Original Article

Disparities in sleep characteristics by race/ethnicity in a population-based sample: Chicago Area Sleep Study

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ABSTRACT

Background: Prior studies report less favorable sleep characteristics among non-Whites as compared with non-Hispanic Whites. However, few population-based studies have used objective measures of sleep duration, especially in more than two racial/ethnic groups. We tested whether objectively estimated sleep duration and self-reported sleep quality varied by race and whether differences were at least partially explained by the variability in clinical, psychological, and behavioral covariates.
Methods: Adults aged 35–64 years who self-identified as White, Black, Asian, or Hispanic were randomly sampled from Chicago, IL, and the surrounding suburbs. Our analytic sample included adults who had an apnea–hypopnea index <15 after one night of screening and who completed seven nights of wrist actigraphy for determination of sleep duration, sleep percentage, minutes of wake after sleep onset, and sleep fragmentation ($n = 495$). Daytime sleepiness was estimated using the Epworth Sleepiness Scale (ESS), and sleep quality was estimated from the Pittsburgh Sleep Quality Index (PSQI).
Results: Following statistical adjustment for age, gender, education, work schedule (ie, day vs. night shift), smoking status, depressive symptoms, body mass index (BMI), hypertension, and diabetes, sleep duration (minutes) was significantly (all $p < 0.01$) shorter in Black (mean = 399.5), Hispanic (mean = 411.7), and Asian (mean = 409.6) participants than in White participants (mean = 447.4). All remaining sleep characteristics were significantly less favorable among Black participants as compared with White participants. Asian participants also reported significantly more daytime sleepiness than did White participants.
Conclusions: Differences in sleep characteristics by race/ethnicity are apparent in a sample of adults with a low probability of sleep apnea and following adjustment for known confounders.

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1. Introduction

There are notable and persistent disparities in the prevalence of major cardiovascular and metabolic disorders by race and ethnicity. Adverse health behaviors such as poor diet and physical inactivity account for a substantial proportion of these disparities [1]. Given research describing the contribution of sleep duration and quality to the development of obesity, hypertension, diabetes, cardiovascular disease, and mortality [2–7], it is equally plausible that differences in sleep characteristics by race/ethnicity contribute to disparities in cardiovascular disease and metabolic disorders.

Variability in short sleep duration and poor-quality sleep by sociodemographic characteristics has been observed in population

research studies. Most often, non-White adults and adults from lower socioeconomic status groups report less favorable sleep characteristics including a higher prevalence of short and long sleep and poorer-quality sleep than White adults [8–16]. The limitations of prior research on sociodemographic variability in sleep include reliance on self-reported versus objectively determined sleep duration, limited racial/ethnic variability within studies (ie, most studies compare two groups), and potential confounding by the prevalence of sleep disorders such as obstructive sleep apnea (OSA), which are independently associated with shorter sleep and poorer-quality sleep [17].

Thus, the objective of our study was to describe and compare objectively measured sleep duration and quality via actigraphy and self-reported sleep quality and sleepiness in a population-based sample of White, Black, Hispanic, and Asian adults with a low probability of sleep apnea. An additional advantage over the previous studies is the ability to statistically adjust for potential behavioral and clinical confounders of the association between race/ethnicity

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and sleep characteristics. Our a priori hypothesis was that we would observe less favorable sleep characteristics in non-Whites versus Whites, but that these differences are at least partially accounted for by differences in clinical and behavioral covariates across groups.

2. Methods

2.1. Study participants and design

Analyses of the Chicago Area Sleep Study (CASS), a cross-sectional population-based epidemiologic study, were conducted. Men and women, ages 35–64 years, who were living in Chicago, IL, or the surrounding suburbs were identified via commercial telephone listings and contacted by mail and telephone. The CASS staff screened the participants via telephone to determine the likelihood of sleep apnea based on the Berlin Sleep Questionnaire [18], a modified STOP-BANG [19] (modified to use self-reported neck circumference for men) and body mass index (BMI). Adults were invited to participate if they met each of the following criteria: BMI < 35 kg/m^2, Berlin score <3 (women) or <2 (men), and a STOP-BANG <2 affirmative responses for women or <3 affirmative responses for men.

