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The onset of pubertal development and actigraphy-assessed sleep during middle childhood: Racial, gender, and genetic effects

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ABSTRACT

Objective: This study (1) examined pubertal development in relation to actigraphy-assessed sleep in twin children, and tested whether associations differed by child race and gender, (2) modeled genetic and environmental influences on pubertal development and sleep indicators, and (3) examined genetic and environmental influences on the covariation of puberty and sleep.
Design: The classic twin design was used to examine genetic and environmental contributions to puberty and sleep and their associations.
Setting: Data were collected from community-dwelling urban and rural families of twins in the southwestern U.S.
Participants: The racially and socioeconomically diverse sample included 596 twin children (M_{age} = 8.41, SD = 0.69; 51.7% female; 66.3% white; 33.7% Hispanic; 170 monozygotic, 236 same-sex dizygotic, 188 opposite-sex dizygotic).
Measurements: Pubertal development was assessed via parent report. Children wore actigraph watches for 7 nights (M = 6.81, SD = 0.67) to capture sleep duration, efficiency, midpoint, onset latency, and duration variability.
Results: In contrast to extant literature with older youth, more advanced pubertal development was associated with longer sleep durations in Hispanic and white girls and higher sleep efficiency in white girls, though Hispanic girls demonstrated later sleep midpoints. Pubertal development was moderately heritable and there was a genetic influence on the covariance between puberty and sleep indicators.
Conclusions: This was the first study to examine the genetic and environmental influences on the covariation between puberty and sleep, and found genetic underpinnings between pubertal development and actigraphy-assessed sleep duration and efficiency, though sleep and puberty were almost entirely independent in twins at this age.

Sleep difficulties are prevalent in childhood and pose risk for poorer physical and emotional health.[1] Pubertal development is a biological process that may impact sleep quality and quantity, as more advanced pubertal development is associated with sleep-wake behaviors (i.e., later bedtimes, shorter sleep duration).[2] Research has focused on youth post-pubertal onset, with less known regarding relations between puberty and sleep at the onset of puberty. Pubertal age of onset is decreasing,[3] suggesting the need to examine these relations at younger ages. Studies have started to consider the effect of race on puberty and sleep, though samples with youth of Hispanic/Latinx race are limited.[4,5] Examining associations between pubertal development and sleep in middle childhood, including phenotypic relations, race and gender differences, and genetic and environmental influences, will allow further understanding of the underlying relations between these important health processes.

Pubertal onset can be detected as early as middle childhood, but there are gender and racial differences in the average course of pubertal development. A review of epidemiological studies concluded that girls progress through development earlier than boys, and Black youth develop earlier than white youth,[4] with initial signs of puberty evident on average at age 7 for Black girls and age 9 for white girls.[6] Hispanic girls have higher body fat distribution and earlier menarche compared to White girls.[7,8] As Black and Hispanic youth tend to enter puberty before white peers,[9] an examination of pubertal onset in Hispanic youth is informative.

Sleep problems, including insufficient sleep, irregular sleep, and insomnia symptoms, increase during adolescence.[10] Less is known about sleep before adolescence, and many studies have relied on self-reported rather than actigraph-measured sleep. Increased development of secondary sex characteristics in girls, but not boys, predicted changes in sleep duration, bedtimes and wake times, and eveningness preference from middle childhood to adolescence.[2] Age and puberty status are also associated with later sleep midpoints, increased variability in sleep-wake patterns, and longer sleep onset

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latencies, due to the biological shift in the sleep-wake cycle and added environmental demands with age.[11-13] Racial differences in sleep outcomes are evident, but studies with significant Hispanic/Latinx representation are limited.[5] Latinx early adolescents reported the highest levels of sleep disturbance relative to Black and Asian youth,[5] and Hispanic and Black adolescents reported shorter sleep durations than Asian and white youth.[14] Gender differences in sleep have also been found, with boys reporting shorter sleep durations and later bedtimes compared to girls during middle childhood; hypothesized explanations include greater biological need for sleep or increased attentiveness to sleep needs by girls and their caregivers.[15]

Heritability of sleep and pubertal development

A recent meta-analysis concluded that the shared environment was an important influence on children's sleep duration.[16] Studies using actigraph sleep assessments in early adolescence found strong genetic influences for the initiation and maintenance of sleep (e.g., sleep onset latency, duration, efficiency), but inconsistent findings for genetic and environmental influences on timing (e.g., sleep midpoint, sleep start and end times).[17] With the current sample, actigraph sleep quantity and quality were highly heritable, while sleep onset latency, midpoint, and midpoint variability were largely influenced by the shared environment.[18] With higher heritability for sleep duration in adolescence compared to childhood,[16] further research in children is needed to determine how genetic and environmental influences on puberty relate to actigraph sleep indicators.

