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Sleep as a Contributor to Socioeconomic Disparities in Hypertension: The Midlife in the United States (MIDUS) Study

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Abstract

Sleep as a Contributor to Socioeconomic Disparities in Hypertension: The Midlife in the United States (MIDUS) Study

By Olivia Barnum

Background: Hypertension is highly prevalent and a risk factor for cardiovascular disease (CVD). Hypertension is more common among low socioeconomic status (SES) individuals and those with poor sleep quality. Given evidence suggesting that SES is associated with poor sleep quality, sleep may link SES and hypertension. Using data from the Midlife in the United States (MIDUS) Study, we tested sleep quality as a partial mediator of socioeconomic disparities in hypertension.

Methods: Participants underwent 7-day actigraphy, a clinic visit for measures of blood pressure, and completed questionnaires. Sleep quality was measured as actigraphy-defined wakefulness after sleep onset (WASO) (higher WASO > 30 minutes) and sleep efficiency (SE) (low SE < 85%). SES was defined with the socioeconomic index score (SEI), which collectively assessed education, income, and occupational prestige (n=274) and educational attainment (n=426). SES indicators were dichotomized into low vs high at the sample's median. Averaged 2nd and 3rd systolic and diastolic blood pressure readings with values ≥ 130 and 80 mmHg, respectively, were classified as hypertensive. Poisson and linear regression models were fit to examine associations between SES and sleep, sleep and hypertension or blood pressure, and SES differences in hypertension or blood pressure with sleep. Covariates included age, gender, race, BMI, and perceived stress. Mediation was tested using Poisson regression models.

Results: The sample had an average age of 55.8 years (average SD: 12.1), and 59.2% female, 53.4% White, 41.4% Black, and 5.3% mixed race; 77.7% had poor WASO, 67.5% had low SE, and 60.8% were hypertensive. In unadjusted analyses SEI was associated with a higher prevalence of hypertension, but was attenuated with adjustment for race.In adjusted analyses, SES was not associated with sleep measures or hypertension after adjustment for covariates. Individuals with high vs. low SE had 19% lower prevalence of hypertension (PR=0.81, 95% CI: 0.66, 0.98), lower systolic blood pressure ($\wr$ = -4.69, 95% CI [(-8.63, -0.75]) and diastolic blood pressure ($\wr$ = -2.59, 95% CI [-5.00, 0.18]) after adjustment for covariates. There was no evidence of mediation.

Conclusions: Effective interventions for decreased hypertension should consider strategies that target SE. Future research should explore the intersectionality of race and SES which may impact sleep quality and hypertension. Improvement of sleep quality may optimize hypertension management.

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Introduction

Cardiovascular disease (CVD) accounts for approximately 1 out of 3 deaths in the United States (US), upholding its position as the leading cause of death from chronic disease since 1921.[1, 2] CVD generally refers to a set of four conditions: coronary heart disease (CHD) also known as coronary artery disease, cerebrovascular disease, peripheral artery disease, and aortic atherosclerosis.[3] CHD covers a broad spectrum of clinical entities, which include myocardial infarction (MI) and sudden cardiac death.[4] CHD and stroke, types of cerebrovascular disease, are the first and second most common causes of death worldwide.[5] An estimated 92.1 million US adults have at least one type of CVD, and by the year 2030, 43.9% of the US adult population is expected to have some form of CVD.[6] Identifying risk factors of CVD are crucial in devising potential interventions that may prevent its onset. Established CVD risk factors include hypertension (also known as high blood pressure), high cholesterol, diabetes, being overweight or obese, and cigarette smoking.[7] Approximately 32% of US adults have hypertension (using blood pressure of >140/90 mmHg), 29% have high cholesterol, 14% have diabetes, 72% are overweight or obese, and 14% smoke cigarettes.[7, 8] Thus, hypertension is the leading risk factor for CVD.[9]

Among risk factors for CVD, hypertension has the strongest evidence for causation of CVD.[9] Hypertension, otherwise known as systemic arterial hypertension, is characterized by persistently high blood pressure in the systemic arteries.[10] Blood pressure is the ratio of the systolic blood pressure (the pressure that the blood exerts on the arterial walls when the heart contracts) and the diastolic blood pressure (the pressure when the heart relaxes).[10] The etiology of hypertension is a complex interplay of environmental and pathophysiological factors.[10] Blood pressure levels rise steadily and continuously with age in both men and women, with age acting

as a proxy for the probability and duration of exposure to numerous environmental factors that increase blood pressure steadily over time, such as excessive sodium consumption, insufficient intake of dietary potassium, overweight and obesity, alcohol intake and physical inactivity.[10] Other factors, such as genetic predisposition or adverse intrauterine environment (such as gestational hypertension or pre-eclampsia), also have associations with high blood pressure in adulthood.[10] Blood pressure also fluctuates daily with a pattern that follows a circadian rhythm (a 24-hour internal clock in the brain that regulates cycles of alertness and sleepiness by responding to light changes in the surrounding environment), with a peak in the early morning hours and a trough during sleep.[11, 12] Nocturnal dipping of blood pressure is a normal circadian pattern, and its absence (also known as non-dipping) is more frequent in hypertensive adults and is associated with more severe end-organ damage and increased risk of CVD events.[11] Hypertension has been consistently associated with cardiovascular mortality and morbidity, as both high systolic and diastolic blood pressure have been shown to be predictive of adverse cardiovascular outcomes.[13] This positive association between hypertension and risk of CVD events starts as low as 115/75mmHg, and is continuous, consistent, and independent of other risk factors.[10, 14] When hypertension is defined as >130/80 mmHg (using the 2017 American College of Cardiology/American Heart Association [ACC/AHA] hypertension clinical practice guideline) or as taking anti-hypertensive medication, nearly half of adults in the US (47%) have hypertension.[8] Reducing the prevalence of hypertension can potentially reduce the prevalence of CVD.

