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Childhood Socioeconomic Status and Race are Associated with Adult Sleep

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Abstract

Race and current socioeconomic status (SES) are associated with sleep. Parental education, a commonly studied component of childhood SES, is predictive of adult health outcomes; yet its impact on adult sleep remains unclear. In this study, the sleep of 128 Black and White adults was investigated. Participants with lower childhood SES (assessed via parental education) slept more time in Stage 2 sleep and less time in slow wave sleep (SWS) than those with higher childhood SES. Additionally, women from low childhood SES backgrounds took longer to fall asleep than women from high SES backgrounds. Black participants slept less time in SWS than their White counterparts and an age by race interaction was detected in the prediction of subjective sleep quality. Results were not mediated via current SES or health practices.

Introduction

Knowledge of factors that promote and detract from optimal sleep is essential in gaining a better understanding of health. Socioeconomic status (SES) refers to one's hierarchical economic position, measured via income, education, and occupation. Each step down in SES confers a host of associated health risks (Adler & Ostrove, 1999; Cohen, Janicki-Deverts, Chen, & Matthews, 2010). An accumulating body of work suggests that SES is related to sleep such that as SES decreases, so does self reported sleep quality (Friedman et al., 2007; Gellis et al., 2005; Mezick et al., 2008; Moore, Adler, Williams, & Jackson, 2002). SES is also related to PSG-assessed sleep. Older, low SES, women have longer sleep onset latency (SOL) and lower sleep efficiency (SE) than their high SES counterparts (Friedman et al., 2007). Low SES has also been associated with longer SOL and more time spent awake after sleep onset in a sample of middle aged Black and White Americans (Mezick et al., 2008). Finally, financial strain has been associated with lower SE in a group of middle aged White, Black, and Chinese women (Hall et al., 2009).

Childhood SES is a potent predictor of adult physical health, one whose influence extends beyond its association with adult social position (Cohen et al., 2010; Galobardes, Lynch, & Davey Smith, 2004; Galobardes, Lynch, & Smith, 2008). Individuals with lower childhood SES are at an elevated risk of suffering from numerous diseases as well as all-cause and specific mortality (Galobardes et al., 2004; Galobardes et al., 2008). Parental education, a commonly measured component of childhood SES, is also associated with adult health outcomes (Lehman et al., 2009; Lehman, Taylor, Kiefe, & Seeman, 2009; Phillips et al., 2009; Thurston &

Matthews, 2009). Despite the link between childhood SES and adult health, the potential relationship between childhood SES and adult sleep has not been examined.

Race is also related to sleep outcomes. A recent review suggests that Black Americans experience poorer sleep quality than White Americans (Durrence & Lichstein, 2006). Research consistently shows that Blacks spend a smaller proportion of total sleep time (TST) in slow wave sleep (SWS) each night (Hall et al., 2009; Mezick et al., 2008; Profant, Ancoli-Israel, & Dimsdale, 2002; Redline et al., 2004; Thomas, Bardwell, Ancoli-Israel, & Dimsdale, 2006). Some work has also demonstrated that Blacks take longer to fall asleep, have poorer SE and report worse sleep quality than Whites (Durrence & Lichstein, 2006). Race and adult SES independently contribute to sleep outcomes (Hall et al., 2009; Mezick et al., 2008); still, it remains unknown whether childhood SES plays a part in the relationship between race and sleep.

Differences in adult health behaviors present a potential pathway between childhood SES, race and sleep. Some work suggests that growing up in a low SES environment is associated with an increased likelihood of smoking, higher risk of substance abuse, lower physical activity and higher body mass index (BMI) (Cohen et al., 2010). In addition to links with adverse health outcomes, smoking, substance use, physical activity and BMI are also associated with poor sleep (Dworak et al., 2008; Feige et al., 2006; Rao et al., 2009; Santos, Tufik, & De Mello, 2007; Vorona et al., 2005; Zhang, Samet, Caffo, & Punjabi, 2006).

