Published in final edited form as:

Psychosom Med. 2008 May ; 70(4): 410–416. doi:10.1097/PSY.0b013e31816fdf21.

Influence of Race and Socioeconomic Status on Sleep: Pittsburgh Sleep SCORE Project

Elizabeth J. Mezick, MA, Karen A. Matthews, PhD, Martica Hall, PhD, Patrick J. Strollo Jr, MD, Daniel J. Buysse, MD, Thomas W. Kamarck, PhD, Jane F. Owens, DrPH, and Steven E. Reis, MD

Departments of Psychology (E.J.M., T.W.K.), Psychiatry (K.A.M., M.H., D.J.B., J.F.O.), and Medicine (P.J.S.), University of Pittsburgh, Pittsburgh, Pennsylvania; and Cardiovascular Institute (S.E.R.), University of Pittsburgh, Pittsburgh, Pennsylvania

Abstract

Objective—To examine the independent and interactive effects of race and socioeconomic status (SES) on objective indices and self-reports of sleep.

Methods—The sleep of 187 adults (41% Black; mean age = 59.5 ± 7.2 years) was examined. Nine nights of actigraphy and two nights of inhome polysomnography (PSG) were used to assess average sleep duration, continuity, and architecture; self-report was used to assess sleep quality. Psychosocial factors, health behaviors, and environmental factors were also measured.

Results—Blacks had shorter sleep duration and lower sleep efficiency, as measured by actigraphy and PSG, and they spent less time proportionately in Stage 3 to 4 sleep, compared with others ($p < .01$). Lower SES was associated with longer actigraphy-measured latency, more wake after sleep onset as measured by PSG, and poorer sleep quality on the Pittsburgh Sleep Quality Index ($p < .05$).

Conclusions—Blacks and perhaps individuals in lower SES groups may be at risk for sleep disturbances and associated health consequences.

Keywords

race; socioeconomic status; sleep; polysomnography; actigraphy

INTRODUCTION

Sleep is a fundamental neurobehavioral state linked to critical domains of health and functioning, including learning and memory consolidation (1), mood (2), and disease risk (3–5). Because sleep is a complex state, it is best described along a number of dimensions, such as duration, continuity (amount and distribution of sleep and wakefulness), architecture (stages of sleep), and quality (perceptions of how soundly one sleeps or how rested one feels). In turn, sleep can be characterized by multimodal assessments that include self-report, behavioral, and physiological methods. Increased knowledge of the factors that affect these domains of sleep may help increase our understanding of the determinants of health and disease.

The current report focuses on the relationships of race and socioeconomic status (SES) with sleep. Race here refers to a self-identified social or cultural group with shared physical features and of common descent and not to the genetic makeup. Reporting relatively short or long sleep

duration may be more common among Blacks than non-Hispanic Whites (hereafter referred to as Whites) (6–8). Several minority groups, including Blacks, take longer to fall asleep and have lower sleep efficiency, as assessed by actigraphy, in comparison with Whites (9,10). Additionally, Blacks spend less time in slow wave or Stage 3 to 4 sleep as assessed by polysomnography (PSG) (11–14). Studies regarding the association between race and perceptions of sleep quality have produced inconsistent results (15).

Although associations among low income, low education, and poor self-reported sleep quality have been well documented (16–18), few studies have examined SES in conjunction with behavioral or physiological measures of sleep, such as actigraphy and PSG. In the study of 669 Blacks and Whites by Lauderdale et al. (10), those with a lower income had longer sleep latency and poorer sleep efficiency as assessed by actigraphy. Fiorentino, Marler, Stepnowsky, Johnson, and Ancoli-Israel (19) found that a lower income was related to more PSG-measured wake after sleep onset (WASO) among a sample of older adults.

Given the disparities in income and education levels that exist across race groups in the United States (20,21), socioeconomic factors have been posited to play a role in explaining some of the observed relationships between race and sleep. However, surprisingly few studies have simultaneously examined the effects of both race and SES on sleep parameters, and those that have reported mixed results. For example, whereas at least one study found that race differences in sleep persisted after adjusting for measures of SES (10), another reported that SES and residential context accounted for some of the increased risk for shorter sleep among Blacks (22), and yet another study reported that race differences in WASO were no longer significant when annual income was taken into consideration (19). Inconsistencies in previous results may be due to differences in sample characteristics and sleep outcomes, but the degree to which race and SES uniquely contribute to dimensions of sleep remains uncertain.

The primary objective of the present study was to quantify the independent effects of race and SES on sleep using a multimethod approach. Based on past reports, we hypothesized that both Blacks and individuals of lower SES would have shorter and poorer sleep, as characterized by shorter sleep duration, poorer sleep continuity, less Stage 3 to 4 sleep, and more complaints on self-report measures. In addition, we explored whether an interaction exists between race and SES in association with sleep, as has been the case with a variety of other health outcomes (23,24).

A second objective was to identify potential pathways linking race and SES with sleep. Health behaviors, such as exercise, alcohol use, and smoking, have been suggested as potential mechanisms, but the few studies examining such factors have failed to provide consistent support (10,13). Other candidate pathways relevant to race and SES include psychosocial factors, such as negative affect (25) or stressful life events (26–28), and unfavorable environmental circumstances, such as noise, extreme temperatures, or crowding (29). Such psychosocial and environmental variables have been related to poorer self-reported sleep quality as well as sleep disturbances assessed by PSG (2,30–33); however, they have not been formally tested as mediators in previous studies of race, SES, and sleep. We hypothesized that adjusting for health behaviors, psychosocial factors, and environmental factors would attenuate observed sociodemographic differences in sleep.