There are strategies aimed at the reduction of the prevalence of hypertension. A 2015 Scientific Statement from the American Heart Association stated that recent breakthroughs in the control of hypertension is rooted in the work of community health workers who are involved in community patients' access to and continuity of care and adherence to treatment.[15] The authors

continue to state that further control and prevention of hypertension will stem from addressing social determinants.[15] Social determinants of health are non-medical factors that influence health outcomes and quality of life.[16] Race or ethnicity, sex/gender, geographic location, and socioeconomic status (SES) are examples of social determinants that contribute to an individual's ability to achieve good health.[17] Certain populations are disproportionately affected by a high prevalence of hypertension, which is likely attributable to social factors.[18]

Hypertension has striking health disparities that can be attributable to the social determinants of health. Long-standing research indicates that minoritized racial groups in the US have a greater burden of hypertension compared to non-racial minorities.[18] For example, African American adults have among the highest rates of hypertension in the world, and Mexican Americans tend to have higher blood pressure than White Americans.[18] Recent research also indicates that hypertension has been shown to contribute more to women's CVD risk than men's CVD risk, and is especially more prevalent in women than in men after the age of 65.[19] This higher contribution of hypertension to CVD risk among women is especially concerning, as across various CVDs, such as coronary heart disease, stroke, heart failure and aortic diseases, women have a higher mortality and worse prognosis after acute cardiovascular events.[20] Geographic location is an additional characteristic that influences hypertension.[21, 22] There is a higher prevalence of hypertension among rural populations compared to urban populations.[22] These factors have been shown to be patterned by SES.

SES is generally defined as the position of an individual or of a household within a society based on a measured combination of occupation, education, income, wealth, or residential neighborhood.[23] SES is widely considered the most influential determinant of health, having the most robust determinants of variations in health outcomes in nearly every society

throughout the world.[23,24] Measures of SES, such as income, education, and occupation, may dictate differential access and control of material and social resources in a society.[25] Racial differences in SES are an important contributor to racial disparities in health.[24] Significantly, high rates of hypertension persist among higher SES African Americans, suggesting that high SES is not always a protective factor against hypertension incidence.[26] A cross-sectional, secondary data analysis of the Cooper Center Longitudinal Study found that African Americans consistently have higher self-reported hypertension compared to White Americans, no matter their SES.[26] A separate study, supporting the effect of race after SES is accounted for, found that, when compared to White peers, Black physicians had a two-fold higher incidence of hypertension.[24] Mixed findings suggest that there may be gender differences in SES inequalities at some ages and not at others, that vary across time and place and across different health measures.[27] However, socioeconomic disparities in hypertension prevalence have been found to be higher in women compared to men.[28] A study with 59,805 participants aged 25-69 years, found education to be the largest gender-specific disparity for women, in which those at the top of the educational hierarchy were more than three times less likely to have hypertension compared to those at the bottom.[28] Spatially, a higher rate of poverty in rural populations exacerbates geographic disparities in cardiovascular health.[22] In addition to the socio-demographic groups described here, research demonstrates that the prevalence and accumulation of psychological and social risk factors associated with hypertension are highly concentrated in individuals of low SES.[29] Consequently, low SES is a likely contributor to hypertension.[29]

Different indices of SES, such as income, educational, and occupational position, are associated with hypertension.[29] One study, using data from the 2011 Behavioral Risk Factor Surveillance System (BRFSS), examined the association of state-level SES indicators and

hypertension within the US.[30] The state median household income was used as a proxy for state SES.[30] Approximately 3 million adults who resided in states with a below median household income ($43,225 or less) had 16% higher odds of hypertension compared with adults from states with an above median household income of $58,814 or more (AOR = 1.16, 95% CI [1.08–1.25]).[30] Similarly, previous research indicates that educational status is independently inversely associated with blood pressure and risk of hypertension.[31] While there is a clear association between SES indicators and hypertension, little is known about the pathways contributing to socioeconomic gradients in hypertension. Thus, it is important to identify and examine potential pathways contributing to socioeconomic disparities in hypertension.

It is hypothesized that sleep is a contributor to hypertension.[32,33] Sleep is defined as a recurring, reversible neuro-behavioral state of suspended unresponsiveness and perceptual disengagement from the surrounding environment.[34] The brain state recurrently alternates between rapid-eye-movement (REM) and non-rapid-eye-movement (NREM) sleep periods, constituting the ultradian NREM-REM or sleep cycle.[35] The sleep cycle is characterized as 5 phases: wake, N1, N2, N3, and REM.[36] The NREM stage refers to phases N1, N2, and N3. Phase N1 is a transition from wakefulness to sleep, N2 represents deeper sleep as heart rate and body temperature drops, and N3 is the deepest stage of sleep.[36] REM sleep are periods of active sleep marked by rapid eye movements and low amplitude electroencephalogram (EEG).[35,37] REM sleep is further characterized, and differentiated from wake and NREM, by neurophysiological and behavioral features such as muscle twitches, autonomic and respiratory activation, and an elevated arousal threshold.[35] Sleep health is defined as a multifaceted biobehavioral process consisting of various dimensions such as sleep duration, continuity, architecture, timing, rhythmicity, regularity, and satisfaction.[38] Sleep can be measured both objectively and

subjectively. For objective measures, sleep can be measured through polysomnography (PSG), which is considered the gold standard measurement of sleep, and actigraphy.[39] PSG assesses various physiologic parameters, such as respiratory effect, sleep stages, electrocardiography, airflow, body position, and limb movements during sleep.[40] PSG is typically performed in a clinic or laboratory setting but can also be measured in-home[41]. Actigraphy is a non-invasive method that can be used over longer periods of time and in the natural sleep environment of an individual.[41] Actigraphic devices, recording the occurrence and degree of limb movement, can be worn on the wrist, ankle, or waist, though, for sleep applications, are typically worn on the wrist or ankle.[39] Actigraphy uses a single channel to collect data on movements during sleep, which is then used to infer time spent asleep and awake.[42] Subjective ways to measure sleep include the use of sleep diaries, or sleep logs, in which individuals record detailed descriptions of their sleep, including bedtime, duration until sleep onset, number of awakenings, duration of awakenings, and nap times.[40] Sleep questionnaires also quantitatively summarize the subjective perception of sleep and are commonly administered as a preliminary evaluation of sleep in primary care, and research.[43]

