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Jaylah Goodson Date

The Role of Sleep Irregularity in Racial Disparities in Hypertension: The Multi-Ethnic Study of Atherosclerosis (MESA)

By

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Master of Public Health

Epidemiology

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By

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B.S.
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Abstract

The Role of Sleep Irregularity in Racial Disparities in Hypertension: The Multi-Ethnic Study of Atherosclerosis (MESA)

By Jaylah Goodson

Background: There are racial differences in hypertension and sleep health, yet the mechanism is unknown. Therefore, we tested whether sleep irregularity contributed to racial differences in hypertension prevalence and control.

Methods: Data from the Multi-Ethnic Study of Atherosclerosis was used to evaluate the research question. Sleep irregularity (SD of sleep duration and sleep onset timing) was measured via 7-day actigraphy. Race was self-reported. Hypertension was defined as systolic blood pressure ≥ 130 mm/Hg or diastolic blood pressure ≥80 mm/Hg, antihypertensive medication use, or self-reported physician hypertension diagnosis. Hypertension control was defined as systolic blood pressure <130 mm/Hg and diastolic blood pressure <80 mm/Hg among those with hypertension. Separate logistic regression and linear regression produced odds ratios (OR) and Beta estimates for sleep irregularity by race. Poisson regression produced prevalence ratios (PR) for hypertension and hypertension control by race and sleep irregularity.

Results: Participants (n=1393) were 68.8 (SD=9.2) years of age, 46% male, 42% Black, 58% non-Hispanic White, and 48% had a college degree or higher. Average sleep irregularity was 83.4 minutes (SD=42) for sleep duration and 82.2 minutes (SD=94.8) for sleep onset timing. The prevalence of hypertension and hypertension control was 70% and 51% respectively. Black compared to non-Hispanic White adults had higher odds of irregular sleep duration (OR=5.71, 95% CI: 3.15, 10.34) and irregular sleep onset timing (OR=3.19, 95% CI: 2.26, 4.52). There was no association between sleep irregularity and hypertension prevalence or hypertension control. In fully adjusted models, Black compared to non-Hispanic White adults had a 24% higher prevalence of hypertension (PR=1.24, 95% CI: 1.09, 1.42). The association persisted with adjustment for sleep irregularity (categorically and continuously). There was no association between race and hypertension control. Exclusion of those who identified as working an 'other' shift and additional adjustments for AHI and average sleep duration produced associations that were consistent with the main analyses.

Conclusions: Racial differences in sleep irregularity and hypertension prevalence were observed. Sleep irregularity did not contribute to racial differences in hypertension prevalence or hypertension control. Further studies should evaluate other dimensions of sleep, to determine whether sleep contributes to racial disparities in hypertension.

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Jaylah Goodson, Dayna A. Johnson

Introduction

Sleep is a restorative and essential function for survival.[1] In fact, optimal sleep health, a multidimensional construct that includes including sleep satisfaction, alertness, timing, efficiency, and duration is associated with physical and mental health.[2] Despite the importance of sleep, as many as 33% of American adults sleep less than 7 hours a night, which is associated with adverse cardiovascular health, including hypertension.[3,4]As highlighted in Healthy People 2030, it is a public health priority to improve sleep, which may subsequently improve overall health, productivity, wellness, and quality of life.[5]

The physiological mechanism of sleep is expansive, consisting of numerous neurochemicals and neurotransmitters in order to support both periods of sleep and wakefulness.[6] Periods of wakefulness are maintained through a network of pathways fitted for the neurochemicals that stimulate what is referred to as the ascending arousal system.[6] Notable participants in this pathway include norepinephrine, serotonin, dopamine, and acetylcholine, among others.[6] In order to both commence and continue periods of sleep, this ascending arousal system must be suppressed, most often achieved through the neurons of the ventrolateral pre-optic area (VLPO).[6] Research has identified extracellular adenosine as responsible for activation of the VLPO.[7] The VLPO also receives input from circadian rhythm influences.[6,8]The circadian rhythm is regulated by light changes in our environment, and is often referred to as the body's 24-hour internal clock.[9] This internal clock helps to regulate cycles of both sleep and wakefulness.[9] Once sleep has been initiated, the body will cycle through two types of sleep. The first type is characterized by non-rapid

eye movement (NREM) by which the body's heart rate will slow, temperature will drop, and the body develops into a deep sleep.[10] NREM sleep has 3 stages, and the third stage, also known as slow wave sleep, is considered the deepest stage and where the body undergoes repairs for tissues, bone, and muscles, which may help us to understand it's connection to physiological processes.[10] The second type of sleep is characterized by rapid eye movement (REM) and is known for being the time where dreaming occurs.[10] When sleep is first initiated the REM period is short, but becomes longer as the sleep period progresses.[10] With these lengthened periods of REM sleep, the amount of time spent in NREM shortens.[10] The typical order of stages in the sleep cycle is N1, followed by N2, then to N3, returning to N2, and ending with REM, with N1-3 corresponding with the 3 stages of NREM sleep.[10] The complete sleep cycle lasts between 90 and 110 minutes, with 4-5 cycles observed in a typical night's sleep.[10]

Similar to how patterns of sleep and wakefulness are influenced by a circadian rhythm, the same can be said for blood pressure which experiences fluctuations depending on the time of day. When sympathetic activity in the body is increased, such as during times of stress, the heart rate elevates along with the force of contraction.[11,12] During slow wave sleep, sympathetic activity is decreased, thus allowing the heart to better stabilize blood pressure throughout the day.[13–15] Nocturnal dipping is a phenomenon that occurs during sleep, typically in slow wave sleep, where blood pressure falls by 10-20% less of daytime pressures.[15,16] Research supports the idea that it is important for the body to reach slow wave sleep, so that these mechanisms for repair and rest may take place and blood pressure can be lowered, known as nocturnal dipping.[17] It is important that this state of lowered blood pressure be reached, as research shows the absence of nocturnal dipping is associated with an increased risk of adverse cardiovascular outcomes.[18] Research also shows that decreased time spent in slow wave sleep is associated with an increased odds of incident hypertension, thus it is important to be mindful of the sleep practices that allow us to prolong our sleep cycles to reach this stage of sleep.[19]

Growing evidence suggests that sleep is an important indicator of cardiovascular health.[20,21] Sleep irregularity, including high deviations in sleep duration and timing, which may disturb sleep-wake cycling, can lead to a number of adverse cardiovascular health outcomes such as hypertension, myocardial infarction, stroke, and coronary heart disease death.[22,23] In fact, the American Heart Association has expanded the Life Simple 7 to the Essential 8, which includes sleep as vital to heart health.[24] Hypertension is one of the main contributing risk factors to cardiovascular disease.[25] Growing research suggests that sleep irregularity is associated with hypertension.[22,26] For example, Scott et al. found that irregularity, as measured by a non-wearable and non-intrusive sleep monitoring sensor, was associated with a 9 to 17% increase in hypertension.[22] Furthermore, Culver et al. found that actigraphy-measured sleep irregularity was associated with both brachial and aortic blood pressure.[26] Based on the prior studies and others, sleep irregularity may be an advantageous intervention target for reducing the burden of hypertension.

