty may be related to racial/ethnic differences in sleep. We expected racial/ethnic differences in both sleep and pubertal timing—with black girls developing earlier, and getting less sleep, than Hispanic, white, and Asian girls. We also hypothesized that earlier timing (Figure 1; hypothesis 1a) and faster rate (hypothesis 2a) would be related to shorter sleep duration in middle childhood, and increasingly less sleep across the transition from middle childhood to adolescence (hypotheses 1b and 2b). Furthermore, we expected that associations between puberty and sleep would be stronger in black girls than other racial/ethnic groups. We also hypothesized that this effect may be apparent in Hispanic girls, given evidence of earlier and faster puberty in Hispanic girls relative to white and Asian girls.

Methods

Participants and procedure

This project was conducted as part of the Puberty Studies of the Breast Cancer and the Environment Research Program funded by the National Institute of Environmental Health Sciences and the National Cancer Institute [31]. A total of 1,239 socioeconomically and ethnically diverse girls, aged 6–8 years, were enrolled between 2004 and 2007 from three locations led by the following institutions: (1) Mount Sinai School of Medicine (MSSM); (2) Cincinnati Children's Hospital Medical Center; and (3) Kaiser Permanente Northern California (KPNC). Girls having a

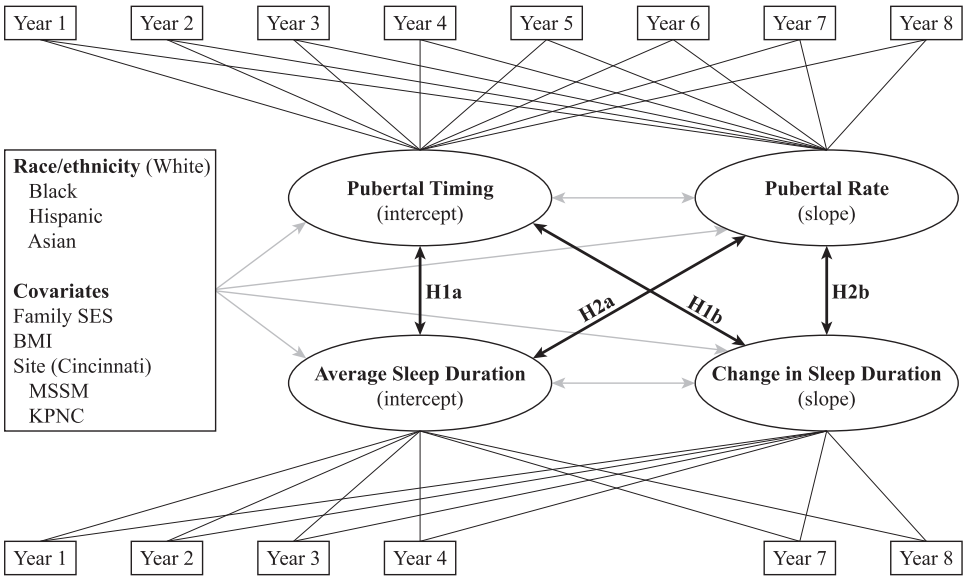

Figure 1. Simplified dual-process model of pubertal development and sleep. Specific study hypotheses represented here as H1a, H1b, H2a, H2b. Puberty was measured by breast development or pubic hair development, in separate models. Covariates included baseline measures of body mass index (BMI), socioeconomic status (SES), and site: Cincinnati Children's Hospital Medical Center, Mount Sinai School of Medicine (MSSM), and Kaiser Permanente Northern California (KPNC).

pre-existing medical condition known to influence puberty or a psychiatric condition that could potentially limit study participation were excluded.

Participants attended annual clinical visits at KPNC and MSSM and biannual visits at Cincinnati, during which anthropometric and pubertal data were collected by clinic staff, trained by a pediatric endocrinologist [6]. Each visit included an interview with the primary caregiver and, starting in 2010 (i.e., year 6), a child interview. The institutional review boards at each institution approved study procedures.

