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High heritability of adolescent sleep–wake behavior on free, but not school days: a long-term twin study

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Abstract

Adolescence development is characterized by significant changes in sleep biology. Despite an overall decline in sleep duration and a delay in bedtime, significant interindividual variation in sleep has been reported. The aim of the current study was to examine genetic and environmental influences on sleep in adolescence using long-term (6 month) actigraphy measurements, differentiating between school and free days. Sixteen monozygotic ($n = 32$) and 10 dizygotic ($n = 20$) twin pairs (mean age 12.8 ± 1.0 years; 25 females) participated in the study. Structural equation modeling was used to compute genetic, shared environmental and unique environmental contributors to sleep behavior. We found significantly more genetic influence on sleep timing (sleep midpoint; school: 14%, free: 90%) and duration (school: 15%; free: 68%) on free compared with school days. On the other hand, the genetic influence on measures of sleep quality (sleep efficiency and sleep onset latency) was high (>60%) and less dependent on the day of measurement. Only wake after sleep onset (WASO) exhibited a strong shared environmental influence (> 52%) on both school and free days, suggesting that behavioral/environmental interventions may help reduce WASO. In addition, self-reported chronotype was also highly genetically influenced (75%). Disrupted, ill-timed, and insufficient sleep in adolescence is associated with poor mental and physical health outcomes. Our findings of a strong genetic contribution to sleep in adolescence suggest that sleep may mark a genetic vulnerability to poor outcomes.

actigraphy, adolescents, twin, genetic, heritability, sleep

Statement of Significance

We use long-term (6 month) measurement of sleep via actigraphy to quantify the heritability of sleep–wake behavior in a sample of early adolescent (mean age 12.8 years) twins. Calculating heritability separately for free and school days, we find that heritability of sleep timing and behavior is high only when measured on free days. Thus, only under conditions which sleep is less constrained by school/work commitments do biological tendencies (i.e. genes) express themselves. Our findings inform the current body of literature and suggest that the genetic influence on sleep is markedly underestimated in previous studies most of which do not differentiate between school/work and free days. Therefore, it is critical that future studies query individuals regarding sleep patterns on free days.

Introduction

Adequate and well-timed sleep is essential for both mental and physical well-being. Disrupted and short sleep is associated with obesity [1, 2], impaired cognitive function [3, 4], and psychiatric disorders [5] to name a few. Although chronic sleep deprivation is common in today's society, adolescents are often the most sleep-deprived members of society, receiving far fewer than the recommended hours of sleep [6–8]. Sleep need in adolescence is significantly greater than in adulthood—studies have shown that adolescents require on average about 9.25 hr [9]. The National Sleep Foundation, however, recognizes interindividual differences in sleep need, thus prescribing a sleep duration between 8 and 10 hr per night as adequate based on individual needs. Many adolescents, however, struggle to get sufficient sleep, particularly on school nights. The National Sleep Foundation poll of 171 US adolescents in 2011 [10] reported that approximately 61% of adolescents in the United States received less than 8 hr of sleep on school nights. This was an increase of 45% compared with the poll conducted in 2006 [11].

This pattern of truncated sleep can be explained by the profound biological, emotional, and social changes that occur during adolescence [12]. During adolescence, a delay in the circadian system that shifts bedtime later has been described not only in humans, but also in other mammalian species such as degus and nocturnal rats [13], suggesting a biological underpinning [14]. This delay in the circadian system is further exacerbated by environmental factors, such as increased academic workload, increased part-time employment, use of technology in the bedroom, and less parental influence on bedtime [15–18]. These biological and behavioral changes combined with early school start times result in the ever diminishing sleep duration amongst adolescents [19, 20].

Nevertheless, large individual differences in the sleep patterns of adolescents, including timing, quality, and duration have long been known [18]. Interindividual variations are not only due to environmental factors, but genetic inheritance also plays an important role. Studies in adult twins have shown that sleep is a complex trait with a strong genetic component. These studies are based on the underlying assumption that identical (monozygotic; MZ) twin pairs share 100% of their genetic material and grow-up in the same familial and social environment, whereas nonidentical twins (dizygotic; DZ) share an environment but on average only half of their genes. Based on this assumption, it is possible to estimate the relative proportions of three sources of variances: additive genetic influences (A), shared environmental influences (C), and nonshared environmental influences (E) [21].

A considerable number of subjective twin studies regarding sleep characteristics have been published in adults. One such study [22] used a large sample to investigate the contribution of genetic factors to sleep length in adults over a 15 year period (time 1; MZ = 3499, DZ = 7542, time 2; MZ = 3017, DZ = 6306, and time 3; MZ = 1540, DZ = 2967). They found that the heritability of sleep length was stable across the three measurement points and ranged from 0.30 to 0.32. Another study of a general population sample of adult Australian twin pairs (n = 3810) aged 18 to 88 also did not find evidence of heterozygosity of twin correlations as a function of age and estimated heritability around 0.40 [23]. Thus, the current evidence suggests that heritability of self-reported sleep duration remains stable across adulthood and ranges between 0.3 and 0.4.

Studies in adolescents have produced mixed results. One study of twins between the ages of 15 and 22 years (MZ = 105 and DZ = 234 twin pairs) estimated the heritability of sleep duration at 0.63 [24], whereas another study of 6319 twins and their nontwin siblings (n = 1359) between the ages of 12 to 20 years [25] found that genes accounted for 33%–47% of the variance.

Despite the large sample sizes, one limitation of the studies above is the use of self- or parent-reported sleep parameters. The gold standard in objective sleep measurement is polysomnography (PSG). Studies using PSG have found substantial genetic influence on sleep architecture [26–30]. However, such studies are expensive and therefore often limited to one night under artificial circumstances (e.g. while wearing sleep recording equipment in a laboratory). Surveying sleep over a longer period in the participant's natural environment is an attractive alternative solution and possible with the advent of modern actigraphy devices. To the best of our knowledge, only one study to date has used actigraphy to examine the relative contributions of genetic and environmental factors on phenotypic sleep–wake patterns in 12-year-old MZ and DZ twins [31]. In this study, 132 adolescent twins (25 MZ and 41 DZ pairs) wore an actigraph and completed a daily sleep diary for 2 weeks. Results showed that additive genetic factors explained 65% of the variance in sleep duration, 83% of the variance in sleep onset latency (SOL), 57% of the variance in sleep efficiency (SE), and 52% of the variance in wake after sleep onset (WASO). In contrast, shared environment had a predominant influence on the timing of sleep (sleep start time, sleep end time) with heritability factors of 67% and 86%, respectively.