Racial and ethnic disparities in hypertension prevalence in the United States are well documented. Using data from the National Health and Nutrition Examination Survey (NHANES), Lopez-Neyman et al. found that 59.3% of non-Hispanic Black adults compared to 43.6% of non-Hispanic White adults had hypertension.[27] Likewise, using data from the Multi-Ethnic Study of Atherosclerosis, Kramer et al. found that the prevalence of hypertension among Black adults was significantly higher in comparison to their White counterparts; 60% and 38%, respectively.[28] Racial and ethnic disparities in hypertension control in the United States are also documented. Uncontrolled hypertension is typically defined as blood pressure above defined normal limits, typically 130 mm/Hg for systolic and 80 mm/Hg for diastolic, with the use of one or two classes of antihypertensive medication.[29] Resistant hypertension is typically defined as blood pressure above normal limits with the use of more than 3 classes of antihypertensive medications, with 1 being a diuretic, or more than 4 classes of antihypertensive medications regardless of blood pressure control.[29] Black adults are more likely to have uncontrolled hypertension than White adults (74.7% vs. 68.3%, respectively).[30] Similarly, among hypertensive populations, resistant hypertension was

more prevalent among Black Americans (10.5%) than European Americans, henceforth referred to as White (7.3%).[31] Given the consistent Black-White disparity in hypertension and hypertension control, it is important to understand the mechanism.

Similar to disparities in hypertension, Black Americans are disproportionately affected by poor sleep health.[32] Research shows that disparities in sleep irregularity by race and ethnicity exist such that Black adults experience greater sleep disparity, namely in sleep timing regularity, than White adults.[33] Among participants of the Multi Ethnic Study of Atherosclerosis (MESA), Black participants had significantly higher variability in sleep duration, 79.1 minutes compared to 59.5 minutes for non-Hispanic White participants.[33] Similar data exists using an alternative definition of sleep regularity, the Sleep Regularity Index (SRI),[34] which is defined as the likelihood that two actigraphy time-points 24 hours apart are the same sleep or wake cycle over the course of the actigraphy wear.[34] Among MESA participants, non-Hispanic White participants had the most regular sleep patterns and non-Hispanic Black participants had the highest sleep irregularity.[34] Emerging data suggests that sleep disparities are contributing to cardiovascular disparities, however data are limited.[35–37]

A small literature supports that sleep may explain racial and ethnic disparities in cardiovascular health. For example, a study by Rasmussen-Torvik et al. found that sleep maintenance mediated racial differences in hypertension by 11.4%.[38] Data from the Coronary Artery Risk Development in Young Adults study, suggests that sleep duration partially mediated the Black-White difference in diastolic blood pressure over 5 years.[39] While those are the only 2 studies focused on blood pressure or hypertension, others have shown sleep partially explained Black-White disparities in cardiovascular health outcomes.[40,41] While the data are promising, additional studies are needed, particularly those with objective measures of sleep over multiple nights, which is done via 7-day actigraphy.

Due to the observed associations between not only race and hypertension, but also the physiological connection of sleep and hypertension, sleep irregularity may be a mechanism through which the racial and ethnic disparities in hypertension are attributed. This hypothesized pathway of sleep irregularity may provide a better understanding of the associations of race, as a social construct, and both hypertension prevalence and hypertension control. With the use of 7-day actigraphy, we can better understand sleep-wake patterns, and how they may influence hypertension prevalence and control.

Using data from the Multi-Ethnic Study of Atherosclerosis, we analyzed objective measures of sleep irregularity through actigraphy as a potential mediator in the difference in hypertension prevalence and hypertension control among non-Hispanic Black and non-Hispanic White adults. We hypothesized that the irregularity in sleep and the inability of cycling through the restorative stages of sleep would mediate the pathway of race as a predictor of the prevalence of hypertension and the lack of hypertension control.

Methods

Study Population

The Multi-Ethnic Study of Atherosclerosis (MESA) is a prospective study that was designed to identify characteristics that are associated with the progression of subclinical cardiovascular disease (CVD) to clinical CVD, as well as other demographic differences that may also be contributing to the progression.[42] Recruited MESA participants included 6,814 men and women free of clinical CVD at baseline from 6 United States regions (Baltimore, MA; Chicago, IL; Forsyth County, NC; Los Angeles County, CA; New York, NY; and St. Paul, MN) whose ages at baseline ranged from 45 to 84 years old.[42] Of these participants, approximately 38% are White, 28% are Black, 23% are Hispanic/Latino, and 11% are Chinese.[42] The participants completed a comprehensive baseline examination from July 2000 to August 2002.[42] Follow up examinations were designed to be completed over 18 or 24-month increments.[42] The present study utilizes data from Exam 5 and includes the MESA Sleep Ancillary Study (2010-2013),

which include 1-night of in home polysomnography (PSG), 7-day wrist actigraphy, and sleep questionnaires.[43] The details of MESA Sleep have been previously published.[44–46] Participants who contributed less than 5 nights of actigraphy, were self-identified as not Black or non-Hispanic White, or had an extreme value of SD of sleep duration (>4 hours) were excluded, leaving 1,393 included for analysis. Institutional Review Board approval was obtained from each study site and all participants provided written informed consent.

Hypertension and hypertension control endpoints

Clinic visits were held for participants and a number of physical metrics were recorded upon examination including blood pressure.[47] Blood pressure was recorded using a Dinamap model Pro 100 automated oscillometric sphygmomanometer (Critikon, Tampa, FL).[47] Three measurements were taken, and the last two readings were averaged.[47] Hypertension was defined as systolic blood pressure ≥130 mm/Hg, diastolic pressure ≥80 mm/Hg, self-report of the use of antihypertensive medications, or self-report of a hypertension diagnosis.[28,29,48] Hypertension control was categorized as controlled and uncontrolled. Controlled hypertension was defined as systolic pressure <130 mm/Hg and diastolic pressure <80 mm/Hg, among those with hypertension.[49] Uncontrolled hypertension was defined as systolic pressure ≥ 130 mm/Hg or diastolic pressure ≥ 80 mm/Hg, among those with hypertension.[49] Hypertension at the time of the study had a higher threshold for systolic blood pressure and diastolic blood pressure, at 140 mm/Hg and 90 mm/Hg respectively.[50] There was 0.2% missing systolic and diastolic blood pressure readings (n=3), but they contained information on the every other metric of the hypertension definition, and were ultimately categorized as not having hypertension.

Sleep assessment

Participants in the MESA Sleep Ancillary Study wore the Actiwatch Spectrum wrist actigraph (Phillips Respironics, Murrysville, PA) for 7 consecutive days on their non-dominant wrist, while also logging their sleep and wake times in a sleep diary.[23] Signals generated from the actigraph were scored as sleep or

wake per each 30-second epoch based on changes in activity count, event markers, the previously referenced sleep diary, and changes in light levels.[23] A certified technician at the Sleep Reading Center of Brigham and Women's Hospital processed this data using Actiware-Sleep version 5.59 analysis software (Mini Mitter Co., Inc. Bend, OR) and scored using the Cole-Kripke algorithm.[23,46,51] Subsamples of rescored data were used to generate an inter-scorer reliability of 0.96 for sleep onset timing and 0.89 for wake timing.[23] Sleep irregularity in this analysis was quantified as the standard deviation (SD) of sleep duration and sleep onset timing, over 7 days. These variables were further categorized as ≤ 60 minutes, 61-90 minutes, 91-120 minutes, and > 120 minutes for sleep duration and ≤ 30 minutes, 31-60 minutes, 61-90 minutes, and > 90 minutes for sleep onset timing. The variables for sleep irregularity had moderate correlation (r=0.49) and were examined separately in all analyses.

