

The Relationship Between Family Socioeconomic Status and Adolescent Sleep and Diurnal Cortisol

Sarah Rocha, MA, David M. Almeida, PhD, Jessica J. Chiang, PhD, Steve W. Cole, PhD, Michael R. Irwin, MD, Teresa Seeman, PhD, and Andrew J. Fuligni, PhD

ABSTRACT

Objective: This study aimed to investigate the associations between indices of family socioeconomic status and sleep during adolescence and to examine whether measures of hypothalamic-pituitary-adrenal (HPA) axis functioning mediate the observed associations.

Methods: A total of 350 ethnically diverse adolescents (57% female; mean [standard deviation] age_{wave 1} = 16.4 [0.7] years) completed a three-wave longitudinal study in which sleep and cortisol data were collected at 2-year time intervals. Sleep duration, latency, and variability were assessed via actigraphy during a period of 8 days per study wave. Salivary cortisol was collected across 3 days per study wave to assess cortisol diurnal slope, area under the curve, and the cortisol awakening response. Adolescents' caregivers reported their education levels, family income, and economic hardship.

Results: A greater family income-to-needs ratio was associated with longer adolescent sleep duration ($b = 2.90, p = .023$), whereas greater parental education was associated with shorter sleep duration ($b = -3.70, p = .030$), less sleep latency ($b = -0.74, p = .016$), and less variability across days ($b = -2.06, p = .010$). Diurnal cortisol slope statistically mediated the association of parental education with sleep duration ($b = -0.48$, 95% confidence interval = -1.099 to -0.042), but not the association of income-to-needs ratio with sleep duration.

Conclusions: Findings suggest that parental education and family resources may have unique impacts upon sleep and HPA axis functioning during the period of adolescence. Future research is needed to examine family and behavioral factors that may underlie socioeconomic status associations with adolescent sleep and HPA axis functioning.

Key words: sleep, adolescence, socioeconomic status, diurnal cortisol, actigraphy.

INTRODUCTION

Scholars have pointed to sleep as a potential source of disparities in education and health that can emerge during adolescence and extend into adulthood (1,2). Sleep plays a critical role in adolescent development, consistently exhibiting associations with learning (3), mood (4), and mental and physical health (5,6). Epidemiologic research suggests that socioeconomic status (SES) may be related to sleep, with higher-SES individuals showing more positive sleep outcomes (7,8). However, the relationship between SES and sleep during adolescence is still somewhat uncertain. Although some studies have found disparities in sleep duration between high- and low-SES adolescents (8–10), null associations are also common (11–13).

Inconsistency in the literature on SES-sleep associations may be due to the diverse ways SES and sleep can be operationalized. Sleep is often measured in terms of duration; however, other components of sleep may also have implications for health. Consistency in bedtime routines has been often cited as a necessary component for good sleep hygiene, and increasing evidence suggests that adolescents with greater variability in nightly sleep duration may be at risk for negative health outcomes (14,15). Sleep latency, or the time it takes for adolescents to fall asleep, can also be an indicator of sleep issues. A short sleep latency may imply feelings of sleepiness,

whereas a long sleep latency can suggest difficulties falling asleep (16). Assessing multiple components of sleep behavior is needed to form a more complete picture of the relationship between SES and sleep because single indicators of sleep do not capture the multiple dimensions of sleep quality.

Furthermore, SES is a multifaceted construct (17), and use of single or composite measures of SES may not fully capture the unique associations different SES measures may have with sleep outcomes. For example, common measures of SES, such as family income and parental education, have the potential to impact youths' sleep in varying ways. Low-income families may be less able to provide a home environment conducive to sleep; previous literature suggests that aspects of the home environment such as noise, light, and family chaos contribute to income associations with youths' sleep quality (13,18). Parental education, on the other hand, maybe be associated with parenting practices that influence youths' sleep. Previous literature suggests that mothers with lower educational attainment are less likely to have consistent bedtime routines for their children (19). Alternatively, highly educated parents

AUC = area under the curve, CAR = cortisol awakening response, EH = economic hardship, HPA = hypothalamic-pituitary-adrenal, INR = income-to-needs ratio, SES = socioeconomic status

From the Department of Psychology (Rocha, Irwin, Fuligni), University of California, Los Angeles, Los Angeles California; Department of Human Development and Family Studies (Almeida), Pennsylvania State University, University Park, Pennsylvania; Department of Psychology (Chiang), Georgetown University, Washington, DC; and Cousins Center for Psychoneuroimmunology, Department of Psychiatry and Biobehavioral Sciences (Cole, Irwin, Fuligni), and Division of Geriatrics, David Geffen School of Medicine (Seeman), University of California, Los Angeles, Los Angeles, California.