One limitation of the above study is the inclusion of only a few free days (mean of 2.96 days), since sleep phenotypes are strongly modulated by free versus school days [18, 32, 33]. Thus, the aim of our study was to estimate the differences between genetic and environmental contributors to sleep/wake behavior in adolescent twins for school nights, free nights (including weekends, public holidays, and vacation), and holidays (public holidays and vacations, excluding weekends). Therefore, actigraphy measurement was performed over a period of 6 months in order to acquire sufficient data to examine this issue. We hypothesize that a stronger genetic influence will be observed on free days since bed and rise times are not restricted by environmental factors (e.g. school start times) and will allow biology a chance to express itself.

Methods

Participants

Fifty-one healthy adolescents (26 boys, 25 girls) aged 12.8 ± 1.0 (mean ± SD) years corresponding to 16 MZ (7 males) and 10 DZ (6 males; one opposite sex pair) twin pairs participated in the study (including one triplet pair which was used for one MZ and DZ measurement). Exclusion criteria included the following: suffering from a chronic or current illness, use of medications affecting sleep and brain function, known sleep disorders, and preterm birth before the 30th gestation week. Zygosity was determined through administration of a zygosity questionnaire to parents, which has been shown to be 95% accurate [34]. Data from two DZ pairs were excluded due to insufficient data, or problems with data collection (i.e. early termination of the study).

Procedures

All participants were asked to wear a commercial actigraph (Jawbone UP) on their nondominant hand daily for 6 months. The actigraphy devices were designed to be worn 24 hr a day and 7 days a week. Participants were instructed to only remove the actigraph when bathing or swimming. Participants and their parents received no instruction on sleep, leaving bed and rise times up to the participant and their parent.

Measures

Jawbone UP (www.jawbone.com/up) powered by MotionX uses micromechanical triaxial accelerometers to track body movements. Participants were instructed to press a button on the band to switch from active to sleep mode when they were in bed with the lights out, and to switch into active mode when they woke up in the morning. Using proprietary algorithms, Jawbone UP calculates the following variables with minute precision: sleep duration (TST), sleep start time, sleep end time, time in bed (TIB), total wake time (TWT), and SOL. In addition to the above variables, WASO (calculated TWT minus SOL), SE (TST divided by TIB), and sleep midpoint (sum sleep start time and sleep end time divided by two) were calculated from the output of the actigraphy device. De Zambotti and colleagues report good agreement between Jawbone UP and PSG for TST (overestimated on average by 10.0 ± 20.5 min), SE (overestimated on average by 1.9% ± 4.2%), SOL (no difference), and WASO (underestimated by 9.3 ± 20.4 min) in healthy adolescents (n = 65; mean age = 15.8 ± 2.5 years) [35]. Furthermore, in a clinical sample of children and adolescents (n = 78; ages 3 to 18 years, mean age = 8.4 ± 4.0 years), good sensitivity (0.92) and accuracy (0.86) were also found when the Jawbone Up was compared with PSG [36]. Actigraphy data were carefully inspected and excluded based on rest/activity patterns (e.g. if band was accidently switched from sleep to active mode or vice versa). Each night of data was individually examined and nights were flagged for possible exclusion when bed or rise time was exceptionally early or late, or TST was very short or long in duration. Nights were then excluded from the analysis based on the activity patterns (i.e. there was no activity following “rise time”). Three hundred ninety-six nights (5% of all measured nights, consisting of 53% school days, and 47% free days) had to be excluded from the analysis for these reasons, and therefore, 6848 nights were analyzed in this study. Compliance differed amongst individuals; therefore, the number of measured nights per individual varied from 50 to 207 nights (134.3 ± 39.4 nights). The proportion of days on which actigraph was not worn was on average 35.3% across participants. Our rate of data loss is similar to what has been reported in a study examining seven nights of actigraphy (28%) and is likely due to noncompliance (e.g. taking the band off to swim and forgetting to put it back on), a failure to switch from sleep to wake mode by pressing the button, and/or device loss or failure [37].

In addition to actigraphy, self-reported sleep duration was also assessed in the study using the Sleep Habits Survey (SHS) at the beginning and at the end of the 6-month data acquisition interval. The questions: “Figure out how long you usually sleep on a normal school night? (Do not include time you spend awake in bed. Remember to mark hours and minutes, even if minutes are zero)” and “Figure out how long you usually sleep on a night when you do not have school the next day (Do not include time you spend awake in bed. Remember to mark hours and minutes, even if minutes are zero.)” from the SHS were used to determine the subjective sleep duration for school and free days.

We also calculated the heritability of chronotype using the Superscience morningness/eveningness scale [38]. This 10-item scale is designed for children and adolescents and is adapted from a Composite Morningness Questionnaire, which is in turn based on the Horne Östberg Morningness–Eveningness Questionnaire (MEQ) and a diurnal type scale by Torsvall and Akerstedt.

Statistical analysis

For each twin, the mean and standard deviation (SD) of all sleep parameters were calculated separately for school days, free days, and holidays, for the 6 month period measured via actigraphy. We define free days as public/school holidays and weekends since on both weekends and holidays sleep

bed and rise times are not dictated by school start times, allowing biological tendencies an opportunity to express themselves. Of the 6848 nights, 3074 nights (45% of all night; mean and *SD* of 60.27 ± 20.61 nights across individuals; range 18–102 nights per individual) were classified as free days. The remaining 3774 nights were school nights (55% of all nights; mean and *SD* of 74.00 ± 21.78; range 28–122 nights per individual).

We perform a separate analysis for sleep on "holidays" excluding weekends because weekend sleep is influenced by weekday sleep, which tends to be truncated in adolescents. For this reason, we exclude the first two holiday nights to allow for a washout of prior weekday sleep/wake history. In this way, holiday nights are an estimation of sleep–wake behavior on nights unaffected by waking for school or recovering from weeknight sleep. Applying this criterion, 23% of the data (1605 nights) consisted of holidays. The number of holiday nights for each individual ranged between 3 and 61 nights, with a mean of 31.5 ± 14.4 nights across individuals.

We examined the influence of measurement day, zygosity, and gender on all sleep parameters using mixed models ANOVA with within-participants factor day of measurement (school day, free day, or holiday) and between-participants factors zygosity (MZ vs. DZ) and gender. We examined main effects and interactions.

In order to compare our results to the existing twin literature, we performed three analyses using the actigraphy data: (1) Pearson correlation, (2) intraclass correlation coefficient (ICC) analysis, and (3) structural equation modeling (SEM) separately for school day, free day, and holiday. Correlation analyses have traditionally been used in twin studies with the underlying assumption that similar MZ and DZ correlations are an indication that shared environmental factors are likely responsible for the variability in the sleep parameters, whereas higher MZ compared with DZ correlations indicates a high genetic impact on the sleep behavior. With regard to ICC analysis, we use absolute agreement, and large and positive ICC values are observed when there is little variation between twin pairs but large variation amongst unrelated pairs. Therefore, ICC is expected to be higher in MZ compared with DZ twins.