For sensitivity analyses purposes, information about sleep apnea were included. The study also administered one night of at-home polysomnography using the Compumedics Somte system (Compumedics, Abbottsville, Australia). Sleep apnea severity was measured via the Apnea-Hypopnea Index (AHI).[43] The AHI was defined as all obstructive apneas and hypopneas associated with 4% or more of oxygen desaturation.[23,43] Moderate to severe sleep apnea was defined as AHI ≥ 15.

A sleep questionnaire wase administered to report sleep habits and sleep-related traits, such as insomnia and daytime sleepiness.[23] Participant's work schedule was grouped into 3 categories of 'day shift', 'other shift', and 'do not work'. 'Other shift' included the responses of 'afternoon shift', 'night shift', 'split shift', 'irregular shift/on-call', and 'rotating shifts'. Given the association between shift work and sleep timing, sensitivity analyses were conducted to explore the associations of the research question while excluding those who reported working other shift for their work schedule.[47]

Exposure

The main exposure of this analysis is race. Race, a social construct and proxy for racism, information was obtained from the participants via self-identification, and categorized as non-Hispanic White, non-Hispanic Black/African American, Hispanic, and Chinese. The focus of this paper is on non-Hispanic White and non-Hispanic Black participants, exclusively.

Covariates

Questionnaires were used to obtain information on participant's age, sex, educational attainment, work schedule, body mass index (BMI), smoking status, and physical activity. Education was categorized as less than high school, high school diploma, bachelor's degree, or graduate degree. Work schedule was categorized as day shift, other shift, or do not work. BMI was calculated using height and weight measurements and reported as kg per m^2. Smoking status was categorized as former or current, based on self-report. Physical activity was summed into total intentional exercise and reported in MET minutes per week. There was 0.3% missing BMI (n=4), 0.8% missing physical activity (n=11), and 0.9% missing work schedule (n=12).

Statistical Analysis

Descriptive statistics were examined by race (Black and non-Hispanic White) using independent-samples t-tests for continuous variables and Chi-squared tests were used for categorical variables. To evaluate the research question, separate logistic regressions, Poisson regressions, and linear regressions were used to examine the association of race and sleep irregularity both categorically and continuously, the association of sleep irregularity measures and hypertension prevalence and control, and the association of race on hypertension prevalence and hypertension control all using a sequential modeling approach. Separate logistic regression was used when categorical measures of sleep irregularity were the outcome, producing odds ratios that would help to observe the probability of sleep irregularity in our sample. Poisson regression was used when hypertension or uncontrolled hypertension were the outcome, as it would produce prevalence ratios that aid in the interpretability of the results, and was appropriate for the high

prevalence of both hypertension (70%) and uncontrolled hypertension (51%). The statistical methods produced odds ratios, prevalence ratios, and Beta estimates that are reported in subsequent tables to display the associations observed. Sleep irregularity measures (SD of sleep duration and sleep onset timing) were analyzed both categorically and continuously in separate models. All statistical analyses were carried out in R v4.3.3.

Model 1 adjusted for age, sex, education, work schedule and study site. Model 2 further adjusts for body mass index, smoking status, and physical activity. Model 3 included Model 2 + 7-day SD of sleep duration. Model 4 included Model 2 and 7-day SD sleep onset timing.

To conduct the cross-sectional mediation analysis, we employed an approach by Valeri and VanderWheele, to test path models by which 7-day SD of sleep duration and 7-day SD of sleep onset timing were modeled separately as a mediator predicted by race and as a predictor of hypertension and hypertension control.[52,53] Complete case analysis was used in the mediation analysis, due to missing values in BMI (n=4), physical activity (n=11), and work schedule (n=12). The approach by Valeri and VanderWheele allows for estimates to be generated by fitting two regression models; 1 Poisson model for the outcome of hypertension, and 1 linear model to test the mediator variable as an outcome.

Given the previous literature that support the associations between sleep and occupation, sensitivity analyses were conducted to understand the associations presented when the research question is tested excluding those who indicated they work an ‘other shift’ for their work schedule.[54–56] Additionally, sensitivity analyses were conducted to adjust for moderate to severe sleep apnea and average sleep duration, given its associations presented in existing literature.[57,58]

Results

The total analytic sample consisted of 1,393 Black and Non-Hispanic White participants. Table 1 shows selected characteristics of the study population (n=1393) both in total and stratified by race. Overall, this sample had an average age of 68.8 years (SD, 9.2), was mostly female (54%), most had a high school diploma (47%), and did not work (57%). Additionally, this sample was overweight, with a mean BMI of 29.1 (SD, 5.6) and almost half of the sample were former smokers (44%). The prevalence of hypertension was 70% and the prevalence of uncontrolled hypertension was 51%. In this sample, 41% of participants had greater than 120 minutes of SD of sleep duration and 15% had greater than 90 minutes of SD in sleep onset timing. Black participants in this study population had a lower educational attainment, a higher mean BMI, more non-workers, and more likely to be current smokers than non-Hispanic White Americans. Also, Black participants had higher sleep irregularity than Non-Hispanic White Americans; 98 vs 70 minutes for the mean SD of sleep onset timing and 94 vs 75 minutes for the mean SD of sleep duration. Among Black participants, 52% had >120 minutes of SD of sleep durations, compared to 33% for non-Hispanic White participants. For SD of sleep onset timing, 20% of Black participants had > 90 minutes compared to 10% for non-Hispanic White participants.

Table 2 provides the odds ratios and beta estimates of the relationship between race and categorical and continuous sleep irregularity, both unadjusted, and adjusted for covariates. Across all models, Black compared to non-Hispanic White Americans had more irregular sleep duration and sleep onset timing. Comparing ≤60 min for Black vs. non-Hispanic White Americans, the ORs (95% CIs) for the SD of sleep duration were 2.30 (1.24 to 4.25) for 61 to 90 min, 4.02 (2.17 to 7.45) for 91 to 120 min, and 5.71 (3.15 to 10.34) for >120 min in the fully adjusted model. Comparing ≤30 min for Black vs. non-Hispanic White Americans, the ORs (95% CIs) for the SD of sleep onset timing were 1.93 (1.43 to 2.61) for 31 to 60 min, 2.70 (1.85, 3.95) for 61 to 90 min, and 3.19 (2.26, 4.52) for >90 min in the fully adjusted model. For continuous sleep irregularity, Black compared to non-Hispanic White Americans had more deviation in their sleep duration and sleep onset timing, 0.31 (95% CI: 0.29, 0.46) and 0.31 (95% 0.29, 0.46), respectively.

Table 3 presents the prevalence ratios for hypertension and hypertension control in relation to sleep irregularity overall and by race. There were no observed associations between SD of sleep duration or SD of sleep onset timing and hypertension or hypertension control overall or by race.

Table 4 provides the prevalence ratios of hypertension prevalence and uncontrolled hypertension, while controlling for demographic characteristics and study site (model 1), health behaviors (model 2), and sleep irregularity (models 3-6). Black adults had 24% higher prevalence of hypertension after adjustment for age, sex, education, work schedule, study site, BMI, smoking status, and physical activity. This association attenuated but remained with adjustment of SD of sleep duration and sleep onset timing. There were no observed associations between race and uncontrolled hypertension. There were no observed associations between sleep irregularity and hypertension, thus there was no evidence for mediation.

