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The Association of Race/Ethnicity with Objectively Measured Sleep Characteristics in Older Men

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Abstract

This study examined the association between race/ethnicity and objectively measured sleep characteristics in a large sample of older men. Black men had significantly shorter total sleep time (6.1 hr vs. 6.4 hr), longer sleep latency (28.7 min vs. 21.9 min), lower sleep efficiency (80.6 % vs. 83.4 %), and less slow-wave sleep (4.9 % vs. 8.8 %) than White men, even after controlling for social status, comorbidities, body mass index, and sleep-disordered breathing. Hispanic men slept longer (6.7 hr) at night than Black (6.1 hr) and Asian American men (6.1 hr). This study supports significant variations in sleep characteristics in older men by race/ethnicity.

Keywords

sleep; race/ethnicity; geriatrics; cohort study

The high prevalence of sleep disturbances in older adults has been well-documented. Older adults complain of waking up more often during the night, experiencing shorter sleep

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duration, taking longer to fall asleep, and taking more naps than younger adults (Feinsilver, 2003; Foley et al., 1995; Klerman, Davis, Duffy, Dijk, & Kronauer, 2004; Kryger, Monjan, Bliwise, & Ancoli-Israel, 2004). Also, changes in sleep architecture with age have been documented: Percentage of stage 1 sleep and stage 2 sleep increase, and percentage of slow-wave sleep (Stages 3 and 4) decreases with age (Bliwise, 2005; Feinsilver, 2003; Ohayon, Carskadon, Guilleminault, & Vitiello, 2004). In a meta-analysis including 65 studies with sleep data collected using polysomnography or actigraphy, it was reported that sleep latency, percentages of stage 1 and stage 2 sleep significantly increased with age, while percent of REM sleep decreased (Ohayon et al., 2004).

Racial/ethnic variations in sleep have gained increasing recognition in recent years. The decrease in slow-wave sleep and the increase in Stages 1 and/or 2 in Black compared with White are the most consistent characteristics across the lifespan (Hall et al., 2009; Mezick et al., 2008; Profant, Ancoli-Israel, & Dimsdale, 2002; Redline et al., 2004; Stepnowsky, Moore, & Dimsdale, 2003). In their population-based Sleep Heart Health Study (SHHS), which used in-home polysomnography for one night, Redline and colleagues (2004) reported that Black participants and American Indians showed lighter sleep (i.e., higher percentage of Stage 2, lower percentage of slow-wave sleep) than Asian American, Hispanic, or White participants. As measured by subjective sleep quality and multiple nights of in-home polysomnography of midlife women (Hall et al., 2009), complaints of sleep quality were higher and slow-wave sleep was lower in Black participants than in White. Studies have also reported that Black participants have either a shorter or a longer sleep duration (Hale & Do, 2007) and take more time to fall asleep with less sleep efficiency than other race/ethnic groups (Mezick et al., 2008). Even among school-aged children, non-White individuals were more likely to have shorter sleep and later bed time than White children (Spilsbury et al., 2004).

Sleep differences across racial/ethnic groups in the older population have been reported, although only a few studies targeted older adults. In a study that used ambulatory monitoring, older Black participants had longer wake time after sleep onset than older White participants. However, when adjustments were made to compensate for socioeconomic status and other health covariates, older White participants woke up significantly more often than their Black counterparts, suggesting that White participants were experiencing more, but short awakenings (Fiorentino, Marler, Stepnowsky, Johnson, & Ancoli-Israel, 2006). Older Black participants also reported less satisfaction with sleep, more difficulty falling asleep, and more frequent and longer napping than their White counterparts (Ancoli-Israel et al., 1995).

Accounting for race/ethnicity in sleep research is important because of its association with specific sleep disorders and other health outcomes. For example, Black participants had a lower prevalence of periodic limb movements during sleep than White participants in a recent study (Scofield, Roth, & Drake, 2008). The prevalence of sleep-disordered breathing (SDB) was higher in Black than White participants in other studies (Ancoli-Israel et al., 1995; Redline et al., 1997). As one of the symptoms of SDB, snoring was more frequent among Black and Hispanic women and Hispanic men than their White non-Hispanic counterparts (O'Connor et al., 2003). Moreover, a recent study reported that Black participants with sleep disturbances had significantly poorer physical health compared with White participants. Hispanic participants with sleep disturbances had significantly poorer mental health than White participants (Baldwin et al., 2010). The different sleep needs of racial/ethnic groups may develop from multidimensional mechanisms involving socioeconomic and cultural factors, genetic/biological differences, and interactions within these factors (Hale & Do, 2007; Rao, Hammen, & Poland, 2009; Villaneuva, Buchanan, Yee, & Grunstein, 2005). Investigating sleep across racial/ethnic groups and identifying the

mediating factors may reduce sleep problems and improve other health outcomes if sleep intervention is tailored to patients' individual needs and characteristics.

Although an increased number of recent studies have evaluated objective measures of sleep in Blacks, White, and other racial groups (e.g., Asians), most of the studies of sleep across race/ethnicity have focused on young and middle-aged populations. Also, the inclusion of more ethnically diverse populations in sleep research is still limited. Finally, the degree to which covariates uniquely contribute to dimensions of sleep across race/ethnicity remains uncertain. Most of the prior studies did not adequately collect data on potential covariates such as socioeconomic status, comorbidities, and other lifestyle factors that might explain the differences in sleep characteristics across racial/ethnic groups. And, even those studies that included a broad range of covariates reported inconsistent results. For example, although recently published studies (Hall et al., 2009; Mezick et al., 2008) have reported that race/ethnicity and sleep relationships remained statistically significant after adjustment for socioeconomic status, another study reported no significance in racial/ethnic difference on wake after sleep onset once data were controlled for health-related covariates and socioeconomic status (Fiorentino et al., 2006).