The present study used data from the first 8 years of the study. Puberty was assessed annually (described in the following section). Sleep data were reported for 6 years of data collection at KPNC and Cincinnati (years 1–4, year 7, and year 8) and 5 years at MSSM (years 1–4 and year 8). Retention was good; about 70% of the sample had at least five (of eight possible) puberty assessments and at least four (of six possible) sleep assessments. The sample (n = 1,239) was almost evenly distributed between black (31.6%), Hispanic (30.0%), and white (33.7%) race/ethnicity, with the remaining 4.6% Asian (and one participant as "other race," .1%). Participants were approximately 7.45 years old at baseline (standard deviation, SD = .68) and 14.50 years old in year 8 (SD = .98). See Table 1 for descriptive information about the sample.

Measures

Puberty. Puberty was assessed using physical examination based on Marshall and Tanner's criteria for breast maturation (with the inclusion of palpation) and pubic hair stages [32,33]. Examiners circled the appropriate stage on a standard form that illustrated and described each stage, where 1 indicates that development has not begun, and then postpubertal scores range from 2 (initial development) to 5 (development complete), with 87% agreement observed between examiners and master trainers (κ statistic = .67). More details on these gold standard sexual maturation rating (SMR; also known as Tanner staging methods) and reliability in the Breast Cancer and the Environment Research Program study are available elsewhere [6].

Sleep. In the first 4 years of data collection, when participants were typically between 7 and 10 years old, the primary caregiver reported their child's bedtime the previous night and wake time that morning in exact hours and minutes; we subtracted wake time from bedtime to get a measure of total sleep duration. Sleep questions were not included in years 5 or 6. Years 7 and 8 incorporated a more detailed set of questions—in the child interview only—asking youth to report their wake/bedtimes for weekdays and weekends separately (over the past week). In years 7–8, we created a weighted weekend/weekday mean score for sleep, reflecting 5/7 weekdays and 2/7 weekend days.

Given that most visits were conducted after school, the majority (75.1%) of sleep reports in years 1–4 reflected weekday sleep, and all youth reported weeknight sleep in years 7–8, we created a second sleep score for weeknights only. The weeknight sleep variables were used for a sensitivity analysis.

Demographic and health characteristics. Caregivers reported girls' race/ethnicity (black, non-black Hispanic, Asian, or non-Hispanic white); these categories were utilized for race/ethnic group analyses. Additionally, childhood obesity and socioeconomic status (SES) have been implicated as key determinants of early pubertal onset and short sleep duration; therefore, the current study controls for family SES and objective measures (i.e., clinical assessment) of body mass index (BMI) before pubertal onset and adolescent sleep changes. Family SES was assessed by total years of education of the child's financial provider and categorized as none (0), grade school (8), some