Research indicates high heritability of youth-reported puberty via the Pubertal Development Scale (PDS) for boys at ages 12 and 14 and girls at age 14, with shared environment more prominent at age 12 for girls.[19] However, pubertal timing as measured by physical development indicators on the Tanner questionnaire was moderately to highly heritable for girls at age 12, with high genetic correlations between menarche, breast development, and pubic hair.[20]

Though sleep and puberty are heritable in childhood and adolescence, and genome-wide association analyses demonstrate genetic correlations between puberty (i.e., age at menarche, growth in height) and sleep indicators (e.g., duration),[21] no quantitative genetic research to our knowledge has examined covariation between sleep and puberty. The biopsychosocial and contextual model of sleep in adolescence supports a biological basis for relations between puberty and sleep, though environmental factors are also important during this developmental stage.[22] Children undergo neurodevelopmental changes during the pubertal process, and alterations in sleep coincide with hormonal changes signaling the onset of puberty, marked by an increase in luteinizing hormone and reactivation of the hypothalamic gonadotropin-releasing hormone network.[23] Understanding why these processes are related is important for understanding adolescent health and development.

The present study

We (1) examined phenotypic associations between pubertal development and actigraph-assessed sleep indicators (efficiency, duration, midpoint, onset latency, and duration variability), and tested whether puberty-sleep associations differed by race and gender; (2) modeled additive genetic, shared environmental, and non-shared environmental influences on pubertal development; and (3) estimated genetic and environmental covariances between pubertal development and related sleep indicators.

To address the first aim, we hypothesized that more Hispanic youth would have started puberty compared to White youth,[7,8] that greater pubertal development would predict poorer sleep in all youth (shorter duration, decreased efficiency, later midpoint, longer onset latency, higher duration variability),[2,15] and that boys would have poorer sleep compared to girls in their respective racial group.[15]

For aims 2 and 3, we examined White and Hispanic children together and did not test differences by race due to power.[24] Regarding the second aim, we hypothesized high genetic influences on pubertal development.[19,20] For the third aim, we hypothesized that duration and efficiency would have largely genetic associations with pubertal development, while midpoint, onset latency, and duration variability would be associated with pubertal development for shared environmental reasons.[18]

Methods

Participants

The Arizona Twin Project is an ongoing longitudinal study of twins across the state of Arizona.[25] Families were initially recruited through birth records and assessed at twin age 12 months. Additional families have been recruited throughout the study to support a sample size necessary for twin modeling. The racially and socioeconomically diverse full sample at the 8 year wave of data collection included 708 twin children (M_{age} = 8.43, SD = 0.68; 51.5% female), who were 55.6% non-Hispanic white, 22.5% Hispanic, 3.0% Asian, 3.7% Black, 0.8% Native Hawaiian or Pacific Islander, 9.5% multiracial, and 4.9% unknown race.

As analyses for this study examined children of Hispanic and white racial backgrounds, children who were neither white nor Hispanic (n = 77) or unknown race (n = 35) were not included in analyses. Children who were both Hispanic and another race (eg, Native American or Black) were included in the Hispanic group. The subsample consisted of 596 twin children (M_{age} = 8.41, SD = 0.69; 51.7% female; 66.3% white; 33.7% Hispanic), including 85 monozygotic (MZ) twin pairs, 118 same-sex dizygotic (DZ) pairs, 94 opposite-sex DZ pairs, and one pair missing zygosity data. Yearly household income ranged from $5,000 to $500,000, with 6% of families living in poverty, 21.7% near the poverty line, 24% lower middle class, and 54.3% middle to upper class.

Procedure

Institutional Review Board approval, caregiver consent, and child assent were received prior to participation. At twin age 8 years, families completed questionnaires and two home visits. Trained research assistants collected physical health assessments, administered questionnaires related to puberty and sleep, and explained actigraphy and daily diary methods. Families completed a week of daily assessments, including twins wearing a wrist-based accelerometer to capture sleep. In addition to indicators of light and temperature, parent-report of twin wake times and bedtimes was used to help determine when children were in bed and attempting to go to sleep, and when they first fell asleep and woke up the next morning, to cross-reference actigraph-assessed measures. Study staff contacted families every evening to ensure procedures were being followed. Families were compensated for their participation.

Measures

Actigraphy. Children wore wrist-based accelerometers (Motion Logger Micro Watch; Ambulatory Monitoring, Inc, Ardsley, NY) on their nondominant wrist for 7 consecutive days and nights (M = 6.81, SD = 0.67). Motion was measured in 1-minute epochs using a zero-crossing mode (i.e., threshold crossing detection where the threshold value is set to a low level of activity and the activity count value is the number of times the activity signal crosses the zero reference point within an epoch)[26] and data was scored using the Sadeh

algorithm in Action W-2 software version 2.7.1 program.[27] Actigraphy is a valid measure in middle childhood.[28] Sleep indicators included duration (total time asleep in hours excluding waking periods), efficiency (ratio of time spent asleep to total time in bed, with total time in bed including true sleep and waking periods), midpoint (midpoint between sleep start and end), sleep onset latency (number of minutes from first attempting to fall asleep to sleep onset), and duration variability (within-person standard deviation estimate of sleep duration, averaged across all nights of the study week). Participant compliance and missing data have been previously reported for the full sample.[18]