Purpose of Study

Using data from the Outcomes of Sleep Disorders in Older Men (MrOS Sleep) Study, we tested whether race/ethnicity was associated with differences in objectively measured characteristics of sleep in older men. Our secondary objective was to test whether the associations between race/ethnicity and sleep characteristics could be explained by specific sleep disorders (e.g., SDB), medical comorbidities, socioeconomic status, or other factors. These findings may elucidate the specific needs of population subgroups and accelerate the development of interventions for sleep promotion and sleep hygiene.

Osteoporotic Fractures in Men Study

Background

Participants and study design—The subjects of our study were participants in the Osteoporotic Fractures in Men (MrOS) Study, a large, multicenter, observational study of community-dwelling men aged 65 and older. The MrOS study was initially designed to examine the risk factors related to bone mass, bone geometry, lifestyle, anthropometric and neuromuscular measures, and falls and to determine the effect of fractures on quality of life in older men (Orwoll et al., 2005). The study population consisted of community-dwelling older men aged 65 years or older who met the following criteria: (a) ambulatory without any assistance, (b) absence of bilateral hip replacements, (c) residence near a clinical site for the duration of the study, (d) no medical condition that would result in imminent death, and (e) the ability to provide an informed consent and self-report data (Orwoll et al., 2005). Recruitment was conducted initially by mailing invitations to potential participants and subsequently by newspaper advertisements and presentations to community groups.

During the baseline clinic visit (from March 2000 through April 2002), 5,994 individuals were enrolled at six clinical centers (Birmingham, AL; Minneapolis, MN; Palo Alto, CA; Monongahela Valley near Pittsburgh, PA; Portland, OR; and San Diego, CA). Further details of the MrOS study and the cohort are published elsewhere (Blank et al., 2005; Orwoll et al., 2005).

Outcomes of Sleep Disorders in Older Men Study

Background

From December 2003 through March 2005 (an average of 3.4 years after baseline), MrOS participants were recruited for an ancillary study, the Outcomes of Sleep Disorders in Older Men (MrOS Sleep) Study. The purpose of the MrOS Sleep Study was to comprehensively investigate the association of sleep characteristics with incident cardiovascular disease and a variety of other age-related outcomes in older men (Mehra et al., 2007).

The inclusion and exclusion criteria for the sleep study were minimal. Men were screened for their use of mechanical devices during sleep such as a pressure mask for sleep apnea (continuous positive airway pressure or bilevel positive airway pressure), a mouthpiece for snoring or sleep apnea, or oxygen therapy. Those who reported nightly use of any of these devices were excluded from the study.

Of the 5,994 participants who initially enrolled in the parent study, 3,135 participants enrolled in the MrOS Sleep Study and completed a clinic visit. The balance of the MrOS participants ($n = 2,860$) were ineligible to participate in the sleep study for these reasons: unwillingness to participate ($n = 1,997$), death before being contacted ($n = 344$), enrollment was met before contact was made ($n = 332$), receiving therapy for sleep apnea ($n = 150$), and termination before the sleep study visit ($n = 37$). Of the 3,135 MrOS Sleep Study participants, 2,862 had complete sleep data (polysomnography and wrist actigraphy) and were included in our analysis sample. The protocols for the MrOS and MrOS Sleep Studies were approved by institutional review boards at each site and informed written consent was obtained from all participants.

Actigraphy—Sleep/wake activity was recorded with the Sleep-Watch-O (Ambulatory Monitoring, Inc., Ardsley, NY), a small actigraph that is worn on the nondominant wrist and measures movement using a piezoelectric bimorph-ceramic cantilevered beam. Participants were asked to wear the device for a minimum of five consecutive 24-hour periods beginning with the date of their clinic visit. Except when participants were bathing or swimming, data were collected continuously and stored in 1-min epochs. Data collected in the digital integration mode were used for this sleep data analysis. This mode of data collection has been validated against polysomnography in older men (Blackwell, Redline, Ancoli-Israel, & Stone, 2007) and women (Blackwell et al., 2008).

Participants were asked to complete a diary while wearing the actigraph; the sleep diary data were used to edit and score the actigraph data. The protocol, which was very similar to that in the Study of Osteoporotic Fractures, has been described previously (Blackwell et al., 2005). Each diary included data on time in and out of bed, estimated time when participants fell asleep and awoke, the number of times they thought they woke during the night, information about naps, time and reasons for removing the actigraph, and times the participants were inactive for long durations as when watching television or movies. Wakeful periods that required the removal of the actigraph (e.g., bathing or water sports) were coded as *awake*. If the data suggested that the actigraph had been removed but no relevant information had been entered into the diary, the timepoints were removed. Data were scored using the Action W-2 software (Ambulatory Monitoring, Inc.), which uses the scoring algorithm to determine sleep/wake status developed by the University of California, San Diego (Cole, Kripke, Gruen, Mullaney, & Gillin, 1992).

On average, the actigraphs were worn for 5.11 days ($SD = 0.86$). Five sleep characteristics were derived from actigraphy and used in this analysis: total sleep time (TST): the average nightly total minutes scored as sleep during the in-bed interval; sleep latency (SL): the

average time to sleep onset after the participant reported going to bed and turning the lights out; sleep efficiency (SE): the average nightly percent of the in-bed interval scored as sleep; wake after sleep onset (WASO): the average nightly minutes of wake during the in-bed interval, after initial sleep onset; and daytime napping: average daily minutes of sleep during the out of bed interval.

