https://doi.org/10.5664/jcsm.9060

JCSM | Journal of Clinical Sleep Medicine

SCIENTIFIC INVESTIGATIONS

Differences in sleep timing and related effects between African Americans and non-Hispanic Whites

Daniel Combs, MD[1,2,3]; Chiu-Hsieh Hsu[4]; Omavi Bailey, MD[1,2]; Salma I. Patel, MD, MPH[1,2]; Saif Mashaqi, MD[1,2]; Lauren Estep, MD[1,2]; Natalie Provencio-Dean, MS[2]; Silvia Lopez, RN[2]; Sairam Parthasarathy, MD[1,2]

[1]UAHS Center for Sleep and Circadian Sciences, University of Arizona, Tucson, Arizona; [2]Division of Pulmonary, Allergy, Critical Care and Sleep Medicine, Department of Medicine, University of Arizona, Tucson, Arizona; [3]Department of Pediatrics, University of Arizona, Tucson, Arizona; [4]Department of Epidemiology and Biostatistics, Mel and Enid Zuckerman College of Public Health, Tucson, Arizona

Study Objectives: Prior studies have shown a morning chronotype for African Americans compared with non-Hispanic Whites, yet self-reported sleep timing is delayed in African Americans compared with Whites.

Methods: We analyzed data from the Multi-Ethnicity Study of Atherosclerosis, a multisite community-based cohort. Self-reported and actigraphic sleep timing, chronotype measured by the modified Horne-Östberg Morningness-Eveningness Questionnaire, and risk of depression measured by the Center for Epidemiologic Studies Depression scale were examined using nonparametric approaches and linear or logistic regression while comparing between African Americans and Whites and evaluating the effects of delayed sleep phase.

Results: In 1,401 participants, there was no difference in chronotype between African Americans and Whites. African Americans were 80% more likely to report a delayed sleep phase (defined as bedtime after midnight) on weekdays and 50% more likely on weekends than were Whites. Actigraphic data showed similar results. Actigraphic midsleep time was delayed 38 minutes on weekdays and 24 minutes on weekends in African Americans compared with Whites. Stratified analysis by chronotype showed that African Americans with a morning or intermediate chronotype had a significantly delayed sleep phase compared with Whites, but there was no difference between African Americans and Whites with an evening chronotype. Delayed sleep phase was associated with depression, but this relationship was only significant in White participants.

Conclusions: African Americans had a delayed sleep phase compared with Whites that was more pronounced in individuals with a morning or intermediate chronotype. Consequences of delayed sleep phase may vary by race and ethnicity.

Keywords: delayed sleep phase, African American, health disparities, depression

Citation: Combs D, Hsu C-H, Bailey O, et al. Differences in sleep timing and related effects between African Americans and non-Hispanic Whites. *J Clin Sleep Med.* 2021;17(5):897–908.

BRIEF SUMMARY

Current Knowledge/Study Rationale: Prior research has shown that African Americans may have a morning chronotype compared with non-Hispanic Whites but that African Americans have a relatively delayed sleep phase compared with non-Hispanic Whites.

Study Impact: In this large cohort, African Americans showed no difference in chronotype compared with non-Hispanic Whites but were much more likely to have both self-reported and actigraphic delayed sleep phase compared with non-Hispanic Whites, and this difference was more pronounced in individuals with a self-reported morning or intermediate chronotype. In addition, depression was associated with delayed sleep phase, but this relationship was only significant in White participants.

INTRODUCTION

Prior research has suggested the presence of racial/ethnic differences in the circadian rhythm of participants with African ancestry when compared with those of European ancestry.[1,2] In the United Kingdom Biobank study, Blacks were more likely to exhibit morning-type sleep behavior than were Whites.[3,4] Consistent with this finding, African Americans have been reported to have a shorter tau than non-Hispanic Whites, which would predict an earlier chronotype.[1] Prior work has shown that evening chronotype is associated with the latest bedtime compared with intermediate and morning chronotypes, whereas morning chronotype is associated with the earliest bedtime compared to intermediate and evening chronotypes.[5,6] Given these findings, it would be anticipated that because prior literature has shown an earlier chronotype in African Americans than in Whites, delayed sleep phase would be less common in African Americans than in Whites. Paradoxically, previous work has shown African Americans to have a delayed sleep phase as compared with Whites. Specifically, in the Sleep Heart Health Study, a large multisite community-based cohort study, African Americans were twice as likely to report delayed sleep timing (defined as a self-reported bedtime of midnight or later) compared with Whites.[7] In addition, midsleep on free days was delayed in African Americans compared with Whites, consistent with a relative delayed sleep phase.

No measures of chronotype were collected in the Sleep Heart Health Study, therefore it was not possible to evaluate any differences in the chronotypes of African American participants compared with those of White participants as well as the potential impact of chronotype on differences in sleep timing in African American participants compared to White participants. The Sleep Heart Health Study also did not collect data on relevant socioeconomic factors that may influence sleep timing such as household income. In addition, we have only been able to evaluate self-reported sleep timing because no objective measures of habitual sleep were included in the Sleep Heart Health Study. Given our prior findings and associated limitations, we sought to compare objective differences of sleep timing and the potential influence of reported chronotype in African Americans compared with Whites in a large community-based cohort.

The Multi-Ethnicity Study of Atherosclerosis (MESA) is a large multisite community-based cohort with available measures including self-reported and objective measures of sleep and self-reported chronotype. We hypothesized that in the MESA cohort, African Americans would be more likely to have both self-reported and objective delayed sleep phase compared with Whites. Given the prior evidence that does not support an underlying biological tendency toward delayed sleep timing in African Americans, we additionally evaluated whether any underlying chronotype differences were present between African Americans and Whites and whether chronotype had an effect on the likelihood of delayed sleep phase.

Because of the known association between delayed sleep timing and depression,[7–9] we evaluated whether there were any racial differences in the MESA cohort, which was designed to evaluate for such differences. Prior research has found that the association between depression and delayed sleep phase is greater in individuals with a longer phase angle between dim-light melatonin onset and sleep onset (ie, sleep onset is delayed relative to circadian rhythm).[10] Because prior research has suggested that African Americans had a tendency toward an early chronotype compared with Whites,[1–4] we hypothesized that a delayed sleep phase in African Americans would be associated with an increased risk of depression compared with Whites. Studies have shown racial differences in the effects of short sleep duration leading to more severe health consequences in African American individuals than in White individuals.[11,12] This finding suggests that not only may sleep disparities be present, but there may also be subsequent disproportion in the effects of these sleep disparities that may put African Americans at higher risk for health effects because of sleep disparities.

METHODS

Study population

The MESA was designed to evaluate predictors of cardiovascular disease in a diverse group of healthy individuals without existing cardiovascular disease.[13] The initial cohort (recruited in 2000–2002) comprised 6,814 White, African American, Hispanic/Latino, and Chinese adults aged 45–84 years in 6 U.S. communities: Baltimore, MD; Chicago, IL; Forsyth County, NC; Los Angeles County, CA; Northern Manhattan and Southern Bronx, NY; and St. Paul, MN.[14] This initial cohort has been followed sequentially for nearly 20 years with intermittent exams. Exam 5 included the optional MESA sleep ancillary study, which included 1 night of home-based polysomnography, 7 days of actigraphy, and sleep questionnaires. The sleep ancillary study was conducted in 2010–2013. A total of 3,789 MESA participants who denied the use of oral devices, nocturnal oxygen, or PAP devices were invited to participate in the sleep ancillary study, 2,261 of whom agreed to participate.[15]

Data sources

Demographic information, including age, sex, and self-identified race/ethnicity, was obtained from questionnaires completed during Exam 5 of the MESA cohort. Race/ethnicity classifications included non-Hispanic White, non-Hispanic Black/African American, Hispanic, and Chinese. For this analysis, only individuals who self-reported as non-Hispanic White or non-Hispanic Black/African American were included.