Pubertal Development. Primary caregivers completed the PDS for each twin and rated puberty indicators from 1 (not yet started) to 5 (development is complete).[29] We used the 5-item composite score, which captures growth in height, growth of body hair, and skin changes for boys and girls, breast growth and menstruation for girls, and voice deepening and growth of facial hair for boys. Adrenal and gonadal puberty composite scores were also formed, in accordance with a well-established coding system (see supplemental material for description of these composites).[30] Male and female composite scores were standardized separately, and all children were analyzed together. However, all analyses were also run with unstandardized scores and results were similar. Parent-reported PDS has good internal consistency and high correlations with a picture-based puberty interview, physical exams of pubertal stages, and basal hormones.[30]

Zygosity. Primary caregivers completed the Zygosity Questionnaire for Young Twins,[31] a 32-item questionnaire about the birth and observed physical differences between the twins, which is over 95% consistent with zygosity determined by genotyping.[32] The questionnaire was supplemented with physical similarity assessments and hospital birth records.

Covariates. For phenotypic analyses, child age, body mass index (BMI)[8], family socioeconomic status (SES), and vacation were included as covariates due to significant correlations with primary study variables. Family SES is a standardized composite of family-income-to-needs ratio using 2016 US poverty thresholds, primary caregiver education, and spouse/partner education.[33] The vacation variable, a binary indicator of whether twins completed their study week during a typical school week (73.4% of children) or during a holiday or summer break (26.6%), helped control for variability in sleep when children are not in school. Twin models included vacation, race, and gender as covariates, but not SES or BMI, to capture broad genetic and environmental influences.

Data Analysis

Analyses for aim 1. Multivariate phenotypic analyses were conducted in MPlus 8.0 using full information maximum likelihood estimation to handle missing data.[34,35] Mixed-model regressions adjusting for twins nested within families were conducted to examine pubertal development, gender, and their interaction, controlling for all covariates and using multigroup analyses to estimate parameters in white (n = 395) and Hispanic (n = 201) groups separately.

Out of the total N = 596 twins, 554 had data on pubertal development and none were missing data on covariates other than BMI (n = 502). Of participants eligible for actigraphy (i.e., residing in state, N = 530), 87.7% completed procedures. Missing data due to loss or malfunction of watch or not participating in the watch portion of the study was low (8.4%). Compliance was high, with 86.7% of twins wearing the watch for all 7 nights, 9.3% for 6 nights, and 4% for 3-5 nights.

Analyses for aims 2 and 3. Quantitative genetic ACE (genetic, A; common environment, C; nonshared environment, E) models were used to estimate genetic and environmental influences on variances and covariances. Because MZ twins share 100% of their segregating DNA and DZ twins share on average 50%, differences between MZ twins are due solely to nonshared environmental factors, while differences between DZ twins may be genetic or environmental. The shared environment includes all environmental influences contributing to similarities between MZ and DZ cotwins, and the nonshared environment encompasses all non-genetic influences contributing to differences between twins, including measurement error. Using this logic, the ACE model estimates additive genetic (A), shared environmental (C), and nonshared environmental (E) contributions to the variance of a phenotype. The correlation of the latent A factors for each twin in a pair is set to 1.0 for MZ twins and 0.5 for DZ twins. The correlation between latent C factors is set to 1.0 for all pairs because C encompasses environmental factors fully shared by cotwins, and E is uncorrelated for all cotwins. Expanding to bivariate models, a Cholesky decomposition is used to estimate genetic and environmental influences common to two phenotypes by examining cross-twin cross-trait covariances.[36] For example, if the relation between one twin's pubertal development and the other twin's sleep is stronger in MZ than DZ pairs, that suggests genetic influences on the covariance. This model estimates the total additive genetic (A_{11}), shared environmental (C_{11}), and nonshared environmental (E_{11}) influences on the first phenotype, the additive genetic, shared environmental, and nonshared environmental influences on the second phenotype that are shared with the first (A_{21}, C_{21}, and E_{21}), and residual influences unique to the second phenotype (A_{22}, C_{22}, and E_{22}).

ACE models were fit using OpenMX, an R-based statistical program that uses maximum likelihood estimation.[37] Due to power, all children were analyzed together.[24] Starting with full bivariate Cholesky decompositions with pubertal development as the first phenotype and sleep as the second, we tested the significance of genetic and environmental variance and covariance by systematically dropping each path in turn and then, for paths which could be dropped without significant loss of fit, dropping them simultaneously. Nonshared environmental influences on pubertal development (E_{11}) and nonshared environmental influences unique to sleep (E_{22}) were always retained because they include measurement error. The full and reduced models were compared to find the most parsimonious solution. Standard fit indices for ACE twin models include the -2 log-likelihood chi-square test of fit (-2LL), with nonsignificant differences indicating that the reduced model did not fit significantly worse than the full model,[36] and the Akaike's Information Criterion (AIC), with lower values indicating better model fit.[38]

Results

Table 1 includes descriptive statistics, zero-order correlations, and twin intra-class correlations for pubertal development and sleep indicators. Most children were just starting to show signs of puberty, represented by lower PDS scores. Pubertal development correlated positively with sleep efficiency and duration, and negatively with onset latency. Twin intraclass correlations indicated greater MZ than DZ twin similarity on pubertal development, sleep efficiency, and sleep duration, supporting genetic influences.