Polysomnography—In-home polysomnography (Safiro, Compumedics, Inc., Melbourne, Australia) was performed for one night in each participant's home. Certified technicians visited the participants' homes to set up and calibrate the sleep study units in the evening and to collect them in the morning. The following channels were recorded: C₃/A₂ and C₄/A₁ electroencephalogram, bilateral electrooculogram, bipolar submental electromyogram, thoracic and abdominal respiratory inductance plethysmography, airflow (by thermister and nasal pressure recordings), finger pulse oximetry, electrocardiogram, body position sensor, and bilateral tibial leg movement by piezoelectric sensors. The collected data were transferred to the Case Western Reading Center and scored by a trained technician. The timing of sleep was based on a participant's self-report, which was recorded on a morning survey administered the morning after the polysomnography study. Sleep stages and arousals were scored using standard criteria (American Sleep Disorders Association Report, 1992). The percentages of total sleep time spent in Stages 1 and 2, slow-wave sleep, and REM were computed from the polysomnographic data. Total sleep time was not included among the polysomnography measures.

SDB was quantified by the respiratory disturbance index (RDI: number of apneas, hypopneas, and other respiratory disturbances associated with a 3% or more oxygen desaturation per hour of sleep). We also examined the arousal index, defined as the total number of arousals during the sleep interval, divided by total number of hours of sleep. Further details concerning methods for polysomnography have been published previously (Mehra et al., 2007).

Although total sleep time and other sleep-wake parameters are available from polysomnography, we used the corresponding variables based on actigraphy because these may provide a more representative estimate of usual sleep experience given that the measurements are less invasive and averaged over several nights.

Demographic, lifestyle, and medical factors—Participants completed a self-administered questionnaire that provided information on their age, race/ethnicity, level of education, marital status, medical history, medications, tobacco use, and physical activity. Race/ethnicity was categorized as White, Black, Asian American, Hispanic, and other, which included American Indian or Alaska Native, Native Hawaiian or other Pacific Islander, and other mixed race/ethnicity. The participants who indicated a Hispanic background were classified as *Hispanic*, regardless of the race category selected.

Educational level, marital status, and smoking status were also assessed during the MrOS Study's baseline examination. Participants were asked about their medical history of selected conditions: angina, congestive heart failure, coronary or myocardial infarction, diabetes, high blood pressure, and pulmonary disease (asthma, chronic bronchitis, chronic obstructive pulmonary disease, or emphysema).

Information about the use of sleep medications (e.g., benzodiazepines and selective serotonin reuptake inhibitors) was also collected during the baseline clinic interview. The participants were asked to bring their current prescription medications to the clinic visit where they were recorded by the study staff.

A Physical Activity Scale for the Elderly was used to assess the level of the participants' physical activity. The instrument is comprised of self-reported occupational, household, and leisure activities over a 1-week period (Washburn, Smith, Jette, & Janney, 1993). Higher scores represent higher frequency of physical activity.

Social status and alcohol intake (number of alcoholic beverages per week) were obtained using an interviewer-administered questionnaire. The MacArthur Scale of Subjective Social Status, which was developed to capture the common sense of social status across socioeconomic status indicators (education, income, occupation), was administered to assess subjective social status (Adler, Epel, Castellazzo, & Ickovics, 2000). The study used the variable from the social ranking that participants perceive in their communities.

Physical and psychological measures—Body mass index (BMI) was calculated as weight (kg)/height (m²). Body weight was measured with a standard balance beam or a digital scale; height was measured using a standard held-expiration technique with a wall-mounted stadiometer.

Depressive symptoms were evaluated by the Geriatric Depression Scale (Sheikh & Yesavage, 1986), a validated self-report questionnaire designed for elderly participants that consists of 15 yes/no questions about depressive symptoms. The scale is analyzed as a dichotomous variable based on the standard cutoff of six or more symptoms that define clinically significant depression; this cutoff has a sensitivity of 91% and a specificity of 65% compared with Diagnostic and Statistical Manual of Mental Disorders-IV (Almeida & Almeida, 1999).

Statistical Analysis—Characteristics of participants were compared across categories of race/ethnicity using analysis of variance for continuous data, the Kruskal-Wallis Test for skewed continuous data, and chi-square tests for categorical data. Fisher's Exact Test was performed for characteristics with very low frequency.

Characteristics associated with race/ethnicity ($p < 0.10$) were tested for association with sleep variables. Those factors associated with any of the sleep variables with $p < 0.10$ were included in multivariate models. General linear models were used to calculate adjusted means (i.e., least squared means reported with 95 % confidence interval) and to assess the significance of the association between race/ethnicity and sleep outcomes. Age-adjusted models and multivariate models with and without adjustment for RDI were analyzed. Posthoc pairwise tests with a Bonferroni correction were used to test differences across groups. Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS Inc., Chicago, IL) 15.0.