Questionnaires from MESA Exam 5 were used to determine relevant variables, including smoking history (pack years), self-reported current alcohol use, and caffeine consumption (total mgs per day as computed from their dietary questionnaire[16]). The Center for Epidemiological Studies Depression scale (CES-D) was used to determine the presence of depression.[17] A score of ≥ 16 on the CES-D indicated depression. Household income was measured by self-report. Annual income was converted to quartiles based on MESA Exam 5 data distribution as follows: < $25,000, $25,000–$49,999, $50,000–$74,999, and > $75,000. Participants' body mass index (BMI) was used as calculated in their MESA Exam 5 visit.

Self-reported bedtime, wake time, and sleep duration on weekdays and weekends were obtained from the MESA sleep questionnaire. Consistent with a prior study, delayed sleep phase was defined as the reported time to fall asleep being midnight or later.[7] Chronotype was measured using the modified Horne-Östberg Morningness-Eveningness Questionnaire (MEQ).[18] Participants with an MEQ ≥ 18 were considered to have a morning preference, those with an MEQ > 11 but < 18 were considered to have an intermediate preference, and participants with an MEQ ≤ 11 were considered to have an evening preference. Employment status and shift work status were also obtained from the MESA sleep questionnaire. Individuals reported their current work schedule as "do not work," "day shift," "afternoon shift," "night shift," "split shift," "irregular shift/on-call," or "rotating shifts." Consistent with previous research,[19] we collapsed these categories into "do not work," "day shift," or "other shift" for analysis. Other shift included "afternoon shift," "night shift," "split shift," "irregular shift/on-call," and "rotating shifts."

Actigraphy

Actigraphy was completed using the Actiwatch Spectrum wrist actigraph (Philips Respironics, Murrysville, PA) on participants' nondominant wrist for 7 consecutive days along with a concurrent sleep diary record. Actiware-Sleep version 5.59 analysis software (Mini Mitter Co, Inc, Bend, OR), was used to score every 30-second epoch of actigraphic data as sleep or wake. Sleep onset was annotated based on changes in activity

count coupled with information from the marker input by participants, environmental light, and sleep timing recorded in the sleep diary.[15] The final activity count for each epoch was generated by incorporating the activity level in the surrounding 2-minute time period (ie, ± 2 minutes) using a validated algorithm.[15] Actigraphic measures of interest included time in bed (analogous to bedtime, not total time in bed), time asleep, time awake (sleep offset), time out of bed, total time in bed, sleep duration, and midsleep time. Mean values of each measure were examined for both weekdays and weekends.

Polysomnography

Polysomnography was conducted using a 15-channel monitor (Compumedics Somte System, Compumedics Ltd, Abbotsville, Australia). The recording montage included electroencephalography, bilateral electrooculograms, chin electromyography, bipolar electrocardiography, thoracic and abdominal respiratory inductance plethysmography, airflow measured by thermocouple and nasal pressure cannula, finger pulse oximetry, and bilateral limb movements. Apneas were scored when the thermocouple signal flattened or nearly flattened for greater than 10 seconds. Hypopneas were scored when the amplitude of the sum of the abdominal and thoracic inductance signals or the nasal pressure flow signal decreased by ≥ 30% or more for ≥ 10 seconds. Events were classified as either central or obstructive according to the presence or absence of respiratory effort. The apnea-hypopnea index (AHI) was calculated based on the average number of all apneas plus hypopneas associated with a 4% desaturation per hour of sleep. Complete polysomnography details have been previously published elsewhere.[15]

Statistical analysis

Statistical analysis was performed using SPSS Version 26 (IBM, Armonk, NY). To assess for differences between groups, Pearson χ^2 tests were used for categorical variables (sex, income quartile, current alcohol use, chronotype). For continuous variables, outcomes data were assessed for normality using the Kolmogorov-Smirnov test. Because all outcomes examined (self-reported sleep variables, actigraphic values, AHI, BMI, caffeine use, smoking history, CES-D score, MEQ score) were found to be nonnormally distributed, the Wilcoxon rank-sum test or Kruskal-Wallis (for evaluating sleep duration by chronotype) test was used for unadjusted comparisons between groups. Prior evidence has shown that linear regression/analysis of covariance is most often not significantly biased compared with nonparametric approaches for evaluation of nonparametric data.[20] In addition, we determined that the use of linear regression would allow direct comparison to a prior study of sleep timing in African Americans compared with Whites.[7] Therefore, linear regression was used for continuous variables with adjustment for age, sex, BMI, smoking, household income, work schedule, AHI, alcohol use, and caffeine consumption.

Logistic regression was used to compare the likelihood of delayed sleep phase (as measured by self-report and actigraphy) between African American and Whites, with adjustment for age, sex, BMI, smoking history, household income, work schedule, AHI, alcohol use, and caffeine consumption. Logistic regression was also used to determine the relationship between delayed sleep time (as measured by self-report and actigraphy) and depression, with adjustment for age, sex, BMI, smoking history, household income, AHI, alcohol use, and caffeine consumption. Finally, logistic regression was used to evaluate the relationship between chronotype and race with adjustment for age, sex, BMI, smoking history, household income, AHI, alcohol use, and caffeine consumption.

Age and sex were included as covariates because sleep duration and timing are known to vary by age and sex.[21] BMI was included because obesity is associated with changes in sleep duration.[22] Similarly, smoking, alcohol use, and caffeine consumption are known to be associated with alterations in sleep quality and duration.[23–26] Household income has been associated with variation in both sleep timing and sleep duration.[27] Finally, the AHI is a measure of obstructive sleep apnea, which is associated with an increased risk of depression and with excessive daytime sleepiness, which may affect both sleep timing and sleep duration.[28]

RESULTS

A total of 1,401 (578 African American and 823 White) participants were included in the analysis. A comparison of the demographic data from African American and White participants is shown in **Table 1.** There were no significant differences in age, sex, smoking history, shift work, or AHI between African Americans and Whites. African Americans were significantly more likely to report a lower income quartile than were Whites. African Americans had a slightly higher BMI than Whites. African Americans were less likely to drink alcohol and consumed less caffeine than were Whites. There was no significant difference in chronotype as measured by the MEQ between African Americans and Whites. CES-D scores were slightly higher in Whites than in African Americans, but there was not a significant difference in the percentage that met the threshold for a diagnosis of depression in Whites as compared with African Americans.

African Americans were more likely to self-report a delayed sleep phase on both weekdays and weekends than were Whites. For weekdays, 27% of African Americans reported a bedtime of midnight or later compared with 17% of Whites (odds ratio [OR], 1.8; 95% confidence interval [CI], 1.4–2.3; $P < .001$; adjusted OR [aOR], 1.9; 95% CI, 1.3–2.8; $P = .001$). For weekends, 34% of African Americans reported a bedtime of midnight or later compared with 25% of Whites (OR, 1.7; 95% CI, 1.3–2.3; $P < .001$; aOR, 1.8; 95% CI, 1.3–2.5; $P < .001$).