Address for correspondence to Sarah Rocha, MA, Department of Psychology, University of California, Los Angeles, 2311 Franz Hall, Los Angeles, CA 90095. E-mail: rocha@ucla.edu

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may have greater academic expectations for their adolescents (20), and greater time spent in engaging in academic activities such as homework can come at the cost of youths' sleep (21). Finally, measures of economic strain, such as perceived hardship and difficulty making ends meet, have been related to sleepiness during adolescence indirectly via family chaos (18). Examining multiple measures of SES simultaneously and estimating their associations with different components of sleep behavior (e.g., duration, latency, variability) can help bring clarity to the literature's inconclusive associations between SES and youths' sleep.

Furthermore, there is still much to be understood about the mechanisms linking SES indices with adolescent sleep, particularly potential physiologic systems. One physiologic system that has shown strong associations with both SES and sleep is the hypothalamic-pituitary-adrenal (HPA) axis. The HPA axis reacts to stressful events, releasing the hormone cortisol (22), and significant evidence suggests that HPA axis activity also is associated with chronic stressors like poverty (23). Cortisol is released by the HPA axis in a typical fashion wherein circulating levels are high in the morning, peak shortly after wake, and then decline gradually across the day until bedtime (24). Consistent literature has linked SES with the pattern of the decline in circulating cortisol across the day, wherein high SES is associated with a steeper decline (25,26). Deviations from typical HPA axis functioning, such as a less steep or "blunted" pattern of diurnal secretion, may indicate dysregulation, and this pattern of cortisol secretion is associated with numerous adverse mental and physical health outcomes (27). SES has also been linked with the total cortisol output over the day, measured as the area under the curve (AUC); however, the direction of the association has been mixed (25,28). Similarly mixed associations have been found in regard to the initial surge in cortisol secretion after wake, known as the cortisol awakening response (CAR) (26). Adding to this complexity, both large and small values of CAR may be indicative of dysregulation (29). Thus, SES seems to be relevant to typical HPA axis functioning, although associations may most consistently be found in relation to the slope of the decline in cortisol across the day.

Given the circadian rhythm of cortisol secretion and its relationship with sleep-wake patterns (24), scholars have investigated whether alterations of typical HPA axis functioning are related to sleep disruption. Evidence suggests that there is likely a reciprocal relationship between cortisol secretion and sleep patterns (30,31). For example, a study

of adolescents found that longer sleep duration was predictive of a steeper decline in cortisol levels the next day, and a steeper decline predicted greater hours of sleep the following day (32). If altered HPA axis functioning disrupts sleep, this could position it as a potential pathway by which SES influences sleep patterns. Considering evidence for a reciprocal relation between diurnal cortisol and sleep duration, altered HPA axis functioning could induce a cascading effect in which initial dysregulation is exacerbated by ongoing sleep difficulties.

The goals of this study were the following: a) to examine the association between SES and sleep using multiple indicators of SES and b) to determine whether HPA axis functioning acts as a statistical mediator of the association between SES and sleep. We focused on parental education, family income-to-needs ratio (INR), and parental economic hardship (EH) as our indicators of SES. We predicted that adolescents from families with greater INR, higher parental education, and less parental EH would have longer sleep duration and less daily variability in their sleep. Given that both short and long sleep latencies can indicate sleep difficulties, we predicted that SES would be related to latency in either direction. Next, we predicted that HPA axis functioning would mediate the association between SES and sleep variables, such that higher SES would be related to better sleep outcomes indirectly through a steeper cortisol diurnal slope. We also explored the mediating role of the CAR and AUC, but because of their less consistent associations with SES, we predicted that mediation would occur through either smaller or higher values of AUC and CAR.