Although the above-mentioned analyses have been used in the past [23, 24, 39], SEM can be used to model the amount of variance due to additive genetics (A), shared environment (C), and unique/nonshared environment, including measurements error (E). We performed SEM in R (version 3.2.3) using the statistical software package OpenMx (version 2.7.4, source; http://openmx.psyc.virginia.edu/getOpenMx.R). Univariate ACE modeling rather than multivariate ACE modeling was performed for each of the seven sleep parameters for the actigraphy data on the basis of the small sample size. The univariate model was adjusted for age and sex. ACE and submodels (AE, CE) were fit to each of the seven variables by the free estimation of variances, covariance, and means to minimize the −2 times log likelihood of the data.

Furthermore, since previous studies on the heritability of sleep have predominately used subjective reports of sleep, we also examine the heritability of self-reported sleep duration using SEM and correlation coefficients (mean from the two time points). Moreover, in order to test whether objective sleep measures are dependent on whether twin pairs shared a bedroom (n = 14) or slept separately (n = 38), a Mann–Whitney U-test was performed. We also examined the heritability of chronotype and also performed a partial correlation, controlling for age and sex, between chronotype and sleep parameters on school and free days.

Results

Mean and standard deviations from the actigraphy data for TIB, TST, sleep start time, sleep end time, WASO, SOL, SE, and sleep midpoint are shown in Table 1. In this table, zygosity and day of measurement (school day, free day, and holiday) are reported separately to verify that biological tendencies were not masked by school schedules. Mixed model ANOVA indicated nonsignificant main effects and interactions for zygosity (i.e. MZ versus DZ) and gender (males versus females) for all sleep parameters. Two exceptions to this were TST, which showed an interaction between gender and day of measurement and TIB which exhibited a significant interaction between day of measurement and zygosity (Table 1). In contrast, sleep parameters differed significantly dependent on the day of measurement. Sleep was longer (i.e. TIB and TST) and later (bed and rise time and midpoint) on free compared with school days. Sleep start times were on average 70 min later on free days compared with school days. Moreover, sleep end time showed a delay of 92 min for free days compared with school days. Participants slept on average 8.20 hr during school days and 8.53 hr on free days. The discrepancy of sleep phase between school days and free days is known as social jetlag [40] and was on average 1.35 ± 0.44 hr. Furthermore, on school days, there was less WASO while SOL was longer compared with free days. Sleep on free days did not significantly differ from holidays, with the exception that the timing (bed and rise time) of holiday sleep was slightly later (Table 1) and SE was lower. TIB, TST, sleep start time, sleep start time, sleep end time, SOL, and SE were not affected by room status as assessed using Mann–Whitney U-test (for school and free day). Shared room status influenced WASO during school (shared bedroom mean = 35.3; nonshared bedroom mean = 23.3; accurate Mann–Whitney U-test; U = 143.000, p = 0.011), but not free days or holidays, indicating that twins in the same bedroom affect each other.

Table 1.

Sleep parameters on school days, free days, and holidays

	School day		Free day		Holiday	
Phenotype	**MZ**	**DZ**	**MZ**	**DZ**	**MZ**	**DZ**
TIB	9 hr 9 min ± 30 min	8 hr 51 min ± 26 min	9 hr 24 min ± 25 min	9 hr 18 min ± 24 min	9 hr 27 min ± 27 min	9 hr 22 min ± 32 min
TST	8 hr 17 min ± 28 min	8 hr 4 min ± 27 min	8 hr 33 min ± 27 min	8 hr 30 min ± 28 min	8 hr 32 min ± 26 min	8 hr 31 min ± 32 min
Sleep start time	22:08 ± 0:41	22:23 ± 0:26	23:13 ± 0:41	23:36 ± 0:36	23:24 ± 0:46	23:44 ± 0:44
Sleep end time	6:43 ± 0:24	6:43 ± 0:18	8:08 ± 0:31	8:27 ± 0:46	8:19 ± 0:37	8:36 ± 0:46
WASO (hr)	0.48 ± 0.28	0.46 ± 0.24	0.72 ± 0.39	0.71 ± 0.29	0.72 ± 0.41	0.72 ± 0.31
SOL (min)	33.95 ± 11.91	30.23 ± 10.32	30.15 ± 10.55	27.94 ± 7.10	32.17 ± 13.71	28.81 ± 9.78
SE (%)	90.58 ± 3.27	91.25 ± 2.83	91.04 ± 3.29	91.29 ± 2.43	90.47 ± 3.66	90.89 ± 2.94
Sleep midpoint	2:26 ± 0:30	2:33 ± 0:18	3:40 ± 0:34	4:01 ± 0:39	3:52 ± 0:40	4:10 ± 0:42

Mean ± standard deviation across 6 months of actigraphy for sleep parameters of MZ and DZ twins, distinguishing between school day, free day, and holiday.

MZ = monozygotic; DZ = dizygotic; TIB = time in bed; TST = total sleep time; WASO = wake after sleep onset; SOL = sleep onset latency; SE = sleep efficiency.

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Pearson correlations using objective sleep data are presented in Table 2 separately for school days, free days, and holidays. Pearson correlations and ICCs (Table 3) revealed an influence of both genetic and environmental factors on sleep behavior. Generally speaking, higher Pearson correlation coefficients were found for MZ than for DZ twins on school days, free days, and holidays. However, on school days, correlation coefficients for TIB, sleep end time, WASO, and sleep midpoint were statistically significant for both MZ and DZ twins. Although correlations were qualitatively higher for MZ, the high and significant correlations for both MZ and DZ twins suggest a strong influence of shared environmental factors for these parameters on school days. Measurements obtained on free days and holidays, however, showed a significant and strong correlation in MZ and DZ twins only for WASO. These findings indicate that shared environmental factors only have a strong influence on the previously mentioned parameters (TIB, sleep end time, sleep midpoint) on school, but not on free days. Findings were similar for the ICC analysis (Table 3), with large and significant ICC values for MZ twins and smaller and nonsignificant values for DZ twin. Exception to this were TIB, TST, sleep end time, WASO, and midpoint on school days and WASO on free days and holidays.

Table 2.