We evaluated 2 actigraphic markers for bedtime: actigraphic time in bed and actigraphic time asleep. A significantly greater prevalence of delayed sleep phase on both weekdays and weekends was seen for both methods in African Americans compared with Whites. For weekdays, 41% of African Americans had an actigraphic time in bed of midnight or later compared with 28% of Whites (OR, 1.7; 95% CI, 1.3–2.3; $P < .001$; aOR, 1.6; 95% CI, 1.2–2.1; $P = .004$). For weekends, 53% of African Americans had an actigraphic time in bed of midnight or later compared with 33% of Whites (OR, 2.2; 95% CI, 1.7–2.9; $P < .001$; aOR, 2.2; 95% CI, 1.6–3.0; $P < .001$). Similar

Table 1—Participant characteristics.

	African American	White	*P* Value
Female sex (%)	56	52	.12
Age (y)	68 (61–76)	68 (61–76)	.62
Income (annual, %)			< .001[a]
< $25,000	35	22	
$25,000–$49,999	31	24	
$50,000–$74,999	14	20	
≥ $75,000	21	35	
Shift work (%)			.48[a]
Do not work	59	55	
Day shift	29	32	
Other shift	13	13	
BMI	29.9 (26.2–33.7)	27.3 (24.3–31.1)	< .001[a]
Smoking (packs per y)	1 (0–10)	0 (1–11)	.37
Current alcohol use (%)	34	49	< .001[a]
Caffeine use (mg/d)	28 (0–97)	97 (15–240)	< .001[a]
AHI (events/h)	8.5 (2.8–19.3)	7.7 (2.9–17.8)	.49
MEQ score	17 (14–20)	17 (15–20)	.73
Chronotype (%)			.19
Morning	67	69	
Intermediate	18	19	
Evening	16	12	
CES-D score	3.5 (2–9)	4 (2–9)	.02[a]
Depression (CES-D score > 16; %)	9	11	.1

Data presented as percentage for categorical data and median (interquartile range) for continuous data. [a]Indicates $P < .05$. BMI = body mass index, CES-D = Center for Epidemiologic Studies Depression scale, MEQ = modified Horne-Ostberg Morningness-Eveningness Questionnaire.

results were seen using actigraphic time asleep. For weekdays, 43% of African Americans had an actigraphic time asleep of midnight or later compared with 29% of Whites (OR, 1.7; 95% CI, 1.3–2.2; $P < .001$; aOR, 1.6; 95% CI, 1.1–2.1; $P = .005$). For weekends, 55% of African Americans had an actigraphic time asleep of midnight or later compared with 35% of Whites (OR, 2.2; 95% CI, 1.7–2.8; $P < .001$; aOR, 2.1; 95% CI, 1.5–2.8; $P < .001$).

Socioeconomic status had a small effect on the odds of delayed sleep phase. For self-reported weekday bedtime after midnight, income in the second quartile was associated with lower odds of delayed sleep phase compared with income in the first (lowest) quartile (aOR, 0.6; 95% CI, 0.4–0.9; $P = .02$). No differences were seen in the odds of delayed sleep phase for the upper 2 quartiles compared with the lowest quartile. No significant association was present with objective measures of weekday sleep timing. For self-reported weekend bedtime after midnight, we found a similar association in which only the second quartile compared with the first was associated with lower odds of delayed sleep phase (aOR, 0.7; 95% CI, 0.4–0.999; $P = .49$). For actigraphic time in bed after midnight, a U-shaped effect was present. The second and third quartiles were associated with lower odds of delayed sleep phase (aOR, 0.6; 95% CI, 0.4–0.9; $P = .01$ and aOR, 0.5; 95% CI, 0.3–0.8; $P = .01$, respectively). For the highest quartile, there was no change in the likelihood of delayed sleep timing (aOR, 0.7; 95% CI, 0.5–1.1; $P = .13$). Using actigraphic time asleep after midnight yielded nearly identical results as using actigraphic time in bed after midnight.

Self-reported sleep timing data are presented in **Table 2.** In the unadjusted analysis, small but statistically significant differences were present. African Americans reported a significantly later weekday and weekend bedtime and an earlier weekday and weekend wake time compared with Whites. Sleep duration was shorter on weekdays and weekends in African Americans compared with Whites. In the adjusted analysis, only weekend bedtime and sleep duration on weekdays and weekends were significantly different between African Americans and Whites.

Actigraphic sleep timing data are presented in **Table 3.** Median weekday time in bed was 31 minutes later in African Americans compared with Whites ($P < .001$). Median weekend time in bed was 41 minutes later in African Americans compared with Whites ($P < .001$). No difference in weekday time out of bed was present, but African Americans were out of bed 14 minutes earlier on weekends compared with Whites ($P = .01$). African Americans had significantly less sleep on weekdays (median, 39 minutes less; $P < .001$) and weekends (median, 51 minutes less; $P < .001$) compared with Whites. Similar results

Table 2—Self-reported sleep timing differences between African American and White participants.

Unadjusted Analysis				
	African American	**White**	***P* Value**	
Weekday bedtime	23:00 (22:00–00:00)	23:00 (22:00–23:30)	.006[a]	
Weekend bedtime	23:00 (22:00–00:00)	23:00 (22:30–23:45)	.009[a]	
Weekday wake time	6:30 (5:30–8:00)	6:45 (6:00–7:30)	.04[a]	
Weekend wake time	7:00 (6:00–8:15)	7:00 (6:30–8:00)	.09[a]	
Weekday sleep duration	7:30 (6:30–9:00)	8:00 (7:00–8:45)	.005[a]	
Weekend sleep duration	8:00 (7:00–9:00)	8:00 (7:30–9:00)	.02[a]	
Adjusted Analysis				
	African American	**White**	**Difference**	***P* Value**
Weekday bedtime	23:03 (22:51–23:16)	22:51 (22:41–23:00)	12 (–3 to 28)	.13
Weekend bedtime	23:27 (23:16–23:38)	23:04 (22:55–23:13)	23 (8 to 38)	.003[a]
Weekday wake time	6:47 (6:32–7:01)	6:56 (6:45–7:07)	–10 (–28 to 9)	.31
Weekend wake time	7:18 (7:04–7:32)	7:25 (7:14–7:36)	–7 (–26 to 11)	.43
Weekday sleep duration	7:25 (7:12–7:37)	8:05 (7:56–8:15)	–41 (–56 to –25)	< .001[a]
Weekend sleep duration	7:51 (7:37–8:04)	8:21 (8:10–8:31)	–30 (–48 to –13)	.001[a]

Unadjusted data are presented as median (interquartile range). Adjusted analysis was performed using linear regression with adjustment for household income, age, sex, BMI, smoking, AHI, alcohol use, and caffeine consumption. Adjusted values are presented as mean (95% confidence interval). [a]Indicates $P < .05$. BMI = body mass index.

were seen in the adjusted analysis, although weekend wake time and midsleep time were not significantly different between African Americans and Whites.

Consistent with a delayed sleep phase in African Americans as compared with Whites, actigraphic midsleep time on both weekdays and weekends was significantly later in African Americans. Median weekday and weekend midsleep time was 15 minutes later in African Americans than in Whites ($P < .001$ and $P = .006$, respectively). Similar results were seen in the adjusted analysis. Mean weekday midsleep time was 27 minutes later in African Americans than in Whites ($P = .02$), and weekend midsleep time was 20 minutes later ($P = .03$).