Eligible potential participants were invited to attend two clinical examinations approximately one week apart. Informed consent was obtained from all participants, and all protocols were approved by the Northwestern University Institutional Review Board. Women were scheduled to attend their first examination during the mid-follicular phase of their menstrual cycle. At the first examination, the staff explained the procedures for wearing the ApneaLink Plus® apnea-screening device and the wrist actigraph. The participants were also given a set of questionnaires to complete prior to the next examination, which was scheduled to take place a minimum of eight days later and a maximum of 14 days later.

2.2. Measurements

2.2.1. Race/ethnicity

The primary aim of the study was to include equal representation of adults from four race/ethnic groups. Because race/ethnicity is primarily a social and cultural construct, we relied on self-report to determine race/ethnicity. Potential study participants were recruited from geographic areas in Chicago, IL, and surrounding suburbs with a high proportion of the targeted racial/ethnic groups. The commercial telephone listings included an indicator of race/ethnicity, and telephone recruiters asked participants to confirm their race/ethnicity. In addition, when participants attended the clinical examination, they were also asked to complete sociodemographic questionnaires indicating their race (Black, White, and Asian) and whether or not they were of Hispanic ethnicity. Very few Hispanic participants reported both their ethnicity and their race, leading to a substantial lack of data on race among Hispanics. This was addressed by classifying all participants indicating Hispanic ethnicity as Hispanic for our study. In cases of disagreement between self-reports of race/ethnicity, participants were classified based on the race/ethnicity that they reported on the sociodemographic questionnaires. Based on prior research reporting disparities in cardiovascular and metabolic disorders in Asian ancestral groups, we attempted to reduce variability by targeting Asians who reported Chinese ethnicity. However, we were unable to identify a sufficient number of eligible Chinese participants, so the inclusion criteria were broadened to other East Asians (eg, Korean, Japanese, and Vietnamese) because South and Southeast Asians (eg, Filipinos and South Asian Indians) have a markedly different cardiovascular and metabolic profile [20].

2.2.2. Sleep characteristics

To restrict the sample to participants with a low likelihood of OSA, at the first visit, participants were asked to wear the ApneaLink Plus® apnea-screening device for one night. Post hoc exclusions for analysis were made for those participants with AHI <15, measured based on a minimum of 4 h of wear time and using a combination of devices including the nasal cannula, a chest belt to detect respiratory effort, and a pulse oximeter to measure oxygen saturation. The analyses were repeated in the subset of 361 participants with AHI ≤ 5 for the sensitivity analysis. High sensitivity (91%) and specificity (95%) were observed between ApneaLink Plus® and the laboratory polysomnograph [21].

At the first examination, participants took home the wrist-worn Actiwatch™ 2 device (Phillips Respironics, Bend, OR, USA). They were asked to wear the watch continuously for seven days and nights until the second clinical examination and to keep a daily sleep log to record their daily bedtime and wake time and any nap times during the preceding 24-h interval. The Actiware software program (version 5) and its built-in algorithm were used to analyze the actigraphy data. Automated scoring was not used because it has not been validated. Instead, a member of the research team identified bedtime and wake time using the sleep logs and the event markers. A proportion of the records (10%) were independently scored by KK, and comparisons were made to ensure data quality. Time in bed was determined based on the Actiwatch™ device marker, which the participants were asked to press when they went to bed to sleep and when they woke. If participants did not use the marker, the study staff estimated bedtimes and wake times based on self-reports recorded on the sleep log. Sleep duration was determined using the device software algorithms, which quantified the amount of movement during time in bed. Minutes of wake after sleep onset (WASO) were calculated. The percentage of time the participant was asleep during the sleep period (sleep onset to sleep end) was calculated (sleep percentage). Sleep fragmentation is an index of restlessness during the sleep period expressed as a percentage. It is calculated by summing the following two percentages: (1) the percentage of the sleep period spent moving (an epoch with >2 activity counts is considered moving) and (2) the percentage of the number of immobile phases (consecutive epochs with no movement) that are only 1 min long or less. The average values for each of the sleep characteristics were calculated for the seven days. Participants were asked to complete the Pittsburgh Sleep Quality Index (PSQI) [22]. Scores range from 0 to 21, with higher scores indicating worse sleep quality. Daytime sleepiness was measured using the eight-item Epworth Sleepiness Scale (ESS) [23], with higher scores (range 0–24) indicating greater sleepiness. Both the PSQI and ESS were treated as continuous variables in analyses.