The objectives of this study were three fold. First, we examined relationships between childhood SES (assessed via highest level of parental education) and adult sleep. We hypothesized that relationships between childhood SES and sleep would be similar to those between adult SES and sleep (e.g., that lower childhood SES would be associated with increased difficulties with sleep initiation and reductions in sleep continuity). We next examined differences between race and adult sleep. We hypothesized that in line with previous findings, Black participants would sleep less time in SWS than their White counterparts. Finally, we explored whether associations between childhood SES, race and sleep were mediated by factors such as current health practices and current social status.

Methods

Subjects were recruited from the local San Diego, California area to participate in a larger study investigating differences in stress and sympathetic nervous system functioning between Black and White Americans. The current investigation was an archival analysis of this data, which was not designed primarily as a sleep study. Recruitment occurred through local papers, online advertisements, community flyers, participation in health fairs and word-of-mouth. Individuals not enrolled were either outside of the inclusion criteria (see below) or could not be excused from work to participate. Exclusion criteria included: diagnosis of and/or ongoing treatment for any clinical illness other than hypertension (e.g., asthma, diabetes), BP $\geq 170/105$ mm Hg, current substance abuse, previous diagnosis of a sleep disorder, or a history of psychosis. Participants were also excluded if they were pregnant or were taking any prescription medication (including hormonal contraception or hormone replacement therapy). Three hypertensive patients were accepted into the study after being weaned off their antihypertensive medications by the study physician and maintaining BP $>140/90$ but $< 170/105$ mm Hg for 3 weeks. All participants in the study reported working ≥ 20 hours/week. This criterion was implemented because we were interested in a wide range of SES. Focusing on only those individuals who are unemployed restricts this range. The project was approved by the Institutional Review Board of the University of California San Diego (UCSD).

Procedure

Once written informed consent was obtained, participants met with a study physician to undergo a physical exam and answer questions about their medical history. If study criteria were met, sleep monitoring was conducted with standard polysomnography (PSG) and occurred over two consecutive nights at the UCSD General Clinical Research Center Gillin Laboratory of Sleep and Chronobiology (GCRC-GLSC). No participants reported taking prescriptions medications during the study and we requested that any over the counter medications, e.g., Tylenol, be suspended for 24 hours prior to admission. Participants were admitted to the GCRC-GLSC at 16:00 hrs on the first night of monitoring. On each night, PSG set-up began at 21:00 hrs and lights off occurred between 22:00 and 24:00 hrs. Each morning participants were awakened at 06:00 hrs and PSG recording equipment was removed. The first night of monitoring served as an acclimation night and data from the second night of sleep recording are the focus of this study. Psychosocial questionnaires were given to participants upon admission to the GCRC-GLSC and were collected prior to discharge.

Measures

Socioeconomic Status

Childhood Socioeconomic Status: Childhood SES status was assessed by querying participants as to the highest level of education attained by each parent. The variable was dichotomously coded with participants classified as having lower childhood SES if neither parent achieved education beyond high school and as having a higher SES if either parent achieved some education beyond high school.

Adult Socioeconomic Status: We assessed current SES with the clinician-rated, Hollingshead 2-Factor Index of Social Position. This scale assesses highest level of formal education attained and current occupation. The two factors are summed, weighted and combined into a continuous measure of social index. Scores range from 11 to 77, with lower social index scores indicating higher SES (Hollingshead, 1957).

Current subjective social status (SSS) was assessed using the MacArthur Scale of SSS, a 10 rung ladder scale. Participants were asked to rate their place on the scale in relation to those who are the best and worst off in terms of money, education and respected jobs in the United States. Lower scores are indicative of lower SSS (Adler, Epel, Castellazzo, & Ickovics, 2000).

Sleep Variables

Pittsburgh Sleep Quality Index (PSQI): Subjective sleep quality was assessed with the Pittsburgh Sleep Quality Index (PSQI). The PSQI is a 19-item self-report questionnaire that assesses habitual sleep quality. PSQI global scores ranges from 0 - 21. Higher scores are indicative of worse sleep quality, scores above 5 suggest poor sleep quality (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989).