We additionally evaluated whether there was an interaction between chronotype (morning, intermediate, or evening as assessed by the MEQ) and race for delayed sleep phase. There was a significant interaction ($P < .001$ for all measures) between race and chronotype for both self-reported and actigraphic measures of delayed sleep phase. Stratified analysis showed that there were no increased odds of delayed sleep phase as measured by either self-report or actigraphy in African Americans compared with Whites in individuals with an evening chronotype. In individuals with an intermediate chronotype, actigraphic measures of delayed sleep phase showed increased odds of delayed sleep phase in African Americans compared with Whites but no difference in self-reported measures. African Americans with a morning chronotype were much more likely to report a delayed sleep phase than were Whites with a morning chronotype. These results are depicted in **Figure 1.**

Actigraphic measures of sleep timing stratified by chronotype showed that there were more significant differences between African American and White individuals with a morning or intermediate chronotype compared with those with an evening chronotype (**Table 4**). There were no significant differences in actigraphic measures of sleep timing in African American individuals compared with White individuals with an evening chronotype. Sleep duration was significantly shorter in African Americans compared to Whites regardless of chronotype. Chronotype was not significantly associated with either weekday or weekend sleep duration in African Americans ($P = .28$ and $P = .11$, respectively) or Whites ($P = .86$ and $P = 0.50$, respectively).

Logistic regression showed that there was no difference in chronotype distribution between African American and White participants. African Americans were not more likely than Whites to have an evening chronotype (OR, 1.4; 95% CI, 0.95–2.0; $P = .09$; aOR, 1.3; 95% CI, 0.8–2.2; $P = .3$) or a morning chronotype (OR, 1.0; 95% CI, 0.8–1.4; $P = .93$; aOR, 1.1; 95% CI, 0.7–1.6; $P = .7$), with an intermediate chronotype as the reference.

Given prior findings that delayed sleep timing was associated with an increased risk of depression,[7] we evaluated the relationship between delayed sleep phase and depression (CES-D score ≥ 16) along with depression symptoms (CES-D raw score). We found that delayed sleep phase was associated with an increased risk of depression, but this association was only significant in White participants. In White participants, self-reported delayed sleep phase was associated with a 1.9-fold greater risk of depression (95% CI, 1.1–3.5; $P = .03$). Similar results were seen in the adjusted analysis (aOR, 2.0; 95% CI, 1.1–3.8; $P = .04$). In White participants, actigraphic sleep onset after midnight was similarly associated with a greater risk of depression. The unadjusted OR was 2.4 (95% CI, 1.4–4.0; $P = .001$) and the aOR was 2.6 (95% CI, 1.5–4.5; $P = .001$). In African American participants, there was no significant association present between delayed sleep phase and depression. For the self-reported measures, the unadjusted OR was 1.7

Table 3—Objective sleep timing differences between African American and White participants.

Unadjusted Analysis				
	African American	**White**	***P* Value**	
Weekday time in bed	23:42 (22:48–00:53)	23:11 (22:25–00:05)	< .001[a]	
Weekday time asleep	23:45 (22:54–00:56)	23:16 (22:30–00:10)	< .001[a]	
Weekend time in bed	00:06 (22:59–01:09)	23:25 (22:37–00:25)	< .001[a]	
Weekend time asleep	00:09 (23:03–01:13)	23:29 (22:41–00:29)	< .001[a]	
Weekday sleep offset	6:49 (5:44–7:45)	6:51 (6:01–7:42)	.55	
Weekday time out of bed	6:51 (5:49–7:58)	6:53 (6:03–7:45)	.62	
Weekend sleep offset	7:05 (6:01–8:22)	7:22 (6:29–8:13)	.009[a]	
Weekend time out of bed	7:10 (6:04–8:25)	7:24 (6:31–8:16)	.01[a]	
Weekday midsleep time	3:20 (2:35–4:25)	3:05 (2:22–3:53)	< .001[a]	
Weekend midsleep time	3:41 (2:45–4:43)	3:26 (2:44–4:16)	.006[a]	
Weekday sleep duration	6:50 (5:28–7:40)	7:29 (6:39–8:12)	< .001[a]	
Weekday in bed duration	6:57 (5:55–7:48)	7:35 (6:45–8:20)	< .001[a]	
Weekend sleep duration	6:53 (5:34–7:55)	7:44 (6:51–8:35)	< .001[a]	
Weekend in bed duration	7:00 (5:38–8:04)	7:51 (6:56–8:42)	< .001[a]	
Adjusted Analysis				
	African American	**White**	**Difference**	***P* Value**
Weekday time in bed	23:55 (23:35–00:15)	23:23 (23:08–23:39)	31 (5 to 58)	.02[a]
Weekday time asleep	23:59 (23:39–00:19)	23:28 (23:12–23:44)	31 (5 to 57)	.02[a]
Weekend time in bed	00:13 (23:55–00:32)	23:42 (23:28–23:56)	31 (7 to 55)	.01[a]
Weekend time asleep	00:17 (23:56–00:35)	23:47 (23:32–00:01)	30 (6 to 54)	.01[a]
Weekday sleep offset	7:13 (6:50–7:37)	7:05 (6:46–7:23)	8 (–22 to 40)	.57
Weekday time out of bed	7:16 (6:52–7:40)	7:07 (6:48–7:26)	9 (–22 to 40)	.56
Weekend sleep offset	7:28 (7:09–7:48)	7:25 (7:10–7:40)	3 (–22 to 28)	.8
Weekend time out of bed	7:34 (7:14–7:53)	7:28 (7:12–7:43)	6 (–20 to 32)	.66
Weekday midsleep time	4:19 (3:50–4:48)	3:23 (3:01–3:46)	55 (18 to 93)	.004[a]
Weekend midsleep time	4:18 (3:53–4:43)	3:49 (3:29–4:09)	29 (–4.2 to 62)	.09
Weekday sleep duration	6:31 (6:18–6:43)	7:27 (7:17–7:36)	–56 (–72 to –40)	< .001[a]
Weekday in bed duration	6:37 (6:25–6:50)	7:33 (7:24–7:43)	–55 (–72 to –40)	< .001[a]
Weekend sleep duration	6:46 (6:30–7:02)	7:34 (7:21–7:47)	–48 (–69 to –27)	< .001[a]
Weekend in bed duration	6:56 (6:36–7:17)	7:41 (7:25–7:57)	–45 (–71 to –19)	.001[a]

Unadjusted data are presented as median (interquartile range). Adjusted analysis was performed using linear regression with adjustment for household income, age, sex, BMI, smoking, AHI, alcohol use, and caffeine consumption. Adjusted values are presented as mean (95% confidence interval). [a]Indicates $P < .05$. BMI = body mass index.

(95% CI, 0.8–3.4; $P = .15$) and the adjusted OR was 1.9 (95% CI, 0.9–4.2; $P = .1$). For the association between actigraphic sleep onset after midnight and depression, the unadjusted OR was 1.4 (95% CI, 0.7–2.8; $P = .4$) and the aOR was 1.6 (95% CI, 0.7–3.3; $P = .3$). We additionally evaluated for any interaction between delayed sleep phase and chronotype, but none was present for either African Americans or Whites. Similar results were seen for the association between depressive symptoms as measured by CES-D score and delayed sleep phase (**Table 5**).

We examined the role of income as well. For White participants, we found that there was no strong association between income quartile and depression. However, for African American participants, we found that increased household income was associated with a decreased risk of depression. Income in the highest quartile was associated with decreased odds of depression compared with income in the bottom quartile (aOR, 0.1; 95% CI, 0.04–0.6; $P = .005$). This relationship was the same regardless of the use of self-reported or actigraphic measures of delayed sleep phase.

DISCUSSION

Consistent with a prior study in the Sleep Heart Health cohort,[7] we found that African Americans were likely to have a delayed sleep phase and reduced sleep duration in comparison to Whites. This racial difference in sleep timing varied by chronotype, with no significant delayed sleep phase in African American participants compared with White participants with an evening chronotype. We also again found, per the previous study,[7] that a

Figure 1—Odds of delayed sleep phase in African Americans compared with Whites, stratified by chronotype.