Sleep is responsible for many regulatory and maintenance functions in human physiology, though the specific mechanisms of these functions and how this physiology relates to relevant clinical outcomes is not entirely understood.[44] Proposed mechanisms with clinical impact include inflammatory, autonomic, and metabolic pathways.[38] The Sleep Research Society (SRS) and American Academy of Sleep Medicine (AASM) recommend that adults should regularly obtain between 7-9 hours of sleep for optimal health and functioning.[38,45] Short sleep duration is considered by AASM as less than 7 hours, while long sleep duration is considered to be more than 9 hours of sleep.[38] Poor sleep is defined by suboptimal sleep duration, <7 hours,

and/or the presence of sleep disorders.[38,46] More than 1/3rd of US adults report sleeping <7 hours in a 24-hour period, which has been associated with an increased risk for obesity, diabetes, high blood pressure, coronary heart disease, stroke, frequent mental distress, and all-cause mortality.[47] Additionally, approximately 30% to 40% of adults in the US report difficulties getting to sleep or staying asleep in a given year, which has been linked to poor outcomes for many diseases, including cardiovascular and cerebrovascular disease, cancer, hypertension, and diabetes.[48] Based on National Health Interview Survey data, the unadjusted prevalence of insomnia or trouble sleeping has increased by 8% from 17.5% (37.5 million adults) in 2002 to 19.2% (46.2 million adults) in 2012.[48] The economic impact of poor sleep is conservatively estimated at $107 billion.[46] There is an ongoing need for effective interventions concerning sleep health, as good sleep is essential for good health.[34] More specifically, improving sleep health may reduce the burden of hypertension.

Growing epidemiologic evidence supports that both short sleep duration and long sleep duration are associated with hypertension prevalence.[49] Data from the National Health Interview Survey, a large national representative sample (n= 71,455) found that short sleep duration (defined here as <6 hours per night) was associated with a 9.2% higher prevalence of hypertension and long sleep duration (defined here as ≥ 10 hours per night) had a 9.3% higher prevalence of hypertension compared to those who slept 8 hours per night.[49] One of the biological explanations for the association of short sleep duration and hypertension, is that short sleep duration increases sympathetic nervous system activity, which is a common pathophysiology for hypertension. In addition to increased sympathetic nervous activity, short sleep duration increases heart rate, vasoconstriction, and salt retention, which are all factors associated with hypertension caused by cardiac overdrive and volume overload.[50] While long

sleep duration could be indicative of underlying sleep disorders or poor sleep quality, biological explanations supporting the association of long sleep duration and hypertension remain purely speculative.[51] Short and long sleep duration are significant risk factors for developing hypertension.

Generally neglected in literature, in addition to sleep duration, poor sleep quality has also been associated with a higher risk of hypertension.[52] A meta-analysis found that poor sleep quality is significantly associated with a greater likelihood of hypertension in 8 studies (odds ratio [OR] = 1.48, 95% confidence interval [CI] [1.13–1.95] [I^2 = 87%]).[52] Additionally, in examining 5 studies, patients with hypertension tended to have significantly worse sleep quality scores, using the Pittsburgh Sleep Quality Index (PSQI), a self-rated questionnaire measuring sleep quality (mean difference = 1.51, 95% CI [1.00–2.02] [I^2 = 64%]).[52] As sleep continues to emerge as a risk factor for hypertension, more research is required on its role in social inequities in hypertension prevalence.

Similar to the social patterning of hypertension, short sleep duration and poor sleep quality are more common in individuals of low SES.[53, 54] In examining social patterns of sleep duration, a study using data from the National Health Interview Survey-Sample Adult Files (NHIS-SAF) 2004–2007, which surveyed adults aged 18 years or older in the US (n=110,441), found that individuals with lower education, income, or few income sources had increased odds of both short and long self-reported sleep duration.[55] In examining social patterns of sleep quality, a study used data from the 2006 Behavioral Risk Factor Surveillance System (BRFSS), US representative sample of adults (n=188,765), found that low income and low educational attainment were significantly associated with more sleep complaints compared to those with high income and high educational attainment among both men and women.[54] Socioeconomic

disparities in sleep duration and sleep quality may be attributable to a greater burden of external stressors among those of low SES.

Research suggests that individuals of lower SES experience greater exposure to occupational and psychosocial stressors, as well as greater environmental exposures to tobacco, allergens, and pollutants, which may adversely impact sleep, and lead to poor health outcomes.[33, 56,57] Individuals who experience socioeconomic adversity across the lifespan report sleep-related problems more frequently than those without disadvantaged experiences.[53] Adult occupational gradient for sleep duration may be due to lower grade occupations having to combine several jobs, work in shifts, and/or live in noisy environments, which contribute to greater levels of stress.[53] Sleep quality is also impacted by occupation, with one study reporting that sleep quality suffers with low workplace social capital, a term used to incorporate interpersonal trust, norms of reciprocity, and mutual aid that facilitate coordination and cooperation for mutual benefit.[58] Additionally, individuals of low SES are more vulnerable to psychological distress. Studies have demonstrated an association between low individual SES or low community SES with higher scores on measures of traumatic and life events, chronic stress, perceived stress, and daily hassles.[59] Furthermore, research has shown that income is inversely correlated with exposure to suboptimal environmental conditions, which includes pollutants, toxins, noise, and crowding across various environmental settings, such as the household, work, schools, and neighborhoods.[60] Sleep may explain socioeconomic disparities in hypertension, however evidence is lacking.