METHOD

Research Participants

Participants were recruited from the Heart Strategies Concentrating On Risk Evaluation (Heart SCORE) study, which is a single center, prospective, community-based participatory research cohort study investigating the mechanisms for racial disparities in cardiovascular risk and

attempting to decrease these disparities via a community-based intervention. Baseline enrollment in Heart SCORE began on June 16, 2003 and was completed on October 11, 2006. Eligibility criteria included age 45 to 75 years, residence in the greater Pittsburgh metropolitan area, ability to undergo baseline and annual follow-up visits, and absence of known comorbidities expected to limit life expectancy to <5 years. Within this full cohort, the mean age at study entry was 59.1 ± 7.5 years; 65% were female, 54% were White, 43% were Black, 3% were of other race; 61% were married; and 81% had at least some college education beyond a high school diploma. The Institutional Review Board at the University of Pittsburgh approved the study protocol and all study participants provided their written informed consent. Data collection included demographics, medical history, anthropometrics, lipids/lipoproteins, physical activity, and psychological status (34).

The sleep study enrolled approximately equal ratios of male to female and Black to White participants from the Heart SCORE study sample. Exclusionary criteria for the current study included pregnancy; use of continuous positive airway pressure treatment for sleep-disordered breathing; medication for sleep problems on a regular basis; nighttime work schedule; medication for diabetes; and prior diagnosis of stroke, myocardial infarction, or interventional cardiology procedures. From a target of 225 participants, the present report is based on the first 187 participants who completed the study.

Measures and Procedure

Overview—Participants were recruited during Heart SCORE assessment visits. Study personnel approached potential participants; provided detailed information to those who expressed interest and met the eligibility criteria for study enrollment; and obtained the subjects' written informed consent that had been approved by the University of Pittsburgh Institutional Review Board. Participants were scheduled for the sleep study within 3 months of providing consent. The study protocol lasted 10 days.

Three methods were used to collect sleep data: PSG, actigraphy, and self-report. Inhome PSG sleep studies were conducted on nights 1 and 2 of the study to obtain unique information regarding the stages of sleep and sleep-disordered breathing that could not be obtained via other methods. Actigraphs are watch-like activity monitors that are worn on the wrist to track rest and activity patterns via physical movement. Because actigraphy is noninvasive and associated with minimal participant burden, it was used throughout the duration of the study (nights 1–9) to provide a more representative sample of typical sleep/wake patterns than 2 nights of PSG alone. Diary reports completed on nights and mornings 1 to 10 assessed subjective sleep quality and were used in conjunction with actigraphy to track sleep and wake times throughout the study (nights and mornings 1–10). Self-report measures of global sleep quality and daytime sleepiness were collected on day 2. Two days and nights of ambulatory blood pressure monitoring and two 15-hour periods of urine collection were also conducted as part of the study; these data are not included in the present report.

Polysomnography—The participants used a Compumedics Siesta monitor (Charlotte, North Carolina) for 2 nights of PSG recording in their homes. The PSG montage included bilateral central and occipital electro-encephalogram (EEG) channels, bilateral electro-oculograms (EOG), bipolar submentalis electromyograms (EMG), and one channel of electrocardiogram (ECG) recording. On the first night of PSG, participants were monitored for sleep-disordered breathing using nasal pressure, inductance plethysmography, and fingertip oximetry. High-frequency filter settings were 100 Hz for EEG and EOG and 70 Hz for EMG. Low frequency filter settings were 0.3 Hz for EEG and 10 Hz for EMG. Trained PSG technologists scored sleep records using standard sleep stage scoring criteria for each 20-second epoch (35). American Academy of Sleep Medicine Task Force (36) definitions were

used to identify apneas and hypopneas; oximetry readings were used to quantify average and minimum oxygen saturation levels.

PSG outcome variables included total time spent asleep (actual sleep time excluding periods of wakefulness during the night), sleep latency (minutes until the first of 10 consecutive minutes of Stage 2 or deeper sleep interrupted by no more than 2 minutes of Stage 1 or wakefulness), WASO (total number of minutes scored as awake, after sleep onset), sleep efficiency (percentage of time in bed spent sleeping), and parameters of sleep architecture (percentage of total sleep time spent in Stage 1, Stage 2, Stages 3–4, and rapid eye movement (REM) sleep). The apnea/hypopnea index (AHI) was defined as number of apneas and hypopneas per hour of sleep. Values from the 2 nights of the study were averaged for each variable, with the exception of AHI. Variables with skewed distributions were transformed using either a log (sleep latency, sleep efficiency, WASO, percentage Stage 1 sleep, AHI) or square root (percentage Stages 3–4 sleep) transformation.

Actigraphy—Participants wore an Actiwatch-64 (Respironics, Inc., Bend, Oregon) on the nondominant wrist continuously for 10 days. Data were stored in 1-minute epochs and validated MiniMitter software (Respironics, Inc.) algorithms were used to estimate sleep parameters. The three outcome variables considered in analyses included time spent asleep (actual sleep time excluding periods of wakefulness during the night), sleep latency (time required for onset of sleep after first attempting to fall asleep), and sleep efficiency (the percentage of time in bed spent sleeping). For each variable, the values were averaged across the 9 nights of the study to obtain the mean value used in analyses. Because of skewed distributions, sleep latency and sleep efficiency were log-transformed.