2.2.3. Covariates

Participants were asked to fast for a minimum of 12 h prior to the second clinic examination and to bring all prescription medications and over-the-counter supplements that they were currently taking. All clinical measurements (ie, phlebotomy, blood pressure, and anthropometry) were collected between 7:30 am and 11:00 am. Blood was drawn from participants in the seated position into citrate Vacutainer tubes, centrifuged at 3000 rpm at 4 °C for 20 min, and stored at –70 °C. The fasting glucose level was determined from plasma using spectrophotometry. Whole blood was assayed for determining hemoglobin A1c using an immunoturbidimetric assay. Diabetes status was determined if fasting glucose was ≥ 126 mg/dL or hemoglobin A1c was ≥ 6.5%, or if participants reported taking medications to control diabetes [24]. Blood pressure was measured using an Omron automated cuff on participants while seated after 5 min of rest. Three measurements were collected and the final two were averaged. Hypertension was defined if participants had systolic blood pressure ≥ 140 or diastolic blood pressure ≥ 90, or if

they self-reported using antihypertensive medications. Height and weight were measured in light examination clothes and no shoes. BMI was calculated as weight in kilograms divided by the height in meters squared.

Age, gender, and race/ethnicity were queried. The years of education completed was captured as the measure of socioeconomic status. Work status (ie, full time, part time, or unemployed) and schedule (ie, regular day shift, night shift, or swing shift) were queried as both an additional measure of socioeconomic status and an indicator of regular sleep patterns. Questionnaires were used to ascertain smoking status, which were categorized into current, former, and never. Physical activity was assessed using the Global Physical Activity Questionnaire (GPAQ), a self-reported surveillance instrument that captures the frequency and duration of engagement in three domains of activity (ie, work, leisure, and transportation) and sedentary behavior [25]. Depressive symptoms were assessed using the Centers for Epidemiologic Studies Depression Scale (CES-D) [26].

2.3. Analysis sample

Among the 630 participants with valid actigraphy data to determine sleep duration and maintenance, 595 completed the clinical examination. Fourteen participants who did not have valid apnea–hypopnea index (AHI) values based on the multichannel ApneaLink Plus® (ResMed Germany Inc., Bremen, Germany) and 52 participants with AHI ≥ 15 were excluded. Thirty-three participants who did not report race/ethnicity or were using sleep medications or hypnotic antidepressants were additionally excluded. After exclusions, 496 participants were available for analysis.

2.4. Statistical methods

The characteristics of the study sample are presented as means and standard deviations for continuous variables or proportions for categorical variables in the total sample and stratified by race/ethnicity. Sleep characteristics are described by race/ethnicity according to their mean and standard deviation, median and interquartile range, and minimum and maximum. Analysis of variance (ANOVA) was used to calculate adjusted means and 95% confidence intervals for each of the sleep characteristics comparing Black, Asian, and Hispanic participants with White participants. Prior to modeling, the presence of effect modification by gender and education level (centered at a mean = 0 and standard deviation = 1) was evaluated by including multiplicative interaction terms in each model. Given the large number of interaction tests, conservative criteria for determining interaction at alpha = 0.05 were maintained. Models were adjusted for age, gender, education, work schedule, smoking status, depressive symptoms, BMI, hypertension, and diabetes. Statistical significance was determined at $p \leq 0.05$. All analyses were conducted using SAS version 9.2 (SAS Institute, Cary, NC, USA).

3. Results

The distribution of participant characteristics is reported in Table 1. A roughly equal distribution of participants self-reported Black ($n = 155$, 31%), Asian ($n = 109$, 22%), Hispanic ($n = 103$, 21%), and White ($n = 129$, 26%) race/ethnicity. The majority (≥60%) of participants were female within each racial/ethnic group except for Whites (50% female). Other sociodemographic characteristics showed considerable variability; namely, White and Asian participants had the highest levels of education and the largest proportions were employed during the day. Cardiovascular risk factor profiles were also more favorable among White and Asian participants – fewer were current smokers, had lower depressive symptom scores and BMI, and had a lower prevalence of overweight, obesity, diabetes, and hypertension than Black and Hispanic participants. Two exceptions were that both Black and Hispanic participants reported more minutes of physical activity per week than Whites and Asians did.