Sex-limitation ACE models using unstandardized PDS scores indicated that the same genetic and environmental factors influenced pubertal development in both genders, but genetic influences were stronger for girls and shared environmental influences were stronger for boys (see Table S3 in the Supplemental Material).

Puberty-sleep multigroup phenotypic analyses

About 135 Hispanic (77.1%) and 261 white (68.9%) participants had initiated puberty (i.e., started development on at least one indicator). Among Hispanic youth, 83.9% of girls and 69.5% of boys had initiated puberty. Among white youth, 75.3% of girls had initiated puberty as compared to 62.1% of boys. The pubertal composite score

Table 1
Descriptive statistics, zero-order correlations, and twin intra-class correlations

1. Pubertal development	1	2	3	4	5	6	7	8	9	MZ	DZ
	–									0.83	0.57
2. Sleep efficiency	0.15**	–								0.74	0.46
3. Sleep duration	0.15**	0.65***	–							0.80	0.47
4. Sleep midpoint	0.01	-0.05	-0.20***	–						0.94	0.93
5. Sleep onset latency	-0.10*	-0.15*	-0.25***	0.15**	–					0.75	0.67
6. Sleep duration variability	-0.03	-0.21***	-0.31***	0.25***	0.27***	–				0.55	0.38
7. Age	0.10*	-0.03	-0.25***	0.25***	0.06	0.12**	–				
8. Body mass index	0.23***	-0.16***	-0.21***	0.02	-0.02	-0.02	0.16**	–			
9. Family SES	-0.12**	0.12**	0.24***	-0.18***	-0.12*	-0.15**	-0.12**	-0.12**	–		
n	554	465	465	465	465	465	596	502	596		
M	1.31	90.05	8.14	2.20	1.21	0.83	8.41	16.74	-0.04		
SD	0.26	5.56	0.70	0.73	0.29	0.34	0.69	2.89	0.77		
Skewness	0.76	-0.88	-0.43	0.60	0.12	0.87	-0.14	2.09	0.40		
Kurtosis	0.71	0.49	0.26	0.48	-0.23	0.97	-0.46	6.73	-0.02		

Note. N = 596. Sleep efficiency = percentage of time asleep out of total time in bed; Sleep duration = time asleep (in hours); Sleep midpoint = median of sleep start and sleep end time (0.00 = midnight, 3.00 = 3:00 AM, etc.); Sleep onset latency = number of minutes from time in bed to sleep onset; Sleep duration variability= individual variation in sleep duration; Pubertal development = composite score from Pubertal Development Scale; Family SES = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education. MZ = monozygotic. DZ = dizygotic
*$p < .05$, **$p < .01$, ***$p < .001$.

of the Hispanic (M = 1.35, SD = 0.27) and white (M = 1.29, SD = 0.26) groups significantly differed (t = 2.72, $p < .01$). Regarding individual indicators, 67.3% of the overall sample had started growth in height, 11.5% had skin changes, and 9.8% had growth in body hair. About 12.8% of girls had started breast growth. No girls had started menstruation.

Table 2 contains mixed-model regression results. Among white and Hispanic participants, greater pubertal development was associated with longer sleep duration (7.2 minutes and 13.2 minutes, respectively) and marginally associated with higher efficiency, with pubertal development also associated with lower duration variability among Hispanic youth. For white, but not Hispanic youth, an interaction between pubertal development and gender was found, with greater pubertal development associated with shorter sleep onset latency for girls (about 4.59 minutes shorter, on average), but not boys (Fig. 1). Hispanic and white girls had longer sleep durations than boys in their respective racial groups (15 minutes and 16.8 minutes, respectively). Other main effects of gender differed by group, with Hispanic girls having a later midpoint (13.2 minutes later) than Hispanic boys, and white girls exhibiting higher sleep efficiency than white boys.

We present results in supplemental material using adrenarche and gonadarche puberty scores (Tables S1 and S2).

Univariate twin ACE models

Table 3 contains fit statistics and parameter estimates for full and reduced univariate ACE models. Pubertal development was moderately heritable, with the full ACE model providing the best fit. For

Table 2
Fixed effects estimates from multigroup mixed models predicting objective sleep from pubertal development, gender, and their interaction