Self-Report—A 10-day diary was used for recording bed and wake times and perceptions of sleep. Every morning, participants rated the previous night's sleep quality and how rested they felt on waking as follows: on a 0 to 5 Likert-type scale, 0 represented “very poor” sleep quality and feeling “not at all rested,” and 5 represented “very good” sleep quality and feeling “extremely rested.” Responses to these two items were averaged across the 10 study days. Average sleep quality and average rested ratings were correlated at $r = .83$; therefore, the two measures were averaged to make one composite variable of subjective sleep quality.

The Pittsburgh Sleep Quality Index (PSQI) (37) is a standardized measure of subjective sleep quality over the previous month. Nineteen individual items on the PSQI are grouped to create seven component scores (e.g., subjective sleep quality, sleep latency, sleep duration). These subscores are summed to generate a global score between 0 and 21, with higher scores indicating worse sleep quality. Participants also completed the Epworth Sleepiness Scale (ESS) (38), an 8-item measure of the likelihood of falling asleep in specific situations. Higher scores on the ESS reflect greater daytime sleepiness. The PSQI and the ESS scores were treated as continuous variables.

Demographic and Biomedical Factors—Race, sex, and age were determined by self-report. Highest education achieved was assessed on a six-level ordinal scale: high school or less, some college/no degree, vocational/technical school/associate (2-year) degree, 4-year degree, Master's degree, and professional degree. Annual income was assessed on a five-level ordinal scale: <\$10,000, \$10,000–<\$20,000, \$20,000–<\$40,000, \$40,000–<\$80,000, and ≥ \$80,000. Values for education and income were standardized and then averaged for each participant to create a composite SES variable. Reports of medication use were collected during inhome PSG studies. Body mass index (BMI) was assessed in the Heart SCORE study protocol.

Candidate Mediators—We considered measures of negative affect, stressful life events, health behaviors, and environmental factors as potential mediators of race and SES differences

in sleep. The Center for Epidemiological Studies Depression Scale (CES-D) (39), the Spielberger Trait Anxiety Inventory (STAI) (40), and the Cook-Medley Hostility Scale (Ho) (41) were completed at the Heart SCORE baseline visit to measure participants' symptoms of depression, anxiety, and hostility, respectively. The sleep item was excluded from the total CES-D score. CES-D, STAI, and Ho scores were standardized and these values were averaged across measures to create one continuous variable indicating negative affect. Ongoing problems were assessed using a 9-item checklist. Participants indicated whether they were currently experiencing a number of problematic events that had been ongoing for at least 12 months, with sample items including "difficulties at work" and "problems in a close relationship." A total count of ongoing problems was obtained by summing the items endorsed on the checklist.

Physical activity was assessed in the daily diary. Each night, participants indicated if they had exercised that day (yes/no) and whether the exercise was light (walking, light housework), medium (golf, hunting), or heavy (running, swimming) in intensity. Light exercise was scored as "1," medium exercise as "2," and heavy exercise as "3." To calculate a physical activity score, we multiplied the number of times exercised during the study by the average level of exercise intensity. For example, if a participant exercised five times over the 10 study days, at an average intensity of 2, her physical activity score would be 10. Self-reported alcohol use and smoking status were also assessed in the diary. Total number of alcoholic beverages consumed over the study period and smoking status were used in analyses.

To explore whether environmental factors might account for race and SES differences in sleep, we included the Sleep Environment Inventory (SEI). An 18-item questionnaire designed for this study, the SEI contains a list of factors that are commonly cited as causes for poor or disturbed sleep. Sample items include "noise outside the house," "uncomfortable bed," and "room temperature." Participants indicated which items disrupted their sleep during the time of the study.

Statistical Analysis

The main effects of race and SES on each of the sleep parameters were tested simultaneously in linear regression models that included race, composite SES, age, sex, and cardiac or hypertensive medication status. Because cardiac and hypertensive medications may be associated with sleep quality, fatigue, and/or sleep apnea, *t* tests were conducted to determine the effects of these medications on sleep. Participants who were taking the following medications at the time of PSG studies differed on at least two of the sleep outcomes: angiotensin-II receptor blockers, angiotensin-converting enzyme inhibitors, α_1 blockers, and α_2 agonists. Use of any one of these four medications was coded dichotomously and included as a covariate. After testing for main effects, a race by SES interaction term was entered into the model; however, the interaction term was not significantly associated with any sleep outcomes and is not discussed below. Finally, we reconducted the above analyses in participants who had an AHI of <15 to remove the potentially confounding effects of sleep-disordered breathing on sleep outcomes. These analyses included 132 participants; 53 were excluded due to AHI of ≥ 15 , and two had missing AHI data.

Mediational analyses tested whether psychosocial factors, health behaviors, or environmental factors attenuated the main effects of race and SES on sleep outcomes. Candidate mediators were first tested for their associations with race and SES in a linear regression model that was adjusted for the covariates listed above. We tested as potential mediators only those variables that differed by race group or were related to SES. The indirect effects of race or SES on sleep outcomes were examined in a series of multiple linear regression analyses in which race, composite SES, and the covariates were entered in the first step, and each of the potential mediating variables was entered in the second step. The statistical significance of the indirect

effects of race or SES on sleep outcomes via an individual mediator was evaluated using the Sobel method (42). All analytic procedures used two-tailed tests of significance and were conducted with SPSS, v. 13 (SPSS Inc., Chicago, IL).