It is plausible that sleep may mediate the association between SES and hypertension. Few research studies have examined this association with hypertension as an outcome, but supporting evidence exists for other cardiovascular outcomes. Using cross-sectional data from eight

European cohorts, a counterfactual mediation model showed a strong inverse association between adult male occupational position and CHD risk (OR = 1.45, 95% CI [1.13; 1.86]) of which short sleep duration (<6 hours per night) mediated 13.4% of the association.[53] An additional study in the US found that sleep quality mediated 20% of the neighborhood socioeconomic disparity in self-rated health.[61] These prior studies are limited by relying on self-reported measures of sleep quality, as opposed to objective measures.[53, 61-63] As questionnaire instruments vary among sleep studies, self-reporting differences in sleep measures (e.g. sleep duration and sleep quality) can limit comparisons made between studies that use subjective measures.[64] Research shows a low-moderate correlation between investigated actigraphy sleep parameters and subjective sleep quality, suggesting that these two methods of measurement capture different dimensions of sleep.[65] Additionally, research is further limited to a narrow focus on one dimension of sleep health, sleep duration, as opposed to research examining sleep quality.[51,49,50] Additional research examining sleep quality through objective measures will provide a more comprehensive understanding of the potential impacts of sleep on health outcomes.

Using data from The Midlife in the United States (MIDUS) Study, we examined sleep quality as a potential contributor to socioeconomic disparities in hypertension. We hypothesized that sleep quality is a partial mediator of the pathway between SES and hypertension. This study may contribute to the literature by offering a more accurate measurement of sleep and expansion of the sleep dimensions typically assessed. The results of this study may provide a better understanding of the association between SES and hypertension, which may allow for tailored public health interventions that aim to improve sleep quality among the socioeconomically disadvantaged in efforts to reduce the burden of hypertension.

Methods

Participants

Midlife in the United States (MIDUS) is a national study on health and aging that began in 1995 and includes over 7,000 sampled US residents aged 25-74 years.[66] Approximately 75% of surviving respondents participated in MIDUS II (n=5895), the second wave of the MIDUS study, 9-10 years later in 2004/05.[66, 67] MIDUS II consisted of five data collection projects: a national survey (project 1), a daily diary study (project 2), cognitive function (project 3), bioindicators (project 4), and neuroscience (project 5).[67] The objective of these continuing projects was to investigate long-term changes across sociodemographic, psychosocial, behavioral, and health characteristics assessed at baseline, as well as add biological assessments to extend the scientific scope of the study.[66] Data for the present study were derived from MIDUS II, project 4 (n=1255), which included comprehensive bioindicator and health assessments data.[66] Respondents participated in overnight, clinic-based biomarker data collection at sites located in Los Angeles, CA, Madison, WI, and Washington, DC.[67] As part of project 4, participants could elect to take part in a sleep substudy, in which actigraphy data was collected to assess sleep.[67] To increase representation of African American adults in project 4, an additional Milwaukee site was added and oversampled African Americans (n=592).[67] Only respondents from the Madison, WI site, which additionally drew from the Milwaukee site, were requested to participate in the week-long sleep substudy (n=1255).[67] The current study examines data from these selected participants.

Measures.

Hypertension.

For MIDUS II, blood pressure was measured by nurses during a large-scale physical examination after participants sat quietly and rested for five minutes.[68] While seated, 3 consecutive blood pressure readings, with a 30-second interval between each assessment, were recorded using a Finometer monitor, which uses a finger cuff for continuous blood pressure measurement by photoplethysmography.[68,69] In keeping arterial size consistent, the air pressure in the cuff adjusts in response to any increase in the size of finger arteries, which are reflective of blood pressure changes.[69] An electric gauge indirectly measures the pressure wave form, and the mean pressure is then calculated by integrating it over a single heart-beat.[69]

Following the 2017 ACC/AHA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults, normal blood pressure was coded for those with systolic blood pressure lower than 120 mmHg and diastolic blood pressure lower than 80 mmHg.[70] Elevated blood pressure (or pre-hypertensive) was defined as readings ranging from 120-129 mmHg systolic and <80 mmHg diastolic.[70] Hypertension was defined as the average of the second and third readings for systolic or diastolic blood pressure with values ≥ 130 or ≥80 mmHg, respectively, encompassing both stage 1 hypertension and stage 2 hypertension.[70] Blood pressure cut points were coded as a single biomarker using these systolic and diastolic values. Blood pressures coded as normal and elevated were collectively considered non-hypertensive, finalizing blood pressure as a dichotomous variable (hypertensive vs non-hypertensive) in line with the 2017 ACC/AHA guideline.[70] Systolic and diastolic blood pressure readings were also independently analyzed as continuous measures.

SES

SES was obtained from questionnaire data and two measures of SES were analyzed. A composite measure of SES was defined by averaging standardized scores (z-scores) for education, household income, and occupational prestige, which were all assessed by coders employed at the University of Wisconsin Survey Center (UWSC) for MIDUS II.[71] The resulting score was termed the Duncan socioeconomic index (SEI) score, the most frequently used assessment of occupational prestige in social research.[72] The Duncan SEI originated from a dataset created by Hauser and Warren (1995), which extracted occupational education and earnings from the 1990 US Census. The Hauser and Warrens' dataset additionally contains 501 values, which correspond to 501 occupational categories, aggregated into nine major occupation groups and 12 major industry groups.[72] This final SEI score, collectively assessing education, income, and occupational prestige, was transformed to range from 0 to 100.[73] A higher SEI score translates to being more socioeconomically advantaged. For categorical, SEI was dichotomized into low (n=137) versus high SEI (n=137) at the median of the final sample (median: 36.23), which is consistent with prior publications.[74]

Educational attainment was measured on a 12-point scale (e.g., 1=no school/some grade school; 5=high school degree; 9=four-year college degree/B.A., 12=advanced graduate/professional degree).[71] For categorical, consistent with prior publications, low educational attainment was considered high school completion or less (n=199), while high educational attainment was considered some college or more (n=227).[75]

Sleep quality

Sleep quality was measured via wrist actigraphy.[66] Participants wore a Mini-Mitter Actiwatch-64 activity monitor (Respironics, Inc.) for 7 days and nights.[66] Actiwatches were set

to collect activity data in 30-second epochs, which were then used for analysis with Actiware software.[76] Rest intervals were determined via diary responses. If diary responses were unavailable, event markers and adjacent data were used to define rest intervals. Actiware software calculated summary statistics using a wake activity threshold of 40.[76]