Epworth Sleepiness Scale (ESS): Daytime sleepiness was assessed with the Epworth Sleepiness Scale (ESS). The eight items on the questionnaire assess the likelihood of falling asleep in everyday situations. Higher scores are indicative of greater daytime sleepiness, with scores above 10 suggesting pathological daytime sleepiness (Johns, 1991).

Sleep Monitoring: Sleep data acquisition was performed using the Embla A10 polysomnography system (Embla Systems Inc. Broomfield, CO USA). We assessed: Electroencephalography (C4/C3), electrooculography, chin electromyography, leg electromyography, airflow with an oronasal thermal sensor, airflow with an air pressure

transducer, snore events with a piezo snore sensor, respiratory effort with piezo thoracic and abdominal belts, oxygen saturation with a pulse oximeter sensor, and body position with a position indicator.

Records were hand scored by experienced polysomnographic technologists using the criteria of Rechtschaffen and Kales (1968) and Somnologica software (Embla, Broomfield, CO). Records were scored for TST, sleep onset latency (SOL), sleep efficiency (SE), wake after sleep onset (WASO) and percentage of Stages 1, Stage 2, SWS (composed of Stages 3 and 4 sleep) and REM sleep. TST was considered the total time asleep (in minutes) minus any waking time and was the sum of all time spent in Stages 1, 2, SWS and REM. SOL was defined as the time from lights out to the first epoch of Stage 2 sleep. SE was calculated as $(TST \times 100 / \text{time in bed})$. The percentages of Stage 1, Stage 2, SWS and REM were calculated at the percentage TST spent in each stage. WASO was defined as $(\text{minutes awake after sleep onset} / TST)$.

Sociodemographic Information—Information about gender, racial identity, age, and years of education, occupation and income were collected via self-report. BMI was calculated from height and weight measurements (to the nearest 0.1 kg and 0.1 cm) taken during the physical exam. BMI was calculated as the ratio of body weight in kilograms divided by square height in meters (kg/m^2).

Health Practices—Participants were classified as smokers if they reported smoking cigarettes daily. Alcohol consumption was defined as the reported average number of alcoholic drinks consumed per week. Physical activity was measured with the Leisure Time Exercise Questionnaire (LTEQ). The LTEQ is a self-administered questionnaire which assesses regular physical activity. A total score is calculated by multiplying the frequency of weekly exercise by differing intensities, strenuous exercise is multiplied by nine, moderate exercise by five, and mild exercise by three (Godin & Shephard, 1985). The total score has been shown to be a valid (Godin & Shephard, 1985; Godin, Jobin, & Bouillon, 1986; Miller, Freedson, & Kline, 1994) and reliable (Sallis, Buono, Roby, Micale, & Nelson, 1993) estimate of regular physical activity; higher scores reflect more regular physical activity.

Results

Participants

The final sample consisted of 128 participants; approximately 43% were women, 42% self identified as Black and 58% as White. Participants were between the age of 18 and 52 years. The mean years of education completed in the sample was 14.8 (SD = 2.2) years. The average PSQI score in the sample was above 5, indicating that some participants were experiencing sleep difficulties. One-way analyses of variance or χ^2 were conducted to test for group equivalence. Subjects in the high childhood SES were significantly younger, more likely to be White and have higher current SES than those in the low childhood SES group. Smoking status, BMI, weekly alcohol consumption and exercise did not differ as a function of childhood SES. Black participants were significantly older, had higher BMI's and were more likely to come from low childhood SES backgrounds than White participants. In addition, Black participants were more likely to smoke, reported less regular exercise and had lower current SES than their White counterparts. White participants reported consuming more alcoholic beverages weekly. See Table 1. Variables that differed between groups were statistically controlled for in later analyses.