Results of Analysis

Participant Characteristics

Of the 3,135 men who attended the sleep visit, 2,862 (91.3 %) had adequate sleep data for analysis. Compared with the men ($n = 3,133$) who were not enrolled for a sleep visit or did not have complete sleep measures, the 2,862 men in the cohort we analyzed tended to be slightly younger (73.0 vs. 74.3; $p < 0.001$), to be more educated (78.5 % vs. 74.1 % reported having completed some college or beyond; $p < 0.001$), to have slightly higher subjective social status (6.96 vs. 6.80; $p < 0.001$), to have a higher proportion who reported excellent or good health (88.6 % vs. 83.0%; $p < 0.001$), and to be less likely to report depressive symptoms (6.4% vs. 10.0%; $p < 0.05$) and smoking (4.0% vs. 6.8%; $p < 0.001$). There were no significant differences in body mass index or alcohol intake. Mean age of the participants was 76.36 ± 5.51 (range 67 to 96 years old). Most were White ($n = 2,598$, 90.8 %).

Participant characteristics by race/ethnicity are shown in Table 1. White participants (76.5 ± 5.6 years old) were significantly older than Black participants (74.0 ± 4.5 years old) and had a significantly higher subjective social status. Also, the level of education was significantly different between some racial/ethnic groups: More than half (56.1%) of Asian American men had high levels of education, including some graduate or graduate school, compared with 26 % of Black men. Average physical activity levels and BMI were the lowest for Asian American participants. Black men were more likely to report a history of diabetes and high blood pressure.

Sleep Characteristics

As shown in Table 2, Black participants had significantly less total sleep time than their White and Hispanic counterparts and lower sleep efficiency than White participants. Black participants also had longer SL, more WASO, and lower percentage of slow-wave sleep than White participants. No significant racial/ethnic differences were found in the RDI or percentage of REM sleep.

Table 3 indicates further exploration of one of significant sleep variables differed by race/ethnicity. We focused on the total sleep time because most epidemiological studies using self-report have focused on total sleep time (Coyne et al., 2003; Gottlieb et al., 2006; Hale & Do, 2007). Participants sleeping 7 to less than 8 hr had higher subjective social status compared with those sleeping less than 5 hr or 5 to less than 7 hr per night. The highest physical activity was shown in the group having 5 to less than 7 hr of total sleep time, although participants sleeping 8 hr or more during nighttime had the lowest physical activity. BMI was highest in the group sleeping less than 5 hr and less so in groups sleeping 5 to 7 hr, more than 8 hr, and 7 to 8 hr, respectively. Older men who slept fewer than 5 hr per night were also more likely to be smokers, to have high blood pressure, and a history of congestive heart failure, heart attack, or myocardial infarction.

Table 4 presents adjusted mean sleep characteristics by categories of race/ethnicity. When adjusting for age only, most of the sleep characteristics showed significant variation across categories of race/ethnicity, with the exception of percentages in Stage 1, 2, and REM sleep. Black participants had less actigraphy-assessed total sleep time than White participants (6.0 hr vs. 6.4 hr) and had longer sleep latency (29 min vs. 22 min), lower sleep efficiency (80.0 % vs. 83.3 %) and more WASO (80.9 min vs. 66.8 min) than White participants. Hispanic participants slept longer during the night than Black participants (6.7 hr vs. 6.0 hr) and spent significantly less napping time (31.43 min) than Black (51.24 min) and Asian American (52.09 min) participants. Mean percentage of slow-wave sleep was significantly higher in White (8.8 %) and Asian American participants (8.6 %) than in Black participants (4.7 %).

In a multivariable model (including age, subjective social status, education, marital status, BMI, physical activity, alcohol drinking, comorbidities such as diabetes, hypertension, and heart attack/myocardial infarction, and clinic site), racial/ethnic differences were found in total sleep time, sleep latency, sleep efficiency, and slow-wave sleep. In particular, Black participants had less total sleep time (6.1 hr vs. 6.4 hr), longer sleep latency (29 min vs. 22 min), lower sleep efficiency (80.6 % vs. 83.4 %), and lower percentage of slow-wave sleep (4.9 % vs. 8.8 %) than White participants. Hispanic participants slept more at night (6.7 hr) than Black (6.1 hr) and Asian American (6.1 hr) participants. Results were similar after further adjustment for the RDI.

Discussion

The results of our study show that compared with other racial/ethnic groups, older Black men experienced significantly shorter nocturnal sleep duration, longer sleep latency, lower

sleep efficiency, and less slow-wave sleep than older White men. Hispanic men had longer sleep duration than Black and Asian American men. Furthermore, these differences persisted after adjusting for demographic factors, comorbidities, and the RDI.. In addition, the average difference in sleep duration between Black men (6.1 hr) and White men (6.4 hr) may be clinically significant, given abundant data showing that even modestly reduced sleep duration is associated with an increased risk of numerous health conditions.

The findings of short sleep duration and increased sleep fragmentation in Black men compared with other racial/ethnic groups are consistent with previous studies. Even in a pediatric cohort, where the influences of comorbidities would be relatively small, non-White (mostly Black) boys slept significantly less than White boys and girls and non-White girls even after adjusting for potential confounders such as chronic health problems and caregiver's educational level, suggesting that population differences in sleep patterns manifest early in life (Spilsbury et al., 2004). A large cohort study, the SHHS reported lower sleep efficiency in Black participants than in Asian American participants (Redline et al., 2004). Using 3 days of actigraphy data from 669 adults, researchers found that Black men and women spent less time in bed and had lower mean sleep duration, lower sleep efficiency, and higher sleep latency than their White counterparts (Lauderdale et al., 2006). Our study confirms that these findings also hold true for older community-dwelling Black men even in analyses that carefully consider a comprehensive set of potential confounders.