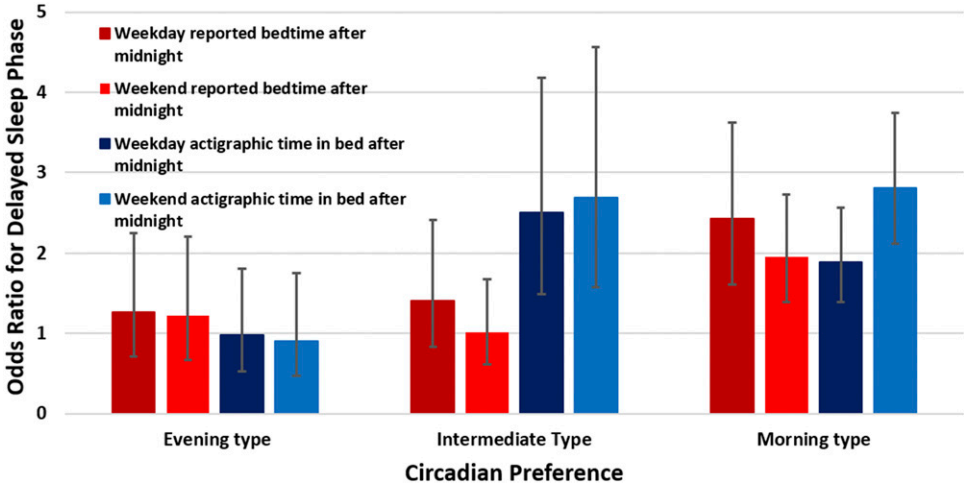


Table 4—Objective sleep timing differences between African American and White participants, stratified by chronotype.

	African American	White	*P* Value
Morning Chronotype			
Weekday time asleep	11:23 (10:37–12:20)	11:01 (10:19–11:46)	< .001[a]
Weekend time asleep	11:50 (10:43–12:36)	11:08 (10:21–11:57)	< .001[a]
Weekday sleep offset	6:27 (5:30–7:22)	6:31 (5:50–7:17)	.26
Weekend sleep offset	6:43 (5:49–7:24)	7:01 (6:18–7:45)	.009[a]
Weekday midsleep time	3:01 (2:21–3:54)	2:47 (2:11–3:27)	.003[a]
Weekend midsleep time	3:19 (2:35–4:06)	3:05 (2:29–3:47)	.007[a]
Weekday sleep duration	6:55 (5:56–7:43)	7:31 (6:42–8:14)	< .001[a]
Weekend sleep duration	7:04 (5:44–8:00)	7:46 (6:54–8:36)	< .001[a]
Intermediate Chronotype			
Weekday time asleep	00:25 (11:18–1:34)	11:43 (11:06–00:25)	< .001[a]
Weekend time asleep	00:42 (11:53–1:45)	11:56 (11:25–1:05)	.001[a]
Weekday sleep offset	7:15 (6:19–8:26)	7:21 (6:34–7:59)	.76
Weekend sleep offset	7:54 (6:48–9:04)	7:58 (7:15–8:35)	.70
Weekday midsleep time	4:04 (3:04–5:08)	3:31 (2:58–4:08)	.003[a]
Weekend midsleep time	4:14 (3:26–5:13)	3:59 (3:27–4:46)	.07[a]
Weekday sleep duration	6:36 (5:29–7:40)	7:27 (6:35–8:08)	< .001[a]
Weekend sleep duration	6:42 (5:05–7:58)	7:40 (6:35–8:32)	< .001[a]
Evening Chronotype			
Weekday time asleep	00:59 (11:45–2:24)	00:38 (23:38–1:43)	.30
Weekend time asleep	1:20 (00:00–2:49)	1:08 (00:04–2:12)	.31
Weekday sleep offset	7:40 (6:50–9:14)	8:13 (7:31–9:10)	.11
Weekend sleep offset	8:22 (6:34–9:36)	8:50 (7:58–10:00)	.02[a]
Weekday midsleep time	4:36 (3:35–5:32)	4:36 (3:42–5:19)	.79
Weekend midsleep time	4:51 (3:31–6:07)	5:00 (3:57–5:53)	.81
Weekday sleep duration	6:44 (5:39–7:37)	7:28 (6:49–8:20)	< .001[a]
Weekend sleep duration	6:32 (5:25–7:32)	7:43 (6:47–8:33)	< .001[a]

Unadjusted data are presented as median (interquartile range). Adjusted analysis was performed using linear regression with adjustment for household income, age, sex, BMI, smoking, AHI, alcohol use, and caffeine consumption. [a]Indicates *P* < .05. BMI = body mass index.

Table 5—DSP and depressive symptoms stratified by race.

CES-D Score	African American			White		
	DSP−	DSP+	*P*	DSP−	DSP+	*P*
Unadjusted self-reported DSP	4 (2–9)	3 (2–10)	.7	4 (2–9)	5.5 (2–11.8)	.1
Adjusted self-reported DSP	6.4 (5.3–7.4)	8.0 (6.5–9.5)	.08	7.2 (6.4–7.9)	9.3 (7.3–11.4)	.049[a]
Unadjusted actigraphic DSP	3.5 (2–9)	3.5 (2–9)	.4	4 (2–9)	5.5 (2–12)	.003[a]
Adjusted actigraphic DSP	6.6 (5.5–7.7)	6.9 (5.5–8.2)	.7	6.7 (5.9–7.6)	9.8 (8.3–11.2)	< .001[a]

Self-reported DSP is defined as a reported bedtime of midnight or later. Actigraphic DSP is defined as a time asleep of midnight or later. Unadjusted data are presented as median (interquartile range). Adjusted analysis was performed using linear regression with adjustment for household income, age, sex, BMI, smoking, AHI, alcohol use, and caffeine consumption. Adjusted values are presented as mean (95% confidence interval). [a]Indicates $P < .05$. BMI = body mass index, CES-D = Center for Epidemiologic Studies Depression scale, DSP = delayed sleep phase.

bedtime after midnight was associated with an increased risk of depression, but this relationship only seemed significant in White participants.