Pearson correlation coefficients for MZ and DZ twins

	School day		Free day		Holiday	
Phenotype	**MZ**	**DZ**	**MZ**	**DZ**	**MZ**	**DZ**
TIB	**0.833** (p < 0.001)	**0.788** (p = 0.007)	**0.736** (p = 0.001)	0.294 (p = 0.41)	**0.577** (p = 0.02)	0.298 (p = 0.40)
TST	**0.689** (p = 0.003)	0.565 (p = 0.09)	**0.682** (p = 0.004)	0.353 (p = 0.32)	**0.549** (p = 0.03)	−0.174 (p = 0.63)
Sleep start time	**0.862** (p < 0.001)	0.518 (p = 0.125)	**0.924** (p < 0.001)	0.497 (p = 0.14)	**0.880** (p < 0.001)	0.207 (p = 0.57)
Sleep end time	**0.880** (p < 0.001)	**0.873** (p = 0.001)	**0.803** (p < 0.001)	0.393 (p = 0.26)	**0.767** (p = 0.001)	0.001 (p = 0.99)
WASO	**0.900** (p < 0.001)	**0.801** (p = 0.005)	**0.892** (p < 0.001)	**0.769** (p =0.009)	**0.776** (p < 0.001)	**0.798** (p = 0.006)
SOL	**0.805** (p < 0.001)	−0.013 (p = 0.97)	**0.761** (p = 0.001)	0.064 (p = 0.86)	**0.594** (p = 0.015)	0.124 (p = 0.73)
SE	**0.826** (p < 0.001)	0.468 (p = 0.173)	**0.764** (p = 0.001)	0.413 (p = 0.236)	**0.620** (p = 0.010)	0.025 (p = 0.95)
Sleep midpoint	**0.872** (p < 0.001)	**0.638** (p = 0.047)	**0.900** (p < 0.001)	0.461 (p = 0.18)	**0.860** (p < 0.001)	0.141 (p = 0.70)

Pearson correlation coefficient for MZ and DZ twins (p-value), separated for school days, free days, and holidays. Significant correlations (p < 0.05) are highlighted in bold.

MZ = monozygotic; DZ = dizygotic; TIB = time in bed; TST = total sleep time; WASO = wake after sleep onset; SOL = sleep onset latency; SE = sleep efficiency.

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Table 3.

Intraclass correlation coefficient (ICC) for MZ and DZ twins

	School day		Free day		Holiday	
Phenotype	**MZ**	**DZ**	**MZ**	**DZ**	**MZ**	**DZ**
TIB	**0.832** (p < 0.001)	**0.783** (p = 0.002)	**0.689** (p = 0.001)	0.264 (p = 0.216)	**0.538** (p = 0.013)	0.280 (p = 0.202)
TST	**0.686** (p = 0.001)	**0.560** (p = 0.036)	**0.679** (p = 0.001)	0.314 (p =0.174)	**0.543** (p = 0.012)	−0.149 (p = 0.669)
Sleep start time	**0.862** (p < 0.001)	0.364 (p = 0.136)	**0.922** (p < 0.001)	0.472 (p = 0.071)	**0.880** (p < 0.001)	0.185 (p = 0.293)
Sleep end time	**0.860** (p < 0.001)	**0.863** (p < 0.001)	**0.802** (p < 0.001)	0.390 (p = 0.118)	**0.764** (p < 0.001)	0.001 (p = 0.499)
WASO	**0.880** (p < 0.001)	**0.800** (p = 0.002)	**0.892** (p < 0.001)	**0.766** (p = 0.003)	**0.776** (p < 0.001)	**0.797** (p = 0.002)
SOL	**0.794** (p < 0.001)	−0.013 (p = 0.515)	**0.740** (p < 0.001)	0.062 (p = 0.428)	**0.542** (p = 0.012)	0.097 (p = 0.389)
SE	**0.826** (p < 0.001)	0.418 (p = 0.100)	**0.754** (p < 0.001)	0.385 (p = 0.121)	**0.620** (p = 0.004)	0.018 (p = 0.480)
Sleep midpoint	**0.872** (p < 0.001)	**0.541** (p = 0.043)	**0.899** (p < 0.001)	0.444 (p = 0.086)	**0.859** (p < 0.001)	0.113 (p = 0.371)

Intraclass correlation coefficient (ICC) and corresponding p-value in parentheses for monozygotic and dizygotic twins, separated for school day, free day, and holiday conditions. Significant correlations (p < 0.05) are highlighted in bold.

MZ = monozygotic; DZ = dizygotic; TIB = time in bed; TST = total sleep time; WASO = wake after sleep onset; SOL = sleep onset latency; SE = sleep efficiency.

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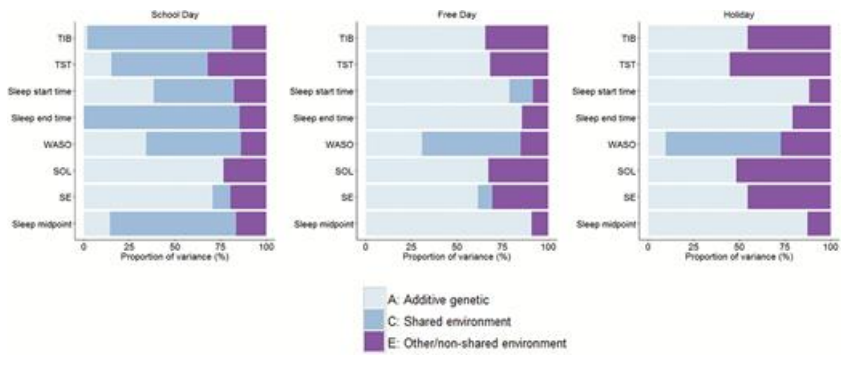
Figure 1 represents results of the univariate ACE model for each objective sleep phenotype adjusted for sex and age, separately for free days, holidays, and school days. Values for each model for school days (Supplementary Table 1), holidays (Supplementary Table 2), and free days (Supplementary Table 3) can be found in the supplements. Large differences between free days/holidays and school days in the estimation of the genetic contribution were found for TIB, TST, sleep start time, sleep end time, and sleep midpoint. Smaller differences (<10%) between free and school days were found for WASO, SOL, and SE, indicating more stable genetic influences on these traits on school and free days. The genetic influence on WASO, SOL, and SE was somewhat lower on holidays compared with school and free days, whereas the influence of other factors and error (i.e. E in the model) was higher.

Figure 1.



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Proportion of variance in sleep parameters accounted for by additive genetic (A; light blue), shared environment (C; dark blue), and nonshared environment (E; purple) for school days, free days, and holidays. TIB = time in bed; TST = total sleep time; WASO = wake

after sleep onset; SOL = sleep onset latency; SE = sleep efficiency.