Sleep characteristics were roughly normally distributed within race/ethnic groups, but they reflect variability by race/ethnicity (Table 2). Black participants had the shortest sleep duration, the lowest sleep percentage, and greater minutes of WASO and sleep fragmentation. Self-reported sleep quality (PSQI) was worse and daytime sleepiness (ESS) was among the highest in Black participants. For each sleep characteristic, Hispanic and Asian participants

Table 1
Distribution of sample characteristics by race/ethnicity.

	Total	Black	Asian	Hispanic	White
N	496	155	109	103	129
Age, years	47.8 (8.2)	49.1 (8.3)	48.9 (7.6)	46.6 (8.1)	46.2 (8.4)
Sex, % female	60%	66%	60%	66%	50%
Education, years	15.6 (3.9)	14.7 (3.2)	18.3 (2.8)	12.2 (4.4)	17.1 (2.5)
Work schedule, %					
Retired/unemployed	31%	45%	19%	36%	19%
Employed – day shift	51%	36%	70%	42%	59%
Employed – other shift	19%	18%	11%	22%	22%
Smoking status, %					
Current	19%	32%	5%	20%	16%
Former	20%	16%	18%	18%	28%
Never	61%	52%	77%	61%	57%
Physical activity, min/week	753.9 (1027.4)	828.5 (1038.2)	463.9 (621.3)	1160.5 (1559.3)	627.3 (669.7)
Depressive symptoms, CES-D score	11.5 (9.6)	12.6 (9.0)	10.4 (9.8)	12.0 (8.9)	10.5 (10.5)
Body mass index, kg/m^2	26.3 (4.5)	28.2 (4.5)	23.0 (3.1)	28.5 (4.3)	25.1 (3.6)
Weight status, %					
Normal weight	44.4	27.7	80.7	18.5	54.3
Overweight	32.3	31.6	16.5	47.6	34.1
Obese	23.4	40.7	2.8	34.0	11.6
Glucose, mg/dL	91.8 (17.4)	91.3 (16.3)	91.7 (9.9)	96.2 (25.3)	89.2 (15.4)
Hemoglobin A1c, %	5.7 (0.6)	5.8 (0.6)	5.7 (0.4)	5.8 (0.8)	5.5 (0.5)
Diabetes, %	5.4	6.5	2.8	11.7	1.6
Systolic blood pressure, mmHg	115.3 (14.3)	121.9 (14.6)	112.3 (14.0)	112.2 (13.2)	112.6 (12.5)
Diastolic blood pressure, mmHg	71.6 (10.4)	76.6 (10.5)	69.5 (9.7)	69.1 (9.5)	69.2 (9.6)
Hypertension, %	16.9	35.5	8.3	13.6	4.7

Table 2
Distribution of sleep characteristics by race/ethnicity.

	Mean (SD)	Median (interquartile range)	Minimum	Maximum
Sleep duration (min)				
Black	409.3 (67.3)	410.6 (91.3)	116.5	589.6
Asian	413.0 (50.9)	416.5 (61.1)	201.7	555.5
Hispanic	417.0 (79.5)	433.7 (107.3)	60.1	592.7
White	444.7 (52.7)	449.6 (59.7)	200.3	660.2
Sleep percentage (%)				
Black	87.6 (5.4)	88.2 (7.3)	68.9	97.4
Asian	90.7 (4.8)	91.7 (5.2)	60.7	96.0
Hispanic	89.7 (4.4)	90.5 (5.6)	71.5	96.8
White	91.5 (3.6)	92.5 (3.9)	72.4	96.9
Wake after sleep onset (min)				
Black	52.5 (27.1)	47.4 (31.4)	9.3	176.3
Asian	38.6 (18.4)	34.9 (21.7)	12.4	140.8
Hispanic	43.4 (19.6)	40.4 (21.7)	11.1	103.9
White	37.8 (16.7)	33.1 (17.7)	15.3	121.2
Sleep fragmentation (%)				
Black	23.1 (7.7)	22.9 (10.5)	5.3	47.5
Asian	18.3 (8.4)	15.8 (9.2)	5.6	60.1
Hispanic	20.3 (7.7)	18.5 (9.6)	7.6	48.0
White	18.3 (7.2)	16.5 (8.6)	6.9	45.1
Pittsburgh Sleep Quality Index				
Black	6.8 (3.3)	7.0 (3.0)	0	18
Asian	5.4 (2.7)	5.0 (4.0)	0	16
Hispanic	5.8 (3.3)	5.0 (4.0)	0	16
White	5.0 (3.0)	4.0 (3.0)	1	15
Epworth Sleepiness Scale				
Black	7.2 (4.0)	6.0 (5.0)	0	19
Asian	7.6 (4.0)	8.0 (5.0)	0	20
Hispanic	6.9 (4.3)	6.0 (5.0)	0	22
White	5.9 (3.9)	5.0 (5.0)	0	18