Table 1
Descriptive statistics for key study variables by race/ethnicity

	Full sample (n = 1,239)	Black (n = 391)	Hispanic (n = 372)	Asian (n = 57)	White (n = 418)
Age at baseline (y)	7.44 (.68)	7.46 (.71)	7.37 (.80)	7.41 (.46)	7.51 (.55)
Family SES (caregiver years of education)	13.93 (2.75)	13.89 (2.06)	11.89 (2.76)	15.38 (1.96)	15.74 (1.90)
BMI at baseline	17.40 (3.22)	17.69 (3.43)	18.14 (3.73)	15.81 (1.60)	16.68 (2.37)
Breast development[a,b]					
Timing (age at midpoint of puberty)	11.61 (1.20)	11.16 (1.01)[d,e,f]	11.62 (1.06)[c,f]	11.98 (.80)[c]	11.94 (1.02)[c,d,*]
Rate (SMR stages/y)	.81 (.32)	.83 (.30)	.79 (.29)	.87 (.26)	.83 (.29)
Pubic hair development[a,b]					
Timing (age at midpoint of puberty)	11.60 (1.23)	10.90 (1.15)[d,e,f]	11.75(1.33)[c,e,f]	12.86 (1.15)[c,d,f]	11.98 (1.02)[c,d,e,*]
Rate (SMR stages/y)	.86 (.32)	.84 (.32)[f]	.91 (.39)	1.01 (.44)[f]	.96 (.33)[c,e,*]
Wake time[a,b]					
Intercept (wake time at year 4)	7:42 A.M. (.74)	7:40 (1.41)[d]	7:51 (1.58)[c,f]	7:49 (3.63)[f]	7:34 (.00)[d,e,*]
Slope (min/y)	3.78 (.24)	4.14 (.49)	3.66 (1.60)	4.02 (.83)	3.84 (.35)
Bedtime[a,b]					
Intercept (bed time at year 4)	22:07 P.M. (.81)	22:16 (1.41)[f]	22:16 (1.41)[f]	22:07 (3.31)[f]	21:50 (.99)[c,d,e,*]
Slope (min/y)	11.76 (.21)	10.86 (.46)[f]	10.44 (.46)[f]	11.88 (.77)	13.92 (19.01) [c,d,*]
Sleep duration (h)					
Intercept (h, centered at year 4)	9.59 (.70)	9.40 (1.41)[d,e,f]	9.60 (1.41)[c,f]	9.71 (3.38)[c]	9.75 (.99)[c,d,*]
Slope (min/y)	−8.04 (.25)	−6.78 (.53)[f]	−6.72 (.53)[f]	−7.74 (.92)	−10.14 (.32)[c,d,*]

Means presented with standard deviations in parentheses.
BMI = body mass index; SES = socioeconomic status; SMR = sexual maturation rating.
[a] These parameters were calculated at year 4 (mean age = 10.45 years, SD = .68) because all analyses were centered at year 4.
[b] Pairwise contrasts (i.e., dummy variables regressed on the latent intercept and slope or timing and rate factors) tested for racial/ethnic differences in sleep and puberty.
[c] Significantly different from the black race/ethnic group.
[d] Significantly different from the Hispanic race/ethnic group.
[e] Significantly different from the Asian race/ethnic group.
[f] Significantly different from the white race/ethnic group.
[*] $p < .01$.

high school (10), high school graduate (12), some college (13), associate's degree (14), bachelor's degree (16), and master's degree or higher (18). Height and weight measurements, taken in clinic using calibrated scales and fixed stadiometers, were used to calculate BMI as weight (kg)/(height [m])^2. BMI values were standardized for age and gender, and percentiles and z-scores were calculated using methods and standard distributions as provided by the Center for Disease Control and Prevention (www.cdc.gov/growthcharts/zscore.htm); standardized BMI was included in all models as a continuous variable. Analyses also included dummy variables to control for study site: KPNC and MSSM (with Cincinnati as the referent group). Age was calculated each year, based on the date of the clinical examination minus the participant's birth date.

Analytic strategy

Puberty and sleep trajectories. Using Mplus (V 7.4; Muthén & Muthén, Los Angeles, CA), we began by modeling puberty (breast and pubic hair development) and sleep duration, separately, using nonlinear (puberty) and linear (sleep) mixed-effects models including individuals' exact ages at each year. The *timing* and *rate* of puberty were assessed using the repeated measures of the SMR at each age with a logistic function and utilizing a first-order Taylor series expansion[1] [34], an approach that has been used in studies assessing individual differences in pubertal timing and rate to account for the nonlinear trajectory of puberty (i.e., sigmoid curve) [29,35–37]. The latent factors of timing and rate were evaluated at the midpoint of development, SMR 3. Thus, *timing* is expressed in terms of the age at the midpoint of puberty (higher values correspond to older ages, or later timing), and *rate* is an index of change in terms of the number of SMR stages per year at the midpoint of puberty (higher values correspond to faster rates of change). Age, assessed continuously, was centered at year 4 to map most closely onto the timing and rate of puberty. Incomplete data were treated using standard missing at random assumptions [38] and modeled using full information maximum likelihood.