RESULTS

Participant Characteristics

Ninety-nine (52.9%) participants were men, 77 (41.2%) were Black, 106 were non-Hispanic White, and four were Asian. White and Asian participants were combined into one group for analytical purposes, referred to as White/Asian hereafter; however, removing the four Asian participants from analyses resulted in identical results. Age ranged from 46 to 78 years (average = 59.5 years). Fifty (26.7%) were taking one of the four cardiac medications that were found to be associated with sleep. Study sample characteristics for the overall sample and for Black and White/Asian participants are shown in Table 1. Race groups were similar in terms of age and medication status ($p > .2$) but differed in composite SES scores ($t(185) = -5.6, p < .001$), with Blacks reporting lower annual incomes ($t(175) = -5.4, p < .001$) and fewer years of education ($t(185) = -3.6, p < .001$). Blacks had higher BMI levels than Whites/Asians ($t(185) = 4.1, p < .001$); however, inclusion of BMI in the analyses did not change the results reported below.

Dimensions of Sleep

One participant had missing actigraphy data and one participant had missing PSG data; therefore, these analyses were based on sample sizes of 186. Several bedtime values that were estimated using actigraphy disagreed with diary reports by >2 hours (a total of 10 data points between two participants) and were dropped from analyses; the remainder of the two participants' data were retained. Including these outliers in analyses did not alter the results for race described below, but it did make the results for SES more significant for latency and efficiency. Table 2 displays the unadjusted means for sleep duration, continuity, and architecture for Blacks and Whites/Asians, as measured by actigraphy and PSG. Also displayed are the standardized regression coefficients for race and composite SES in models adjusted for covariates.

Sleep Duration—Estimates of time spent asleep as assessed by both actigraphy and PSG were shorter among Blacks than Whites/Asians. Composite SES was not associated with time spent asleep as measured by actigraphy or PSG. Repeating the analyses in the subsample of participants who had an AHI of <15 reduced the main effect of race on PSG-measured time spent asleep ($\beta = 0.20, p = .05$), whereas actigraphy estimates were unaltered.

Sleep Continuity—Blacks took longer to fall asleep and had less efficient sleep as measured by actigraphy than Whites/Asians. Results based on PSG were similar, with Blacks having marginally longer sleep latency and significantly poorer efficiency compared with Whites/Asians. Lower composite SES was associated with longer actigraphy-measured latency and more PSG-measured WASO but was not significantly related to any other continuity measures. Repeating the analyses in the subsample of participants who had an AHI of <15 did not change the reported results.

Sleep Architecture—Blacks had a higher percentage of Stage 2 sleep and a lower percentage of Stage 3 to 4 sleep than Whites/Asians. There were no race differences in the percentage of REM sleep. Composite SES was not associated with stages of sleep architecture. Repeating the analyses in the subsample of participants who had an AHI of <15 did not alter these results.

Sleep Quality—Table 3 displays the unadjusted means for self-reported sleep quality and daytime sleepiness as well as the standardized regression coefficients for race and composite SES in models adjusted for covariates. Race was not associated with self-reported sleep quality or daytime sleepiness as measured by the PSQI, diary reports, and the ESS. Lower composite SES was associated with poorer self-reported sleep quality on the PSQI. When the analyses were repeated in the subsample of participants who had an AHI of <15, composite SES was no longer associated with PSQI scores ($p = .20$).

Mediational Analyses

Race—The association between race and each potential mediating variable was tested in a linear or logistic regression model that was adjusted for covariates. Blacks endorsed more ongoing problems ($\beta = -0.15, p = .05$) and less physical activity ($\beta = 0.14, p = .06$); however, race was not associated with any of the other potential mediators. Neither ongoing problems nor physical activity were statistical mediators of the associations between race and sleep outcomes (all $z < 1.96, p > .05$).

SES—Lower composite SES was independently associated with increased negative affect ($\beta = -0.22, p = .006$) and less physical activity ($\beta = 0.20, p = .008$). Individuals with lower composite SES were also more likely to cite noise outside the house ($\beta = -0.98, p = .001$), room temperature ($\beta = -0.43, p = .05$), and worries about health ($\beta = -0.43, p = .09$) as disturbing their sleep on the SEI; therefore, these three environmental items were summed in a subscale before being tested as mediators. When tested individually, the subscale of SEI environmental factors ($z = -4.68, p = .001$) and negative affect ($z = -2.33, p = .02$) were statistical mediators of the relationship between SES and PSQI scores. When these two variables were tested simultaneously in the same double mediation model, both SEI environmental factors ($z = -2.55, p = .01$) and negative affect ($z = -2.19, p = .03$) remained significant mediators. The indirect effect of environmental factors and negative affect on the association between SES and PSQI scores represented 82% of the total effect. None of the potential mediators attenuated associations between SES and sleep latency or WASO (all $z < 1.51, p > .13$).

DISCUSSION

This study investigated the influences of race and SES on multiple dimensions of sleep in a community sample. Substantial race differences in both actigraphy and PSG-measured sleep were observed, such that Blacks slept for a shorter duration, took longer to fall asleep and had less continuous sleep, and spent a smaller percentage of time in Stage 3 to 4 sleep than Whites and Asians, all of which are consistent with prior reports (9–14). Importantly, Blacks' likelihood of having shorter and more disturbed sleep persisted even after taking into account SES measures. Although it has been hypothesized that socioeconomic factors may be responsible for race disparities in sleep, our results show that the race-sleep relationship persisted independently of SES. The above results were consistent across both PSG and actigraphy measures of sleep. However, Blacks and Whites/Asians in the present sample did not differ in their reports of sleep quality or daytime sleepiness. Given that past studies have produced inconsistent findings regarding race differences in subjective sleep quality (15), further investigation of factors that may influence such perceptions within race groups may be beneficial.