Sensitivity analyses included exclusion for workers who identified as “other” shift, additional adjustments for moderate to severe sleep apnea and average sleep duration, and prevalence ratios for hypertension (systolic blood pressure ≥140 mm/Hg or diastolic blood pressure ≥ 90 mm/Hg or self-reported diagnosis of hypertension or use of antihypertensive medications) as defined at the time of data collection. All associations were consistent with the main analyses.

Discussion

This paper aimed to understand the role of sleep irregularity in the relationship between race and hypertension. We observed an association between race and sleep irregularity and race and hypertension prevalence, such that Black participants were more likely to have irregular sleep and hypertension compared to White participants. No association was observed between race and uncontrolled

hypertension or sleep irregularity and hypertension prevalence or uncontrolled hypertension. These findings support racial disparities in both sleep irregularity and hypertension. While sleep irregularity did not contribute to racial disparities in hypertension, there is a need for future studies to identify the mechanism by which these disparities occur.

There were racial differences in sleep irregularity. Black individuals had higher odds of sleep irregularity (SD of sleep duration and SD of sleep onset timing) than non-Hispanic White participants. The magnitude of this association increased as sleep irregularity increased, reflecting a dose-response relationship. This association is consistent with previous literature, by which Chung et al. found that Black participants were more likely to have higher variability in both sleep timing and duration.[33] This identified disparity highlights an important area that should be further studied, to identify interventions to reduce variability and irregularity in sleep patterns or the determinants contributing to irregular sleep.

There was no association between sleep irregularity and hypertension both overall and by race. These findings were unexpected and was contrary to our proposed hypothesis. In a sample of 12,287 participants of which the average was 50 and 88% of participants were male, Scott et. al found that irregularity in sleep duration was associated with a 9 to 17% increase in hypertension. There are a few key differences between the current sample and Scott et al.'s that may explain the difference in findings.[22] First, the sleep metrics were gathered by different means. This study evaluates actigraphy data, whereas Scott et. al used contactless sleep technology, which is placed underneath the participant's mattress and uses ballistography to capture the parameters of sleep.[22] This signaling captures breathing and cardiac activity, in addition to movement.[59] However, a study by G Ravindran et al found that contactless sleep technologies do not estimate all night sleep summary measures as well as actigraphy.[59] Participants in the Scott et al. study also had to complete at least 28 nights of sleep recordings, whereas this study used actigraphy for only 7 days.[22] Having more nights to gather data on sleep patterns is a more favorable means of establishing sleep irregularity, despite the method used.[22] For example, the mean SD of sleep

onset timing in this sample was 82 minutes vs 70 minutes in the Scott et al. study. Overall, these differences may explain the lack of association for sleep irregularity and its relationship to hypertension. A study by Nikbakhtian et al. found an association between sleep onset timing and risk of cardiovascular disease.[60] Cardiovascular disease is different than hypertension, which is likely why there is a difference in findings between this study and Nikbakhtian et al.'s. Häusler et al. studied the relationship between irregular sleep duration through 14 days of actigraphy and hypertension, and did not find an association.[61] With Scott et al. being the only current study to observe an association between sleep irregularity and hypertension, further studies should seek to validate the methods used to gather sleep irregularity data and confirm the associations found. There was no association between sleep irregularity and uncontrolled hypertension, which was also unexpected and contrary to the proposed hypothesis. A previous study has observed an association between sleep duration and hypertension control, though this study did not test the association between sleep duration and hypertension control.[62]

Racial differences in hypertension were observed. Black participants had as much as a 29% higher prevalence of hypertension, compared to Non-Hispanic White participants. This association is consistent with previous studies, which have found a higher prevalence of hypertension in Black adults than non-Hispanic White adults.[27,28] Associations between race and hypertension prevalence may be due to social determinants of health that disproportionately affect Black Americans. One of these determinants is insurance coverage, which may impact one's ability to access preventative care for hypertension. A study by Buchmueller et. al sought to understand how the Affordable Care Act (ACA) impacted health insurance coverage by race and ethnicity. Decreased attainment of insurance coverage was seen for Black individuals compared to White individuals; 25.8% uninsured vs 14.8% uninsured.[63] They found that the ACA did help to decrease this difference in insurance attainment, yet disparities in coverage still remain.[63] Furthermore, racial discrimination as a social determinant of health may also be contributing to the difference seen in hypertension prevalence by race. As such, fear of racial discrimination may also be feeding the apprehension of Black Americans to seek healthcare. A study by Bleich et al. sought to

understand the experiences of Black Americans and the discrimination they face.[64] They found that 32% of Black adults reported experiences of discrimination in clinical settings and 22% reported that they avoided seeking health care due to the anticipation of experiencing discrimination.[64] It is important to note that these experiences of discrimination do not only occur with lower levels of income or educational attainment. A study by Stepanikova and Oates found that at higher incomes and educational attainment, Black Americans reported more discrimination than White Americans.[65] Thus, while efforts should be made to increase access to healthcare by way of insurance coverage, efforts should also be made to reduce discrimination that is experienced by Black Americans in healthcare, which may in turn help to reduce the burden of hypertension prevalence that is increased among Black individuals. We did not find an association between race and hypertension control. A previous study by Kramer et. al found an association between race and hypertension control.[28] It is possible this discrepancy can be explained by the difference in sample size between the present study, and the aforementioned study by Kramer et al. Though both studies use data from MESA, this study restricted the sample to those contributing sleep data at Exam 5. The Kramer et al utilized data from Exam 1 of MESA, and of the 6,814 adults included for analysis, 3,068 had hypertension. The 3,068 participants with hypertension were self-identified as either White, Black, Asian, or Hispanic. The present study had a total of 983 participants with hypertension out of 1,393 total participants, of which were self-identified as either Black or White. Of these 983 participants with hypertension, 486 were Black and 497 were White. Of the Black participants with hypertension, 227 had controlled hypertension. It is possible this difference in sample size may explain the lack of association observed in this study between race and hypertension control.

There was no evidence of an association between sleep irregularity and hypertension prevalence or hypertension control, thus no mediation analysis was conducted. No other studies have tested sleep irregularity as a mediator, though one study did find that sleep duration could be attributed to racial differences in diastolic blood pressure.[39] However, sleep duration and irregular sleep duration are two different dimensions of sleep.[2] Though this study could not attribute the racial differences in hypertension

prevalence to sleep irregularity, this hypothesized pathway remains an area that should be further studied. Evaluating other metrics of sleep that are known to differ by race could also be evaluated a mediator on this pathway, such as short sleep duration.[66]

The findings of this study help us to further understand the relationship between race and both sleep irregularity and hypertension prevalence, as understanding this relationship may help us to decrease these adverse events in sleep and cardiovascular health and improve overall outcomes in both sleep health and cardiovascular health. These observed associations are of importance when understanding the way in which social determinants of health operate, and relate to overall measures of health, such as hypertension and sleep health. The findings of this study, in tandem with previous literature, further proves the disparities Black Americans face in both sleep irregularity and hypertension prevalence. Future works should strive to understand the means by which these associations present and focus on interventions that would in turn reduce their impact on sleep and cardiovascular health outcomes.

There are several strengths in this study. The use of an objective measure of sleep, such as actigraphy, is recognized as a more reliable and superior measure of sleep than self-report methods.[67] The availability of objective measures of sleep through both actigraphy and polysomnography in this data help to garner confidence in the results produced. The MESA study took great care to ensure a sample that was representative of many races and ethnicities, and as a result generated a sufficient sample of Black and White adults to be included in these analyses, as disparities by race were evaluated. The MESA data was comprehensive, and the availability of many important variables of which could be controlled for and assessed in the analyses provided for robust results to be obtained and evaluation of possible mechanisms by which the study findings may be explained by.