Of particular note in our study were low levels of slow-wave sleep among Black men. On average, they had 4 % lower slow-wave sleep than White men, with a corresponding increase in Stage 2 sleep, findings that are similar to those reported in other studies (Profant et al., 2002; Rao et al., 1999; Redline et al., 2004; Stepnowsky et al., 2003). Recent research has highlighted the importance of slow-wave sleep in the pathogenesis of metabolic dysfunction and obesity (Spiegel, Tasali, Leproult, & Van Cauter, 2009; Tasali, Leproult, Ehrmann, & Van Cauter, 2008). For example, a laboratory study showed that slow-wave sleep suppression in healthy young adults resulted in significant decreases in insulin release, leading to reduced glucose tolerance and an increased risk of type 2 diabetes (Tasali et al., 2008). Moreover, a higher prevalence of metabolic syndrome (e.g., diabetes) or obesity in Black people compared with White people was reported in a nationally representative United States survey studies (Centers for Disease Control and Prevention, 2003, 2009). These suggest the importance of further research to understand the contributing role of slow-wave sleep reduction in the pathogenesis of diabetes and metabolic diseases in minority populations.

As reported by the SHHS (Redline, et al., 2004), REM sleep did not significantly vary by race/ethnicity. This suggests that intersubject variations in REM sleep may be less than variations in the proportion of time within stages of non-REM sleep.

In addition to differences between Black and White participants, we also observed that Hispanic men have longer nightly sleep duration than Black and Asian American men. Although data on Hispanic men is limited, one small study (Rao et al., 1999) reported that they had higher REM density than did Black and White individuals. A recent study of National Health and Nutrition Examination Surveys data also reported that Hispanic adults, specifically Mexican Americans had longer self-reported sleep duration than the general U.S. sample, an association hypothesized to reflect cultural influences on sleep behaviors (Seicean, Neuhauser, Strohl, & Redline, 2011).

Several demographic, lifestyle, and medical/physical factors were examined as potential mediators of the relationship between race/ethnicity and sleep. However, the relationship was not explained by subjective social status, SDB, or diseases such as diabetes,

hypertension, and cardiovascular disease. The bases for these observed race/ethnic differences in objectively measured sleep parameters are still not clear. Environmental, behavioral, and genetic factors, which were not directly measured in the study, may explain our findings. The relationship between race/ethnicity and sleep is most likely complex and may reflect both population differences in environmental exposures and differences in response to the environment. For example, a study has shown that Black people experience greater reductions in slow-wave sleep in a hospital environment than White people (Stepnowsky et al., 2003). Another recent study suggested that residential context could play a role in explaining sleep difference by race/ethnicity (Hale & Do, 2007). Studies have shown racial difference in genetic associations for sleep apnea traits (Buxbaum, Elston, Tishler, & Redline, 2002; Redline et al., 1997), and it is possible that there are genetic differences in sleep homeostatic processes that track with race/ethnicity. In addition, a recent study reported that Black participants had a shorter endogenous period of the circadian clock, with larger phase advances and smaller phase delays than White participants (Smith, Burgess, Fogg, & Eastman, 2009). Thus, racial/ethnic differences in sleep patterns may be explained by the differences in endogenous circadian rhythms.

The strengths of our study include its large sample size and comprehensive and objective measures of sleep. To our knowledge, this study is the first of its kind to explore racial/ethnic differences in objectively measured sleep characteristics, using both actigraphy and polysomnography, among older community-dwelling men. Most of the participants in the majority of earlier studies of sleep and race/ethnicity were young or middle aged adults (Hale & Do, 2007; Hall et al., 2009; Lauderdale et al., 2006; Mezick et al., 2008).

This study has some limitations, the major ones being the relatively small number of non-White men and generalizability of the study population. Our study is comprised of only older men, and therefore we were unable to examine whether the race/ethnicity associations with sleep are similar in men and women. To our knowledge, no other study with objective measures of sleep has examined this possible interaction between gender and race/ethnicity in relation to sleep. The SHHS ($N = 2,685$, participants aged 37 to 92 years) reported that men had lighter sleep with a higher percentage of Stage 1 and 2 and a lower percentage of slow-wave sleep than women (Redline, 2004). Similar to the findings in our study, a recent study of middle-aged women found more poor sleep in Black than White women in both subjective and objective sleep measures (Hall et al., 2009). Additional studies are needed to determine whether associations between race/ethnicity and sleep are similar by gender. Because the study's findings are largely drawn from volunteer samples of the MrOS parent study, our study sample is not completely representative of *men aged 65 and older living in the United States*. For instance, MrOS participants had slightly higher BMIs than participants in the National Health and Nutrition Examination Survey III (Looker et al., 1995), a cohort randomly selected from the U.S. population. Moreover, each of racial/ethnic groups in our study showed a high proportion of graduate education and low percentage of smokers. This suggests that the study participants should not be considered entirely representative of the general population of older U.S. men.

We had small numbers of participants in minority racial/ethnic groups (e.g., Black, Asian American, and Hispanic individuals), thus limiting the statistical power to detect differences in these groups. Because race/ethnicity was not assessed separately in this study, we do not know the race of those participants who marked only *Hispanic*. Although total sleep time and other sleep-wake parameters are available from polysomnography, our study did not include them. A single night of polysomnography may be particularly problematic because participants' sleep may be affected by equipment that they are not accustomed to. However, data from the SHHS have shown that RDI and sleep architecture can be accurately estimated based on a single night of unattended nonlaboratory polysomnography (Quan et al., 2002).