METHODS

Participants

Participants were 350 adolescents recruited from four public high schools in the Los Angeles area who took part in a three-wave longitudinal study with their primary caregivers (88% mothers, 8% fathers, 4% others) in which data were collected on daily experiences at 2-year time intervals. Adolescents were majority female (57%) and ethnically diverse (42% Latinx, 30% European American, 22% Asian American, and 6% identifying with another ethnicity). Forty-one percent of adolescents had at least one parent born outside the United States, and 2% did not provide information on caregiver country of origin. In the first wave, which took place between October 2011 and June 2012, a total of 316 adolescents (mean [standard deviation {SD}] age = 16.40 [0.74] years) agreed to participate in the study (see Table 1

TABLE 1. Descriptive Statistics for Sociodemographic, Sleep, and Diurnal Cortisol Variables

Variable	Wave 1 (n = 311)		Wave 2 (n = 223)		Wave 3 (n = 159)	
	Range	Mean (SD)	Range	Mean (SD)	Range	Mean (SD)
Age, y	14.50 to 20.50	16.39 (0.74)	14.50 to 22.17	18.31 (0.77)	16.50 to 22.09	20.29 (0.74)
Parental education	1 to 11	7.18 (1.87)	1 to 11	7.42 (1.96)	1 to 11	7.44 (2.05)
Family INR	0 to 14.32	2.80 (2.13)	0 to 17.69	3.58 (2.74)	0 to 14.73	3.82 (2.83)
Parental EH	1 to 3.95	2.15 (0.76)	1 to 4	2.09 (0.79)	1 to 4	2.02 (0.73)
Duration, min	177.4 to 603.0	447.7 (57.84)	221.4 to 575.4	446.5 (64.23)	161.0 to 592.4	430.77 (75.14)
Latency, min	0 to 57.50	10.38 (9.85)	0 to 95.00	10.10 (11.31)	0 to 64.00	11.30 (11.83)
Variability, min	0.75 to 175.33	57.13 (28.99)	0.75 to 166.46	60.28 (29.61)	0.75 to 164.05	62.03 (35.89)
Diurnal slope	-2.58 to 0.22	-1.01 (0.44)	-3.34 to 1.01	-1.26 (0.63)	-4.75 to 0.67	-1.49 (0.96)
CAR	-22.16 to 37.25	4.73 (9.19)	-24.64 to 35.58	4.47 (10.81)	-29.51 to 50.77	4.18 (13.05)
AUC	32.98 to 499.64	173.07 (70.76)	28.70 to 531.05	218.64 (87.00)	17.02 to 710.02	225.76 (112.92)

n = number of adolescents completing at least some measurement of sleep or cortisol during study wave; SD = standard deviation; INR = income-to-needs ratio; EH = economic hardship; CAR = cortisol awakening response; AUC = area under the curve.

for descriptive statistics). In the following wave of data collection, conducted in October 2013 to June 2014, 214 of the original participants returned, and 34 new, grade-matched participants were added to the sample to account for attrition, for a total of 248 participants (mean [SD] age = 18.31 [0.77] years). In the final wave of data collection (October 2015–August 2016), 180 participants who had participated in one or both of the previous two waves (mean [SD] age = 20.29 [0.74] years) returned. Given that a subset of adolescents was recruited at the second wave, study completion was determined by the percent of possible waves in which an adolescent collected some sleep or cortisol data. Study completion varied by adolescent ethnicity ($F(3,346) = 5.408, p = .001$), with Asian adolescents completing the fewest waves of data collection. In addition, female adolescents were more likely to complete all possible waves of data collection ($r(347) = 0.11, p = .05$), as were those with higher family incomes ($r(306) = 0.14, p = .01$), higher parental education ($r(346) = 0.14, p = .01$), and whose parents were born in the United States ($r(340) = 0.11, p = .03$).

Procedures

Adolescents and their caretakers were recruited via flyers and presentations at school. Those interested in participating in the study were contacted by phone, and written consent was obtained in-person during the first study visit at participants' homes. Adolescents and their caregivers reported demographic information and completed surveys during the initial visit. At each wave of data collection, adolescents were instructed to wear an actigraph watch (Micro Motionlogger Sleep Watch; Ambulatory Monitoring, Inc., Ardsley, New York) for eight nights to record their sleep. In addition, for the first three nights of each study wave, adolescents were asked to collect saliva samples to measure diurnal cortisol at designated time points throughout the day. Adolescents were compensated after each wave of data collection, receiving \$50 after the first wave, \$75 after the second, and \$125 after the third. To further incentivize retention, adolescents were given two movie theater passes for completing the daily protocols correctly. All study procedures were approved by the University of California, Los Angeles Institutional Review Board.

Measures

SES Variables

Parent Education

Caregivers reported the highest level of education they achieved (1, some elementary school; 2, completed elementary school; 3, some junior high school; 4, completed junior high school; 5, some high school; 6, graduated from high school; 7, trade or vocational school; 8, some college; 9, graduated from college; 10, some medical, law, or graduate school; 11, graduated from medical, law, or graduate school) and, if applicable, the highest level of education of the adolescents' other caregiver. Responses for both caregivers were averaged for one composite score of parental education. Education reported at study entry (i.e., wave 1 or Wave 2) was used in all analyses.