Table 3
Adjusted means (95% confidence intervals) of association between race and sleep characteristics.

	Black	Asian	Hispanic	White (Referent)
Sleep duration, min	399.5** (388.3, 410.7)	409.6** (394.7, 424.5)	411.7** (397.4, 426.1)	447.4 (435.7, 459.1)
Sleep percentage, %	87.9** (87.1, 88.8)	89.7 (88.6, 90.8)	89.5 (88.5, 90.6)	90.9 (90.0, 91.8)
Wake after sleep onset, min[a]	50.2** (46.4, 54.0)	43.0 (38.0, 48.1)	43.5 (38.6, 48.4)	41.2 (37.2, 45.1)
Sleep fragmentation	22.9** (21.5, 24.3)	20.0 (18.2, 21.8)	20.9 (19.1, 22.7)	19.5 (18.0, 20.9)
Pittsburgh Sleep Quality Index	6.6** (6.0, 7.2)	5.6 (4.8, 6.4)	6.0 (5.3, 6.8)	5.2 (4.6, 5.9)
Epworth Sleepiness Scale[a]	7.1* (6.4, 7.8)	7.4** (6.5, 8.4)	6.9 (6.0, 7.9)	5.8 (5.1, 6.6)

Adjusted for age, gender, education, work schedule, smoking status, depressive symptoms, BMI, hypertension, and diabetes.
* $p < 0.05$, ** $p < 0.01$.
[a] Interaction term between education and race included in the model.

had values between those of Black (least favorable) and White (most favorable) participants.

Prior to modeling, the absence of statistical interaction by gender for the relationships of race with sleep duration ($F = 1.2$, $p = 0.31$), sleep percentage ($F = 1.32$, $p = 0.26$), WASO ($F = 1.28$, $p = 0.28$), fragmentation ($F = 0.87$, $p = 0.45$), PSQI ($F = 0.67$, $p = 0.57$), and ESS ($F = 1.78$, $p = 0.15$) was tested and confirmed. All analyses were pooled by gender. There was no evidence of statistical interaction by education level for sleep duration ($F = 2.21$, $p = 0.09$), sleep percentage ($F = 1.78$, $p = 0.15$), or fragmentation ($F = 1.14$, $p = 0.33$). A significant interaction of race with WASO ($F = 2.81$, $p = 0.04$) and daytime sleepiness ($F = 3.6$, $p = 0.01$) was found. Among Black and Hispanic participants, there was an inverse association of education with WASO, but there was no such association among Asian or White participants. Education was positively associated with daytime sleepiness among Black participants, but there was no association or an inverse association in the other race/ethnic groups. When the associations adjusted for sociodemographic characteristics, health behaviors, and cardiovascular disease risk factors were modeled, an interaction term for education was included in the models with WASO and daytime sleepiness as outcomes (Table 3). Black, Asian, and Hispanic participants each had significantly ($p < 0.01$) shorter average sleep duration than did White participants. However, only Black participants had significantly lower sleep percentage, more minutes of WASO, greater sleep fragmentation, and poorer self-reported sleep quality (PSQI) than White participants; the differences in those same measures comparing Hispanic and Asians with Whites were not statistically significant. Asians did report significantly ($p < 0.01$) higher daytime sleepiness (ESS) than Whites did.

Restricting the sample to 403 day-shift workers and unemployed/ retired workers did not substantially change these associations (data not shown). Similarly, restricting to the subset of participants with AHI ≤ 5 did not change the results (data not shown).