In comparison with race, associations between SES and sleep were fewer in number and less consistent across types of sleep measures. Lower SES was related to longer sleep latency as measured by actigraphy and more time spent awake after sleep onset as measured by PSG. Although these findings are in agreement with two other studies that have reported relationships between dimensions of sleep continuity and SES, independent of race (10,19), they must be

interpreted with some caution. It is possible that actigraphy estimates were more representative of typical sleep patterns in this study, as they were collected over 9 nights using a relatively noninvasive method. Our sample size was also considerably smaller than one of the previous studies in this area (10), which may have resulted in our being able to detect only trends in SES effects in other continuity outcomes. Finally, consistent with past reports (16–18), we observed an association between low SES and poor self-reported sleep quality on the PSQI.

A number of psychosocial, health, and environmental variables were examined as potential mediators of the race-sleep relationship. Although Blacks reported more ongoing problems and less physical activity than Whites/Asians, these factors did not account for obtained differences. It is possible that cultural norms or other psychosocial variables that were not assessed, such as social support or discrimination, are more influential in Blacks' sleep (43). Physiological pathways, such as autonomic functioning, also may be relevant mechanisms for future study (44). Finally, recent evidence suggests that residential context may play a role in explaining race differences in sleep duration (22). We attempted to capture environmental factors that affect sleep using the SEI, but self-reports of such factors did not differ by race. It is possible that differences in sample size, environmental measures, or the degree of variability in neighborhoods may account for disparate findings between these two studies.

Although SEI items did not mediate race differences in sleep, they were helpful in identifying several of the factors that were perceived to contribute to poor sleep quality among individuals of lower SES in our sample. In particular, outside noise, room temperature, and health worries mediated the association between SES and PSQI scores. Additionally, negative affect—as characterized by depression, anxiety, and hostility—was also responsible for the link between SES and self-reported sleep. A better understanding of the role that environmental and emotional factors play in the link between SES and sleep may hold important implications for improving perceptions of sleep and well-being among those in lower socioeconomic groups.

Indices of sleep disordered breathing did not vary by race or SES in this sample; however, we wanted to confirm the extent to which the obtained relationships would be apparent in the subset of participants with AHI scores of <15. Race differences in PSG-measured time spent asleep were reduced in participants with an AHI of <15, and SES differences in PSQI scores were eliminated among participants with low apnea scores. In the aggregate, these findings suggest that undiagnosed sleep-disordered breathing may account for some of the differences in the literature on self-reported sleep quality and short or poor sleep.

This study has several limitations. First, our sample was drawn from a large study of volunteers in the Pittsburgh community screened for cardiovascular risk, and, therefore, is not representative of the general population. There have been few studies examining the sleep of individuals from other races, and the inclusion of such individuals would have strengthened the generalizability of the findings. Second, the large number of analyses conducted may have raised Type I error rates and increased the chance of reporting spurious findings. However, our findings linking race and SES with sleep duration and continuity are in agreement with larger, population-based epidemiological reports. Although we attempted to examine the interactive effect between race and SES, few Black participants fell into the highest SES categories and few White/Asian participants fell into the lowest categories, potentially causing interactions to be difficult to detect. Finally, conclusions regarding causality cannot be made due to the cross-sectional design of the study. For example, the direction of the association between low SES and poor sleep cannot be determined, and although we excluded diabetics and assessed medication status, it is possible that sleep disturbances may reflect subclinical or other unmeasured disease.

In summary, these findings are consistent with the hypothesis that substantial race differences in sleep exist, and these differences are not accounted for by SES or the measured psychosocial and health factors. Blacks may be at particularly high risk for shorter and less efficient sleep and their health consequences, including obesity, hypertension, and diabetes (3–5). Low SES is associated with poor self-reported sleep quality, perhaps via adverse environmental factors and negative affect, and may also contribute to poor sleep continuity as measured by actigraphy and PSG. Pathways that account for sociodemographic differences in sleep remain largely unknown. Identification of mediating factors may advise interventions and increase our understanding of sleep's associations with physical and psychological health.

Acknowledgments

This research was supported by Grants HL076369, HL065111, HL065112 (K.A.M.), and HL07560 (E.J.M.) from the National Institutes of Health, Bethesda, Maryland, and a grant (Contract ME-02-384) with the Pennsylvania Department of Health (S.E.R.). The Department specifically disclaims responsibility for any analyses, interpretations, or conclusions.