However, there are some limitations. The most immediate limitation is the type of data used to answer this research question. Because the ancillary sleep study was included only at Exam 5, this data is cross

sectional, therefore no assessments of temporality can be made when evaluating the hypothesized relationships, and causality cannot be established. This data was collected at one time point, and as such may not be most representative of the associations tested. This sample is not representative of Black and White adults in the United States (US), the mean age was 69 (SD, 9) and most participants did not work. Further research should be done to evaluate a sample with more variation in age and work schedule. Lastly, the MESA study only has 6 sites across the United States that were included in this analysis. This may lead to a lack of generalizability, as there may be differences among the sites not included.

In conclusion, the results presented suggest that there are racial differences in sleep irregularity and hypertension, however, there was no evidence that sleep irregularity contributed to the racial differences in hypertension or hypertension control. Though our hypothesis sought to find that variability in sleep may be contributing to racial disparities, this question should be further tested with more robust and longitudinal data to better understand the mechanisms by which this hypothesis may occur.

Table 1. Descriptive of Black Americans, BA (n=590) and non-Hispanic Whites, NHW (n=803) in the MESA Study (n=1393)

Variables	Overall *n* = 1393	Black *n* = 590	NHW *n* = 803
Age in years (mean, SD)	68.8 (9.2)	68.6 (9.0)	68.9 (9.2)
Male (n, %)	636 (46)	262 (44)	374 (47)
Education (n, %)			
Less than high school	69 (5.0)	44 (7.5)	25 (3.1)
High school diploma	649 (47)	332 (56)	317 (39)
Bachelor's degree	322 (23)	113 (19)	209 (26)
Graduate degree	353 (25)	101 (17)	252 (31)
Work Schedule (n, %)			
Day shift	414 (30)	165 (28)	249 (31)
Other shift	183 (13)	80 (14)	103 (13)
Afternoon shift	34 (2.5)	22 (3.7)	12 (1.5)
Night shift	24 (1.7)	16 (2.7)	8 (1.0)
Split shift	14 (1.0)	8 (1.4)	6 (0.8)
Irregular shift/on-call	98 (7.1)	26 (4.4)	72 (9.1)
Rotating shifts	13 (0.9)	8 (1.4)	5 (0.6)
Do not work	784 (57)	342 (58)	442 (56)
BMI (kg/m^2) (mean, SD)	29.1 (5.6)	30.4 (5.6)	28.1 (5.4)
Smoking Status (n, %)			
Former	612 (44)	235 (40)	377 (47)
Current	113 (8.1)	67 (11)	46 (5.7)
Physical Activity (mean, SD)			
Total Intentional Exercise, MET-min/week	3053 (3935)	3292 (4901)	2879 (3041)
Sleep Irregularity			
7-day SD in sleep duration			
≤ 60 min (n, %)	91 (6.5)	16 (2.7)	75 (9.3)
61-90 min (n, %)	389 (28)	117 (20)	272 (34)
91-120 min (n, %)	345 (25)	153 (26)	192 (24)
> 120 min (n, %)	568 (41)	304 (52)	264 (33)
Per hour (mean, SD)	1.39 (0.70)	1.57 (0.68)	1.25 (0.69)
7-day SD in sleep onset timing			
≤ 30 min (n, %)	736 (53)	240 (41)	496 (62)
31-60 min (n, %)	297 (21)	142 (24)	155 (19)
61-90 min (n, %)	157 (11)	89 (15)	68 (8.5)
> 90 min (n, %)	203 (15)	119 (20)	84 (10)
Per hour (mean, SD)	1.37 (1.58)	1.64 (1.64)	1.17 (1.51)
Hypertensive (n, %)	983 (70)	486 (82)	497 (62)
Hypertensive, as defined by MESA	832 (60)	427 (72)	405 (50)
Hypertension Control (n, %)	483 (49)	227 (47)	256 (52)

BMI = Body Mass Index; SD = standard deviation

Table 2. Odds ratios and Beta estimates of sleep irregularity in the MESA Study (n=1393)

	7-day SD of sleep duration					7-day SD of sleep onset timing				
	≤60 min	61-90 min	91-120 min	>120 min	Per hour	≤30 min	31-60 min	61-90 min	>90 min	Per hour
Unadjusted										
Race										
NHW (REF)										
BA	REF	2.02* (1.13, 3.61)	3.74** (2.09, 6.67)	5.40** (3.07, 9.49)	0.32** (0.25, 0.39)	REF	1.89** (1.44, 2.49)	2.70** (1.90, 3.84)	2.93** (2.13, 4.03)	0.46** (0.30, 0.63)
Adjusted										
Race										
NHW (REF)										
BA	REF	2.30** (1.24, 4.25)	4.02** (2.17, 7.45)	5.71** (3.15, 10.34)	0.31** (0.24, 0.39)	REF	1.93** (1.43, 2.61)	2.70** (1.85, 3.95)	3.19** (2.26, 4.52)	0.31** (0.24, 0.39)

* P < 0.05; ** P < 0.01

BA = Black American; NHW = non-Hispanic White

SD = standard deviation; OR = odds ratio; CI = confidence interval

Values reported as OR (95% CI) or β (95% CI)

Adjusted for age, sex, study site, BMI, physical activity, and education.

Table 3. Prevalence ratios of hypertension (n= 1393) and hypertension control (n=979) in the MESA study overall and stratified by race.