Moreover, although it was recorded in the participants' homes, where first-night effects are fewer than in the laboratory (Edinger et al., 1997; Edinger et al., 2001), additional nights of testing may have improved the data's validity. In addition, certain racial/ethnic groups may have had a larger first-night effect than other groups (Stepnowsky et al., 2003). Because medical conditions were assessed by self-report, these data may underestimate the prevalence of these conditions and further their relationships with sleep in this sample.

Results are not applicable to younger men, women, or institutionalized elderly men. We did not have information on certain cultural factors or recent life events that could have been potential confounding factors for the associations we observed. Our sample size was insufficient to test interactions between race/ethnicity and other variables (e.g., age and the RDI).

Conclusion

In summary, this study shows significant differences in sleep characteristics across race/ethnic groups that persist after adjusting for a broad range of behavioral and health factors, including the RDI. Overall, Black men demonstrated worse sleep behavior than their White counterparts, whereas Hispanic men had longer sleep at night than Black and Asian American men. Future research is needed to delineate the role of these population differences in sleep and their affect on health disparities, and to better understand the extent to which they reflect environmental and/or genetic determinants.

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Table 1

Participant Characteristics by Race/Ethnicity ($N = 2,862$)

	White	Black	Asian American	Other	Hispanic	P-Value
N (%)	2,598 (90.8)	96 (3.4)	82 (2.9)	31 (1.1)	55 (1.9)	
Age, mean	76.49 ± 5.56	74.03 ± 4.50*	76.10 ± 5.17	74.55 ± 5.09	75.29 ± 4.26	< 0.001
Subjective social status, mean	7.00 ± 1.66	6.23 ± 1.76*	6.63 ± 1.75	6.97 ± 1.94	6.98 ± 1.56	< 0.001
Marital Status, %						< 0.001
Married	84.1	68.8	96.3	90.3	80.0	
Not married	15.9	31.3	3.7	20.0	9.7	
Education, %						< 0.001
High school or below	21.3	42.7	9.8	19.4	14.5	
Some college/college	40.7	31.3	34.1	48.4	50.9	
Some graduate/graduate	38.0	26.0	56.1	32.3	34.5	
Number of alcoholic beverages per week	4.36 ± 6.71	3.35 ± 7.12	2.14 ± 4.24*	2.68 ± 4.88	5.85 ± 7.38 [‡]	< 0.001
Smoking, %	1.9	5.2	0	0	1.8	0.148
Physical Activity, mean	145.50 ± 70.72	150.18 ± 78.33	122.01 ± 62.49*	157.64 ± 82.00	172.71 ± 86.07 [‡]	0.001
BMI, mean	27.23 ± 3.74 [‡]	27.94 ± 4.69 [‡]	24.58 ± 3.10	27.21 ± 4.19 [‡]	27.36 ± 3.88 [‡]	< 0.001
Depression (GDS ≥ 6), %	6.4	10.4	6.1	3.2	5.5	0.527
Comorbidities						
Diabetes, %	12.6	25.0	20.7	22.6	14.6	< 0.001
Pulmonary/lung disease, %	5.2	8.3	4.9	3.2	1.8	0.534
CHF, %	6.1	5.2	4.9	12.9	0	0.128
HTN, %	49.2	62.5	54.9	67.7	54.5	0.020
Heart attack, MI, %	18.1	17.7	8.5	9.7	10.9	0.089
Angina, %	15.5	14.6	11.0	22.6	10.9	0.498
Benzodiazepine, %	4.7	5.2	3.7	3.2	1.8	0.943
SSRI, %	4.6	1.0	1.2	0	5.4	0.207

Abbreviations: BMI, body mass index; GDS, geriatric depression scale, congestive heart failure; HTN, hypertension; MI, myocardial infarction; SSRI, selective serotonin reuptake inhibitors

 $n = 2,861$ for smoking, physical activity, BMI, comorbidities (diabetes, pulmonary/lung disease, CHF, HTN, MI, and angina), Benzodiazepine, and SSRI, $n = 2,860$ for alcohol use, $n = 2,859$ for depression, $n = 2,851$ for subjective social status* $P < 0.05$ compared with Caucasians,

[†] compared with African Americans,
[‡] compared with Asians

Table 2

Sleep Characteristics by Race/Ethnicity ($N = 2,862$)

	White	Black	Asian American	Other	Hispanic	P-Value
N (%)	2,598 (90.8)	96 (3.4)	82 (2.9)	31 (1.1)	55 (1.9)	
Actigraphy						
TST, hours	6.43 $\pm$ 1.23	6.03 $\pm$ 1.29*	6.22 $\pm$ 1.14	6.49 $\pm$ 1.21	6.65 $\pm$ 1.23 [†]	0.006
SL, minutes	21.84 $\pm$ 1.18	29.37 $\pm$ 1.15*	21.91 $\pm$ 0.99	22.34 $\pm$ 1	21.09 $\pm$ 1.18	0.009
SE, %	82.66 $\pm$ 10.40	79.70 $\pm$ 10.15*	82.55 $\pm$ 9.09	82.09 $\pm$ 9.52	83.79 $\pm$ 8.16	0.004
WASO, minutes	77.85 $\pm$ 44.35	90.44 $\pm$ 44.37*	76.84 $\pm$ 38.95	83.37 $\pm$ 47.15	75.97 $\pm$ 41.14	0.023
Napping, minutes	68.51 $\pm$ 59.10	77.51 $\pm$ 72.38	75.72 $\pm$ 73.52	64.49 $\pm$ 55.11	47.43 $\pm$ 40.36 [‡]	0.059
Polysomnography						
Stage 1, %	6.81 $\pm$ 4.15	6.59 $\pm$ 5.48	7.17 $\pm$ 3.57	6.21 $\pm$ 2.19	7.92 $\pm$ 4.58	0.129
Stage 2, %	62.55 $\pm$ 9.63	64.79 $\pm$ 10.32	61.89 $\pm$ 8.79	63.56 $\pm$ 7.76	61.87 $\pm$ 10.10	0.246
Slow-wave sleep, %	11.43 $\pm$ 8.99	7.99 $\pm$ 7.88*	11.03 $\pm$ 8.64	10.35 $\pm$ 8.70	9.02 $\pm$ 7.32	0.001
REM sleep, %	19.24 $\pm$ 6.59	20.71 $\pm$ 7.00	19.79 $\pm$ 5.70	20.03 $\pm$ 6.25	20.51 $\pm$ 7.03	0.147
RDI $\geq$ 15, %	43.4	42.7	42.7	51.6	45.5	0.913
Arousal Index	23.95 $\pm$ 11.88	19.77 $\pm$ 10.83*	21.29 $\pm$ 10.14	21.90 $\pm$ 9.29	22.58 $\pm$ 9.25	0.003