Sleep quality was operationalized as wakefulness after sleep onset (WASO) and sleep efficiency. WASO refers to the periods of wakefulness that occur after sleep onset, excluding wakefulness occurring before sleep onset.[77] Sleep efficiency refers to the percentage of total time in bed spent asleep.[77]

Studies aimed at developing standardized definitional criteria for insomnia found that a WASO of > 30 minutes represented the optimal severity cutoff for discriminating insomnia groups from normal-sleeper groups.[78] Therefore, lower WASO was defined as ≤ 30 minutes and higher WASO was defined as > 30 minutes. In agreement with a study that empirically derived cutoff values for sleep health dimensions after assessing extensive sleep health literature, high sleep efficiency was defined as ≥ 85% and low sleep efficiency was defined as having an actigraphy score < 85%.[79]

Covariates

Age, gender, race, body mass index (BMI), and perceived stress have been shown to be associated with hypertension or SES in previous studies and were therefore considered covariates for all analyses.[59, 80-82] Age and gender were self-reported using self-administered questionnaires.[67] Race groups included White, Black and/or African American, Native American or Alaska Native Aleutian Islander/Eskimo and Native Hawaiian or Pacific Islander. Due to small sample sizes, Native American or Alaska Native Aleutian Islander/Eskimo and Native

Hawaiian or Pacific Islander were grouped in a category referred to as “mixed”. Height and weight were measured using a standardized procedure at a clinic visit, in which BMI was calculated by dividing body weight in kilograms by height in meters squared.[67, 83] Perceived stress was measured through the Perceived Stress Scale (PSS), the most widely used psychological instrument for measuring the perception of stress.[84] Participants were asked about the frequency of their feelings and thoughts within the past month regarding 10 prompts, on a scale of 1-5 (1 = never, 5 = very often).[85] The prompts assessed the degree to which situations in their life were appraised as stressful.[84] PSS scores were then obtained by reversing responses to the four positively stated items and summing scores across all scale items.[84]

Analysis

This analysis included two samples, one with complete data for a socioeconomic index score (SEI) (n=274, see Figure 1), and another with complete data for educational attainment, (n=426, see Figure 2). All participants for both samples had complete valid blood pressure readings. Participants were excluded from the final sample if they did not have valid actigraphy data for at least five days of sleep (n=106 for both samples).

SAS 9.4 was used for all data analyses. Descriptive statistics were reported on covariates, blood pressure, and actigraphy-measured sleep measures by SES categories for the SEI and educational attainment sample (Tables 1a, 1b). All variables were separately tested and found to follow a normal distribution. Spearman’s correlation measured the strength and direction of monotonic association between sleep measures, WASO and sleep efficiency (Table 2). Poisson with robust variance and linear regression models were performed to examine associations between SES and sleep measures (Table 3), and sleep and hypertension or blood pressure (Table

4). Analyses were adjusted for age, gender, race, BMI, and perceived stress. Prevalence ratios (PRs) were estimated rather than odds ratios because hypertension was not rare in the sample.[86]

For the overall findings, Poisson regression models with robust error variance and linear regression models were performed for each path in the mediation model to examine SES differences in hypertension or blood pressure and the role of sleep quality (Tables 5a, 5b, 6a, 6b). Model 1 is the unadjusted effect of SEI or educational attainment on hypertension. Model 2 further adjusted for age, gender, race, BMI, and perceived stress. Model 3 further added WASO to model 2 and model 4 further added sleep efficiency to Model 2. See Figure 3 for a schematic diagram of the analyses.

Using the SAS PROC CAUSALMED procedure, mediation was tested in path models in which each sleep measure was separately modeled as a mediator predicted by SES and as a predictor of hypertension or blood pressure. Mediation models were adjusted for age, gender, race, BMI, and perceived stress.

Equation 1. M = intercept(1) + aX + residual(1)
Equation 2. Y = intercept(2) + c'X + bM + hXM + residual(2)

Based on formulas of VanderWeele, the SAS PROC CAUSALMED procedure uses the estimation of two regression equations as input to estimate casual effects for the single mediator model.[87-89] The procedure first estimates the parameters of equation 1, the effect of *X* on *M* (*a* coefficient) and equation 2, the effect of *M* on *Y* adjusted for X (*b* coefficient) and the effect of the *XM* interaction on (*h* coefficient).[87] The *h* coefficient magnitude signifies how much the effect of *X* on *Y* (c') differs across mediator levels (*M*) and how much the effect of *M* on *Y* (*b*) differs across treatment levels (*X*). It is assumed that both residuals, X and residual(1), X and residual(2), and M and residual(2) are uncorrelated, that the variables are measured without error, that temporal order follows X→M→Y, and that there are no common causes of M and Y.

The procedure uses estimated parameters from equations 1 and 2 to compute the causal mediation effects using regression-based estimators of the causal indirect, direct, and total effects for models.[87] Mediation was conducted with SEI (high vs low) signifying X, sleep quality indicators (high vs low) signifying M, and hypertension signifying Y. Mediation was also conducted with education (high vs low) signifying X, sleep quality indicators (high vs low) signifying M, and blood pressure (hypertensive vs non-hypertensive) signifying Y.

Results

Demographics were similar across SES samples. Study characteristics by SEI categories are shown in Table 1a. The SEI study population (n = 274) had a mean age of 52.0 years (SD: 11.8) and was 58.8% female. The population identified as 55.1% White, 40.2% Black and/or African American, and a mix of racial minorities (4.7%). Overall, 60.6% of the total population had hypertension. Of those who had hypertension, 44.6% had low SEI (55.4% had high SEI). Participants with a low SEI had an average WASO of 47.3 min (SD: 23.2), with 75.9% of the population having higher WASO (> 30 minutes). Participants of lower SEI had an average sleep efficiency of 79.3% (SD: 10.6), with 67.1% of the population having low sleep efficiency (< 85%).