Income-to-Needs Ratio

At each study wave, adolescents' primary caregivers reported their total household income over the past year and how many people lived in their household. Given the greater fluctuation in income as compared with parental education, we used income at each wave to predict the sleep and cortisol parameters at the same wave. Each family's INR was computed by dividing total reported household income over the US Department of Health and Human Services' federal poverty threshold by family size. For waves 1 through 3, the 2012, 2014, and 2016 guidelines were used, respectively. An INR = 1 represents a family living at the poverty level. Descriptive statistics for family INR across study waves are reported in Table 1.

Parental Economic Hardship

A modified version of a well-established questionnaire used by Conger et al. (33) for measuring family economic pressure was completed by the primary caregiver at each wave. The questionnaire was broken down into

three subsections concerning their ability to: a) pay bills, 2) save, and 3) afford material items over the past 3 months. First, caregivers were asked to rate their difficulty paying bills on a 4-point Likert scale with end points: "no difficulty at all" to "a great deal of difficulty." Second, caregivers were asked to estimate their savings on a 4-point scale from "with more than enough money left over" to "very short on money." Third, caregivers were provided with a list of seven affirmative statements on their ability to purchase essential material needs (e.g., food, clothing, medical care) and rated their agreement on a 4-point scale from "not at all true" to "very true." Responses to the three subsections are averaged to create a composite score of perceived EH (18,33). Mean parental EH scores are reported in Table 1.

Sleep Variables

Sleep variables were based on adolescents' sleep actigraphy data. Adolescents were instructed to wear the actigraph watch on their nondominant hand and to keep it on for the duration of the time they spent in bed at night. An event marker was pressed to indicate when adolescents turned off the lights to go to bed, when they woke up, and when/if they got out of bed during the night. Data were scored using software package Action 4 (Ambulatory Monitoring, Inc.). Sleep statistics for each night were calculated using 1-minute epochs and the Sadeh actigraph scoring algorithm, which has been validated and used in studies with children and adolescents (15,34). Time in bed began when the adolescent pressed the event marker indicating lights out and ended when pressed again the next morning. Sleep onset began after at least 3 consecutive minutes of sleep were scored, and sleep offset was determined after the last 5 or more consecutive minutes of sleep. A total of 343 participants completed sleep actigraphy measurement during at least one study wave, 206 completed actigraphy for at least two waves, and 101 completed actigraphy for all three waves. During data collection periods, adolescents, on average, wore the watch for 6.19 nights. Descriptive statistics on sleep parameters are reported in Table 1.

Sleep Duration (in Minutes)

Nightly sleep duration was computed as the total amount of time spent in bed that was scored as sleep for each 8 days of sleep assessment. Sleep duration was averaged across the 8 days to determine mean weekly sleep duration.

Sleep Latency (in Minutes)

Time to fall asleep (latency) was computed as the minutes between the start of time in bed (as determined by event marker press) and sleep onset (as determined by actigraphy scoring), representing the time it took for adolescents to fall asleep.

Daily Sleep Variability (in Minutes)

Variability was computed to represent daily-level variations in sleep duration. To do so, we computed the absolute value of the difference between each individual day's sleep duration and the average sleep duration for the week.

Salivary Cortisol

At each study wave, adolescents collected five saliva samples using Salivettes (Sarstedt, Nümbrecht, Germany) at designated time points (wake, 15 minutes after wake, 30 minutes after wake, dinner, and bedtime) for 3 consecutive days. Text messages were sent before each collection time to encourage compliance. Adolescents were asked to refrain from drinking, brushing teeth, or using tobacco products 30 minutes before saliva collection. Participants used electronic time stampers (Dymo Corporation, Stamford, Connecticut) and stamping booklets to record sampling time and completion. Samples were stored in adolescents' household refrigerators until they were picked up by research staff 1 to 3 weeks later. Research staff stored the collected samples at -80°C before assay. Saliva samples were shipped to the Laboratory of Biological Psychology at the Technical University of Dresden, Germany, where they were assayed for cortisol concentrations (in nanomoles per liter) using high-sensitivity chemiluminescence immunoassays (IBL International, Hamburg, Germany). In the first wave

of data collection, 308 adolescents collected saliva during the 3-day period. In the following two waves, 221 and 158 adolescents, respectively, collected saliva during the 3-day study period. Adolescents collected an average of 4.63 of the five daily saliva samples at each wave.