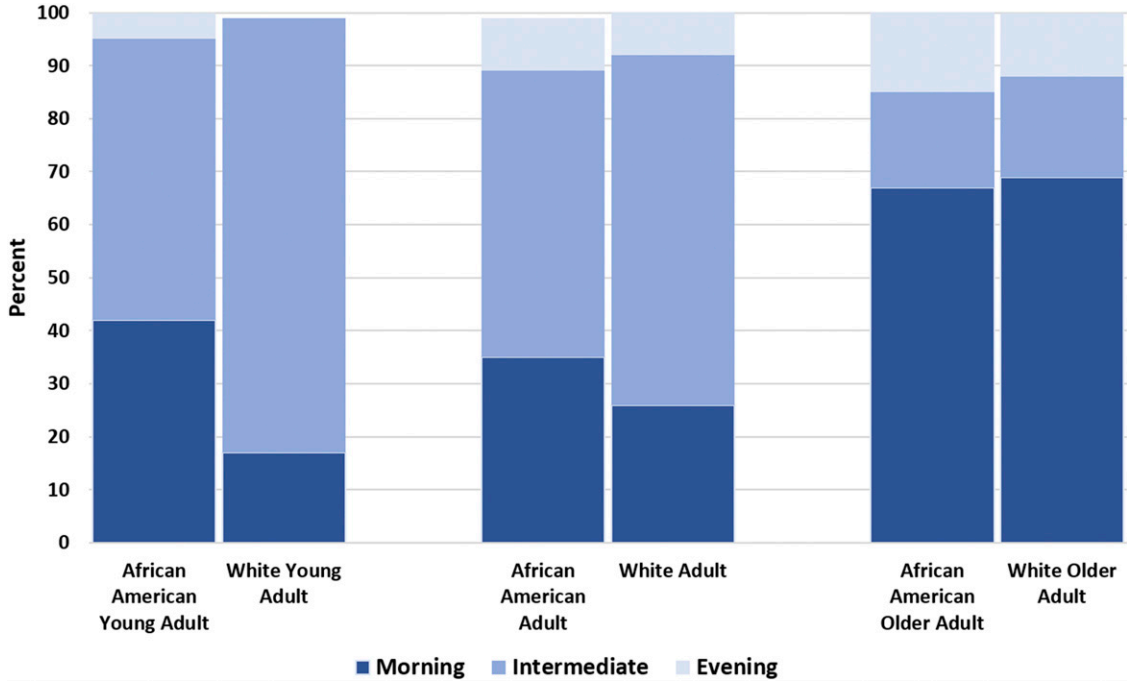
Differences in self-reported bedtime tended to be small. For example, the mean adjusted weekday bedtime for African Americans was only 12 minutes later compared with Whites. However, these differences seemed much greater when objectively measured by actigraphy: Median weekday bedtime was 30 minutes delayed in African Americans compared with Whites, with similar differences seen in the adjusted mean weekday bedtime and that on weekends. This finding suggests that there may be a relative underreporting of delayed sleep phase in African Americans relative to Whites. Future studies would benefit from objective sleep measurement rather than self-reported measurement to reduce the risk of bias because of underreporting. This finding also suggests that the previous ~15-minute delay in bedtime in African Americans compared with Whites in the prior study[7] may have underestimated the true relative delay in sleep phase. Consistent with this finding, prior research has shown variation by race/ethnicity in the accuracy of self-reported sleep duration compared with the accuracy of objective measures such as actigraphy and polysomnography. Specifically, Whites generally overestimated total sleep time per self-reports compared with results from using objective measures of sleep time to a significantly greater degree than African Americans,[29,30] and 1 study of African Americans found that self-reported measurements of sleep actually underestimated objective sleep.[31]

Similar to earlier findings,[7] African Americans had significantly shorter sleep duration (> 30 minutes shorter on weekdays and nearly an hour shorter on weekends as measured by actigraphy). There was no difference in wake time on weekdays or weekends regardless of measurement technique. This suggests that the difference in sleep duration is predominantly driven by a delayed bedtime. Based on our results, African Americans obtain nearly 5 hours less sleep per week than do Whites. Given that sleep restriction associated with delayed sleep onset of 30 minutes per day over a 5-day period decreased performance on neurocognitive testing,[32] this trend may conceivably lead to significant adverse cognitive effects. Conversely, because sleep extension of 30 minutes has also been shown to improve cognitive performance,[32] it is conceivable that the potential cognitive impact of delayed sleep phase may be reversible.

In our study, contrary to prior results in the United Kingdom Biobank study showing a morning chronotype in Blacks, there was no difference in self-reported chronotype between African Americans and Whites.[3,4] Note that the MESA participants included in this study are approximately 15 years older than the sample reported on in the Biobank study, so it is conceivable that there may be an African American tendency toward a morning chronotype relative to Whites that decreases with increased age and the general tendency toward a morning chronotype in older adults.[33] Similarly, a prior study showing a shorter tau in African Americans included participants much younger than seen in our study, with an age range of 20–43 years.[1] Consistent with our hypothesis that this chronotype difference may be age-dependent, in the prior study measuring tau,[1] 17% of White participants reported a morning chronotype on the MEQ compared with 69% of White participants in the current study. Forty-two percent of African Americans in the prior study[1] reported a morning preference, compared with 67% in the current study. This finding suggests that age-related changes in chronotype may explain why no difference in chronotype was seen in this study but was present in studies with younger populations. Previous research has shown that ethnic differences in sleep timing and duration change with age in children,[34] further supporting this hypothesis. **Figure 2** displays the reported chronotype by age and race across the prior 2 studies[1,4] and this study.

In the stratified analysis, we found that African American participants were significantly more likely to have delayed sleep phase compared with White participants if they had a morning or intermediate chronotype, but there was no difference in sleep timing between African American and White participants with an evening chronotype. African American individuals with a morning or intermediate chronotype may be driven by other factors toward a later sleep schedule than they would otherwise choose based on their chronotype, whereas African American individuals with an evening chronotype may maintain a schedule more typical of their chronotype. In addition, the differences in sleep timing were still present after adjustment for household income. This finding suggests that at least this measure of socioeconomic status is not a significant driver in the observed differences in sleep timing.

Figure 2—Self-reported chronotype of African Americans and Whites across the lifespan.



Data for young adults (mean age 33 years for African Americans and 30 years for Whites) and adults (mean age 52 years for African Americans and 57 years for Whites) are derived from prior studies[1,4] and data for older adults (median age 69 years for African Americans and 70 years for Whites) are from the current study.

Other potential factors that may contribute to the relative delayed sleep phase in African American participants compared with White participants may include social factors such as neighborhood, job-related stresses, or social stresses. Prior work has shown that poor neighborhood and housing conditions are associated with decreased sleep quality and short sleep duration.[35] Studies have also shown racial/ethnic differences in the likelihood of short sleep by occupation, with evidence that African American individuals are more likely to have short sleep duration than are White individuals as professional responsibility increases.[36] Similarly, racial/ethnic discrimination, one form of social stress, has been associated with short sleep duration and insomnia.[37] Unfortunately, these prior studies have not evaluated delayed sleep phase, but given prior findings that delayed sleep phase was closely associated with short sleep duration,[7] a similar effect is likely present. Many of these prior studies have included actigraphy, which means that data related to sleep timing would be available for future secondary analyses. Future work is needed to determine the underlying etiology of delayed sleep phase in African American individuals compared with White individuals.

Previous research has identified that delayed sleep phase is associated with an increased risk of depression, although this finding was limited by the lack of a validated questionnaire for depression.[7] In a prior study, there were no differences in this association in African American participants as compared with White participants, but the Sleep Heart Health Study cohort used for that study was not specifically designed to measure racial or ethnic differences in outcomes and did not have a balanced recruitment of African American and White participants.[7] In our current study, using a cohort designed to evaluate racial and ethnic differences in outcomes, we again found an association between delayed sleep phase and depression. However, unlike many sleep-related health disparities that are more significant in African American individuals,[38] we found that the relationship between delayed sleep phase and depression was only significant in White participants. In this cohort, African American participants seemed to not be at increased risk of depression in the setting of delayed sleep phase.

Prior research using the American Time Use Survey has shown that African Americans and Whites with short sleep duration spent their increased wake time engaged in different activities.[27] Although no analysis has specifically examined this difference in regard to delayed sleep phase, given the high association of delayed sleep phase with short sleep duration, it is conceivable that this same association may be present in African Americans with delayed sleep phase compared with Whites with delayed sleep phase. It is possible that the different activities chosen may have protective effects against depression in African American individuals, but we are unable to evaluate this hypothesis in the current article. More research is needed to better understand the effects of race and ethnicity on health disparities. In particular, further research to understand the resilience of African American participants that mitigated the association between delayed sleep phase and depression seen in White participants in the MESA cohort may shed a light on possible targets for interventions to improve sleep-related health disparities in African American individuals.