Study characteristics by educational attainment category are shown in Table 1b. The mean age was 53.5 years (SD: 12.4) and 59.6% were female. The population identified as 51.6% White, 42.5% Black and/or African American, and a mix of racial minorities (5.9%). Overall, 61.0% of the total population had hypertension. Of those who had hypertension, 45.4% had low educational attainment (54.6% had high educational attainment). Participants with low educational attainment had an average WASO of 48.7 min (SD: 23.0), with 77.9% of the

population having higher WASO. Participants with low educational attainment had an average sleep efficiency of 79.4% (SD: 10.9), with 66.3% of the population having low sleep efficiency.

Figures 4 and 5 show the prevalence of blood pressure groups by SEI (Fig. 4) and educational attainment (Fig. 5). Of those with low SEI, 54.0% were hypertensive. Of those with high SEI, 67.2% were hypertensive. Of those with low educational attainment, 59.3% were hypertensive. Of those with high educational attainment, 62.6% were hypertensive.

Table 2 demonstrates the Spearman correlation coefficients for sleep measures. The Spearman correlation between WASO and sleep efficiency signified a strong negative association in both samples (SEI, r = -0.67; educational attainment, r=-0.68). As sleep efficiency increased, the WASO decreased.

There were no associations between SEI or education attainment and WASO or sleep efficiency (Table 3). However, sleep measures were associated with hypertension and blood pressure (Table 4). Among the SEI sample, lower WASO was associated with lower systolic blood pressure (β = -5.19, 95% CI [-10.25, 0.14]). The association was attenuated and no longer significant after adjustment for age, gender, race, BMI, and perceived stress. In the educational attainment sample, individuals with high sleep efficiency had a 19% lower prevalence of hypertension (PR = 0.81, 95% CI [0.66–0.98]) after adjustment for age, gender, race, BMI, and perceived stress. Also, high sleep efficiency was associated with lower systolic blood pressure (β = -4.69, 95% CI [(-8.63, -0.75]) and lower diastolic blood pressure (β = -2.59, 95% CI [-5.00, -0.18]) after adjustment for age, gender, race, BMI, and perceived stress.

High SEI was associated with hypertension (PR = 1.24, 95% CI [1.02, 1.51] Table 5a). This association was attenuated and no longer significant after adjustment for age, gender, race, BMI, and perceived stress. Further analyses determined race as the covariate that attenuated the

association between SEI and hypertension (Supplementary Table 1). Further adding WASO or sleep efficiency did not change the association between SEI and hypertension. Educational attainment was not associated with hypertension (Table 5b). The null association persisted with adjustment for sleep quality. There were no associations between SES and systolic or diastolic blood pressure readings (Tables 6a, 6b).

There is no evidence that WASO or sleep efficiency mediated the relationships between socioeconomic indicators and hypertension (Table 7).

Discussion

In a cross-sectional analysis of the MIDUS longitudinal study among White, Black and/or African American, Native American or Alaska Native Aleutian Islander/Eskimo, and Native Hawaiian or Pacific Islanders, we examined associations of SES, sleep quality (e.g., WASO and sleep efficiency), and hypertension. We found that 1) sleep quality was associated with hypertension; and 2) The association between SEI and hypertension was attenuated with adjustment for race. SES and sleep quality were not associated, thus, there was no evidence of mediation. There was a high prevalence of hypertension (averaged 60.8% across both samples) and low sleep efficiency (averaged 67.5% across both samples). Our results suggest sleep efficiency may be an appropriate target for effective reduction of hypertension prevalence.

The present study found no direct association between SES and sleep quality, which is not consistent with prior studies conducted within the US.[33, 54] Among a sample of predominately White (67.1%), Hispanic/Latino (17.4%), and Black/African American (8.9%) participants, Grandner et al, found higher rates of self-reported sleep complaints were associated with lower SES.[54] Our results may differ due to a difference in racial composition. Our study had a large sample of Black/African American adults, which may have masked effects due to potential

opposite SES-sleep gradients by race. Racial/ethnic disparities in reports of sleepiness and sleep complaints have been found to be inconsistent, further complicated by the added role of SES.[56] Jackson et al found that shorter sleep duration increases with professional responsibility for Blacks, while the opposite effect occurs for Whites.[33] An additional study sampling Black adults also found that the association between stress and short sleep duration was more pronounced among those of higher educational attainment.[90] This sleep-SES gradient for Blacks and Whites may be due to John Henryism, a stressor in which marginalized populations are strongly motivated to combat negative stereotypes associated with their social identity group.[56] As the sleep-SES association may vary by race, race should therefore be further explored as an effect modifier. Additionally, while Native American or Alaska Native Aleutian Islander/Eskimo, and Native Hawaiian or Pacific Islanders were included in the present study, low sample size for these population groups suggest that more studies are needed to evaluate these populations in regard to sleep and the added dimensionality of SES. As these listed populations, and the Black population, are heterogenous groups, within-group studies should also be further explored.

Sleep disorders, which can affect sleep quality, such as obstructive sleep apnea (OSA) and insomnia, have been linked to increased hypertension risk.[91] Cross-sectional studies generally find that self-reported poor sleep quality is associated with higher blood pressure or higher prevalence of hypertension.[92] In contributing to objective measures of sleep quality, the present study found lower sleep efficiency to be associated with hypertension. Additionally, high sleep efficiency was associated with a lower systolic blood pressure and diastolic blood pressure. Our finding is consistent with a study by Ramos et al, who found that a 10% reduction in sleep efficiency, measured via actigraphy, was significantly associated with a 7.5% (95% CI [-12.9 – -2.2]) greater hypertension prevalence in a sample of US Latinos.[93] These findings are supported

by research demonstrating a significant relationship between sleep efficiency and hypertension.[33,93] Jackson et al reported evidence that symptoms of insomnia are associated with the activation of the hypothalmic-pituitary-adrenal axis and the sympathetic nervous system which may increase hypertension risk.[33] Individuals with poor sleep quality have more interruptions and wakefulness during the sleep period that can cause higher blood pressure at night due to a lack of nocturnal dip in blood pressure, resulting in higher blood pressure during the day.[94] Based on these findings, sleep is a viable target for hypertension reduction.