The first set of models was unconditional to obtain initial estimates for breast development, pubic hair development, and sleep parameters for the full sample (i.e., three separate models). In the second set of models, intercept and slopes were allowed to vary across racial/ethnic groups (run separately) to obtain estimates of pubertal *timing* and *rate* and sleep *intercept* and *slope* for each racial/ethnic group. To examine racial/ethnic differences, a final set of models that included contrasts (i.e., dummy variables regressed on the latent *timing* and *rate* or *intercept* and *slope* factors) tested for racial/ethnic differences in puberty and sleep.

Dual-process models. To test the next set of hypotheses (modeling puberty and sleep together), we fit a series of dual-process models in Mplus (Figure 1). Separate models were tested for breast/pubic hair stages to index pubertal maturation. The latent factors of *pubertal timing*, *pubertal rate*, *sleep intercept*, and *sleep slope* were also regressed onto race/ethnicity (i.e., black, Hispanic, and Asian vs. white) and the full set covariates (i.e., centered dummy variables indicating site [MSSM and KPNC vs. Cincinnati], centered age-standardized BMI scores, and caregiver's years of education) to control for potential confounds related to site, body size, or SES. To test associations of pubertal timing and rate with sleep intercept and slope, and pubertal timing with

[1] Equations and Mplus code are available in a previous publication [34].

pubertal rate, covariances among the *pubertal timing*, *pubertal rate*, *sleep intercept*, and *sleep slope* latent factors were estimated. As a robustness check, we ran each dual-process model for weeknight sleep duration only (Appendix S1). Finally, we ran two additional sets of models for wake/bedtimes, which are presented in Appendix S2.

Exploration of race/ethnicity in the dual-process model. Because of known racial/ethnic differences in both pubertal maturation [6,7,29] and sleep duration [8–10], we also explored whether there were differences in the association between puberty and sleep for each racial/ethnic group. This analysis was intended to be a first step in exploring whether patterns of pubertal development (i.e., early/late timing, fast/slow rate) might help explain racial/ethnic differences in sleep trajectories across adolescence. We conducted multigroup analyses in which means, variances, and factor loadings for pubertal timing and rate, and sleep intercept and slope, were freely estimated (i.e., allowed to vary) across race/ethnicity, and also freely estimated the covariances among the *pubertal timing*, *pubertal rate*, *sleep intercept*, and *sleep slope* latent factors for each racial/ethnic group. Because there were differences in these parameters across racial/ethnic groups, and thus factorial invariance assumptions were not met, we did not explicitly test for differences in associations of the latent factors. Instead, we examined the multigroup analyses to explore whether associations differed by group. Given the small size of the Asian subgroup, Asian girls (n = 57), and the single participant of "other" race/ethnicity, only black (n = 391), Hispanic (n = 372), and white (n = 418) girls were included in the multigroup analyses (n = 1,181). Finally, site dummy variables were excluded from this analysis because of the collinearity between race/ethnicity and site (e.g., there were no white participants in the MSSM site). Additionally, we conducted racial/ethnic group analyses for wake/bedtimes (Appendix S2).

Results

Puberty

The average midpoint (i.e., SMR 3) for breast development for the entire sample occurred at age 11.61 years (SD = 1.20) and breast development progressed at an average rate of .81 stage per year (SD = .32), with similar estimates for pubic hair timing (M = 11.60, SD = 1.23) and rate (M = .86, SD = .32). Timing and rate were negatively correlated for breast development ($r = -.11$, $p = .010$), indicating that girls who developed earlier tended to move through sexual maturity stages more quickly, but there was no significant correlation between timing and rate for pubic hair development in the full sample.

Racial/ethnic differences in puberty. There were significant racial/ethnic differences in pubertal timing (means and SDs are presented in Table 1). Black girls had significantly earlier breast and pubic hair development than any other group. There was variability in pubertal rate: white girls had faster pubic hair development rate than black and Asian girls. Furthermore, although the overall sample had similar estimates for breast development and pubic hair timing, black girls had more advanced pubic hair development at a younger chronological age (breast: M = 11.16, SD = .83; pubic hair: M = 10.90, SD = .84).