MANOVA Findings

In order to control for Type I error inflation associated with investigating multiple outcomes, we first conducted two multivariate analyses of variance (MANOVA) tests with (1) race (Black

vs White) and (2) childhood SES (low vs high) predicting a joint multivariate outcome defined by the 8 PSG-assessed sleep variables described above. Hotelling's Trace (a statistic used to assess mean differences between two groups on a multivariate outcome) from these models suggested a significant relationship between race ($F = 4.19$, Hotelling's Trace P value < 0.01) and childhood SES ($F = 3.41$, Hotelling's Trace P value < 0.01) with sleep.

We next conducted a series of ANCOVAs to assess differences between (1) childhood SES, (2) race and individual sleep measures, controlling for covariates. No violations of assumptions of normality, linearity or homoscedasticity were detected. In two cases, violations of the homogeneity of regression of slopes (whereby covariates do not interact with predictor variables) were detected; the interactions were interpreted separately and are presented in the results. The variables Stage 1 sleep, SOL, SE, WASO, exercise and alcohol consumption were log transformed to obtain a normal distribution.

Is Childhood SES associated with Adult Sleep?

The covariates age, BMI, gender and race (Ancoli-Israel, Ayalon, & Salzman, 2008; Rao et al., 2009; Redline et al., 2004; Vorona et al., 2005) were included as covariates because group differences were detected and/or previous work has demonstrated their relationship to measures of sleep quality.

Neither subjective sleep (PSQI Global Score) nor daytime sleepiness (ESS) significantly differed between childhood SES group. Childhood SES was associated with sleep architecture, predicting percentage of time spent in Stage 2 sleep ($F [1, 122] = 6.74$, $p < 0.05$, $\eta^2 = 0.05$) and SWS ($F [1, 122] = 8.92$, $p < 0.01$, $\eta^2 = 0.07$). Holding covariates constant, individuals with lower childhood SES slept more TST in Stage 2 sleep (53.7%; $CI_{.95} = 51.2, 56.2$ vs. 49.5%; $CI_{.95} = 47.8, 51.3$) and less time in SWS (17.7%; $CI_{.95} = 15.0, 20.4$ vs. 22.8%; $CI_{.95} = 20.9, 24.6$) than participants from higher childhood SES backgrounds. See Table 2.

A gender by childhood SES interaction was detected when investigating the outcome, SOL ($F [1, 121] = 5.21$, $p < 0.05$, $\eta^2 = 0.04$), thus violating the homogeneity of the regression assumption in ANCOVA. We investigated the interaction by conducting separate ANCOVA analyses by gender. Estimated marginal means showed that women from low childhood SES backgrounds took 17.61 ($CI_{.95} = 12.3, 22.9$) minutes to fall asleep each night, approximately 10 minutes longer than women from the high childhood SES group who fell asleep in an average of 7.32 ($CI_{.95} = 3.53, 11.1$) minutes ($F [1, 50] = 8.09$, $p < 0.01$, $\eta^2 = 0.14$). Significant SOL differences were not observed in men from different childhood SES backgrounds ($F [1, 68] = 0.35$, $p = 0.56$, $\eta^2 < 0.01$).

TST, SE, WASO, Stage 1 sleep and REM sleep did not significantly differ between childhood SES groups. See Table 2.

Is Race associated with Sleep?

Next, we examined relationship between race and sleep. In order to isolate the specific effect of race on sleep, we included childhood SES as an additional covariate in these analyses. Black participants endorsed significantly worse subjective sleep than Whites. However, race significantly interacted with the covariate age in the prediction of subjective sleep ($F [1, 119] = 6.63$, $p < 0.05$, $\eta^2 = 0.05$), violating the homogeneity of the regression assumption. To further investigate the interaction, separate regression analyses were conducted by race with age predicting PSQI Global scores. We found that age significantly predicted PSQI Global scores in White ($B = 0.08$, $SE = 0.03$, $t = 2.46$, $p = 0.02$, $\Delta R^2 = 0.08$), but not in Black participants ($B = -0.08$, $SE = 0.06$, $t = -1.29$, $p = 0.20$, $\Delta R^2 = 0.03$). Black participants had significantly higher PSQI Global scores (indicating worse subjective sleep) than Whites at younger ages but

PSQI scores were approximately equivalent in the two racial groups in the age range of 45 – 50 years old.