The present study found an unadjusted association between SEI and hypertension. This association was attenuated and no longer significant after the addition of race as a confounder. Growing research demonstrates that SES and race/ethnicity can function jointly and independently to affect health.[95] Different approaches in conceptualizing SES result in different conclusions about the role of SES in accounting for racial/ethnic health outcomes.[96] Further research should therefore additionally consider race as a potential effect modifier as the magnitude of the effect of SES on hypertension may differ by race. The present study additionally found no direct association between educational attainment and hypertension, which is not consistent with prior studies conducted within the US.[28-31] Multiple studies have indicated that low SES is a risk factor for hypertension, with a recent meta-analysis reporting multiple indicators of SES (income, occupation, and education) were associated with hypertension.[29] Discrepancies between our findings and prior studies may be due to the operationalization of our SES indicators and hypertension. The operationalization of socioeconomic indicators may have skewed the differential impact of SES. The current study analyzed binary measures of socioeconomic indicators rather than using more common scale or alternative ordinal categorizations.[97] Through dichotomizing SES into high vs low, differences across continuous

levels of SES may have been lost. As a prior study found that Black physicians had a higher incidence of hypertension compared to White counterparts, examining the intersectionality of race and SES on health outcomes is necessary.[24]

The present study found no evidence that sleep mediated the association between SES and hypertension. Few studies have considered sleep quality as a mediator between SES and hypertension. Piccolo et al tested restless sleep as a mediator for SES disparities in multiple outcomes, including hypertension, in which no mediation was observed for SES and hypertension.[98] Specifically, the authors found that while social disparities in sleep and in the incidence of hypertension were highly significant and mirrored one another, sleep did not have a significant role in mediating SES differences in hypertension.[98] Further contributing to the intersectionality of race, SES, and sleep, the Piccolo et al study noted significant differences in the prevalence of sleep-related problems by both race and SES; Black and Hispanic adults had a higher prevalence of restless sleep than White participants and lower- and middle-class adults were more likely to report restless sleep compared to higher-class adults. The prior study showed an association between SES and sleep quality, which was not observed in the current study. Given these findings, while sleep is an acceptable target as an effective intervention for reduction of hypertension prevalence, further research should consider the potential influence of race-SES disparities on hypertension.

Our results are important in supporting prevention of hypertension through targeting sleep. Poor sleep quality in adults typically manifest through primary sleep disorders, such as sleep apnea or insomnia, or are secondary to comorbidities.[99] Early identification of sleep disorders is important for the prevention of poor health consequences, which can be actualized through routine screening of sleep disorders by primary care physicians[100]. In conjunction, there is a

continuing need for further research to examine the treatment of sleep disorders as a form of hypertension management.[101] Apart from disordered sleep, sleep quality has been suggested as a general therapeutic target for the prevention of hypertension, with potential interventions including disseminating sleep hygiene education, participating in sleep scheduling, and undergoing cognitive behavioral therapy.[102] The high prevalence of poor sleep quality and hypertension in the present study indicate that there is an immediate need to identify strategies to reduce this societal burden.

The present study has many strengths. There has been limited exploration of sleep quality as a potential mediator between SES and hypertension. This contributes to literature by examining pathways through which social factors are related to hypertension. Also, there was a large sample of Black individuals included in the sample increasing diversity of research and yielding a sufficient sample size for possible race comparisons. An additional strength was the use of actigraphy to objectively measure sleep quality. This contributes to a lack of literature on objective measures of sleep quality as a risk factor for long-term health outcomes.[103] The majority of studies relating sleep with disease risk rely on self-reporting measures due to ease of measuring and reduced participant burden.[104] However, questionnaire design, response formats, and social desirability may affect responses to questionnaires, as well as rounding or heuristic strategies when asked to give a single estimate of sleep traits.[103] Further impacting discrepancies are reported positive associations between perceived stress and subjective sleep quality, but not between perceived stress and objective sleep quality[103]. These extreme deviations between objective and subjective sleep measures are commonly found in those who have sleep disorders, such as insomnia[105]. As self-reported sleep does not always corroborate with objective sleep indicators, relying on self-reporting measures may mask different dimensions of sleep-related

health outcomes. An additional strength is that this study included a sample that operationalized SES through a composite measure, which is beneficial for capturing a comprehensive understanding of SES on health.

There are several limitations to this study. This is a cross-sectional study design. There are predicational limitations in analyzing cross-sectional studies because the exposure and outcome are simultaneously assessed.[106] Thus, there is insufficient evidence of a temporal relationship between exposure and outcome.[106] This study was also limited by the small population sample. A larger diverse sample could more effectively study the intersection of race and SES on sleep and hypertension. Additionally, participants were sampled from Madison, WI and Milwaukee, WI, which are both urban cities within Wisconsin and not nationally representative. This limits the generalizability of the present study.

For our educational attainment sample, educational attainment was used as a sole indicator of SES, which has additional limitations. For example, if participants obtained education outside of the US, different educational regimes may have different implications for educational levels.[107] Also, measuring levels of attainment does not indicate the quality of the educational experience, which is likely to be important in conceptualizing the role of education as a socioeconomic indicator for health outcomes.[107]

The present study found that low sleep efficiency was associated with a higher prevalence of hypertension. Consequently, improvements in sleep efficiency may be important in the potential reduction of hypertension in US adults. Effective interventions for the reduction of hypertension should consider strategies that improve sleep efficiency. Additional research should examine contributors to poor sleep quality, such as undiagnosed or untreated sleep disorders as well as

risk factors associated with poor sleep efficiency, such as stress and chronic pain management. The improvement of sleep quality may optimize hypertension management.