Sleep

Based on linear growth models, participants got 9 hours and 36 minutes of sleep (SD = 42.24 minutes) on average at year 4[2] (when most youth were 10 or 11 years old). Participants woke up at 7:42 A.M. (SD = 44.35 minutes) on average at year 4, and wake times were approximately 3.78 minutes later each year across the study period (SD = 14.60 minutes). Average wake time (intercept) and rate of wake time change (slope) were positively correlated ($r = .12$, $p < .001$): girls with later average wake times tended to have greater increases in (i.e., progressively later) wake times across the transition from childhood to adolescence compared with girls with earlier wake times. Bedtimes were 10:07 P.M. (SD = 48.58 minutes) on average at year 4, becoming significantly later at a rate of 11.76 minutes/year (SD = 12.67 minutes).

Racial/ethnic differences in sleep. Black girls reported significantly shorter sleep at year 4 than all other race/ethnic groups, and Hispanic girls reported significantly less sleep than white girls (Table 1). There were also racial/ethnic differences in bedtime slope. Contrasts revealed that both black girls ($\beta = .06$, $p < .001$) and Hispanic girls ($\beta = .06$, $p < .001$) had progressively shorter sleep duration across the transition from childhood to adolescence compared with white girls.

White girls reported significantly earlier wake times at year 4, followed by black girls, Asian girls, and Hispanic girls (Table 1). The rate of change in wake times did not differ by race/ethnicity. White girls, on average, also reported earlier bedtimes compared with all other groups. There were also racial/ethnic differences in bedtime slope. Contrasts revealed that both black girls ($\beta = -.05$, $p = .004$) and Hispanic girls ($\beta = -.06$, $p < .001$) had progressively later bedtimes across the transition from childhood to adolescence compared with white girls.

Dual-process models of pubertal development and sleep

Parameter estimates for dual-process models of sleep duration (intercept and slope) and puberty (timing and rate; breast and pubic hair development) are presented in Table 2; separate analyses for wake/bedtimes are presented in Appendix S2.

Sleep duration. Across both models, early pubertal timing (i.e., younger chronological age at the midpoint of puberty) was marginally associated with shorter sleep duration (breast development: $\beta = .05$, $p = .051$; pubic hair development: $\beta = .06$, $p = .023$; hypothesis 1a). There was also a marginal association between pubertal timing and sleep slope (hypothesis 1b), but only for pubic hair ($\beta = .02$, $p = .028$). There was no evidence of any association between pubertal rate (breast or pubic hair development) and sleep duration; thus, hypotheses 2a and 2b were not supported. There was also no evidence of any association between pubertal rate (breast or pubic hair development) and bedtimes; thus, hypotheses 2a and 2b were not supported. The racial/ethnic differences in sleep hours reported previously remained significant in the full model: black girls ($\beta = -.30$, $p < .001$)

[2] These parameters are calculated at year 4 (mean age = 10.45 years, SD = .68) because all analyses were centered at year 4. All estimates accounted for individuals' exact ages.

Table 2
Parameter estimates for dual-process model of puberty and sleep (n = 1,239)