With regards to subjective data (i.e. questionnaire data) on sleep duration on school days, genetic factors accounted for 18.7% of the variance, while shared environmental factors accounted for 62% of the variance. For sleep duration on free days, genetic factors contributed to 1.8% of the variance, 41.5% was due to shared environmental factors, and unique environment/error explained 56.8% of the variance. Pearson correlation coefficient between subjective and objective data was 0.53 for school days and 0.25 for free days for sleep duration.

Chronotypes ranged between the values of 21–35 and the sample had an average value of 29.8 ± 3.5, indicating mixed chronotypes within the sample. We found a large contribution of genes (A = 75.5%) on chronotype, with no contribution of shared environmental factors (C = 0%) and a moderate influence of unique environment/error (E = 24.5%). As expected, later chronotype was associated with later bed and wake times, sleep midpoint, and SOL on school day, free days, and holidays (Table 4).

Table 4.

Partial correlation coefficient between chronotype and sleep parameters

Phenotype	School day	Free day	Holiday
TIB	−0.043 (p = 0.768)	−0.281 (p = 0.050)	−0.248 (p = 0.085)
TST	−0.003 (p = 0.985)	−0.179 (p = 0.219)	−0.121 (p = 0.406)
Sleep start time	**−0.515** (p < 0.001)	**−0.376** (p = 0.008)	**−0.352** (p = 0.013)
Sleep end time	**−0.539** (p < 0.001)	**−0.439** (p = 0.002)	**−0.405** (p = 0.004)
WASO	0.281 (p = 0.050)	0.107 (p = 0.463)	0.020 (p = 0.891)
SOL	**−0.383** (p = 0.007)	**−0.389** (p = 0.006)	**−0.400** (p = 0.004)
SE	0.066 (p = 0.653)	0.075 (p = 0.607)	0.159 (p = 0.275)
Sleep midpoint	**−0.578** (p < 0.001)	**−0.430** (p = 0.002)	**−0.397** (p = 0.005)

Partial correlation coefficients, adjusted for sex and age, between chronotype and sleep parameters for school day, free day, and holiday separately (df = 47). p-Values are shown in parentheses and significant values are highlighted in bold.

TIB = time in bed; TST = total sleep time; WASO = wake after sleep onset; SOL = sleep onset latency; SE = sleep efficiency.

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Discussion

To the best of our knowledge, this is the first study providing objective data on the contribution of genes and environment on sleep behavior that distinguishes between school and free days in early adolescents (aged 11–14 years) and includes sufficient data to accurately estimate sleep on free days [37]. We show a strong genetic impact on sleep timing, duration, and quality in early adolescence when measured on days which sleep is less constrained by school obligations (i.e. weekends and holidays). Disrupted sleep is a common feature in most psychiatric disorders [41–44], many of which have their onset during adolescence [45]. This comorbidity has been hypothesized to be in part due to genes that code for both sleep/circadian and psychiatric disorders [5]. Our findings bolster this idea by showing that sleep quality and duration have a large (>60%) genetic component in adolescence. Furthermore, the association between disrupted/short sleep and psychiatric illness is bidirectional, with poor sleep in adolescence being a unique predictor of depression in adulthood [46]. Thus, our finding of significant genetic contribution to sleep duration and quality when measured on free days and holidays indicates that sleep may mark a genetic vulnerability to psychiatric illness. One exception to this is WASO, which is influenced by shared environmental more than genetic factors. This suggests that behavioral interventions, such as cognitive behavioral therapy for insomnia (CBT-I), may be effective in treating sleep continuity problems in adolescence.

Interestingly, we find the largest genetic contribution to sleep midpoint, a proxy for circadian phase [47, 48]. During adolescence, a shift towards evening-type occurs, and the strong genetic influence on sleep midpoint on free days suggests a biological substrate to this phenomenon. In line with

this, we find a strong genetic contribution to chronotype in our sample. Aberrations in the circadian timing system have been reported in a number of psychiatric illness, including but not limited to, depression, and schizophrenia [5, 41]. Indeed, polymorphisms of the CLOCK gene, a gene that affects circadian rhythms, have been associated with bipolar disorder and alcohol addiction. Therefore, our finding of a heritability of 90% with regards to sleep midpoint suggests that this measure may be a fruitful avenue for future research examining overlapping genes between psychiatry and sleep.

We note that compared with free and school days, significantly less data were available for the calculation of holidays and may in part explain the greater amount of variance explained by the factor E in the model, which includes measurement error.

Comparing the results from our objective and long-term measurement of sleep to most other twin studies is difficult given that most studies subjectively assessed sleep parameters and used different methodologies. Most previous twin studies only assessed sleep duration and used subjective categorical surveys in which participants reported sleep duration within a range (e.g. 5 to 6 hr). In addition to the difficulties of transforming a continuous variable (i.e. sleep duration) into a categorical one, the discrepancy between objective and subjective assessment of sleep is well known. For example, Short et al. showed substantial and significant differences between parents and adolescents with regards to sleep estimated by actigraphy, sleep diary, and self-report [49]. Parents reported 35–45 min more sleep during school days and 40–92 min on free days than adolescents did. Another study, comparing actigraphy, sleep diary, and questionnaires assessed in children, found that questionnaire answers are highly influenced by memories, experiences, and expectations [50]. A further limitation of questionnaires was demonstrated in a twin study of 100 MZ and 199 DZ school-aged twins (aged 8 years). In this study, estimates of genetic and environmental influences on sleep differed based on who filled out the sleep questionnaires [51]. No genetic influence on sleep duration was obtained when children were queried. In contrast, 71% of the variance of sleep length was explained by genetic factors when parents answered questions about their children's sleep. Given this discrepancy between subjective and objective measures, it is not surprising that we find widely varying influence of genes when comparing subjective (18.7% school day; 1.8% free days) to objective (15.2% school day; 68.2% free day; 45% holiday) long-term assessment of sleep duration. We note that the estimation of genetic factors is similar on school days for objective (i.e. actigraphy) and subjective (i.e. questionnaires) measures of sleep. We hypothesize that this may be because rise times (and bedtimes to an extent) are determined by school start times, allowing for less variability and a more accurate subjective assessment of sleep on school compared with free days.

Our study questions the utility and accuracy of questionnaires in genetic studies of sleep [25, 49–51]. For example, due to the feasibility of data collection, genome-wide association studies (GWAS) [52, 53] with one exception [54] have used questionnaires to assess sleep duration. The genes identified in such studies typically account for a small portion of the observed variance in sleep duration, and this may in part be due to the inaccuracy of self-reported sleep.