	Overall		Black Americans		Non-Hispanic White Americans	
	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Hypertension						
7-day SD of sleep duration						
61-90 min	0.95 (0.72, 1.25)	0.94 (0.71, 1.24)	1.04 (0.56, 1.91)	1.04 (0.57, 1.92)	0.88 (0.64, 1.21)	0.87 (0.63, 1.20)
91-120 min	1.05 (0.79, 1.38)	1.02 (0.77, 1.35)	1.10 (0.60, 2.00)	1.10 (0.60, 2.02)	0.94 (0.67, 1.31)	0.92 (0.66, 1.29)
>120 min	1.09 (0.83, 1.42)	1.06 (0.81, 1.38)	1.09 (0.61, 1.97)	1.10 (0.61, 1.99)	0.97 (0.70, 1.33)	0.96 (0.70, 1.32)
Per hour	1.08* (0.99, 1.18)	1.07 (0.98, 1.18)	1.01 (0.89, 1.16)	1.02 (0.89, 1.17)	1.06 (0.94, 1.21)	1.06 (0.93, 1.20)
7-day SD of sleep onset timing						
31-60 min	1.10 (0.94, 1.30)	1.10 (0.93, 1.29)	1.03 (0.82, 1.30)	1.05 (0.83, 1.32)	1.11 (0.88, 1.41)	1.09 (0.87, 1.38)
61-90 min	1.15 (0.94, 1.41)	1.13 (0.92, 1.39)	1.01 (0.77, 1.32)	1.02 (0.77, 1.34)	1.19 (0.87, 1.62)	1.17 (0.86, 1.60)
>90 min	1.18 (0.98, 1.42)	1.15 (0.95, 1.39)	1.04 (0.81, 1.34)	1.04 (0.81, 1.34)	1.14 (0.85, 1.53)	1.16 (0.86, 1.56)
Per hour	1.03 (0.99, 1.07)	1.02 (0.99, 1.06)	1.00 (0.95, 1.06)	1.00 (0.95, 1.06)	1.02 (0.97, 1.08)	1.03 (0.97, 1.09)
Uncontrolled Hypertension						
7-day SD of sleep duration						
61-90 min	1.00 (0.67, 1.50)	1.00 0.67, 1.51)	0.98 (0.41, 2.31)	1.00 (0.42, 2.37)	1.02 (0.63, 1.63)	1.03 (0.64, 1.65)
91-120 min	1.09 (0.72, 1.64)	1.09 (0.73, 1.64)	1.09 (0.47, 2.53)	1.10 (0.47, 2.57)	1.02 (0.62, 1.67	1.04 (0.63, 1.71)
>120 min	1.05 (0.71, 1.55)	1.04 (0.70, 1.53)	0.97 (0.43, 2.23)	0.96 (0.42, 2.20)	1.08 (0.68, 1.71)	1.10 (0.69, 1.76)
Per hour	1.04 (0.92, 1.18)	1.03 (0.90, 1.17)	1.00 (0.83, 1.21)	0.98 (0.81, 1.18)	1.05 (0.88, 1.26)	1.06 (0.88, 1.27)
7-day SD of sleep onset timing						
31-60 min	1.05 (0.84, 1.32)	1.03 (0.82, 1.30)	0.97 (0.71, 1.33)	0.96 (0.70, 1.32)	1.10 (0.79, 1.54)	1.09 (0.78, 1.53)
61-90 min	1.10 (0.83, 1.45)	1.10 (0.83, 1.46)	1.04 (0.72, 1.50)	1.04 (0.72, 1.52)	1.09 (0.70, 1.71)	1.11 (0.71, 1.73)
>90 min	1.09 (0.84, 1.41)	1.07 (0.82, 1.39)	0.98 (0.69, 1.38)	0.96 (0.67, 1.38)	1.16 (0.78, 1.75)	1.18 (0.78, 1.79)
Per hour	1.01 (0.95, 1.06)	1.00 (0.95, 1.06)	0.99 (0.91, 1.07)	0.99 (0.90, 1.08)	1.01 (0.94, 1.10)	1.01 (0.94, 1.10)

* $P < 0.1$

SD = standard deviation; PR = prevalence ratio; CI = confidence interval

Values reported as PR (95% CI)

≥60 min is referent group for 7-day SD in sleep duration.

≥30 min is referent group for 7-day SD in sleep onset timing.

N=983 for hypertension overall, n=486 for BA and n=497 for NHW
N=500 for uncontrolled hypertension overall, n=259 for BA and n=241 for NHW
Model 1 adjusts for age, sex, education, work schedule, and study site.
Model 2 adjusts for Model 1 + body mass index, smoking status, and physical activity.
Separate models were run for the categorical and continuous variables representing sleep irregularity.

Table 4. Prevalence ratios of hypertension (n= 1393) and uncontrolled hypertension (n=983) in the MESA Study

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Hypertension						
Race						
NHW (REF)						
BA	1.29** (1.13, 1.47)	1.24** (1.08, 1.42)	1.23** (1.07, 1.41)	1.22** (1.07, 1.40)	1.22** (1.06, 1.40)	1.23** (1.07, 1.41)
7-day SD of sleep duration						
61-90 min			0.90 (0.69, 1.20)			
91-120 min			0.97 (0.73, 1.28)			
>120 min			0.98 (0.75, 1.29)			
Per hour				1.04 (0.95, 1.15)		
7-day SD of sleep onset timing						
31-60 min					1.07 (0.91, 1.26)	
61-90 min					1.09 (0.89, 1.34)	
>90 min					1.10 (0.91, 1.33)	
Per hour						1.01 (0.98, 1.06)
Uncontrolled Hypertension						
Race						
NHW (REF)						
BA	1.09 (0.91, 1.31)	1.07 (0.89, 1.29)	1.07 (0.88, 1.29)	1.07 (0.88, 1.29)	1.06 (0.88, 1.28)	1.07 (0.89, 1.29)
7-day SD of sleep duration						
61-90 min			0.99 (0.66, 1.49)			
91-120 min			1.07 (0.71, 1.62)			
>120 min			1.01 (0.68, 1.50)			
Per hour				1.02 (0.89, 1.16)		
7-day SD of sleep onset timing						
31-60 min					1.02 (0.81, 1.29)	
61-90 min					1.09 (0.82, 1.45)	

>90 min	1.06 (0.81, 1.38)	
Per hour		1.00 (0.94, 1.06)

* P < 0.05; ** P < 0.01
BA = Black American; NHW = non-Hispanic White
SD = standard deviation; PR = prevalence ratio; CI = confidence interval
Values reported as PR (95% CI)
≥60 min is referent group for 7-day SD in sleep duration.
≥30 min is referent group for 7-day SD in sleep onset timing.
N=983 for hypertension; N=500 with uncontrolled hypertension
Model 1 adjusts for age, sex, education, work schedule, and study site.
Model 2 adjusts for Model 1 + body mass index, smoking status, and physical activity.
Model 3 adjusts for Model 2 + 7-day SD of sleep duration (categorically).
Model 4 adjusts for Model 2 + 7-day SD of sleep duration (continuously).
Model 5 adjusts for Model 2 + 7-day SD of sleep onset timing (categorically).
Model 6 adjusts for Model 2 + 7-day SD of sleep onset timing (continuously).

Supplemental Table 1. Odds ratios and Beta estimates of sleep irregularity in the MESA Study (n=1210) excluding shift workers (n=183)

	7-day SD of sleep duration					7-day SD of sleep onset timing				
	≤60 min	61-90 min	91-120 min	>120 min	Per hour	≤30 min	31-60 min	61-90 min	>90 min	Per hour
Unadjusted										
Race										
NHW (REF)										
BA	REF	2.28* (1.21, 4.31)	3.91** (2.06, 7.40)	5.61** (3.01, 10.43)	0.30** (0.22, 0.38)	REF	2.06** (1.54, 2.77)	2.68** (1.84, 3.91)	2.60** (1.83, 3.70)	0.40** (0.23, 0.57)
Adjusted										
Race										
NHW (REF)										
BA	REF	2.60** (1.33, 5.08)	4.43** (2.25, 8.72)	5.97** (3.10, 11.50)	0.30** (0.22, 0.38)	REF	2.06** (1.50, 2.83)	2.91** (1.93, 4.39)	2.85** (1.94, 4.18)	0.30** (0.22, 0.38)

* P < 0.05; ** P < 0.01

BA = Black American; NHW = non-Hispanic White

SD = standard deviation; OR = odds ratio; CI = confidence interval

Values reported as OR (95% CI)

Adjusted for age, sex, study site, BMI, physical activity, and education

Supplemental Table 2. Odds ratios and Beta estimates of sleep irregularity in the MESA Study (n=1393) with additional adjustment for AHI and average sleep duration

	7-day SD of sleep duration					7-day SD of sleep onset timing				
	≤60 min	61-90 min	91-120 min	>120 min	Per hour	≤30 min	31-60 min	61-90 min	>90 min	Per hour
Adjusted										
Race										
NHW (REF)										
BA	REF	1.85 (0.94, 3.66)	2.84** (1.46, 5.52)	4.79** (2.55, 8.98)	0.30** (0.22, 0.38)	REF	1.85** (1.34, 2.55)	2.39** (1.55, 3.69)	1.94** (1.30, 2.91)	0.31** (0.12, 0.50)

* P < 0.05; ** P < 0.01

BA = Black American; NHW = non-Hispanic White

SD = standard deviation; OR = odds ratio; CI = confidence interval

Values reported as OR (95% CI)

Adjusted for age, sex, study site, BMI, physical activity, education, AHI and average sleep duration.