	Breast development	Pubic hair development
Sleep[a]		
Sleep intercept	9.59* (.05)	9.58* (.02)
Sleep slope	–.13* (.01)	–.14* (.01)
Puberty		
Pubertal timing	11.58* (.03)	11.59* (.03)
Pubertal rate	.80* (.01)	.86* (.01)
Associations		
Pubertal timing with sleep intercept	.04** (.02)	.06** (.03)
Pubertal timing with sleep slope	.01 (.01)	.02** (.01)
Pubertal timing with pubertal rate	–.05* (.01)	–.02 (.02)
Pubertal rate with sleep intercept	.00 (.01)	.00 (.01)
Pubertal rate with sleep slope	.00 (.00)	–.00 (.00)
Sleep intercept with sleep slope[a]	.01* (.01)	.01* (.01)
On sleep intercept[a]		
Black	–.30* (.06)	–.30* (.06)
Hispanic	–.09 (.07)	–.09 (.07)
Asian	–.14 (.09)	–.14 (.09)
BMI	–.04** (.02)	–.04** (.02)
Family SES	.00 (.01)	.01 (.01)
MSSM (site)	.01 (.07)	.01 (.07)
KPNC (site)	.14* (.05)	.14* (.05)
On sleep slope[a]		
Black	.05** (.02)	.05** (.02)
Hispanic	.03 (.02)	.03 (.02)
Asian	.02 (.03)	.02 (.03)
BMI	.02* (.01)	.02* (.01)
Family SES	–.00 (.00)	–.00 (.00)
MSSM (site)	.03 (.02)	.03 (.02)
KPNC (site)	.03 (.02)	.03 (.02)
On pubertal timing		
Black	–.90* (.09)	–1.18 (.10)
Hispanic	–.50* (.11)	–.40 (.13)
Asian	–.20 (.16)	.62 (.18)
BMI	–.33* (.03)	–.33 (.03)
Family SES	–.05* (.02)	–.08 (.02)
MSSM (site)	.29* (.11)	–.02 (.12)
KPNC (site)	.21** (.08)	.01 (.10)
On pubertal rate		
Black	.02 (.03)	–.10* (.04)
Hispanic	.00 (.03)	–.02 (.04)
Asian	–.07 (.05)	–.18* (.06)
BMI	–.01 (.01)	.03* (.01)
Family SES	.01 (.01)	.00 (.01)
MSSM (site)	–.01 (.03)	–.05 (.04)
KPNC (site)	.16* (.03)	–.01 (.03)
Model fit		
Free parameters	49	49
–2 Res log likelihood	–18,377.33	–18,294.08
Akaike Information Criterion (AIC)	41,595.41	41,428.91

Analyses were centered at year 4, but all estimates accounted for individuals' exact ages at each year. Race/ethnic variables, black, Hispanic, and Asian are dummy codes comparing against white participants. Covariates include body mass index (BMI) and family socioeconomic status (SES), measured as caregiver years of education. Site variables, MSSM (New York) and KPNC (California), are dummy codes comparing against Cincinnati participants. Unstandardized parameter estimates and standard errors are provided. Sleep intercept is presented in hours (e.g., military time with fractions of the hour rather than minutes), and slope is the rate of change in sleep in hours/year. Timing is presented in years of age at sexual maturation rating (SMR) 3, and rate is the change in SMR stages/year at SMR 3.

[a] The two sleep intercept and slope estimates for pubic hair, and the two for breast development timing, were estimated from the same data and are therefore nearly identical (within rounding error) across models.

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

had significantly shorter sleep duration than white girls. Finally, as a robustness check, all models were run for weeknight sleep only, showing the same pattern of results and similar effect sizes (Table A1 in Appendix S1).

Wake times and bedtimes. Earlier pubertal timing was marginally associated with a smaller wake time slope, or progressively earlier wake times across development (breast development: $\beta = .02$, $p = .033$; pubic hair development: $\beta = .02$, $p = .022$). Across both models, earlier pubertal timing was associated with later bedtimes (breast development: $\beta = –.09$, $p < .001$; pubic hair development: $\beta = –.08$, $p = .004$), confirming hypothesis 1a for bedtime. There were no other significant associations between pubertal timing/rate and wake/bedtimes (Table B1 in Appendix S2).

Exploration of racial/ethnic differences

Sleep duration. Earlier breast development timing and pubic hair timing were associated with later bedtime for black girls only (Table 3; hypothesis 1a: breast development: $\beta = .13$, $p = .005$; pubic hair development: $\beta = .14$, $p = .012$). None of the pubertal development indicators were associated with sleep intercept or slope in any other racial/ethnic group.