We computed three indices of HPA axis functioning: diurnal cortisol slope, the CAR, and AUC. Daily-level slope, CAR, and AUC values were averaged across the 3 days to characterize the typical diurnal patterns of adolescents and minimize the influence of daily-level fluctuations in cortisol levels. Sample values greater than 60 nmol/L (2.1% of the total samples provided) were removed from the sample to reduce the impact of outliers on diurnal profiles.

Diurnal Slope

Diurnal slope was computed by subtracting the bedtime cortisol concentration from the waking cortisol concentration and dividing the value by the hours passed between the wake and bed samples (35,36). Average diurnal slope was computed during at least one study wave for $n = 334$ adolescents, during at least two waves for $n = 216$ adolescents, and $n = 110$ for all three waves.

Cortisol Awakening Response

The CAR was computed by subtracting the salivary cortisol concentration from the sample taken 30 minutes after wake from the cortisol concentration of the wake sample. The CAR can be affected by the timing of saliva collection (37), and so daily-level CAR values were flagged where the wake and +30-minute sample were taken <15 minutes apart ($n = 25$) or >60 minutes apart ($n = 24$). As a sensitivity check, all analyses with the CAR were repeated wherein flagged wake and +30-minute samples were removed from the data set. Average CAR was computed during at least one study wave for $n = 343$ adolescents, during at least two waves for $n = 221$ adolescents, and $n = 119$ for all three waves.

Area Under the Curve

To measure total cortisol output, we computed AUC with respect to ground using the trapezoid equation established by Pruessner and colleagues (38). Daily-level AUC was computed for each day across the 3-day period where participants collected all five saliva samples. Average AUC was computed during at least one study wave for $n = 328$ adolescents, during at least two waves for $n = 208$ adolescents, and $n = 94$ for all three waves.

Analytic Strategy

Using STATA 16.1, multilevel models were run to estimate the associations between SES variables and sleep. Study waves (level 1) were nested within participants (level 2). These models allowed for the inclusion of adolescents with missing data, such that adolescents were included in the analytic data set if they participated in at least one wave of data collection. Individual observations within adolescents were dropped if missing information on age, sex, ethnicity, immigration status, or sleep actigraphy data.

To assess the unique contribution of each SES characteristic on sleep outcomes, each multilevel model included all three SES variables: parental education, family INR, and parental EH. Parental education (a level 2 or person-varying SES variable, given the lack of change in parental education over time) was grand-mean centered. Family INR and parental EH (level 1 or wave-varying SES variables) were also grand-mean centered, pooling the model estimates across within- and between-person associations. Separate multilevel models were run for each sleep outcome variable: duration, latency, and variability. Model covariates included age, sex (reference = female), ethnicity (reference = Latinx), parents' immigration status (reference = US born), and average wake time (in minutes). Age was centered around the youngest age in the sample (14.5). Grand-mean centered sleep duration was added as an additional covariate for the model examining sleep variability as the dependent variable, given the associations between less sleep duration and greater variability (39). The final analytical sample for the multilevel models included $n = 585$ observations from 311 adolescents.

Next, we repeated analyses with the addition of three SES-by-age interaction terms to the model (one for each SES predictor variable) to investigate whether the relationship between SES and sleep varied as a function of age.

Finally, for models that demonstrated a significant association between SES and sleep, we estimated mediation by indices of HPA axis functioning. Separate mediation models were run to examine mediation by diurnal slope, the CAR, and AUC. The framework outlined by Krull and colleagues (40) was used to run multilevel mediation models. Multilevel estimation offers an advantage over single-level procedures for assessing mediation when there are nested data (41). Confidence intervals (CIs) for the indirect effects were obtained using percentile-based bootstrapping with 10,000 replications. Model covariates

TABLE 2. Bivariate Correlations Between Key Study Variables, as Measured at Study Entry