Supplemental Table 3. Prevalence ratios of hypertension (n=1210) and hypertension control (n=876) in the MESA Study overall and stratified by race, excluding shift workers (n=183).

	Overall		Black Americans		Non-Hispanic White Americans	
	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Hypertension (n=983)						
7-day SD of sleep duration						
61-90 min	0.99 (0.74, 1.33)	0.97 (0.72, 1.31)	1.16 (0.58, 2.31)	1.16 (0.58, 2.32)	0.90 (0.64, 1.26)	0.89 (0.64, 1.25)
91-120 min	1.05 (0.78, 1.42)	1.03 (0.76, 1.39)	1.22 (0.61, 2.43)	1.21 (0.61, 2.43)	0.93 (0.66, 1.33)	0.92 (0.4, 1.31)
>120 min	1.09 (0.82, 1.45)	1.06 (0.79, 1.41)	1.21 (0.62, 2.38)	1.22 (0.62, 2.40)	0.95 (0.68, 1.33)	0.95 (0.68, 1.33)
Per hour	1.07 (0.98, 1.18)	1.07 (0.97, 1.18)	1.03 (0.90, 1.19)	1.04 (0.90, 1.21)	1.04 (0.91, 1.19)	1.05 (0.91, 1.20)
7-day SD of sleep onset timing						
31-60 min	1.13 (0.95, 1.34)	1.12 (0.94, 1.33)	1.06 (0.83, 1.35)	1.07 (0.84, 1.36)	1.13 (0.88, 1.44)	1.10 (0.86, 1.42)
61-90 min	1.14 (0.92, 1.42)	1.13 (0.91, 1.41)	1.03 (0.77, 1.37)	1.03 (0.77, 1.38)	1.16 (0.83, 62)	1.15 (0.82, 1.62)
>90 min	1.17 (0.95, 1.43)	1.14 (0.93, 1.40)	1.04 (0.79, 1.37)	1.04 (0.78, 1.37)	1.17 (0.86, 1.60)	1.20 (0.88, 1.64)
Per hour	1.03 (0.98, 1.07)	1.02 (0.98, 1.06)	1.01 (0.94, 1.07)	1.00 (0.94, 1.07)	1.02 (0.96, 1.08)	1.03 (0.97, 1.09)
Uncontrolled Hypertension						
7-day SD of sleep duration						
61-90 min	1.01 (0.65, 1.55)	1.01 (0.65, 1.56)	1.09 (0.39, 3.05)	1.10 (0.39, 3.09)	1.01 (0.61, 1.66)	1.02 (0.62, 1.68)
91-120 min	1.12 (0.72, 1.73)	1.13 (0.73, 1.75)	1.22 (0.44, 3.40)	1.24 (0.45, 3.47)	1.06 (0.63, 1.77)	1.08 (0.64, 1.82)
>120 min	1.08 (0.71, 1.64)	1.06 (0.70, 1.62)	1.14 (0.42, 3.12)	1.13 (0.41, 3.08)	1.04 (0.64, 1.70)	1.07 (0.65, 1.75)
Per hour	1.05 (0.92, 1.20)	1.04 (0.91, 1.19)	1.03 (0.85, 1.26)	1.01 (0.82, 1.24)	1.04 (0.87, 1.26)	1.05 (0.87, 1.27)
7-day SD of sleep onset timing						
31-60 min	1.09 (0.86, 1.38)	1.07 (0.84, 1.36)	1.04 (0.75, 1.45)	1.03 (0.73, 1.43)	1.09 (0.77, 1.55)	1.08 (0.75, 1.54)
61-90 min	1.08 (0.80, 1.46)	1.09 (0.80, 1.47)	1.05 (0.71, 1.56)	1.07 (0.72, 1.58)	1.04 (0.64, 1.69)	1.05 (0.65, 1.70)
>90 min	1.06 (0.80, 1.40)	1.03 (0.77, 1.38)	0.93 (0.63, 1.38)	0.92 (0.61, 1.37)	1.17 (0.77, 1.78)	1.21 (0.78, 1.86)
Per hour	1.00 (0.94, 1.06)	1.00 (0.94, 1.06)	0.98 (0.89, 1.08)	0.98 (0.89, 1.09)	1.01 (0.93, 1.10)	1.02 (0.94, 1.10)

*** P < 0.01; ** P < 0.05; * P < 0.1

SD = standard deviation; PR = prevalence ratio; CI = confidence interval

Values reported as PR (95% CI)

≥60 min is referent group for 7-day SD in sleep duration.

≥30 min is referent group for 7-day SD in sleep onset timing.

N=876 for hypertension overall, n=426 for BA and n=450 for NHW
N=446 for uncontrolled hypertension overall, n=226 for BA and n=220 for NHW
Model 1 adjusts for age, sex, education, work schedule, and study site.
Model 2 adjusts for Model 1 + body mass index, smoking status, and physical activity.
Separate models were run for the categorical and continuous variables representing sleep irregularity.

Supplemental Table 4. Prevalence ratios of hypertension (n= 1393) and hypertension control (n=983) in the MESA Study overall and stratified by race, with additional adjustments for AHI and average sleep duration

	Overall	Black Americans	Non-Hispanic White Americans
Hypertension			
7-day SD of sleep duration			
61-90 min	0.93 (0.68, 1.26)	1.05 (0.55, 1.99)	0.85 (0.60, 1.21)
91-120 min	1.02 (0.75, 1.39)	1.12 (0.60, 2.12)	0.92 (0.63, 1.33)
>120 min	1.03 (0.77, 1.39)	1.12 (0.60, 2.07)	0.90 (0.63, 1.30)
Per hour	1.06 (0.96, 1.17)	1.02 (0.88, 1.18)	1.03 (0.89, 1.20)
7-day SD of sleep onset timing			
31-60 min	1.07 (0.90, 1.27)	1.04 (0.81, 1.32)	1.05 (0.81, 1.36)
61-90 min	1.12 (0.89, 1.40)	1.00 (0.74, 1.35)	1.20 (0.84, 1.70)
>90 min	1.12 (0.90, 1.38)	1.04 (0.78, 1.39)	1.11 (0.80, 1.54)
Per hour	1.02 (0.98, 1.06)	1.00 (0.94, 1.07)	1.03 (0.97, 1.09)
Uncontrolled Hypertension			
7-day SD of sleep duration			
61-90 min	1.02 (0.64, 1.60)	0.88 (0.37, 2.12)	1.09 (0.64, 1.87)
91-120 min	1.11 (0.71, 1.76)	1.01 (0.43, 2.37)	1.09 (0.62, 1.92)
>120 min	1.04 (0.67, 1.61)	0.85 (0.37, 1.96)	1.14 (0.66, 1.98)
Per hour	1.03 (0.89, 1.19)	0.96 (0.78, 1.18)	1.05 (0.86, 1.29)
7-day SD of sleep onset timing			
31-60 min	0.99 (0.77, 1.26)	0.93 (0.66, 1.30)	1.04 (0.72, 1.50)
61-90 min	1.03 (0.75, 1.41)	1.02 (0.67, 1.55)	1.00 (0.61, 1.66)
>90 min	1.00 (0.74, 1.36)	0.91 (0.60, 1.38)	1.08 (0.68, 1.71)
Per hour	0.99 (0.93, 1.06)	0.98 (0.89, 1.08)	1.00 (0.92, 1.09)

*** P < 0.01; ** P < 0.05; * P < 0.1

SD = standard deviation; PR = prevalence ratio; CI = confidence interval

Values reported as PR (95% CI)

≥60 min is referent group for 7-day SD in sleep duration.