Wake times and bedtimes. For Hispanic girls, earlier pubertal timing was moderately associated with a smaller wake time slope, meaning progressively earlier waking (breast development: $\beta = .04$, $p = .039$; pubic hair development: $\beta = .04$, $p = .047$). None of the pubertal development indicators were associated with wake time intercept or slope in any other racial/ethnic group (Table B2 in Appendix S2). Similar to the findings for sleep hours reported previously, pubertal timing was only significantly associated with bedtimes for black girls (Table B3). Earlier breast development timing (and pubic hair timing at $p < .05$) was associated with later bedtime for black girls (hypothesis 1a: breast development: $\beta = –.14$, $p = .003$; pubic hair development: $\beta = –.13$, $p = .025$).

Discussion

This is the first known study to examine the longitudinal associations between pubertal timing, pubertal rate, and sleep time trajectories across the transition from middle childhood to adolescence. Given growing evidence of the importance of sleep in mental and physical health, the primary goal of this study was to gain a better understanding of the normal maturational changes regarding sleep timing and duration across pubertal development. We found that earlier pubertal timing in girls (measured in clinic using SMR for breast and pubic hair development) was associated with shorter sleep duration (primarily driven by later bedtimes) across the transition from middle childhood to adolescence; there were no associations with pubertal rate.

Furthermore, we examined these developmental processes among racial/ethnic minority youth in the United States, who remain vastly underrepresented in the puberty literature. This diverse sample of girls from three large cities across the United States allowed us to explore racial/ethnic differences in pubertal timing/rate and sleep, as well as to provide the first exploratory evidence for whether puberty may be related to racial/ethnic differences in sleep. Despite some racial/ethnic differences in pubic hair development rate, we found no evidence that pubertal rate was associated with sleep trajectories. We found that the association between early pubertal timing and shorter sleep duration from the full sample was only statistically significant for black girls. Therefore, the psychosocial processes described previously may be more apparent for black girls, who could face more

Table 3
Multigroup analysis of racial/ethnic differences in associations of pubertal development and sleep duration

	Model 1: Breast development			Model 2: Pubic hair development		
	Black (n = 391)	Hispanic (n = 372)	White (n = 418)	Black (n = 391)	Hispanic (n = 372)	White (n = 418)
Sleep duration[a]						
Intercept	9.40* (.04)	9.60* (.04)	9.75* (.03)	9.40* (.04)	9.60* (.04)	9.75* (.03)
Slope	–.11* (.02)	–.11* (.02)	–.17* (.01)	–.11* (.02)	–.11* (.02)	–.17* (.01)
Pubertal development						
Timing	11.13* (.06)	11.65* (.06)	11.92* (.05)	10.90* (.06)	11.77* (.07)	11.97* (.05)
Rate	.82* (.02)	.80* (.02)	.83* (.01)	.84* (.02)	.91* (.02)	.96* (.02)
Associations						
Timing with intercept	.13* (.05)	–.04 (.03)	.04 (.03)	.14* (.06)	.02 (.05)	.04 (.03)
Timing with slope	.02 (.02)	.01 (.01)	.00 (.01)	.03 (.02)	.03 (.02)	.01 (.01)
Timing with rate	–.06* (.02)	–.00 (.01)	.00 (.01)	–.07** (.03)	.03 (.04)	.03 (.03)
Rate with intercept	–.00 (.01)	.03* (.01)	–.03 (.02)	–.01 (.02)	.01 (.02)	–.01 (.01)
Rate with slope	.00 (.01)	.00 (.05)	.01 (.01)	–.00 (.01)	–.01 (.01)	–.00 (.00)
Intercept with slope[a]	.00 (.01)	.03 (.02)	.00 (.00)	.00 (.01)	.03* (.01)	.01 (.01)
On sleep duration intercept[a]						
BMI	–.03 (.04)	–.07** (.03)	–.04 (.03)	–.03 (.04)	–.07** (.03)	–.04 (.03)
Family SES	.02 (.02)	.01 (.01)	–.00 (.02)	.02 (.02)	.01 (.01)	–.00 (.02)
On sleep duration slope[a]						
BMI	.01 (.01)	.01 (.01)	.02* (.01)	.01 (.01)	.02 (.01)	.02* (.01)
Family SES	.00 (.01)	–.00 (.01)	.00 (.01)	.00 (.01)	–.00 (.01)	.00 (.01)
On pubertal development timing						
BMI	–.36* (.05)	–.29* (.06)	–.34* (.05)	–.36* (.06)	–.27* (.06)	–.35* (.06)
Family SES	–.11* (.03)	–.04 (.02)	–.03 (.03)	–.10* (.03)	–.08 (.03)	–.04 (.03)
On pubertal development rate						
BMI	–.00 (.02)	.01 (.02)	–.01 (.02)	.05** (.02)	.03 (.02)	.02 (.03)
Family SES	.02** (.01)	.02* (.01)	–.02* (.01)	.03* (.01)	.01 (.01)	–.03 (.01)
Model fit						
–2 Res log likelihood		–16,992.59			–16,815.56	
AIC		34,159.17			33,805.12	