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. Age												
2. Female	0											
3. US-born parent	-0.04	-0.19**										
4. Parent education	-0.13*	-0.11*	0.44**									
5. Family INR	0.03	-0.06	0.39**	0.49**								
6. Parental EH	0.02	0.08	-0.20**	-0.28**	-0.43**							
7. CAR	-0.17**	0.19**	0.01	0.05	-0.04	-0.10						
8. Diurnal Slope	-0.13*	-0.05	-0.06	0.09	-0.05	-0.07	0.44**					
9. AUC	0.06	0.21**	0.11	0.01	0.05	-0.09	0.51**	-0.18**				
10. Sleep duration	-0.16**	0.16*	0.05	0.01	0.08	0.04	0.05	-0.09	-0.12			
11. Sleep latency	-0.10	-0.06	-0.11	-0.12*	-0.08	0.05	-0.14*	-0.12*	-0.06	-0.06		
12. Sleep variability	0.04	0.09	0.05	0.03	0	-0.03	0.02	0.12*	-0.08	-0.11	0.05	
13. Wake time	-0.02	0.03	-0.07	0.03	0.03	0.01	0.07	0.07	-0.11	0.38**	0.15*	0.31**

INR = income-to-needs ratio; EH = economic hardship; CAR = cortisol awakening response; AUC = area under the curve.

Results of bivariate correlations between study variables.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

TABLE 3. Multilevel Associations Between SES and Sleep

Variable	Duration, <i>b</i> (SE)	Latency, <i>b</i> (SE)	Daily Variability, <i>b</i> (SE)
Intercept	495.01 (7.54)***	11.80 (1.38)***	71.04 (3.93)***
Age	−12.21 (1.46)***	−0.10 (0.27)	−1.14 (0.82)
Parent EH	4.53 (3.64)	0.57 (0.67)	0.44 (1.78)
Family INR	2.90 (1.28)*	0.12 (0.23)	0.12 (0.62)
Parent education	−3.70 (1.75)*	−0.74 (0.30)*	−2.06 (0.80)**
Male	−24.07 (5.38)***	0.60 (0.97)	−7.86 (2.56)**
Asian American	−18.55 (7.89)*	−1.41 (1.43)	3.88 (3.71)
European American	9.78 (7.05)	−2.92 (1.28)*	−1.76 (3.29)
Other ethnicity	7.43 (11.80)	2.23 (2.13)	−5.18 (5.47)
Immigrant parent	−2.21 (7.14)	−0.04 (1.28)	−9.82 (3.28)**
Wake time	0.34 (0.03)***	0.02 (0.01)**	0.13 (0.02)***

SES = socioeconomic status; SE = standard error; EH = economic hardship; INR = income-to-needs ratio.

Results of multilevel models predicting sleep outcomes from SES indices, controlling for age, sex, ethnicity, parent immigration, and wake time. Age was centered at 14.5 years. Parent EH, family INR, parental education, and wake time were grand-mean centered. Reference variables were female, Latinx, and adolescents of US-born parents.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

remained the same as mentioned previously. The final analytical sample for the mediation models included $n = 562$ observations from 311 adolescents.

RESULTS

Descriptive Statistics

Table 1 presents descriptive statistics for age and socioeconomic information by study wave, as well as descriptive statistics for sleep and diurnal cortisol variables. Across study waves, adolescents' average sleep duration decreased and their daily sleep variability became greater (Table 1). Bivariate correlation coefficients between key study variables at study entry are presented in Table 2. Parental education was associated with lower sleep latency ($r(291) = -0.12, p = .04$). None of the other indicators of SES were associated with sleep parameters in a bivariate manner.

SES-Sleep Associations

As shown in Table 3, multilevel models indicated that a higher family INR was significantly associated with longer adolescent

sleep duration ($b = 2.90, p = .023$), but not latency or variability. In contrast, greater parental education was significantly associated with shorter sleep duration ($b = -3.70, p = .030$), shorter sleep latency ($b = -0.74, p = .016$), and less daily variability in duration ($b = -2.06, p = .010$), the last when additionally controlling for average sleep duration. Lastly, parental EH was not associated with adolescent sleep, and follow-up analyses suggested that associations between SES sleep parameters did not vary by age.

Mediation Analyses

Parental Education

The observed associations of parent education with adolescent sleep duration and sleep variability were tested for mediation by diurnal cortisol indices. As shown in Figure 1, mediation analyses suggested that greater parental education was significantly associated with a flatter (less negative) diurnal cortisol slope ($b = 0.05, p = .008$). A flatter diurnal slope, in turn, was associated with shorter sleep duration ($b = -9.74, p = .009$), and the indirect effect was significant ($b = -0.48, 95\% \text{ CI} =$