Race was also associated with SWS ($F [1, 122] = 6.63, p = 0.01, \eta^2 = 0.05$). Black participants slept less time in SWS (18.5%; $CI_{.95} = 16.0, 21.0$ vs. 23.0%; $CI_{.95} = 20.9, 25.1$) than their White counterparts.

TST, Stage 1 sleep, Stage 2 sleep, REM sleep, SOL, WASO, and SE were not significantly different between races. See Table 2.

Are Health Practices and Current SES Mediators of Observed Associations?

Having observed that race and childhood SES were associated with aspects of sleep, we next examined potential pathways of the association. We acknowledge that a study design such as this cannot decisively examine mediation processes (experimental manipulation would be required to do this). However, these analyses can provide an indication as to whether or not covariates related to race, childhood SES and sleep are associated in a way consistent with mediation. Mediation (M) examines the way in which an independent variable, X , e.g., Race affects a dependent variable, Y , e.g., SWS, through one or more potential intermediates (i.e., health practices) (Baron & Kenny, 1986). Mediation was assessed using a bootstrapped (5000 times) multivariate test of mediation, which allows for the consideration of covariates as well as the simultaneous testing of multiple mediators (Preacher & Hayes, 2004; 2008). The procedure parses apart the total effect - the effect of the X on Y not considering mediators - into (1) the direct effect - the effect of the X on the Y controlling for mediators - and (2) the indirect effect - the effect of X on Y (through M).

Potential mediators included exercise, smoking, alcohol consumption and current SES (assessed with the Hollingshead and the MacArthur scale of SSS). Covariates included in the previous ANCOVA analyses were controlled for in each model.

Childhood SES—Childhood SES and sleep relationships were not mediated by health practices or current SES. The direct effects of childhood SES on adult SWS ($\beta = 0.05, SE = 0.02, t = 2.74, p < 0.01$), Stage 2 sleep ($\beta = -0.05, SE = 0.02, t = -2.80, p < 0.01$) and the childhood SES, gender interaction in the prediction of SOL ($\beta = -0.37, SE = 0.17, t = -2.09, p = 0.04$) were significant after indirect effects were considered. The total indirect effects of mediators were not significant in the above models, nor were significant individual mediators detected (p 's > 0.05),

Race—Here again, potential mediators did not account for the relationships between race and sleep. The direct effects of race on SWS ($\beta = 0.02, SE = 0.01, t = 2.90, p < 0.01$), and the race, age interaction in the prediction of PSQI global scores ($\beta = 0.08, SE = 0.03, t = 2.75, p < 0.01$) were significant after indirect effects were considered. Again, the total indirect effects of mediators were not significant in the above models, nor were significant individual mediators detected (p 's > 0.05),

Discussion

Our findings revealed that childhood SES (defined as parental education of high school and below vs. high school and beyond) was associated with significant differences in time slept in Stage 2 and SWS. Adult participants with lower childhood SES slept more time in Stage 2 sleep and less time in SWS than their high childhood SES counterparts. Additionally, an interaction between gender and childhood SES was detected whereby women with lower childhood SES took longer to fall asleep each night than women with higher childhood SES. Contrary to our expectations childhood SES was not associated with sleep maintenance

throughout the night. Racial factors also exerted a prominent effect on sleep. In line with our hypothesis, Black participants in our sample slept less time in SWS each night than their White counterparts. Additionally, race interacted with age in the reporting of subjective sleep problems. Young Black participants had worse subjective sleep than young White participants; however with increases in age, the amount of subjective sleep problems reported by Black participants did not change, whereas older white participants reported more sleep problems than younger white participants. Findings persisted after controlling for demographic information (age, BMI, race, gender) and were not mediated by health practices (smoking, alcohol consumption, exercise) or current SES. Results suggest that both childhood SES and race are important factors to consider when investigating adult sleep outcomes.