Another limitation of previous studies is that they did not differentiate between school/work and free days [22–25, 39, 55, 56], despite well-documented differences in sleep duration on school/work days (TST; e.g. 5–8 hr) compared with free days (TST; e.g. 9–12 hr) in adolescents [8]. The same holds true for adults, where sleep is truncated by work and social schedules resulting in a considerable sleep debt on work days [40]. To the best of our knowledge, only one study to date has used actigraphy to examine the relative contributions of genetic and environmental factors on phenotypic variance in sleep–wake patterns over a period of 2 weeks [31]. Although they found significant differences for the parameters bedtimes, rise times, sleep start time, sleep end time, and SOL between school and free days (based on mean and standard deviation) in their sample of 12-year-old MZ ($n = 50$) and DZ ($n = 82$) twins, their further analysis showed a relatively consistent sleep pattern when school days and free days were separate. Due to the small numbers of free days (maximum of 4 days) in their data set, their SEM analysis was performed without distinguishing between school and free days. A study regarding the number of actigraphy nights needed to reliably estimate sleep found that five nights are adequate; however, seven nights or more may be required to obtain reliable estimates of interindividual differences in sleep [37]. Thus, the long-term actigraphy recordings in our sample are a significant strength of our analyses.

Our findings for school days are globally in line with the results reported by the above-mentioned study. We find similar genetic and environmental influences on sleep start and end times. This similarity between the studies despite the combination of free and school days in the Sletten [31] study may be either due to the small number of free days in the Sletten study which would not significantly affect the mean, or the difference between a European and Australian sample. To wit, parental influence on bedtimes is more pronounced in young Australian adolescents [16] and data show that Australian teens are long sleepers.

In contrast to the above findings, we find large difference in our estimation of the genetic and environmental contributions to TST. Sletten et al. [31] find that 52% of the variance in TST is driven by genetic factors. This value lies in between our estimation of the genetic influence of TST free days and holidays (free days = 68%; holidays = 45%) and could be driven by the fact that their study combined measurement days. Another parameter for which our findings diverge is WASO (school day; A = 34 %; free day A =31 %; holiday = 10%) for which we find a lower genetic impact compared with 57% of variance explained by genetic factors in the study of Sletten et al. [31]. This disparate finding could be due to the use of two different actigraphy advices. The actigraphy (Actiwatch-64, Bend, OR) used in the Sletten study is able to detect WASO with an accuracy of 56% [57],

which is lower than the 83% accuracy found for the device used in the current study (Jawbone Up [35]).

Given that sleep timing and duration on school days is dictated by school start times, we believe that sleep on free days allows an opportunity for genes to express themself and more accurately reflects biological processes. Focusing on free days, we found a slightly higher genetic impact of 68% on sleep duration compared with what has been reported in the literature for adults (i.e. 17%–44% [22, 23, 39, 58–60]). Given the aforementioned difficulties of comparing questionnaire data to subjective measures, it remains unclear whether the difference between our findings and those in adults is due to methodology or biology. Based on the current evidence, the association between heritability of sleep and age may be inverse U-shaped. Two twin studies on early childhood assessed children at 15 (n = 3862) and 18 months (n = 624) of age using parental-rated questionnaires and estimated a modest additive genetic effect of 26%–31% on sleep duration [55, 56]. Our data, an Australian sample [31] and a Croatian one [24], place the heritability of sleep duration in adolescent slightly above 60%. As suggested by others [31, 61], nonshared environmental influences may exert more influence on adulthood in which work and family responsibilities dictate sleep/wake times. Such age-dependent changes in heritability have been reported for other traits, such as IQ [62]. Indeed, there is growing evidence that the genetic impact on sleep is age dependent, as has been shown for a polymorphism in the clock gene, PER 3 [63]. Therefore, any conclusions about the etiology of a trait should be limited to the age group being studied.

Similar to sleep duration, we find higher heritability of chronotype (75%) in our sample of adolescents compared with adults (34%–56%) [64–68]. A similar U-shape pattern has been reported for chronotype; a study which examined heritability of chronotype between the ages of 19 to 93 years found that heritability was higher in younger and older adults (A = 44%) compared with middle-aged adults (A = 34%) [69]. We note that the pubertal process, which is both heritable [70, 71] and affects the circadian timing system [72], may in part drive our high values of heritability given the age of our sample.

Despite the ability of actigraphy to provide estimates of sleep in participants' home environment over a longer time period, it also has some limitations. The actigraphs used in this study overestimate TST and underestimate WASO by approximately 10 min compared with PSG. No significant difference between PSG and the actigraphy device was found for SOL [35]. Despite this limitation, we do not anticipate a large impact on our findings due to the large number of nights measured for each participant in the current study and the use of the same device across participants.

Another limitation of this study is the modest sample size of only 16 MZ and 10 DZ twin pairs. Despite this, our results on weekdays and model fits are similar to that of Sletten [31]; therefore, we believe that we have sufficient statistical power. A further limitation of the study is the use of questionnaires to assess Zygosity. Studies have shown that such parent-report questionnaires of zygosity in adolescent samples are only 95% to 97.4% accurate compared with 99% accuracy obtained from DNA tests [73]. Nonetheless, we show a strong genetic impact on sleep timing, duration, and quality in early adolescence when measured on days which sleep is less constrained by environmental factors. Adolescence is a unique biological and behavioral milieu and sleep reflects and interacts with this new phase of development. Therefore, our findings have broad implications for understanding adolescent development.

Supplementary Material

Supplementary material is available at *SLEEP* online.

Notes

Conflict of interest statement. None declared.

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References

1. Jarrin DCet al. Beyond sleep duration: distinct sleep dimensions are associated with obesity in children and adolescents. *Int J Obes (Lond)* . 2013; 37(4): 552–558.
   Google Scholar Crossref PubMed

2. Patel SRet al. ; Osteoporotic Fractures in Men (MrOS); Study of Osteoporotic Fractures (SOF) Research Groups. The association between sleep patterns and obesity in older adults. *Int J Obes (Lond)* . 2014; 38(9): 1159–1164.
   Google Scholar Crossref PubMed

3. Scullin MKet al. Sleep, cognition, and normal aging: integrating a half century of multidisciplinary research. *Perspect Psychol Sci* . 2015; 10(1): 97–137.
   Google Scholar Crossref PubMed

4. Burke TMet al. Sleep inertia, sleep homeostatic and circadian influences on higher-order cognitive functions. *J Sleep Res* . 2015; 24(4): 364–371.
   Google Scholar Crossref PubMed

5. Wulff Ket al. Sleep and circadian rhythm disruption in psychiatric and neurodegenerative disease. *Nat Rev Neurosci* . 2010; 11(8): 589–599.
   Google Scholar Crossref PubMed

6. Carskadon MA. Sleep in adolescents: the perfect storm. *Pediatr Clin North Am* . 2011; 58(3): 637–647.
   Google Scholar Crossref PubMed

7. Hysing Met al. Sleep patterns and insomnia among adolescents: a population-based study. *J Sleep Res* . 2013; 22(5): 549–556.
   Google Scholar Crossref PubMed

8. Gradisar Met al. Recent worldwide sleep patterns and problems during adolescence: a review and meta-analysis of age, region, and sleep. *Sleep Med* . 2011; 12(2): 110–118.
   Google Scholar Crossref PubMed

9. Carskadon MA. The second decade. In: Guilleminault C, *ed. Sleep and Waking Disorders: Indications and Techniques* . 1982: 99–125.