Fixed effects	Sleep efficiency (percentage)		Sleep duration (hours)		Sleep midpoint (24-hour clock)		Sleep onset latency (minutes)		Sleep duration variability (hours)	
	Est.	SE	Est.	SE	Est.	SE	Est.	SE	Est.	SE
White										
Pubertal development	0.86†	0.47	0.12*	0.06	-0.01	0.06	-0.01	0.03	.02	.04
Gender	2.08**	0.65	0.28***	0.08	0.02	0.09	0.01	0.04	-.05	.04
Pubertal development x Gender	-0.15	0.65	-0.04	0.09	0.05	0.08	-0.08*	0.04	.02	.05
Age	0.55	0.50	-0.14*	0.07	0.21**	0.08	0.07*	0.03	.03	.03
Body mass index	-0.38**	0.14	-0.06***	0.01	0.00	0.02	-0.00	0.01	.00	.01
Family SES	0.89†	0.52	0.15**	0.06	-0.05	0.07	-0.04	0.03	-.01	.03
Vacation	-1.04	0.77	-0.08	0.09	0.48***	0.14	0.03	0.04	-.01	.05
Hispanic										
Pubertal development	1.36†	0.73	0.22*	0.09	-0.10	0.07	-0.02	0.04	-.10*	.04
Gender	0.63	0.97	0.25*	0.11	0.22*	0.11	-0.08†	0.05	-.09	.06
Pubertal development x Gender	-0.54	1.01	-0.14	0.13	0.17	0.12	0.03	0.05	.09	.06
Age	-1.01	0.91	-0.31**	0.11	0.30*	0.13	-0.07†	0.04	.04	.06
Body mass index	-0.30†	0.15	-0.02	0.02	-0.03*	0.02	-0.00	0.01	-.02*	.01
Family SES	1.28	0.82	0.22*	0.10	-0.29**	0.09	-0.09*	0.04	-.12*	.05
Vacation	0.93	1.41	0.14	0.15	0.48**	0.15	0.12	0.07	.13†	.08

Note. Sleep efficiency = percentage of time asleep out of total time in bed; Sleep duration = time asleep (in hours); Sleep midpoint = median of sleep start and sleep end time (0.00 = midnight, 3.00 = 3:00 AM, etc.); Sleep onset latency = number of minutes from time in bed to sleep onset; Sleep duration variability= individual variation in sleep duration; Pubertal development= standardized composite score from Pubertal Development Scale; Gender: 1 = Female, 0 = Male; Family SES = mean composite of family income-to-needs ratio, primary caregiver education, and secondary caregiver education; Vacation = participation during summer or holiday break (0 = not on vacation, 1 = on vacation); Est. = partial regression coefficient estimate (unstandardized); SE = robust standard error.
†$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$.

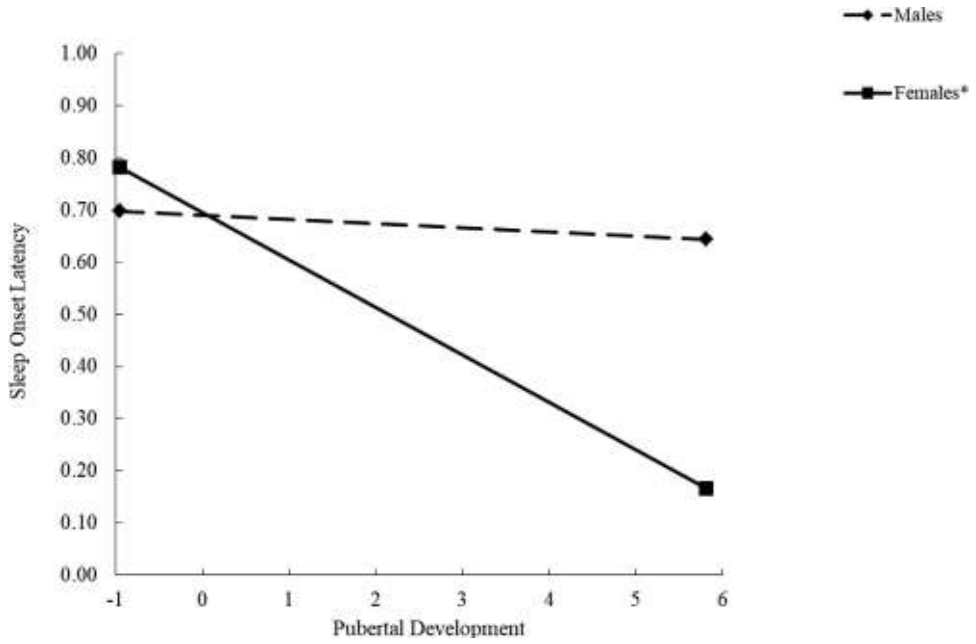

Figure 1. Simple slopes for association between pubertal development (Pubertal Development Scale composite scores were standardized within gender; minimum = -0.96, maximum = 5.81) with sleep onset latency (log transformed) by gender. *p < .05.

sleep duration and efficiency, the reduced AE model fit best, with high heritability and modest nonshared environmental influence. The reduced CE model fit best for sleep midpoint, onset latency, and duration variability, with high shared environmental variance for midpoint and onset latency and moderate shared environmental variance for duration variability. The univariate models for sleep indicators other than duration variability have been reported using the full sample, with similar results.[18]

Bivariate twin ACE models

Following recommendations[36] and based on phenotypic correlations (Table 1), two bivariate models were fit for pubertal development with sleep efficiency and duration, respectively. Table 4 contains fit statistics and standardized path estimates for the full Cholesky decomposition and most parsimonious reduced models. As C was nonsignificant in univariate sleep