Glossary

SES	socioeconomic status
PSG	polysomnography
WASO	wake after sleep onset
Heart SCORE	Heart Strategies Concentrating On Risk Evaluation
PSQI	Pittsburgh Sleep Quality Index
ESS	Epworth Sleepiness Scale
EOG	electro-oculogram
EMG	electromyogram
ECG	electrocardiogram
REM	rapid eye movement
AHI	apnea/hypopnea index
BMI	body mass index
CES-D	Center for Epidemiological Studies Depression Scale
STAI	Spielberger Trait Anxiety Inventory
Ho	Cook-Medley Hostility Scale
SEI	Sleep Environment Inventory

References

1. Maquet P. The role of sleep in learning and memory. *Science* 2001;294:1048–52. [PubMed: 11691982]
2. Pilcher JJ, Ginter DR, Sadowsky B. Sleep quality versus sleep quantity: relationships between sleep and measures of health, well-being and sleepiness in college students. *J Psychosom Res* 1997;42:583–96. [PubMed: 9226606]
3. Gangwisch JE, Heymsfield SB, Boden-Albala B, Bujis RM, Kreier F, Pickering TG, Rundle AG, Zammit GK, Malaspina D. Short sleep duration as a risk factor for hypertension: analyses of the first national health and nutrition examination study. *Hypertension* 2006;47:833–9. [PubMed: 16585410]
4. Gangwisch JE, Malaspina D, Boden-Albala B, Heymsfield SB. Inadequate sleep as a risk factor for obesity: analyses of the NHANES I. *Sleep* 2005;28:1289–96. [PubMed: 16295214]

5. Mallon L, Broman J, Hetta J. High incidence of diabetes in men with sleep complaints or short sleep duration. *Diabetes Care* 2005;28:2762–7. [PubMed: 16249553]
6. Kripke DF, Brunner R, Freeman R, Hendrix SL, Jackson RD, Masaki K, Carter RA. Sleep complaints of postmenopausal women. *Clin J Women Health* 2001;1:244–52.
7. Qureshi AI, Giles WH, Croft JB, Bliwise DL. Habitual sleep patterns and risk for stroke and coronary heart disease: a 10-year follow-up from NHANES I. *Neurology* 1997;48:904–11. [PubMed: 9109875]
8. Schoenborn CA. Health habits of U.S. adults, 1985: the “Alameda 7” revisited. *Public Health Rep* 1986;101:571–80. [PubMed: 3097736]
9. Jean-Louis G, Kripke DF, Ancoli-Israel S, Klauber MR, Sepulveda RS. Sleep duration, illumination, and activity patterns in a population sample: effects of gender and ethnicity. *Biol Psychiatry* 2000;47:921–7. [PubMed: 10807965]
10. Lauderdale DS, Knutson KL, Yan LL, Rathouz PJ, Hulley SB, Sidney S, Liu K. Objectively measured sleep characteristics among early-middle-aged adults: the CARDIA study. *Am J Epidemiol* 2006;164:5–16. [PubMed: 16740591]
11. Profant J, Ancoli-Israel S, Dimsdale JE. Are there ethnic differences in sleep architecture? *Am J Hum Biol* 2002;14:321–6. [PubMed: 12001088]
12. Rao U, Poland RE, Lutchmansingh P, Ott GE, McCracken JT, Lin KM. Relationship between ethnicity and sleep patterns in normal controls: implications for psychopathology and treatment. *J Psychiatr Res* 1999;33:419–26. [PubMed: 10504010]
13. Redline S, Kirchner HL, Quan SF, Gottlieb DJ, Kapur V, Newman A. The effects of age, sex, ethnicity, and sleep-disordered breathing on sleep architecture. *Arch Intern Med* 2004;164:406–18. [PubMed: 14980992]
14. Stepnowsky CJ Jr, Moore PJ, Dimsdale JE. Effect of ethnicity on sleep: complexities for epidemiologic research. *Sleep* 2003;26:329–32. [PubMed: 12749554]
15. Durrence HH, Lichstein KL. The sleep of African Americans: a comparative review. *Behav Sleep Med* 2006;4:29–44. [PubMed: 16390283]
16. Gellis LA, Lichstein KL, Scarinci IC, Durrence HH, Taylor DJ, Bush AJ, Riedel BW. Socioeconomic status and insomnia. *J Abnorm Psychol* 2005;114:111–8. [PubMed: 15709817]
17. Hall M, Bromberger J, Matthews K. Socioeconomic status as a correlate of sleep in African-American and Caucasian women. *Ann N Y Acad Sci* 1999;896:427–30. [PubMed: 10681942]
18. Moore PJ, Adler NE, Williams DR, Jackson JS. Socioeconomic status and health: the role of sleep. *Psychosom Med* 2002;64:337–44. [PubMed: 11914451]
19. Fiorentino L, Marler M, Stepnowsky C, Johnson S, Ancoli-Israel S. Sleep in older African Americans and Caucasians at risk for sleep-disordered breathing. *Behav Sleep Med* 2006;4:164–78. [PubMed: 16879080]
20. Bauman, KJ.; Graf, NL. Educational Attainment: 2000. Census 2000 Brief. 2003 [Accessed June 1, 2007]. Available at <http://www.census.gov/prod/2003pubs/c2kbr-24.pdf>
21. DeNavas-Walt, C.; Proctor, BD.; Lee, CH. U.S. Census Bureau, Current Population Reports, P60-231. Income, poverty, and health insurance coverage in the United States. 2005 [Accessed June 1, 2007]. Available at <http://www.census.gov/prod/2006pubs/p60-231.pdf>
22. Hale L, Do DP. Racial differences in self-reports of sleep duration in a population-based study. *Sleep* 2007;30:1096–103. [PubMed: 17910381]
23. Bardwell WA, Burke SC, Thomas KS, Carter C, Weingart K, Dimsdale JE. Fatigue varies by social class in African Americans but not Caucasian Americans. *Int J Behav Med* 2006;13:252–8. [PubMed: 17078776]
24. Rodriguez CJ, Sciacca RR, Diez-Roux AV, Boden-Albala B, Sacco RL, Homma S, DiTullio MR. Relation between socioeconomic status, race-ethnicity, and left ventricular mass: the Northern Manhattan study. *Hypertension* 2004;43:775–9. [PubMed: 14981073]
25. Gallo LC, Matthews KA. Understanding the association between socioeconomic status and physical health: do negative emotions play a role? *Psychol Bull* 2003;129:10–51. [PubMed: 12555793]
26. Brady SS, Matthews KA. The influence of socioeconomic status and ethnicity on adolescents’ exposure to stressful life events. *J Pediatr Psychol* 2002;27:575–83. [PubMed: 12228329]