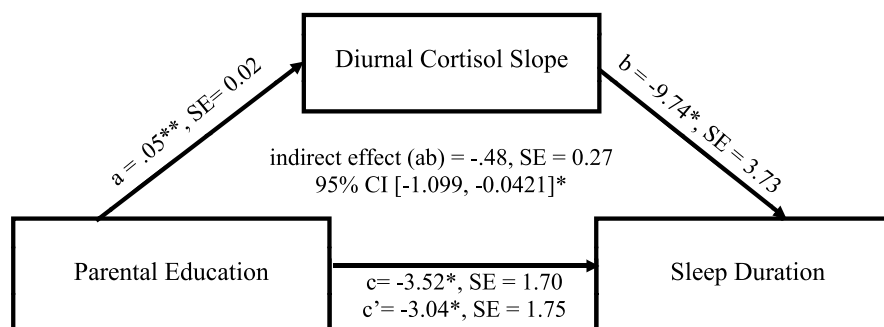


FIGURE 1. Parental education and sleep duration: mediation by diurnal cortisol slope. Mediation pathway of the association between parent education and sleep duration. Models control for wake time, age, sex, ethnicity, family INR, parent EH, and parent immigration. β Coefficients are provided with standard errors. Significant indirect, direct, and total pathways are denoted with an asterisk if the bootstrapped confidence interval of the β coefficient did not include zero. INR = income-to-needs ratio; EH = economic hardship; CI = confidence interval; SE = standard error.

−1.099 to −0.042), suggesting that parental education was related to shorter sleep duration indirectly via a flatter diurnal slope. However, diurnal cortisol slope was not a significant mediator for the association of parental education with sleep variability ($b = 0.24$, 95% CI = −0.006 to 0.567) or latency ($b = -0.05$, 95% CI = −0.140 to 0.016). In addition, neither the CAR nor AUC was associated with parent education, and neither emerged as significant statistical mediators.

Income-to-Needs Ratio

A greater family INR was significantly associated with a steeper (more negative) diurnal cortisol slope ($b = -0.03$, $p = .033$). However, the indirect effect was nonsignificant, indicating that diurnal cortisol slope did not mediate the association between family INR and sleep duration ($b = 0.29$, 95% CI = −0.005 to 0.711). Neither the CAR nor AUC was associated with family INR, and neither statistically mediated the association between family INR and sleep duration.

DISCUSSION

The present study examined the association between SES and sleep and the mediating role of HPA axis functioning in a longitudinal study of adolescents. Study results suggest that the relationship between SES and sleep during adolescence is complex. As hypothesized, higher family INR was associated with greater minutes of sleep duration. Greater parental education was associated with less sleep variability and shorter sleep latency. Contrary to our hypothesis, parental education was also associated with fewer minutes of sleep duration. Finally, evidence was found supporting HPA axis functioning, particularly a flatter (less negative) diurnal cortisol slope, as a statistical mediator of the high-parental education association with shorter sleep duration. In contrast, diurnal cortisol slope was not found to statistically mediate the association between family INR and sleep duration.

Although the association of greater parental education with shorter sleep is inconsistent with findings from a large previous epidemiologic study in adolescents (11), that study relied on a less precise measure of duration (i.e., self-reported frequency of receiving <7 hours of sleep per night). In contrast, actigraphy-based studies in adolescent populations have not found an association between parental education and sleep duration (11,42). Our finding that parental education is associated with less sleep variability is consistent with actigraphy-based work (11), suggesting that youth with more highly educated parents may have more consistent bedtime routines. Although less variability in nightly sleep duration is generally thought to be a positive sleep outcome (43), it may also indicate that these youth are sleeping less on a consistent basis. Likewise, the observed association of parental education with shorter sleep latency may reflect greater feelings of sleepiness for those with more highly educated parents (16). Taken together, these findings suggest that higher parental education may be related to insufficient sleep during adolescence. Information on the types of activities youth engage in instead of sleeping can help clarify why higher parental education might be associated with less sleep. School or academic-related obligations are common contributors to insufficient sleep during adolescence (44). Enrollment in advanced coursework may vary by SES and has been associated with reduced sleep among high-school aged adolescents (45). Higher SES youth may also be more likely to participate in extracurricular activities (46), and these obligations can similarly

come at the cost of sleep (47). Future research should consider collecting information on academic and extracurricular obligations to assess whether they may explain parental education associations with sleep.