Table 3
Univariate ACE model fit and parameter estimates

Scale	Model	-2LL	df	Δ-2LL	Δdf	*p*	AIC	A	C	E
Pubertal development	**ACE**	**1409.17**	**570**				**269.17**	**0.53**	**0.31**	**0.16**
	AE	1418.41	571	9.24	1	.002	276.41			
	CE	1434.14	571	24.97	1	<.001	292.14			
	E	1592.71	572	183.53	2	.001	448.71			
Sleep efficiency	ACE	2735.33	446	–	–	–	1843.33	0.53	0.20	0.27
	AE	**2737.43**	**447**	**2.1**	**1**	**.15**	**1843.43**	**0.74**	–	**0.26**
	CE	2744.87	447	9.54	1	<.001	1850.87			
	E	2819.41	448	84.08	2	<.001	1923.41			
Sleep duration	ACE	837.22	446	–	–	–	-54.78	0.70	0.11	0.19
	AE	**837.99**	**447**	**0.77**	**1**	**.38**	**-56.01**	**0.81**	–	**0.19**
	CE	856.90	447	19.68	1	<.001	-37.10			
	E	933.91	448	96.69	2	<.001	37.91			
Sleep midpoint	ACE	499.66	446	–	–	–	-392.34	0.05	0.90	0.05
	AE	664.11	447	164.45	1	<.001	-229.89			
	CE	**502.75**	**447**	**3.10**	**1**	**.08**	**-391.25**	–	**0.93**	**0.07**
	E	932.05	448	36.05	2	<.001	432.39			
Sleep onset latency	ACE	15.43	446	–	–	–	-876.57	0.22	0.55	0.23
	AE	39.47	447	24.04	1	<.001	-854.53			
	CE	**18.51**	**447**	**3.08**	**1**	**.08**	**-875.49**	–	**0.69**	**0.31**
	E	160.48	448	145.04	2	<.001	-735.52			
Sleep duration variability	ACE	258.98	446	–	–	–	-633.02	0.22	0.27	0.51
	AE	261.70	447	2.73	1	.1	-632.30			
	CE	**259.93**	**447**	**0.95**	**1**	**.33**	**-634.07**	–	**0.42**	**0.58**
	E	300.00	448	41.02	2	<.001	-596.00			

-2LL = -2 log likelihood; Δ = change; AIC = Akaike's information criterion.
Note. Bolded models denote the best-fitting models for each variable. A, C, and E are standardized squared parameter estimates for additive genetic (A), common environment (C), and nonshared environment (E) factors. Univariate ACE models using the full sample for some of the sleep parameters were previously reported in.[18] ACE models using the unstandardized puberty scores produced similar results.

Table 4
Bivariate ACE model fits and parameter estimates

Scale	Model	-2LL	df	Δ-2LL	Δdf	*p*	AIC
Pubertal development and sleep efficiency	ACE-ACE-ACE	4206.69	1018	–	–	–	2170.69
	ACE-ACE-AE	4208.30	1019	1.61	1	.21	2170.30
	ACE-AE-AE	**4208.35**	**1020**	**1.66**	**2**	**.44**	**2168.35**
Pubertal development and sleep duration	ACE-ACE-ACE	2275.63	1018	–	–	–	239.63
	ACE-AE-ACE	2275.63	1019	<.001	1	>.99	137.63
	ACE-AE-AE	**2275.93**	**1020**	**0.30**	**2**	**.86**	**235.93**
Model	Phenotype	A1	C1	E1	A2	C2	E2
ACE-ACE-ACE	Pubertal development	0.49/0.52	0.30/0.32	0.15/0.16			
	Sleep efficiency	0.29/0.01	0.10/0.00	0.08/0.00	17.14/.56	5.12/.17	7.99/0.26
ACE-AE-AE	Pubertal development	0.49/0.52	0.30/0.32	0.15/0.16			
	Sleep Efficiency	0.58/0.02	–	0.06/0.00	22.09/0.73	–	7.44/0.25
ACE-ACE-ACE	Pubertal development	0.50/0.52	0.32/0.33	0.15/0.15			
	Sleep duration	0.01/0.02	0.00/0.00	0.00/0.00	0.36/0.74	.03/.07	0.08/0.17
ACE-AE-AE	Pubertal development	0.50/0.52	0.32/0.33	0.15/0.15			
	Sleep duration	0.01/0.02	–	0.00/0.00	0.39/0.81	.00/.00	0.17

-2LL=-2 log likelihood; Δ= change; AIC= Akaike's Information Criterion.
Note. Bolded models denote the most parsimonious models for each variable. The names of reduced models indicate the genetic and environmental variance paths retained in the first phenotype, followed by the retained covariance paths shared between the first and second phenotype, and lastly the genetic and environmental paths retained in the second phenotype after accounting for covariance with the first phenotype. A, C, and E are standardized variance components for additive genetic (A), common environment (C), and nonshared environment (E) factors for phenotype 1 (pubertal development), phenotype 2 (sleep), and shared between the 2 phenotypes. For all models, unstandardized parameter estimates are reported first, followed by standardized estimates.

models, it was dropped from bivariate models. The most parsimonious models for pubertal development and sleep efficiency (Fig. 2a) and duration (Fig. 2b) indicated genetic influences on covariance between pubertal development and the sleep parameters. The ACE-AE-AE model fit best for both bivariate analyses according to the chi-squared difference test and the AIC, with 2% of the total variance in the sleep parameters accounted for by genetic influences shared with puberty. We could not distinguish between the ACE-A-AE and ACE-E-AE models given our sample size, so we accepted the ACE-AE-AE model as our final model.