Our study has multiple strengths. Although the MESA cohort was not originally designed specifically to evaluate sleep measures, it was explicitly designed to assess racial and ethnic differences in health. We were therefore able to evaluate a sample that included similar numbers of African Americans (41%) and Whites (59%). In addition, unlike the prior study that used the Sleep Heart Health Study,[7] actigraphy was used to obtain objective sleep measures in addition to self-reported measures. We were also able to evaluate the role of chronotype because of the inclusion of a self-reported measure of chronotype, the MEQ. Finally, we were able to include the covariate of household income as a measure of socioeconomic status and its effect on racial differences in sleep timing, and unlike in previous research, we were able to use the CES-D, a validated epidemiologic measure of depression.[17]

Our study has limitations as well. The use of linear regression for adjusted analysis in our sample likely had reduced statistical efficiency and consequently larger (less significant) *P* values as compared to the unadjusted nonparametric analysis. This detail may explain the differences in some of the *P* values in the adjusted vs the unadjusted analysis, which resulted in some outcomes having statistical significance in the unadjusted analysis but not in the adjusted analysis. However, our use of this approach for the adjusted analysis allowed a direct comparison to the prior study of delayed sleep timing in African Americans relative to Whites in the Sleep Heart Health Study.[7] In addition, we used a self-reported measure of chronotype, the MEQ, rather than the direct measurement of tau to objectively determine circadian rhythm. However, given the high participant burden associated with study protocols to directly measure tau, it is unlikely that the measurement of tau would be feasible in a large multisite community cohort such as the MESA. Furthermore, although we provide information on midsleep differences, data on the use of alarm clocks were not available. Given that alarm clock use may advance calculated midsleep timing by leading to an earlier wake time than chronotype would predict, midsleep timing results in this cohort may not be an accurate reflection of true midsleep timing. In particular, given the delayed bedtime and shorter sleep duration in African American participants compared with White participants but no difference in wake time, it is possible that the difference in midsleep time was smaller than what it may have been if participants had midsleep time assessed in the known absence of an alarm. Finally, there is an important distinction between race as a primary factor vs race as a marker of interest within the context of other social, cultural, and environmental factors.

Note that we also focused on only African Americans and Whites for this analysis and did not include Asian and Hispanic MESA participants. We chose this approach because we felt that a single analytic approach would not be appropriate for comparisons between all 4 ethnic groups included in the MESA study. Specifically, certain covariates such as acculturation (as measured by language or other metrics) are highly relevant to Asian and Hispanic participants but are not relevant to African American or White participants. Similarly, for Hispanic participants, information on place of origin (eg, Mexico, Puerto Rico) may be an important covariate to include that does not have any direct correlates for African American participants. Future analyses are needed to examine whether there are similar disparities in sleep timing in Asian and Hispanic participants in the MESA cohort.

CONCLUSIONS

African Americans were more likely to experience delayed sleep phase and short sleep duration when compared with Whites. Specifically, African American individuals with a morning or intermediate chronotype seemed specifically more likely to have a delayed sleep phase than did White individuals with the same chronotype. We also found that delayed sleep phase was associated with depression, but this relationship was only significant in White participants. These results highlight the need for further research to better understand sleep and health disparities that vary by race/ethnicity.

ABBREVIATIONS

AHI, apnea-hypopnea index
aOR, adjusted odds ratio
BMI, body mass index
CES-D, Center for Epidemiologic Studies Depression scale
CI, confidence interval
MEQ, modified Horne-Östberg Morningness-Eveningness Questionnaire
MESA, Multi-Ethnicity Study of Atherosclerosis
OR, odds ratio

REFERENCES

1. Eastman CI, Suh C, Tomaka VA, Crowley SJ. Circadian rhythm phase shifts and endogenous free-running circadian period differ between African-Americans and European-Americans. *Sci Rep.* 2015;5(1):8381.
2. Paech GM, Crowley SJ, Eastman CI. Sleep and cognitive performance of African-Americans and European-Americans before and during circadian misalignment produced by an abrupt 9-h delay in the sleep/wake schedule. *PLoS One*. 2017;12(10):e0186843.
3. Malone SK, Patterson F, Lu Y, Lozano A, Hanlon A. Ethnic differences in sleep duration and morning-evening type in a population sample. *Chronobiol Int.* 2016;33(1):10–21.
4. Malone SK, Patterson F, Lozano A, Hanlon A. Differences in morning-evening type and sleep duration between Black and White adults: results from a propensity-matched UK Biobank sample. *Chronobiol Int.* 2017;34(6):740–752.
5. Vitale JA, Roveda E, Montaruli A, et al. Chronotype influences activity circadian rhythm and sleep: differences in sleep quality between weekdays and weekend. *Chronobiol Int.* 2015;32(3):405–415.
6. Martin JS, Hébert M, Ledoux E, Gaudreault M, Laberge L. Relationship of chronotype to sleep, light exposure, and work-related fatigue in student workers. *Chronobiol Int.* 2012;29(3):295–304.
7. Bailey O, Combs D, Sans-Fuentes M, et al. Delayed sleep time in African Americans and depression in a community-based population. *J Clin Sleep Med.* 2019;15(6):857–864.
8. Saxvig IW, Pallesen S, Wilhelmsen-Langeland A, Molde H, Bjorvatn B. Prevalence and correlates of delayed sleep phase in high school students. *Sleep Med.* 2012;13(2):193–199.
9. Reis C, Paiva T. Delayed sleep-wake phase disorder in a clinical population: gender and sub-population differences. *Sleep Sci.* 2019;12(3):203–213.