Abbreviations: TST, total sleep time; SL, sleep latency; SE, sleep efficiency; WASO, wake after sleep onset; REM, rapid eye movement; RDI, respiratory disturbance index

Data are given as mean $\pm$ SD $n = 2,823$ for arousal index, $n = 2,848$ for napping, $n = 2,727$ for REM sleep, $n = 2,700$ for slow-wave sleep, $n = 2,668$ for stage 2, $n = 2,583$ for stage 1* $P < 0.05$ compared with whites,[†] compared with blacks,[‡] compared with Asian Americans

Table 3

Participant Characteristics Associated with Total Sleep Time ($N = 2,862$)

	< 5 hours	5 to < 7 hours	7 to < 8 hours	8 + hours	p-value
N (%)	344 (12)	1,602 (56)	704 (24.6)	212 (7.4)	
Age, mean	76.43 ± 5.64	76.12 ± 5.35	76.48 ± 5.72	77.59 ± 5.64 [†]	0.003
Subjective social status, mean	6.75 ± 1.73	6.94 ± 1.67	7.14 ± 1.58* [†]	6.92 ± 1.82	0.003
Marital status, %					0.001
Married	77.9	84.4	87.1	79.7	
Not married	22.1	15.6	12.9	20.3	
Education, %					0.004
High school or below	28.5	21.7	17.2	23.1	
Some college/college	39.0	39.7	42.9	41.0	
Some graduate/graduate	32.6	38.6	39.9	35.8	
Number of alcoholic beverages per week	4.78 ± 8.06	3.91 ± 6.19	4.67 ± 6.74	4.91 ± 7.34	0.012
Smoking, %	4.4	1.6	1.7	0.9	0.005
Physical Activity, mean	140.92 ± 74.03	150.07 ± 72.37	139.87 ± 68.28 [†]	138.99 ± 67.70*	0.003
BMI, mean	29.24 ± 4.66	27.06 ± 3.63*	26.54 ± 3.41* [†]	26.87 ± 3.55*	< 0.001
Depression (GDS ≥ 6), %	7.6	5.4	7.8	7.5	0.112
Comorbidity					
Diabetes, %	17.7	12.9	12.5	13.2	0.091
Pulmonary/lung disease, %	7.6	5.2	4.0	4.7	0.105
CHF, %	11.0	5.6	5.5	2.4	< 0.001
HTN, %	57.6	49.2	49.0	49.5	0.035
Heart attack, MI, %	21.8	15.7	19.3	18.4	0.023
Angina, %	19.5	14.7	15.6	11.8	0.069
Benzodiazepine, %	4.4	3.1	4.9	2.8	0.231
SSRI, %	3.3	3.2	2.0	5.1	0.200

Abbreviations: BMI, body mass index; GDS, geriatric depression scale, congestive heart failure; HTN, hypertension; MI, myocardial infarction; SSRI, selective serotonin reuptake inhibitors

 $n = 2,861$ for smoking, physical activity, BMI, comorbidities (diabetes, pulmonary/lung disease, CHF, HTN, MI, and angina), Benzodiazepine, and SSRI. $n = 2,860$ for alcohol use, $n = 2,859$ for depression, $n = 2,851$ for subjective social status* $P < 0.05$ compared with total sleep time <5 hours,