Discussion

Phenotypic and genetic associations between puberty and actigraphy-assessed sleep in middle childhood, when children are reaching the earliest stages of pubertal development, offer a novel contribution to the child health and development literature. Unexpectedly, better sleep outcomes were observed for children further in pubertal development, and this was consistent across Hispanic and white youth. There were gender differences in puberty-sleep associations, with white girls demonstrating higher sleep efficiency and longer duration than white boys, and Hispanic girls having longer sleep

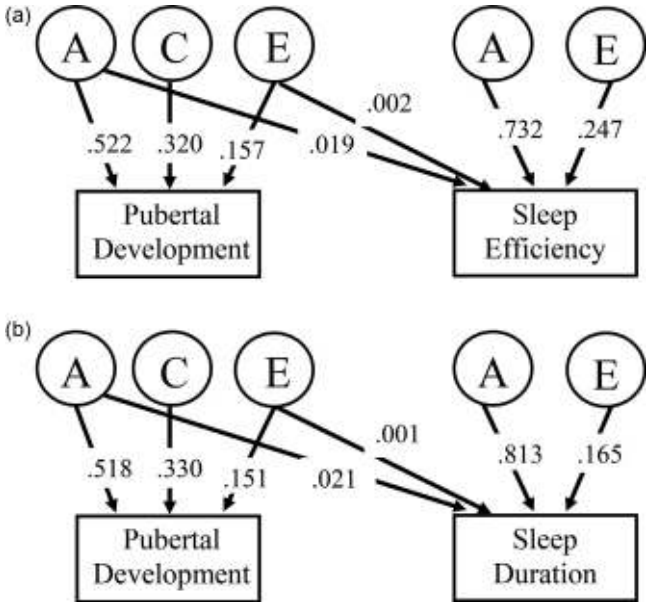

Figure 2. (a) Most parsimonious bivariate model for pubertal development and sleep efficiency. (b) Most parsimonious bivariate model for pubertal development and sleep duration. Note. Most parsimonious bivariate Cholesky decompositions are shown for associations between puberty and sleep, after nonsignificant paths were dropped from the full model without significant loss of fit to the data (see Table 4 for model fit indices and standardized path estimates for full models and best-fitting models). A, C, and E are standardized variance components for additive genetic (A), common environment (C), and nonshared environment (E) factors for phenotype 1 (pubertal development), phenotype 2 (sleep), and their covariance. Standardized path estimates for the second phenotype in each model are adjusted after accounting for the covariance between phenotypes.

duration and later midpoints than Hispanic boys. Pubertal development had genetic and environmental influences, and sleep efficiency and duration were highly heritable, but midpoint, onset latency, and duration variability were neither heritable nor related to pubertal development. Puberty and sleep were almost entirely genetically distinct, but genetic influences largely explained the modest but significant correlation between pubertal development and sleep duration and efficiency.

Hispanic and white children had similar relations between pubertal development and sleep, suggesting that developing earlier may be beneficial for sleep, at least at the start of puberty. This finding is opposite our hypotheses, as puberty is a stressor for youth and stress typically results in poorer sleep.[39] Instead, pubertal onset may require greater sleep to compensate for physical and psychological changes. Additionally, children in this sample may not yet be experiencing common environmental factors that negatively impact sleep in adolescence (e.g., early school start times, increased homework and extracurricular demands, difficulties in interpersonal relationships).[40] Advanced pubertal development and such stressors may both be needed for poorer sleep. The onset of puberty is an important focus, but associations between puberty and sleep may differ across the pubertal transition. Although growth in height was the most prominent indicator at this age, other indicators may become more relevant, and growth in height may slow at advanced stages of puberty. It will be beneficial to examine the puberty-sleep relation longitudinally across development.[2,9]

Gender differences were identified, such that white girls had better sleep quality than white boys, and Hispanic and white girls both had longer sleep duration than their respective male group. Hispanic girls had a later sleep midpoint than their male counterparts, suggesting later bedtimes and wake times compared to Hispanic boys. Although previous research indicated poorer sleep for male children ages 5-10,[15] this association may not hold across racial groups or across development. Importantly, significant gender differences were not found across all sleep indicators, limiting clear explanations for findings in each racial group, though environmentally-influenced sleep midpoint suggests greater environmental demands for Hispanic girls than boys (i.e., family assistance behaviors).[41] Further, racial differences are not biologically or genetically based, but rather reflect differential environmental opportunities and experiences.[42] Therefore, these findings call for the continued examination of proximal predictors of the puberty-sleep relation across multiple demographics.