In our sample, Black participants and those with lower childhood SES slept approximately 20% less time in SWS each night than White participants and those with higher childhood SES. SWS is associated with a number of advantageous physiological outcomes including reduced heart rate, lowered BP, and altered sympathetic nervous system activity (Dijk, 2008; Van Cauter et al., 2008). Short-term suppression of SWS (but not TST) in healthy adults leads to reduced insulin sensitivity, increased risk of type 2 diabetes and decreased BP dipping (Dijk, 2008; Sayk et al., 2010; Tasali, Leproult, Ehrmann, & Van Cauter, 2008). These studies point to the importance of SWS in nocturnal physiological functioning. The health impact of the SWS disparities between racial and childhood SES groups deserves further attention.

Neither health practices, nor measures of current socioeconomic status accounted for the relationship between childhood SES, race and adult sleep, leaving questions regarding mechanisms behind the associations unanswered. The possibility exists that self-report of health practices did not accurately assess participants 'actual' exercise levels, smoking, and alcohol consumption. Another potential explanation is that differences in exposure to and appraisal of stress underlie the findings. Children from low SES environments are exposed to more life stressors and tend to appraise those stressors as more severe than children from higher SES circumstances (Chen, 2004). Exposure to stressful childhood circumstances can lead to disrupted emotion regulation (e.g., higher anxiety, depression and hostility) in adulthood (Cohen, et al., 2010) which could in turn influence adult sleep. Additionally, Black Americans are exposed to more stressors (e.g., discrimination) than their White counterparts. In fact, one study has shown that the experience of ethnic discrimination mediated racial differences in Stage 4 sleep (Thomas et al., 2006). Stress exposure throughout the lifespan should be considered as a potential pathway linking childhood SES, race and adult sleep (Akerstedt, 2006; Kim & Dimsdale, 2007).

Findings from this study must be interpreted in light of several limitations. First, we conceptualized childhood SES as a dichotomous variable. This limits our ability to detect whether there is a graduated effect of childhood SES on sleep or if there is a critical level of childhood SES (i.e. High vs. Low) that is most predictive of adult sleep outcomes. In addition, childhood SES ratings were based on both parents educational achievement. This is a commonly used metric of childhood SES used in health psychology; however, there may be utility in future studies examining maternal and paternal education alone, particularly in families where there were not two parents. Parental education is indicative of family social position and related to income and occupation, but these measures do not fully overlap and potentially each variable may predict different sleep variables (Kaufman, Cooper, & McGee, 1997; Krieger, Williams, & Moss, 1997). Additionally, because we assessed only highest level of parental education, it is unknown whether there is a critical period when childhood SES is most impactful on sleeping patterns. Finally, the population under investigation was uniquely healthy and free from chronic illness; thus we are unsure how our results will generalize into wider populations including those with ongoing medical problems.

The study protocol may have also influenced our findings. Work from our lab has demonstrated that Black participants sleep less time in SWS than Whites, but that this result is most pronounced when PSG is conducted in the hospital, versus when it is conducted at home (Stepnowsky, Moore, & Dimsdale, 2003). Additionally, standardized sleep and wake times enforced in the hospital could have curtailed the normal sleep patterns of some participants. Future studies should replicate these results using at home PSG with subjects who are following their own habitual bed and rise times.

Despite these limitations, our findings provide evidence that race and childhood SES are independently associated with adult sleep outcomes. Black participants spent less time in SWS each night than their White counterparts. Additionally, participants with lower childhood SES slept more time Stage 2 sleep and less time in SWS than participants with higher childhood SES. Further, the relationship between childhood SES, race and adult sleep was independent of demographic information, health practices, and current SES.