10. National Sleep Foundation. 2011 Sleep in America Poll: Summary of Findings. https://sleepfoundation.org/sites/default/files/sleepinamericapoll/SIAP_2011_Summary_of_Findings.pdf.

11. National Sleep Foundation. 2006 Sleep in America Poll: Summery of Findings. https://sleepfoundation.org/sites/default/files/2006_summary_of_findings.pdf.

12. Tarokh Let al. Physiology of normal sleep in adolescents. *Adolesc Med State Art Rev* . 2010; 21(3): 401–17, vii.
   Google Scholar PubMed

13. Hagenauer MHet al. The neuroendocrine control of the circadian system: adolescent chronotype. *Front Neuroendocrinol* . 2012; 33(3): 211–229.
   Google Scholar Crossref PubMed

14. Carskadon MAet al. Association between puberty and delayed phase preference. *Sleep* . 1993; 16(3): 258–262.

Google Scholar Crossref PubMed

15. Cain Net al. Electronic media use and sleep in school-aged children and adolescents: A review. *Sleep Med* . 2010; 11(8): 735–742.
Google Scholar Crossref PubMed

16. Carskadon MA. Patterns of sleep and sleepiness in adolescents. *Pediatrician* . 1990; 17(1): 5–12.
Google Scholar PubMed

17. Short MAet al. Time for bed: parent-set bedtimes associated with improved sleep and daytime functioning in adolescents. *Sleep* . 2011; 34(6): 797–800.
Google Scholar Crossref PubMed

18. Wolfson ARet al. Sleep schedules and daytime functioning in adolescents. *Child Dev* . 1998; 69(4): 875–887.
Google Scholar Crossref PubMed

19. Fallone Get al. Sleepiness in children and adolescents: clinical implications. *Sleep Med Rev* . 2002; 6(4): 287–306.
Google Scholar Crossref PubMed

20. Iglowstein Iet al. Sleep duration from infancy to adolescence: reference values and generational trends. *Pediatrics* . 2003; 111(2): 302–307.
Google Scholar Crossref PubMed

21. Boomsma Det al. Classical twin studies and beyond. *Nat Rev Genet* . 2002; 3(11): 872–882.
Google Scholar Crossref PubMed

22. Hublin Cet al. Genetic factors in evolution of sleep length–a longitudinal twin study in Finnish adults. *J Sleep Res* . 2013; 22(5): 513–518.
Google Scholar Crossref PubMed

23. Heath ACet al. Evidence for genetic influences on sleep disturbance and sleep pattern in twins. *Sleep* . 1990; 13(4): 318–335.
Google Scholar Crossref PubMed

24. Butkovic Aet al. Sleep duration and personality in Croatian twins. *J Sleep Res* . 2014; 23(2): 153–158.
Google Scholar Crossref PubMed

25. te Velde SJet al. Genetic and environmental influences on individual differences in sleep duration during adolescence. *Twin Res Hum Genet* . 2013; 16(6): 1015–1025.
Google Scholar Crossref PubMed

26. De Gennaro Let al. The electroencephalographic fingerprint of sleep is genetically determined: a twin study. *Ann Neurol* . 2008; 64(4): 455–460.
Google Scholar Crossref PubMed

27. Landolt HP. Genetic determination of sleep EEG profiles in healthy humans. *Prog Brain Res* . 2011; 193: 51–61.
Google Scholar Crossref PubMed

28. Linkowski P. EEG sleep patterns in twins. *J Sleep Res* . 1999; 8 Suppl 1: 11–13.
Google Scholar Crossref PubMed

29. Webb WBet al. Relationships in sleep characteristics of identical and fraternal twins. *Arch Gen Psychiatry* . 1983; 40(10): 1093–1095.
Google Scholar Crossref PubMed

30. Linkowski Pet al. Genetic determinants of EEG sleep: a study in twins living apart. *Electroencephalogr Clin Neurophysiol* . 1991; 79(2): 114–118.
Google Scholar Crossref PubMed

31. Sletten TLet al. Genetic and environmental contributions to sleep-wake behavior in 12-year-old twins. *Sleep* . 2013; 36(11): 1715–1722.
Google Scholar PubMed

32. Strauch Iet al. Sleep need in adolescents: a longitudinal approach. *Sleep* . 1988; 11(4): 378–386.
Google Scholar Crossref PubMed

33. Szymczak JTet al. Annual and weekly changes in the sleep-wake rhythm of school children. *Sleep* . 1993; 16(5): 433–435.
Google Scholar Crossref PubMed

34. Goldsmith HH. A zygosity questionnaire for young twins: a research note. *Behav Genet* . 1991; 21(3): 257–269.
Google Scholar Crossref PubMed

35. Zambotti Met al. Validation of sleep-tracking technology compared with polysomnography in adolescents. *Sleep* . 2014; 38(9): 1461–1468.
Google Scholar Crossref

36. Toon Eet al. Comparison of commercial wrist-based and smartphone accelerometers, actigraphy, and PSG in a clinical cohort of children and adolescents. *J Clin Sleep Med* . 2016; 12(3): 343–350.
Google Scholar Crossref PubMed

37. Acebo Cet al. Estimating sleep patterns with activity monitoring in children and adolescents: how many nights are necessary for reliable measures?*Sleep* . 1999; 22(1): 95–103.
Google Scholar Crossref PubMed

38. Wolfson ARet al. Sleep schedules and daytime functioning in adolescents. *Child Dev* . 1998; 69(4): 875–887.
Google Scholar Crossref PubMed

39. Partinen Met al. Genetic and environmental determination of human sleep. *Sleep* . 1983; 6(3): 179–185.
Google Scholar Crossref PubMed

40. Wittmann Met al. Social jetlag: misalignment of biological and social time. *Chronobiol Int* . 2006; 23(1-2): 497–509.
Google Scholar Crossref PubMed