10. Obeysekare JL, Cohen ZL, Coles ME, et al. Delayed sleep timing and circadian rhythms in pregnancy and transdiagnostic symptoms associated with postpartum depression. *Transl Psychiatry.* 2020;10(1):14.
11. Spaeth AM, Dinges DF, Goel N. Resting metabolic rate varies by race and by sleep duration. *Obesity (Silver Spring)*. 2015;23(12):2349–2356.
12. Bertisch SM, Sillau S, de Boer IH, Szklo M, Redline S. 25-hydroxyvitamin D concentration and sleep duration and continuity: Multi-Ethnic Study of Atherosclerosis. *Sleep*. 2015;38(8):1305–1311.
13. Bild DE, Bluemke DA, Burke GL, et al. Multi-Ethnic Study of Atherosclerosis: objectives and design. *Am J Epidemiol.* 2002;156(9):871–881.
14. Olson JL, Bild DE, Kronmal RA, Burke GL. Legacy of MESA. *Glob Heart*. 2016; 11(3):269–274.
15. Chen X, Wang R, Zee P, et al. Racial/ethnic differences in sleep disturbances: the Multi-Ethnic Study of Atherosclerosis (MESA). *Sleep*. 2015;38(6):877–888.
16. Miller PE, Zhao D, Frazier-Wood AC, et al. Associations of coffee, tea, and caffeine intake with coronary artery calcification and cardiovascular events. *Am J Med.* 2017;130(2):188–197.e5.
17. Andresen EM, Malmgren JA, Carter WB, Patrick DL. Screening for depression in well older adults: evaluation of a short form of the CES-D (Center for Epidemiologic Studies Depression Scale). *Am J Prev Med.* 1994;10(2):77–84.
18. Huang T, Redline S. Cross-sectional and prospective associations of actigraphy-assessed sleep regularity with metabolic abnormalities: the Multi-Ethnic Study of Atherosclerosis. *Diabetes Care*. 2019;42(8):1422–1429.
19. Huang T, Mariani S, Redline S. Sleep irregularity and risk of cardiovascular events: the Multi-Ethnic Study of Atherosclerosis. *J Am Coll Cardiol.* 2020;75(9): 991–999.
20. Vickers AJ. Parametric versus non-parametric statistics in the analysis of randomized trials with non-normally distributed data. *BMC Med Res Methodol.* 2005;5(1):35.
21. Jonasdottir SS, Minor K, Lehmann S. Gender differences in nighttime sleep patterns and variability across the adult lifespan: a global-scale wearables study [published online ahead of print, 2020 Sept 4]. *Sleep*.
22. Benaich S, Mehdad S, Andaloussi Z, et al. Weight status, dietary habits, physical activity, screen time and sleep duration among university students [published online ahead of print, 2020 Oct 12]. *Nutr Health*. doi: 10.1177/ 0260106020960863
23. Zhu G, Catt M, Cassidy S, et al. Objective sleep assessment in >80,000 UK mid-life adults: associations with sociodemographic characteristics, physical activity and caffeine. *PLoS One*. 2019;14(12):e0226220.
24. Zandy M, Chang V, Rao DP, Do MT. Tobacco smoke exposure and sleep: estimating the association of urinary cotinine with sleep quality. *Health Promot Chronic Dis Prev Can.* 2020;40(3):70–80.
25. Goodhines PA, Desalu JM, Zaso MJ, Gellis LA, Park A. Sleep problems and drinking frequency among urban multiracial and monoracial adolescents: role of discrimination experiences and negative mood. *J Youth Adolesc.* 2020;49(10): 2109–2123.
26. Spadola CE, Guo N, Johnson DA, et al. Evening intake of alcohol, caffeine, and nicotine: night-to-night associations with sleep duration and continuity among African Americans in the Jackson Heart Sleep Study. *Sleep*. 2019;42(11):zsz136.
27. Basner M, Spaeth AM, Dinges DF. Sociodemographic characteristics and waking activities and their role in the timing and duration of sleep. *Sleep*. 2014; 37(12):1889–1906.
28. Ishman SL, Cavey RM, Mettel TL, Gourin CG. Depression, sleepiness, and disease severity in patients with obstructive sleep apnea. *Laryngoscope*. 2010; 120(11):2331–2335.
29. Jackson CL, Patel SR, Jackson WB 2nd, Lutsey PL, Redline S. Agreement between self-reported and objectively measured sleep duration among white, black, Hispanic, and Chinese adults in the United States: Multi-Ethnic Study of Atherosclerosis. *Sleep*. 2018;41(6):zsy057.
30. Lauderdale DS, Knutson KL, Yan LL, Liu K, Rathouz PJ. Self-reported and measured sleep duration: how similar are they? *Epidemiology*. 2008;19(6):838–845.
31. Jackson CL, Ward JB, Johnson DA, Sims M, Wilson J, Redline S. Concordance between self-reported and actigraphy-assessed sleep duration among African-American adults: findings from the Jackson Heart Sleep Study. *Sleep*. 2020;43(3): zsz246.
32. Sadeh A, Gruber R, Raviv A. The effects of sleep restriction and extension on school-age children: what a difference an hour makes. *Child Dev.* 2003;74(2):444–455.
33. Czeisler CA, Dumont M, Duffy JF, et al. Association of sleep-wake habits in older people with changes in output of circadian pacemaker. *Lancet.* 1992;340(8825): 933–936.
34. Combs D, Goodwin JL, Quan SF, Morgan WJ, Parthasarathy S. Longitudinal differences in sleep duration in Hispanic and Caucasian children. *Sleep Med.* 2016; 18:61–66.
35. Troxel WM, Haas A, Ghosh-Dastidar B, et al. Broken windows, broken zzs: poor housing and neighborhood conditions are associated with objective measures of sleep health. *J Urban Health*. 2020;97(2):230–238.
36. Jackson CL, Redline S, Kawachi I, Williams MA, Hu FB. Racial disparities in short sleep duration by occupation and industry. *Am J Epidemiol.* 2013;178(9): 1442–1451.
37. Gaston SA, Feinstein L, Slopen N, Sandler DP, Williams DR, Jackson CL. Everyday and major experiences of racial/ethnic discrimination and sleep health in a multiethnic population of U.S. women: findings from the Sister Study. *Sleep Med.* 2020;71:97–105.
38. Adenekan B, Pandey A, McKenzie S, Zizi F, Casimir GJ, Jean-Louis G. Sleep in America: role of racial/ethnic differences. *Sleep Med Rev.* 2013;17(4):255–262.

ACKNOWLEDGMENTS

The authors thank the investigators, the staff, and the participants of the MESA study for their valuable contributions. A full list of participating MESA investigators and institutions can be found at https://www.mesa-nhlbi.org. Dr. Combs had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. This article was prepared using MESA research materials obtained from the National Heart, Lung, and Blood Institute Biologic Specimen and Data Repository Information Coordinating Center and the National Sleep Research Resource and does not necessarily reflect the opinions or views of the MESA study investigators or the National Heart, Lung, and Blood Institute.

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication July 23, 2020
Submitted in final revised form December 2, 2020
Accepted for publication December 2, 2020
Address correspondence to: Daniel Combs, MD, 1501 North Campbell Avenue, P.O. Box 245073, Tucson, AZ 85724; Tel: (520) 626-7780; Fax: (520) 626-9465; Email: dcombs@peds.arizona.edu

DISCLOSURE STATEMENT

All authors have seen and approved this manuscript. The Multi-Ethnicity Study of Atherosclerosis study was supported by contracts HHSN268201500003I, N01-HC-95159, N01-HC-95160, N01-HC-95161, N01-HC-95162, N01-HC-95163, N01-HC-95164, N01-HC-95165, N01-HC-95166, N01-HC-95167, N01-HC-95168, and N01-HC-95169 from the National Heart, Lung, and Blood Institute and by grants UL1-TR-000040, UL1-TR-001079, and UL1-TR-001420 from the National Center for Advancing Translational Sciences. The National Sleep Research Resource was supported by the National Institutes of Health/National Heart, Lung, and Blood Institute (R24 HL114473). Dr. Combs was supported by an American Heart Association Career Development Award (19CDA34740005), the National Institutes of Health (HL151254), and a University of Arizona Health Sciences Career Development Award. Dr. Patel was supported by grants from the American Academy of Sleep Medicine Foundation, the National Institutes of Health (HL126140), and a University of Arizona Health Sciences Career Development Award. Dr. Parthasarathy was supported by the National Institutes of Health/National Heart, Lung, and Blood Institute (HL126140, AG059202, OD028307, HL151254, HL138377, HL140144, and HL128954), the Patient-Centered Outcomes Research Institute (DI-2018C2-13161 and COVID supplement, PPRND-1507-31666, PCS-1504-30430, PCS-1504-30430, and EADI-16493), and the American Academy of Sleep Medicine Foundation (169-SR-17). Dr. Combs reports a grant from the LuMind-

IDSC Foundation and a prior grant from the American Academy of Sleep Medicine Foundation. Dr. Parthasarathy reports grants from the National Institutes of Health, the American Academy of Sleep Medicine Foundation, the Patient Centered Outcomes Research Institute, and the Johrei Institute, and personal royalty fees from UpToDate Inc. Outside the submitted work, Dr. Parthasarathy has a patent (UA 14-018 U.S.S.N. 90 61/884,654; PTAS 502570970 [home breathing device]) issued. The above-mentioned conflicts, including the patent, are unrelated to the topic of this article. The remaining authors have no conflicts of interest to report.