[†] compared with total sleep time 5 to < 7 hours

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Table 4

Adjusted Mean Sleep Characteristics by Race/Ethnicity

Race/Ethnicity							
		White	Black	Asian American	Other	Hispanic	p-Value
Actigraphy	TST, hr	Model 1 6.4 (6.4–6.5)	6.0 * (5.8–6.3)	6.2 (6.0–6.5)	6.5 (6.1–6.9)	6.7 † (6.3–7.0)	0.009
	Model 2	6.4 (6.4–6.5)	6.1 * (5.8–6.3)	6.1 (5.8–6.3)	6.5 (6.1–7.0)	6.7 †‡ (6.4–7.0)	0.001
	Model 3	6.4 (6.4–6.5)	6.1 * (5.8–6.3)	6.1 (5.8–6.4)	6.5 (6.1–7.0)	6.7 †‡ (6.4–7.0)	0.001
SL, min	Model 1	21.86 (21.18–22.55)	29.27 * (24.94–34.32)	21.91 (18.36–26.1)	22.28 (16.74–29.55)	21.03 (16.99–26.04)	0.015
	Model 2	21.86 (21.18–22.50)	28.72 * (24.41–33.67)	23.43 (19.61–27.97)	22.17 (16.7–29.34)	21.28 (17.2–26.29)	0.025
	Model 3	21.86 (21.18–22.5)	28.72 * (24.47–33.75)	23.27 (19.46–27.77)	21.96 (16.54–28.99)	21.23 (17.16–26.16)	0.024
SE, %	Model 1	83.3 (83.0–83.7)	80.0 * (78.1–82.0)	83.0 (81.0–85.0)	82.4 (79.0–85.7)	84.0 (81.6–86.5)	0.02
	Model 2	83.4 (83.0–83.7)	80.6 * (78.7–82.5)	81.4 (79.3–83.4)	82.5 (79.2–85.6)	84.1 (81.7–86.4)	0.017
	Model 3	83.4 (83–83.7)	80.6 * (78.7–82.4)	81.6 (79.5–83.6)	82.7 (79.5–85.8)	84.1 (81.8–86.4)	0.019
WASO, min	Model 1	66.8 (65.4–68.2)	80.9 * (72.3–90.6)	68.2 (60.4–77.2)	74.0 (60.7–90.2)	67.6 (58.2–78.4)	0.02
	Model 2	66.8 (65.2–68.2)	77.9 (69.8–87.1)	74.0 (65.4–83.5)	73.5 (60.5–89.2)	67.7 (58.4–78.3)	0.034
	Model 3	66.8 (65.4–68.2)	78.1 (70.0–87.3)	73.3 (64.9–82.8)	72.6 (60.0–88.0)	67.6 (58.4–77.9)	0.039
Napping, min	Model 1	45.99 (44.2–47.9)	51.24 (41.6–63.0)	52.09 (41.9–64.9)	48.32 (33.8–68.8)	31.43 †‡ (24.0–41.2)	0.039
	Model 2	46.0 (44.3–47.8)	47.1 (38.3–57.8)	52.5 (42.1–65.5)	47.0 (33.0–66.5)	33.0 (25.2–42.9)	0.109
	Model 3	46.0 (44.3–47.8)	47.1 (38.3–57.8)	52.5 (42.1–65.4)	46.9 (33.0–66.5)	33.0 (25.2–42.9)	0.109
Polysomnography							
Stage 1, %	Model 1	5.9 (5.8–6.1)	5.7 (5.0–6.5)	6.4 (5.6–7.2)	6.0 (4.9–7.4)	6.9 (5.9–8.0)	0.241
	Model 2	5.9 (5.8–6.0)	5.6 (4.9–6.3)	6.5 (5.8–7.4)	6.1 (4.9–7.5)	6.9 (5.9–8.0)	0.140
	Model 3	5.9 (5.8–6.0)	5.6 (4.9–6.4)	6.5 (5.7–7.3)	6.0 (4.9–7.4)	6.9 (5.9–8.0)	0.194
Stage 2, %	Model 1	62.5 (62.2–62.9)	65.1 (63.1–67.2)	61.9 (59.8–64.0)	63.8 (60.4–67.2)	62.0 (59.4–64.7)	0.135
	Model 2	62.5 (62.1–62.9)	64.8 (62.8–66.9)	62.3 (60.1–64.5)	63.8 (60.4–67.2)	62.1 (59.4–64.7)	0.258
	Model 3	62.5 (62.1–62.9)	64.9 (62.8–66.9)	62.2 (60.0–64.3)	63.7 (60.3–67.0)	62.0 (59.3–64.6)	0.246
Slow-wave sleep, %	Model 1	8.8 (8.4–9.2)	4.7 * (3.5–6.3)	8.6 † (6.7–10.8)	7.1 (4.5–10.4)	6.5 (4.6–8.9)	<0.001
	Model 2	8.8 (8.5–9.2)	4.9 * (3.6–6.5)	7.9 (6.0–10.1)	6.9 (4.4–10.2)	6.5 (4.6–8.9)	<0.001
	Model 3	8.8 (8.5–9.2)	4.9 * (3.6–6.5)	8.0 (6.1–10.2)	7.0 (4.4–10.3)	6.6 (4.6–9.0)	<0.001

	Race/Ethnicity					p-Value	
	White	Black	Asian American	Other	Hispanic		
Stage REM, %	Model 1	19.3 (19.0–19.5)	20.4 (19.0–21.7)	19.8 (18.3–21.2)	19.8 (17.4–22.1)	20.33 (18.6–22.1)	0.383
	Model 2	19.3 (19.0–19.5)	20.7 (19.3–22.0)	19.8 (18.3–21.3)	19.8 (17.4–22.1)	20.3 (18.5–22.0)	0.252
	Model 3	19.3 (19.0–19.5)	20.7 (19.3–22.0)	19.9 (18.5–21.4)	19.9 (17.6–22.2)	20.3 (18.6–22.1)	0.204

Model 1: Adjusted for age

Model 2: Adjusted for age, subjective social status, education, marital status, BMI, physical activity, alcohol drinking, comorbidities (diabetes, hypertension, and heart attack/MI), and site

Model 3: Adjusted for age, subjective social status, education, marital status, BMI, physical activity, alcohol drinking, comorbidities (diabetes, hypertension, and heart attack/MI), site, and RDI

* P < 0.05 compared with whites,
† compared with blacks,
‡ compared with Asian Americans

Numbers in parentheses are 95% CI for adjusted means