4. Discussion

We observed patterns of objectively determined sleep duration and self-reported sleep quality that were consistent with our primary hypothesis that non-Whites in our sample (ie, Black, Asian, and Hispanics) would have shorter sleep duration, worse subjective sleep quality, and more daytime sleepiness than Whites. However, contrary to our secondary hypothesis, accounting for differences in social, psychological, behavioral, and clinical characteristics across groups did not attenuate these differences. Findings from our population-based observational study show that racial/ ethnic differences in sleep duration and quality cannot be attributed to common covariates measured in the present study.

Our findings are consistent with prior reports that are based on self-reported measures of both sleep and sleep symptoms by race/ ethnicity and socioeconomic status. In one of the largest studies of 32,749 adults from the National Health Interview Survey (NHIS), Hale et al. [8] reported that Black participants were 41% more likely to self-report being short sleepers and 62% more likely to report being long sleepers than White participants were; Hispanics and "others" were 26–35% more likely to report being short sleepers. In a 2013 report from the National Health and Nutrition Examination Survey (NHANES), race/ethnic minorities were more likely to report adverse sleep symptoms [27]. For example, Black participants were more likely to report longer sleep latency than White participants were, and Hispanics/Latinos reported snoring more often than White participants did. Grandner et al. [27] indicated that the reporting varied by how questions were worded, which favors the objective assessment of sleep. The findings were similar to other studies that relied on self-report [9,10,12,14,16,27,28].

Despite relatively low correlations between self-reported and objective measures of sleep duration as described in the Coronary Artery Risk Development in Young Adults (CARDIA) study ($r = 0.47$) [29], findings are similar when sleep is objectively determined. The CARDIA study captured sleep using wrist actigraphy over 6 nights in 669 participants, and sleep duration ranged from a high of 6.71 h (standard deviation [SD] = 0.89) per night in White women to a low of 5.10 h (SD = 1.30) per night in Black men [11]. Disparities in CARDIA were also apparent by self-reported income (which in the US is closely correlated with race/ethnicity), whereby household income was positively associated with sleep duration and sleep efficiency and inversely associated with latency and time in bed. Although the absolute difference between groups amounts to a little

over an hour and a half, meta-analyses suggest that crossing the threshold into "short" sleep is associated with significantly increased risks of hypertension, diabetes, and mortality [2,3,7].

One strength of our study over prior studies that used actigraphy to capture sleep is that most prior studies compare only two racial/ethnic groups – most commonly Black and White adults. By contrast, we additionally included Asians and Hispanics whose sleep has been less frequently characterized. As expected, Black participants in our study had less favorable sleep characteristics than Whites, according to each objective and self-reported measure. However, there is ample evidence that the rates of metabolic disorders are higher in Hispanics than in Whites and lower in adults of Asian ancestry than in Whites; however, few differences in sleep characteristics other than duration and sleepiness (Asians only) were seen in comparison with Whites. Our findings contradict those of the Study of Women's Health Across the Nation (SWAN) and NHIS, which describe variability in sleep characteristics across these groups [8,9,15].

Unexpectedly short sleep duration and more reports of daytime sleepiness were observed in Asian participants than in Whites. Prior studies have observed similar or even higher rates of sleep apnea among Asians despite lower BMI [30], which may be due to differences in their craniofacial structure resulting in a smaller airway [31]. However, participants with apnea were excluded using questionnaires to identify participants with a high likelihood of apnea and using apnea-screening equipment. Thus, the shorter sleep duration and greater daytime sleepiness observed in Asians as compared to Whites may not be attributable to apnea but rather to other unknown, and thus unmeasured, factors. Furthermore, the previously identified correlates of short sleep were not observed in Asians. For example, Asians had more education, lower BMI, and fewer cardiovascular risk factors at baseline. The only notable difference was that physical activity levels were lower in Asians than for other racial/ethnic groups. Additional research on the contribution of cultural factors, attitudes towards sleep and family, and household environment is warranted to explore the racial/ethnic differences in sleep.