≥30 min is referent group for 7-day SD in sleep onset timing.

N=983 for hypertension overall, n=486 for BA and n=497 for NHW

N=500 for uncontrolled hypertension overall, n=259 for BA and n=241 for NHW

Model 1 adjusts for age, sex, education, work schedule, study site, BMI, smoking status, physical activity, AHI, and average sleep duration

Separate models were run for the categorical and continuous variables representing sleep irregularity.

Supplemental Table 5. Prevalence ratios of hypertension (n= 1210) and uncontrolled hypertension (n=876) in the MESA Study, excluding other shift workers (n=183)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Hypertension						
Race						
NHW (REF)						
BA	1.26** (1.09, 1.44)	1.21** (1.05, 1.39)	1.20** (1.04, 1.39)	1.19* (1.03, 1.38)	1.19* (1.03, 1.37)	1.20** (1.04, 1.39)
7-day SD of sleep duration						
61-90 min			0.94 (0.70, 1.27)			
91-120 min			0.98 (0.72, 1.32)			
>120 min			0.99 (0.74, 1.33)			
Per hour				1.04 (0.94, 1.15)		
7-day SD of sleep onset timing						
31-60 min					1.09 (0.92, 1.30)	
61-90 min					1.09 (0.87, 1.36)	
>90 min					1.10 (0.89, 1.35)	
Per hour						1.01 (0.97, 1.06)
Uncontrolled Hypertension						
Race						
NHW (REF)						
BA	1.09 (0.90, 1.32)	1.07 (0.88, 1.31)	1.06 (0.87, 1.30)	1.06 (0.87, 1.30)	1.06 (0.87, 1.30)	1.07 (0.88, 1.31)
7-day SD of sleep duration						
61-90 min			1.00 (0.64, 1.54)			
91-120 min			1.10 (0.71, 1.72)			
>120 min			1.04 (0.68, 1.60)			
Per hour				1.03 (0.90, 1.18)		
7-day SD of sleep onset timing						
31-60 min					1.06 (0.83, 1.35)	

61-90 min	1.08 (0.80, 1.46)	
>90 min	1.02 (0.76, 1.37)	
Per hour		1.00 (0.94, 1.06)

* P < 0.05; ** P < 0.01
BA = Black American; NHW = non-Hispanic White
SD = standard deviation; PR = prevalence ratio; CI = confidence interval
Values reported as PR (95% CI)
≥60 min is referent group for 7-day SD in sleep duration.
≥30 min is referent group for 7-day SD in sleep onset timing.
N=876 for hypertension; N=446 for uncontrolled hypertension
Model 1 adjusts for age, sex, education, work schedule, and study site.
Model 2 adjusts for Model 1 + body mass index, smoking status, and physical activity.
Model 3 adjusts for Model 2 + 7-day SD of sleep duration (categorically).
Model 4 adjusts for Model 2 + 7-day SD of sleep duration (continuously).
Model 5 adjusts for Model 2 + 7-day SD of sleep onset timing (categorically).
Model 6 adjusts for Model 2 + 7-day SD of sleep onset timing (continuously).

Supplemental Table 6. Prevalence ratios of hypertension (n= 1393) and uncontrolled hypertension (n=983) in the MESA Study with additional adjustment for AHI and average sleep duration

	Model 1	Model 2	Model 3	Model 4
Hypertension				
Race				
NHW (REF)				
BA	1.23** (1.06, 1.43)	1.23** (1.06, 1.43)	1.23** (1.06, 1.43)	1.24** (1.07, 1.43)
7-day SD of sleep duration				
61-90 min	0.91 (0.67, 1.23)			
91-120 min	0.98 (0.72, 1.34)			
>120 min	0.97 (0.72, 1.32)			
Per hour		1.03 (0.93, 1.14)		
7-day SD of sleep onset timing				
31-60 min			1.04 (0.87, 1.24)	
61-90 min			1.08 (0.86, 1.36)	
>90 min			1.08 (0.87, 1.34)	
Per hour				1.01 (0.97, 1.06)
Uncontrolled Hypertension				
Race				
NHW (REF)				
BA	1.00 (0.81, 1.24)	1.00 (0.81, 1.23)	1.01 (0.82, 1.24)	1.01 (0.82, 1.24)
7-day SD of sleep duration				
61-90 min	1.02 (0.64, 1.60)			
91-120 min	1.11 (0.70, 1.76)			
>120 min	1.04 (0.66, 1.62)			
Per hour		1.03 (0.89, 1.19)		
7-day SD of sleep onset timing				
31-60 min			0.99 (0.77, 1.26)	
61-90 min			1.03 (0.75, 1.41)	
>90 min			1.00 (0.74, 1.36)	
Per hour				0.99 (0.93, 1.06)

* P < 0.05; ** P < 0.01

BA = Black American; NHW = non-Hispanic White
SD = standard deviation; PR = prevalence ratio; CI = confidence interval
Values reported as PR (95% CI)
≥60 min is referent group for 7-day SD in sleep duration.
≥30 min is referent group for 7-day SD in sleep onset timing.
N=983 for hypertension; N=500 for uncontrolled hypertension
Model 1 adjusts for age, sex, education, work schedule, BMI, smoking status, physical activity, 7-day SD of sleep duration (categorically) + AHI and average sleep duration
Model 2 adjusts for age, sex, education, work schedule, BMI, smoking status, physical activity and 7-day SD of sleep duration (continuously) + AHI and average sleep duration
Model 3 adjusts for age, sex, education, work schedule, BMI, smoking status, physical activity and 7-day SD of sleep onset timing (categorically) + AHI and average sleep duration
Model 4 adjusts for age, sex, education, work schedule, BMI, smoking status, physical activity and 7-day SD of sleep onset timing (continuously) + AHI and average sleep duration

Supplemental Table 7. Prevalence ratios of hypertension as defined at time of study (n= 1393)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Hypertension						
Race						
NHW (REF)						
BA	1.38** (1.20, 1.59)	1.32** (1.14, 1.53)	1.33** (1.14, 1.54)	1.32** (1.13, 1.53)	1.31** (1.13, 1.52)	1.32** (1.14, 1.53)

* P < 0.05; ** P < 0.01
BA = Black American; NHW = non-Hispanic White
SD = standard deviation; PR = prevalence ratio; CI = confidence interval
Values reported as PR (95% CI)
N=832 for hypertension defined at time of study
Model 1 adjusts for age, sex, education, work schedule, and study site.
Model 2 adjusts for Model 1 + body mass index, smoking status, and physical activity.
Model 3 adjusts for Model 2 + 7-day SD of sleep duration (categorically).
Model 4 adjusts for Model 2 + 7-day SD of sleep duration (continuously).
Model 5 adjusts for Model 2 + 7-day SD of sleep onset timing (categorically).
Model 6 adjusts for Model 2 + 7-day SD of sleep onset timing (continuously).

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