27. McLeod JD, Kessler RC. Socioeconomic status differences in vulnerability to undesirable life events. *J Health Soc Behav* 1990;31:162–72. [PubMed: 2102495]
28. Myers, HF.; Lewis, TT.; Parker-Dominguez, T. Stress, coping and minority health: biopsychosocial perspective on ethnic health disparities. In: Bernal, G.; Trimble, J.; Burlew, K.; Leong, F., editors. *Handbook of Racial and Ethnic Minority Psychology*. Thousand Oaks, CA: Sage; 2003.
29. Evans GW, Kantrowitz E. Socioeconomic status and health: the potential role of environmental risk exposure. *Annu Rev Public Health* 2002;23:303–31. [PubMed: 11910065]
30. Akerstedt T. Psychosocial stress and impaired sleep. *Scand J Work Environ Health* 2006;32:493–501. [PubMed: 17173205]
31. Hall M, Buysse DJ, Nowell PD, Nofzinger EA, Houck P, Reynolds CF 3rd, Kupfer DJ. Symptoms of stress and depression as correlates of sleep in primary insomnia. *Psychosom Med* 2000;62:227–30. [PubMed: 10772402]
32. Miedema HM, Vos H. Associations between self-reported sleep disturbance and environmental noise based on reanalyses of pooled data from 24 studies. *Behav Sleep Med* 2007;5:1–20. [PubMed: 17313321]
33. Owens JF, Matthews KA. Sleep disturbance in healthy middle-aged women. *Maturitas* 1998;30:41–50. [PubMed: 9819782]
34. Aiye AN, Kip KE, Marroquin OC, Mulukutla SR, Edmundowicz D, Reis SE. Racial differences in coronary artery calcification are not attributed to differences in lipoprotein particle sizes: the Heart Strategies Concentrating on Risk Evaluation (Heart SCORE) study. *Am Heart J* 2007;153:328–34. [PubMed: 17239697]
35. Rechtschaffen, A.; Kales, A. *A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects*. Washington, DC: US Government Printing Office, Department of Health Education and Welfare; 1986. NIH Publication 204
36. The Report of an American Academy of Sleep Medicine Task Force. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. *Sleep* 1999;22:667–89. [PubMed: 10450601]
37. Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res* 1989;28:193–213. [PubMed: 2748771]
38. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep* 1991;14:540–5. [PubMed: 1798888]
39. Radloff LS. The CES-D scale: a self-report depression scale for research in the general population. *Appl Psychol Measurement* 1977;1:385–401.
40. Spielberger, CD.; Gorsuch, RL.; Lushene, RE. *Manual for the State-Trait Anxiety Inventory*. Palo Alto: Consulting Psychologists Press; 1970.
41. Cook WW, Medley DM. Proposed hostility and pharisaic-virtue scales for the MMPI. *J Appl Psychol* 1954;38:414–8.
42. MacKinnon DP, Lockwood CM, Hoffman JM, West SG, Sheets V. A comparison of methods to test mediation and other intervening variable effects. *Psychol Methods* 2002;7:83–104. [PubMed: 11928892]
43. Thomas KS, Bardwell WA, Ancoli-Israel S, Dimsdale JE. The toll of ethnic discrimination on sleep architecture and fatigue. *Health Psychol* 2006;25:635–42. [PubMed: 17014281]
44. Liao D, Barnes RW, Chambless LE, Simpson RJ, Sorlie P, Heiss G. Age, race, and sex differences in autonomic cardiac function measured by spectral analysis of heart rate variability—the ARIC study. *Am J Cardiol* 1995;76:906–12. [PubMed: 7484830]