Given the high rate of metabolic disorders in the Hispanic population overall and in this sample, it was unexpected that most sleep characteristics, with the exception of duration, were similar between Whites and Hispanics. A prior study in SWAN identified greater reporting of sleep complaints among US-born Latinas and Asians and among immigrant Latinas and Japanese (but not Chinese) with greater English-language acculturation [15]. We did not capture details regarding acculturation in our cohort, so we may have missed an important source of variability in perceptions of sleep among Hispanics and Asians. Although the findings reported in SWAN relied on self-reported complaints, which may be more susceptible to bias arising from social or cultural factors, the present study captured objective measures of sleep.

A primary advance of our study over previous research is that we attempted to exclude participants with moderate to severe OSA using screening equipment in addition to questionnaires. These findings were additionally confirmed in the subset of adults with a very low likelihood of apnea (AHI < 5). Prior research has consistently demonstrated that sleep apnea is associated with adverse metabolic and cardiovascular disease risk. However, OSA is estimated to affect 2–4% of the population [32], which indicates that the very large remaining proportion of the population show variability in sleep duration and quality that can be attributed to psychological, social, behavioral, and environmental factors.

It is possible that genetic factors could contribute to differences in sleep duration. However, the majority of research on sleep genetics is related to sleep disorders [33] or genetically "short sleepers" who do not have a biological need for >5 h of sleep per night [34]. Studies on genes related to the range of regular sleep duration are scarce. Further, if genes were the source of racial/ethnic

variability in sleep duration and efficiency, then they would be differentially distributed by race/ethnicity in the population. Few studies have a large enough sample size of non-Whites to explore the plausibility of the genetic hypothesis. In addition, focusing attention on an immutable characteristic such as genes does not permit an opportunity for intervention to modify sleep behaviors.

Although the additional strengths of our study include population-based sampling required to describe the distribution of sleep in the general population, seven days of actigraphy, and standardized data collection in a research clinic setting, our findings should be interpreted in light of some limitations. The primary aim of our study was to study sleep characteristics in adults without OSA, so adults with a high likelihood of apnea were excluded based on symptom screeners (ie, Berlin and STOP-BANG). These symptom screeners indicate that adults with common cardiovascular disease risk factors such as hypertension are at high risk. Given that the rates of hypertension are much higher in Black and Hispanic adults than in White adults [35], our sample distribution by race and ethnicity may have been biased towards a healthier sample of Black and Hispanic study participants and may not be representative of Black and Hispanic adults in the US. However, even with this limitation, shorter sleep duration was still observed among Black and Hispanic participants as compared with White participants. Although our sample size is relatively large compared with other studies that included objective determination of sleep, we did not have sufficient power to include more potential confounders in our statistical models. Similarly, we were underpowered to evaluate interaction in our sample. We did observe interactions by education level for two outcomes, WASO and daytime sleepiness, but those primarily indicated an isolated effect in Black and Hispanic adults, who were also more likely to have lower levels of education. Future studies with larger sample sizes should investigate whether education exerts an independent effect on these sleep characteristics across race/ethnic groups. Although findings from other studies indicated the role of psychological stressors such as perceived discrimination on sleep [36], we did not include a comprehensive battery of scales to assess discrimination, perceived stress, anxiety, or other psychological factors that could vary by race/ethnicity and interfere with sleep. Depressive symptoms were assessed; despite the differences in the presence of symptoms by race/ethnicity, statistical adjustment for depressive symptoms did not change the patterns observed in this study. Finally, only a single measure of socioeconomic status, years of education completed, was included. Prior studies have consistently demonstrated that numerous markers of socioeconomic status including income, occupational classification, and hours worked were associated with sleep duration [9,12,37,38]. It is possible that the racial/ethnic differences observed here can be attributed to residual confounding by socioeconomic status.

In summary, shorter objectively determined sleep duration was observed in non-Whites than in Whites in this population-based sample of adults with a low likelihood of sleep apnea that persisted following statistical adjustment for known correlates of short sleep and poor-quality sleep. Future studies with larger sample sizes and more comprehensive measurements of psychological and social characteristics may be able to extend beyond our findings by investigating factors that contribute to variability in sleep by race/ethnicity. These observed patterns in sleep differences remain important, given the potential for racial/ethnic differences sleep characteristics to contribute to disparities in the onset of cardiovascular and metabolic disease.

Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2015.07.005.

Acknowledgments

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