The univariate models for the full sample are most similar to findings from sex-limitation models for girls, with high heritability and moderate shared environmental influences, whereas boys show mostly environmental influences. Thus, results from bivariate models more likely reflect these associations for girls than boys, likely because fewer boys have matured enough for heritable influences on pubertal development to be expressed. As boys and girls continue to develop with age, qualitatively different genetic influences may emerge.

Partially consistent with hypotheses, pubertal development was moderately heritable, in line with studies of older youth demonstrating higher co-twin correlations in age of menarche for monozygotic than dizygotic pairs.[43] Heritability of puberty increases from 12 to 14 years, and the shared environment may be more integral earlier in adolescence, especially for girls.[19] Our results align with the idea that the environment influences pubertal development at the onset of puberty, though genetic influences also matter. Most children in this sample were early in pubertal maturation, so the full range of genetic influences on puberty may not have had the opportunity to be fully expressed. Our findings also suggest that children may be exposed to shared environmental factors influencing pubertal development. Previously identified environmental factors linked to pubertal development include endocrine-disrupting chemicals, family structure and stress, and socioeconomic status.[44,45] As genetic and environmental influences on pubertal development are present during childhood, it remains critical to identify important environmental factors potentially affecting pubertal onset and sleep.

Our findings are consistent with prior analyses on the full twin sample,[18] including high genetic influences on sleep duration and efficiency, and more prominent shared and nonshared environment for midpoint, onset latency, and duration variability. Our younger sample may not yet be exposed to environmental factors linked with disrupted sleep in adolescence (i.e., increasing school demands, social media use, autonomy from parents).[46,47] While the heritability of sleep duration has been found to increase from childhood to adolescence, the heritability of sleep quality did not differ by age,[16] demonstrating that some sleep parameters may always be more environmentally influenced.

This was the first study to examine behavioral genetic covariances between pubertal development and actigraphy-assessed sleep. Bivariate models demonstrated primarily genetic links between pubertal development and sleep duration and efficiency, with possible modest nonshared environmental influences, though correlations were small. These results suggest a biologically-based relation between puberty and sleep, perhaps due to hormonal changes, supporting the biopsychosocial and contextual model of sleep. However, puberty and sleep are only modestly related at this age, and shared genetic factors may become more prominent with age. Regardless of magnitude of associations, results suggest that promoting healthy behaviors includes emphasizing the importance of improved sleep patterns and emotional adjustment to pubertal changes. Results also suggest that contextual factors potentially associated with disturbances in sleep and other health behaviors, such as discrimination and socioeconomic status,[42] require further examination.[22,23] Attention to these and other factors potentially related to genetic and environmental influences (e.g., BMI) may aid in understanding puberty-sleep relations.

Limitations, strengths, and future directions

This study has multiple limitations. First, cross-sectional analyses of pubertal development and sleep are uninformative about directionality. Second, we lacked power to conduct twin analyses separately by race and gender, though this is an important next step for future studies. Although samples sizes are small, we present twin intra-class correlations separately by race and gender in supplemental material for illustrative, hypothesis-generating purposes (see Table S4). Third, the genetic covariances between puberty and sleep were small, and should be examined in samples representing the full range of puberty development. Last, parents may rate their twins more similarly than objective measures (i.e., hormonal indicators, practitioner rated Tanner stages), leading to higher shared environmental influences. However, when considering boys and girls separately, shared environmental influences are mainly evident for boys, likely due to lower PDS scores. Further, Tanner stages are moderately to highly associated with both self- and parent-reported PDS.[48] Additional work using actigraphy-assessed sleep and multiple assessments of puberty is warranted.

The present study has numerous strengths, setting the stage for future research. The focus on the onset of puberty builds on existing literature examining puberty-sleep relations in older youth, and we importantly found more developed children to have better sleep. While most research relies on self-report, this study focused on actigraphy-assessed sleep, including within-week variability in sleep timing. Future studies may consider how parent- or self-report of child sleep and sleep problems differ in genetic and environmental influences,[18] potentially uncovering distinctions in the relation between disordered sleep and pubertal development. This was the first study to examine genetic and environmental influences on covariation between puberty and sleep, and identified shared genetic underpinnings between pubertal

development and sleep duration and efficiency. Examining whether highly environmentally-influenced sleep indicators (i.e., onset latency, midpoint, and duration variability) are linked to pubertal development for older youth is an important future direction.

Conclusions

Puberty and sleep duration and efficiency were positively associated, and twin analyses supported genetic influences underlying this relation, though associations are small during middle childhood and likely become more important during adolescence. As sleep is a critical adolescent health process,[10] public health efforts focused on interventions for improved sleep quality and quantity may be especially important for youth at the onset of puberty to provide a foundation of healthy sleep habits in preparation for the transition into pubertal development and sleep changes that occur during adolescence.

Declaration of conflict of interest

The authors have no conflicts of interest to disclose.

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Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.sleh.2021.12.006.

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