TABLE 1

Sample Characteristics

Measure	Total Sample	Black	White/Asian
Total, <i>n</i> (%)	187	77 (41.2)	110 (58.8)
Men, <i>n</i> (%)	99 (53)	33 (43.0)	66 (60.0)
Age, mean $\pm$ SD	59.5 $\pm$ 7.2	58.7 $\pm$ 7.1	60.1 $\pm$ 7.2
Body mass index, mean $\pm$ SD	29.4 $\pm$ 5.0	31.2 $\pm$ 5.2	28.2 $\pm$ 4.5
Taking cardiac/hypertensive medications, <i>n</i> (%) ^a	50 (26.7)	22 (28.6)	28 (25.5)
Years education, <i>n</i> (%)			
High school or less	31 (16.6)	16 (20.8)	15 (13.6)
Some college, no degree	31 (16.6)	18 (23.4)	13 (11.8)
Other degree, non-4 yr (vocational/technical, associate)	31 (16.6)	17 (22.1)	14 (12.7)
4-yr. degree	39 (20.9)	14 (18.2)	25 (22.7)
Advanced degree	55 (29.4)	12 (15.6)	43 (39)
Annual income, <i>n</i> (%) ^b			
<\$20,000	30 (16.9)	22 (29.7)	8 (7.8)
\$20,000–<\$40,000	51 (28.8)	25 (33.8)	26 (25.2)
\$40,000–<\$80,000	64 (36.2)	23 (31.1)	41 (39.8)
$\geq$ \$80,000	32 (17.1)	4 (5.4)	28 (27.2)
Psychosocial factors and health behaviors			
CES-D, mean $\pm$ SD	9.8 $\pm$ 9.5	8.7 $\pm$ 8.6	10.6 $\pm$ 10.0
STAI, mean $\pm$ SD	6.0 $\pm$ 5.1	5.7 $\pm$ 4.9	6.3 $\pm$ 5.3
Ho, mean $\pm$ SD	8.5 $\pm$ 4.3	8.9 $\pm$ 4.4	8.3 $\pm$ 4.2
Ongoing problems, mean $\pm$ SD	12.9 $\pm$ 3.9	14.0 $\pm$ 4.3	12.1 $\pm$ 3.3
Physical activity rating, mean $\pm$ SD	7.8 $\pm$ 6.6	5.9 $\pm$ 6.1	9.2 $\pm$ 6.5
Alcoholic drinks consumed, mean $\pm$ SD	4.8 $\pm$ 8.2	3.6 $\pm$ 7.4	5.6 $\pm$ 8.7
Current smokers, <i>n</i> (%)	19 (10.2)	12 (15.6)	7 (6.4)

The income categories, <\$10,000 and \$10,000–<\$20,000, and the education categories, Master's degree and advanced degree, were assessed separately but combined in the table for ease of presentation.

^aThis category includes those cardiac/hypertensive medications that were associated with two or more sleep outcomes.

^bTen participants failed to report annual income (*n* = 3 for Blacks and *n* = 7 for Whites/Asians).

SD = standard deviation; CES-D = Center for Epidemiological Studies Depression Scale; STAI = Spielberger Trait Anxiety Inventory; Ho = Cook-Medley Hostility Scale.

Unadjusted Means of Actigraphy and Polysomnography Measures Among Blacks and Whites/Asians and β Coefficients for Race and Composite SES

TABLE 2

	Unadjusted Mean $\pm$ SD		White/Asian		Composite SES	
	Black	White/Asian	β	p	β	p
Duration (hr)						
Actigraphy	5.4 $\pm$ 0.83	6.0 $\pm$ 0.90	0.44	<.001	-0.11	.13
PSG	5.8 $\pm$ 1.08	6.2 $\pm$ 0.93	0.25	.001	-0.06	.46
Sleep continuity latency (min)						
Actigraphy	25.4 $\pm$ 23.7	16.3 $\pm$ 19.2	-0.25	.001	-0.17	.03
PSG	40.7 $\pm$ 40.2	26.1 $\pm$ 26.7	-0.15	.06	-0.11	.18
Efficiency (%)						
Actigraphy	77.4 $\pm$ 9.6	82.1 $\pm$ 8.5	-0.30	<.001	-0.10	.19
PSG	73.6 $\pm$ 11.8	78.7 $\pm$ 9.8	-0.23	.003	-0.13	.08
WASO (min)						
PSG	86.9 $\pm$ 47.8	77.3 $\pm$ 45.2	-0.09	.22	-0.19	.01
Sleep architecture (%)						
Stage 1	9.6 $\pm$ 6.2	10.2 $\pm$ 6.2	0.02	.81	-0.12	.10
Stage 2	65.1 $\pm$ 8.0	60.3 $\pm$ 8.2	-0.29	<.001	-0.01	.98
Stage 3 to 4	3.4 $\pm$ 5.0	6.8 $\pm$ 7.0	0.34	<.001	0.07	.32
REM	21.9 $\pm$ 5.0	22.6 $\pm$ 5.3	0.10	.21	0.03	.70
Sleep disordered breathing						
Apnea/Hypopnea Index	14.8 $\pm$ 17.9	12.8 $\pm$ 13.1	-0.10	.19	0.01	.99

Note: Reported β coefficients are from analyses adjusted for age, sex, and medication use. Race coded +1 for White/Asian and 0 for Black.

β coefficients for actigraphy and PSG-measured sleep efficiency are reversed due to data transformations.

SES = socioeconomic status; SD = standard deviation; PSG = polysomnography; WASO = wake after sleep onset; REM = rapid eye movement.

Unadjusted Means of Sleep Quality Measures among Blacks and Whites/Asians and β Coefficients for Race and Composite SES

TABLE 3

	Unadjusted Mean $\pm$ SD		White/Asian		Composite SES	
	Black	White/Asian	β	p	β	p
Sleep quality						
Pittsburgh Sleep Quality Index	7.0 $\pm$ 3.6	5.8 $\pm$ 3.1	-0.07	.41	-0.16	.04
Epworth Sleepiness Scale	8.2 $\pm$ 4.5	8.3 $\pm$ 3.4	-0.04	.61	0.10	.21
Diary Sleep Quality ^a	2.3 $\pm$ 0.5	2.3 $\pm$ 0.5	-0.11	.17	0.01	.98

^aDiary Sleep Quality is the average value of morning ratings of feeling rested and previous night's sleep quality. Race coded +1 for White/Asian and 0 for Black.
SES = socioeconomic status; SD = standard deviation.