41. Benca RMet al. Sleep and psychiatric disorders. A meta-analysis. *Arch Gen Psychiatry* . 1992; 49(8): 651–68; discussion 669.
Google Scholar Crossref PubMed

42. Shahid Aet al. Peeking into the minds of troubled adolescents: the utility of polysomnography sleep studies in an inpatient psychiatric unit. *J Affect Disord* . 2012; 139(1): 66–74.
Google Scholar Crossref PubMed

43. Tesler Net al. Developmental changes in sleep and their relationships to psychiatric illnesses. *Curr Opin Psychiatry* . 2013; 26(6): 572–579.
Google Scholar Crossref PubMed

44. Tarokh Let al. Sleep in child and adolescent psychiatry: overlooked and underappreciated. *Eur Child Adolesc Psychiatry* . 2014; 23(6): 369–372.
Google Scholar Crossref PubMed

45. Paus Tet al. Why do many psychiatric disorders emerge during adolescence?*Nat Rev Neurosci* . 2008; 9(12): 947–957.
Google Scholar Crossref PubMed

46. Urrila ASet al. Sleep in adolescent depression: physiological perspectives. *Acta Physiol (Oxf)* . 2015; 213(4): 758–777.
Google Scholar Crossref PubMed

47. Martin SKet al. Sleep logs of young adults with self-selected sleep times predict the dim light melatonin onset. *Chronobiol Int* . 2002; 19(4): 695–707.
Google Scholar Crossref PubMed

48. Burgess HJet al. The relationship between the dim light melatonin onset and sleep on a regular schedule in young healthy adults. *Behav Sleep Med* . 2003; 1(2): 102–114.
Google Scholar Crossref PubMed

49. Short MAet al. Estimating adolescent sleep patterns: parent reports versus adolescent self-report surveys, sleep diaries, and actigraphy. *Nat Sci Sleep* . 2013; 5: 23–26.
Google Scholar Crossref PubMed

50. Werner Het al. Agreement rates between actigraphy, diary, and questionnaire for children's sleep patterns. *Arch Pediatr Adolesc Med* . 2008; 162(4): 350–358.
Google Scholar Crossref PubMed

51. Gregory AMet al. A twin-study of sleep difficulties in school-aged children. *Child Dev* . 2006; 77(6): 1668–1679.
Google Scholar Crossref PubMed

52. Hu Yet al. GWAS of 89,283 individuals identifies genetic variants associated with self-reporting of being a morning person. *Nat Commun* . 2016; 7: 10448.
Google Scholar Crossref PubMed

53. Jones SEet al. Genome-Wide Association Analyses in 128,266 Individuals Identifies New Morningness and Sleep Duration Loci. *PLoS Genet* . 2016; 12(8): e1006125.
Google Scholar Crossref PubMed

54. Spada Jet al. Genome-wide association analysis of actigraphic sleep phenotypes in the LIFE Adult Study. *J Sleep Res* . 2016; 25(6): 690–701.
Google Scholar Crossref PubMed

55. Brescianini Set al. Genetic and environmental factors shape infant sleep patterns: a study of 18-month-old twins. *Pediatrics* . 2011; 127(5): e1296–e1302.
Google Scholar Crossref PubMed

56. Fisher Aet al. Genetic and environmental influences on infant sleep. *Pediatrics* . 2012; 129(6): 1091–1096.
Google Scholar Crossref PubMed

57. Rupp TLet al. Comparison of motionlogger watch and actiwatch actigraphs to polysomnography for sleep/wake estimation in healthy young adults. *Behav Res Methods* . 2011; 43(4): 1152–1160.
Google Scholar Crossref PubMed

58. Gottlieb DJet al. Genome-wide association of sleep and circadian phenotypes. *BMC Med Genet* . 2007; 8Suppl 1: S9.
Google Scholar Crossref PubMed

59. de Castro JM. The influence of heredity on self-reported sleep patterns in free-living humans. *Physiol Behav* . 2002; 76(4–5): 479–486.
Google Scholar Crossref PubMed

60. Watson NFet al. Sleep duration and area-level deprivation in twins. *Sleep* . 2016; 39(1): 67–77.
Google Scholar Crossref PubMed

61. Barclay NLet al. Moderation of genetic and environmental influences on diurnal preference by age in adult twins. *Chronobiol Int* . 2014; 31(2): 222–231.
Google Scholar Crossref PubMed

62. Haworth CMet al. The heritability of general cognitive ability increases linearly from childhood to young adulthood. *Mol Psychiatry* . 2010; 15(11): 1112–1120.
Google Scholar Crossref PubMed

63. Jones KHet al. Age-related change in the association between a polymorphism in the PER3 gene and preferred timing of sleep and waking activities. *J Sleep Res* . 2007; 16(1): 12–16.
Google Scholar Crossref PubMed

64. Drennan MDet al. Morningness/eveningness is heritable. *Soc Neurosci Abstracts* . 1992; 18: 196.

65. Hur Yet al. Genetic and environmental influence on morningness–eveningness. *Pers Individual Differences* . 1998; 25: 917–925.
Google Scholar Crossref

66. Vink JMet al. Genetic analysis of morningness and eveningness. *Chronobiol Int* . 2001; 18(5): 809–822.
Google Scholar Crossref PubMed

67. Koskenvuo Met al. Heritability of diurnal type: a nationwide study of 8753 adult twin pairs. *J Sleep Res* . 2007; 16(2): 156–162.
Google Scholar Crossref PubMed

68. Klei Let al. Heritability of morningness-eveningness and self-report sleep measures in a family-based sample of 521 hutterites. *Chronobiol Int* . 2005; 22(6): 1041–1054.
Google Scholar Crossref PubMed

69. Barclay NLet al. Moderation of genetic and environmental influences on diurnal preference by age in adult twins. *Chronobiol Int* . 2014; 31(2): 222–231.
Google Scholar Crossref PubMed

70. Wehkalampi Ket al. Genetic and environmental influences on pubertal timing assessed by height growth. *Am J Hum Biol* . 2008; 20(4): 417–423.
Google Scholar Crossref PubMed

71. Hoekstra RAet al. Heritability of testosterone levels in 12-year-old twins and its relation to pubertal development. *Twin Res Hum Genet* . 2006; 9(4): 558–565.
Google Scholar Crossref PubMed

72. Hagenauer MHet al. Adolescent changes in the homeostatic and circadian regulation of sleep. *Dev Neurosci* . 2009; 31(4): 276–284.
Google Scholar Crossref PubMed

73. Chen WJet al. Diagnosis of zygosity by questionnaire and polymarker polymerase chain reaction in young twins. *Behav Genet* . 1999; 29(2): 115–123.
Google Scholar Crossref PubMed

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