Notably, we also found that HPA axis functioning, as indicated by a flatter diurnal slope, mediated the association between higher parental education and shorter sleep duration. A potential interpretation of this finding could be that chronic stressors associated with higher parental education are able to indirectly influence sleep through dysregulation of the stress-response system. Although research on the relationship between parental education and diurnal cortisol in adolescence is limited, a recent study of first-year college students found that students whose parents had an academic degree had higher levels of hair cortisol concentrations than those whose parents did not have an academic degree (48). The authors of the study suggested that youth with educated parents may feel greater pressure to succeed academically, potentially pursuing academic studies that are too difficult for them. High academic demands in turn can contribute to youths' feelings of stress (49), which may be reflected in diurnal cortisol rhythms. However, this study does not have sufficient data to confirm whether parental education was associated with differences in academic demands or participation in advanced coursework. Further research is needed to examine the relationship between academic-related stressors and diurnal cortisol during adolescence and assess if these factors may explain part of the association between parental education and sleep behavior.

The relationship between income-based measures of SES and sleep duration during adolescence is somewhat inconsistent in previous research, with studies showing a positive association, no association, or a positive association only for children but not adolescents (9,11,12,42). Relatively few studies have examined the relationship between income and actigraphy-based measures of sleep during adolescence using actigraphy (10,11,13,42), and our study contributes evidence to the literature suggesting that family financial resources may impact how long adolescents are sleeping. In terms of HPA activity, we did find a significant association between family INR and diurnal cortisol slope, such that higher family INR was related to a steeper decline in cortisol levels across the day. This finding is consistent with previous literature and may reflect a theory that youth with fewer economic resources show a more blunted pattern of diurnal cortisol secretion as a result of exposure to chronic stressors. Diurnal cortisol, however, did not explain the association between family INR and sleep duration, suggesting the role of other factors. Household stressors, such as family chaos, can potentially explain part of the association between income and sleep quality in adolescents (18). Feelings of stress or worries that occur before bedtime have also been shown to disrupt low-SES children's sleep (13). Alternatively, aspects of the home sleep environment, such as noise and light levels, could be stressors that contribute to low-income youths' sleep problems (13,50).

The present study had several strengths. To our knowledge, this is the first study to assess associations of multiple measures of SES simultaneously with sleep and cortisol during adolescence. This study provides a step forward in understanding physiologic systems that may underlie SES differences in sleep by providing evidence that altered cortisol secretion may explain part of the association between parental education and sleep. Next, this study benefits from its use of sleep actigraphy, which estimates sleep from

participants' movement. Much of the literature on SES disparities in adolescent sleep has relied on subjective or self-reported sleep (8,12,18), which is often only loosely correlated with objectively measured sleep behavior (51). Actigraphy-based sleep assessment eliminates the potential reporting bias of survey-based measures of sleep and provides a minimally invasive alternative to the criterion standard of sleep assessment, polysomnography. Finally, the broad age span of the study allowed for an examination of the association between SES and sleep over the transition from adolescence to young adulthood, an understudied developmental period.

The study also had several limitations. Study results may be affected by attrition bias as higher-SES adolescents (i.e., those with higher family incomes and greater parental education) were more likely to complete all study waves. Further completion differences by sex, ethnicity, and parent immigration status may have also affected results and might limit generalizability of findings. In addition, all measures of SES used in this study were parent reported, which may not encompass adolescents' perceptions of their SES. Previous research suggests that adolescent-perceived financial strain is associated with physical and mental health outcomes even after controlling for parent SES measures (52). The lack of information regarding youths' perceived EH is a limitation of this study and may provide context for the null associations of parent EH with adolescent sleep behavior. Future research assessing socioeconomic differences during adolescence should consider including questions regarding youths' perceptions of EH, which may offer additional insight into the potentially complex relationship that SES has with sleep during this developmental period. Finally, and importantly, our study design cannot establish causal relationships between SES, HPA axis activity, and sleep. Previous research suggests that adolescents moving into poverty may be at risk for shorter sleep duration, whereas those maintaining their poverty status do not show changes in sleep (53). Additional longitudinal research following youth from childhood into adulthood is needed to capture SES trajectories and examine whether HPA axis dysregulation is an underlying mechanism linking changes in SES with sleep disruption.

CONCLUSIONS

This study finds evidence that differing measures of SES have divergent relationships with sleep during late adolescence. Consistent with previously observed patterns, family INR was associated with longer sleep duration. By contrast, parental education was associated with shorter sleep duration, shorter sleep latency, and less sleep variability. Findings suggested that parental education was linked with sleep duration via flatter diurnal cortisol slopes. As such, parental education may be reflective a complex system of behavioral or microcultural processes that induce distinctive patterns of stress in adolescence that manifest as disturbances in sleep and HPA axis functioning. These results highlight the importance of assessing multiple SES indicators when studying sleep behavior, with particular care for how indicators might operate distinctively in the lives of adolescents versus adults.

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