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Sample Demographics and Characteristics

Table 1

	Black (n = 54)	White (n = 74)	p	Low Childhood SES (n = 43)	High Childhood SES (n = 85)	p
Age (yrs ± SD)	39.0 (8.6)	31.9 (9.5)	<0.01	38.0 (9.2)	33.3 (9.9)	<0.01
Women, n (%)	24 (44.4)	31 (41.9)	0.86	19 (44.1)	36 (42.4)	0.85
BMI (mean ± SD)	27.5 (3.4)	24.8 (3.6)	<0.01	26.7 (4.0)	25.5 (3.6)	0.10
White, n (%)	---	---	--	16 (37)	58 (68)	<0.01
Low Childhood SES, n (%)	27 (50.0)	16 (21.6)	<0.01	---	---	--
Smokers, n (%)	12 (22.2)	6 (8.1)	0.04	8 (18.6)	10 (11.8)	0.29
Average drinks per week (mean ± SD)	1.7 (4.9)	2.6 (4.2)	0.01	1.5 (2.0)	2.5 (5.4)	0.33
Exercise [†]	37.0 (28.2)	63.2 (46.7)	0.02	44.6 (36.5)	56.7 (44.4)	0.14
Hollingshead Total Score (mean ± SD)	45.8 (12.5)	37.1 (15.7)	<0.01	45.9 (12.9)	38.2 (15.4)	<0.01
MacArthur Scale of SSS (mean ± SD)	5.0 (2.2)	5.9 (1.9)	0.01	4.8 (2.1)	5.9 (2.0)	<0.01

SD = standard deviation; BMI = body mass index; SSS = subjective social status

[†] Leisure Time Exercise Questionnaire

Note: Means and standard deviations for the variables average drinks per week and exercise presented as untransformed values; however *p* values reflect analyses conducted with transformed variables as the outcome measure.

Table 2

Sleep Measures among Blacks and Whites and High and Low Childhood SES Groups

	Black (n = 54) (mean ± SE)	White (n = 74) (mean ± SE)	p	Low Childhood SES (n = 43) (mean ± SE)	High Childhood SES (n = 85) (mean ± SE)	p
PSQI Global Score	6.12 (3.46)	4.31 (2.42)	0.02	5.11 (0.47)	5.11 (0.33)	0.94
Epworth Sleepiness Scale	9.03 (0.58)	7.60 (0.49)	0.08	8.98 (0.61)	7.83 (0.44)	0.14
Total Sleep Time (min)	387.01 (7.57)	401.86 (6.34)	0.17	405.20 (8.15)	390.44 (5.69)	0.15
Sleep Efficiency	91.15 (0.82)	92.41 (0.69)	0.27	91.50 (0.89)	92.07 (0.62)	0.63
Sleep Latency (min)	15.59 (2.26)	10.02 (1.89)	0.08	14.22 (2.43)	11.43 (1.70)	0.24
WASO (%)	5.95 (0.84)	6.05 (0.70)	0.97	6.20 (0.90)	5.91 (6.31)	0.84
Sleep Architecture (%)						
Stage 1	6.11 (0.48)	5.34 (0.40)	0.24	5.67 (0.52)	5.67 (0.36)	0.99
Stage 2	52.8 (1.2)	49.6 (1.0)	0.06	53.67 (1.27)	49.54 (0.89)	0.01
SWS	18.5 (1.3)	23.0 (1.1)	0.01	17.73 (1.35)	22.78 (0.95)	<0.01
REM	22.6 (0.9)	22.1 (0.7)	0.64	22.92 (0.92)	22.02 (0.64)	0.43

Mean = estimated marginal mean; SE = standard error; SES = socioeconomic status; WASO = wake after sleep onset; REM = rapid eye movement

Note: Reported race means are adjusted for age, gender, BMI and childhood SES. Childhood SES means are adjusted for age, gender, BMI, and race. Untransformed estimated marginal means and standard errors are presented for the variables sleep latency, Stage 1 sleep and WASO; however *p* values reflect ANCOVA's conducted with transformed variables as the outcome measure.