Model fit: Free parameters = 87 in each model. Covariates include body mass index (BMI) and family socioeconomic status (SES), measured as caregiver years of education. Analyses were centered at year 4, but all estimates accounted for individuals' exact ages at each year.

[a] The sleep intercept and slope are estimated from the same data for models including breast and pubic hair development timing, and thus are nearly identical (within rounding error) across models.

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

stress (e.g., early exposure to a sexualized society) or discrimination (e.g., lower academic expectations from teachers) if they are perceived to be more mature (or older) than their less developed same-aged peers. Research with adult samples suggests that discrimination is correlated with sleep quantity and quality [14,15].

Study strengths include the prospective design, the large ethnically and socioeconomically diverse sample, and the objective measures of pubertal maturation. Importantly, all models included baseline measures of SES and BMI. Our study also had several limitations. Measures of wake/bedtime were subjectively reported, first by the caregiver (years 1–4) and then by the adolescent (years 7–8), with sleep duration reflecting time in bed (vs. hours asleep). Additionally, sleep data were unavailable for 2 of the 8 years, and we could not run separate analyses for weekend sleep, given limited data on weekend sleep in years 1–4. Further, the participants were recruited in three distinct areas of the United States, and geographic area is inextricably intertwined with demographic characteristics. Of note, Asian youth were underrepresented in the sample and thus could not be included in the race/ethnic subgroup analyses. Finally, during the 8-year follow-up period, it is likely that a large proportion of girls changed schools, leading to changes in class schedules, school start times, and lengths of commute, directly affecting wake time, and there is likely variation in school start times among the school districts attended by our study participants. Future research should examine these important contextual factors and environmental contributors to sleep (e.g., housing, noise pollution) during the transition to adolescence.

The tendency toward later bedtimes as girls mature, combined with earlier school start times in middle and high school, often means that adolescents obtain much less sleep than children [21,39]. Later bedtimes, in particular, driven by puberty-related biological (e.g., phase delay) and psychosocial (e.g., stress) influences, could be a key behavioral factor linking early pubertal timing with poor health, and health disparities, in some youth. Pediatricians and other clinicians should consider using pubertal onset as a "teachable moment" for health behavior change to help promote positive sleep attitudes and practices among girls before the emergence of major sleep deficits. Providers can educate youth about the importance for sleep, particularly in adolescence, to perform well in school, sports, or other activities. Awareness and early intervention may be especially significant for black youth, who experience puberty-related changes in sleep before their same-aged peers. Health professionals can work with families, schools, and communities to make them aware of common barriers to sleep. Although pubertal processes are not easily malleable, recent research [18,40] suggests that behavioral changes (e.g., improved bedtime routines) or contextual changes (e.g., later school start times) may promote positive health and reduce racial/ethnic health disparities, across the life course.

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Supplementary Data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.jadohealth.2017.10.014.

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