Tables and Figures

Table 1a. Sample characteristics, by categories of SEI, Midlife in the United States Study (MIDUS II) 2004-2006

Characteristics	**Overall Population (n=274)**	**Low SEI (n= 137)**	**High SEI (n= 137)**
	% / Mean (SD)	**% / Mean (SD)**	**% / Mean (SD)**
Demographic covariates			
Age (years)	52.0 (11.8)	51.4 (11.7)	52.6 (12.0)
Gender (% women)	58.8	62.8	54.7
Race %			
White	55.1	43.1	67.2
Black and/or African American	40.2	54.0	26.3
Mixed†	4.7	2.9	6.6
Other covariates			
Body Mass Index (BMI)*	29.01 (6.5)	29.4 (7.1)	28.6 (5.81)
Perceived Stress Scale (PSS)**	23.01 (6.67)	23.18 (6.65)	22.85 (6.71)
Socioeconomic indicators			
Socioeconomic index	37.7 (13.5)	26.5 (5.68)	49.0 (8.9)
Educational attainment (continuous)	6.6 (2.3)	5.5 (1.7)	7.7 (2.3)
Household income ($, continuous)***	42,422.69 (54,431.18)	26,254.13 (45,634.02)	59,890.50 (57,864.16)
Cardiovascular indicators			
Systolic Blood Pressure, mm Hg	131.6 (17.8)	129.4 (18.3)	133.9 (17.0)
Diastolic Blood Pressure, mm Hg	77.3 (10.7)	76.8 (11.0)	77.7 (10.3)
Normal %	24.4	29.2	19.7
Elevated %	15.0	16.8	13.1
Hypertension %	60.6	54.0	67.2
Actigraphy Sleep Measures			
WASO (min)	49.9 (25.5)	47.3 (23.2)	52.4 (27.5)
WASO (% high)	77.7	75.9	79.6

Characteristics	Overall Population (n=274)	Low SEI (n= 137)	High SEI (n= 137)
	% / Mean (SD)	% / Mean (SD)	% / Mean (SD)
Sleep Efficiency (%)	79.0 (10.8)	79.3 (10.6)	78.7 (10.8)
Sleep Efficiency (% low)	67.9	67.1	68.6

†Mixed includes Native American or Alaska Native Aleutian Islander/Eskimo, Native Hawaiian or Pacific Islander, and Other

*Sample size 274 except: BMI = 228 for overall population, BMI = 118 for low SEI, and BMI = 110 for high SEI; **PSS = 273 for overall population, PSS = 136 for low SEI; ***Household income = 233 for overall population, Household income = 121 for low SEI, and Household income = 112 for high SEI

Table 1b. Sample characteristics, by categories of educational attainment, Midlife in the United States Study (MIDUS II) 2004-2006

Characteristics	Overall Population (n=426)	Low Education (n= 199)	High Education (n= 227)
	% / Mean (SD)	% / Mean (SD)	% / Mean (SD)
Demographic covariates			
Age (years)	53.5 (12.4)	53.1 (13.4)	53.8 (11.5)
Gender (% women)	59.6	58.8	60.4
Race %			
White	51.6	42.2	59.9
Black and/or African American	42.5	53.3	33.0
Mixed†	5.9	4.5	7.1
Other covariates			
Body Mass Index (BMI)*	29.71 (9.63)	30.11 (11.76)	29.4 (7.1)
Perceived Stress Scale (PSS)**	22.65 (6.51)	22.38 (6.28)	22.89 (6.71)
Socioeconomic indicators			
Socioeconomic index (continuous)***	37.7 (13.5)	26.5 (5.68)	49.0 (8.9)
Educational attainment (continuous)	6.24 (2.4)	4.31 (1.07)	7.94 (1.85)

Characteristics	**Overall Population (n=426)**	**Low Education (n= 199)**	**High Education (n= 227)**
	% / Mean (SD)	**% / Mean (SD)**	**% / Mean (SD)**
Household income ($, continuous)****	34,126.94 (49,039.08)	20,402.43 (37,002.77)	46,500.33 (55,008.16)
Cardiovascular indicators			
Systolic Blood Pressure, mm Hg	132.4 (18.0)	131.7 (16.9)	133.0 (18.9)
Diastolic Blood Pressure, mm Hg	77.3 (10.6)	77.8 (11.4)	76.8 (9.8)
Normal (%)	23.7	21.1	26.0
Elevated (%)	15.3	19.6	11.4
Hypertension (%)	61.0	59.3	62.6
Actigraphy Sleep Measures			
WASO (min)	49.0 (24.1)	48.7 (23.0)	49.4 (25.1)
WASO (% high)	77.7	77.9	77.5
Sleep Efficiency (%)	79.4 (10.5)	79.4 (10.9)	79.1 (10.2)
Sleep Efficiency (% low)	67.1	66.3	67.8

†Mixed includes Native American or Alaska Native Aleutian Islander/Eskimo, Native Hawaiian or Pacific Islander, Other, and Don't Know

*Sample size 426 except: BMI = 365 for overall population, BMI = 176 for low educational attainment, and BMI = 189 for high educational attainment; **PSS = 424 for overall population, PSS = 197 for low educational attainment; ***Socioeconomic index = 274 for overall population, Socioeconomic index = 109 for low educational attainment, Socioeconomic index = 165 for high educational attainment; ****Household income = 367 for overall population, Household income = 174 for low educational attainment, and Household income = 193 for high educational attainment

Table 2. Spearman correlation coefficients between WASO and Sleep Efficiency measured continuously in both samples, Midlife in the United States Study (MIDUS II) 2004-2006

	Sample: SEI		**Sample: Educational Attainment**	
	WASO	Sleep Efficiency	WASO	Sleep Efficiency
WASO	1.00	-0.67*	1.00	**-0.68***
Sleep Efficiency	**-0.67***	1.00	**-0.68***	1.00

*P-values <0.001

Table 3. Associations of SEI or Educational Attainment and WASO or Sleep Efficiency, Midlife in the United States Study (MIDUS II) 2004-2006

	WASO (continuous)				**WASO (high = ref)**				**Sleep Efficiency (continuous)**				**Sleep Efficiency (low = ref)**			
	Model 1		**Model 2**		**Model 1**		**Model 2**		**Model 1**		**Model 2**		**Model 1**		**Model 2**	
	Beta	**95% CI**	**Beta**	**95% CI**	**PR**	**95% CI**	**PR**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**PR**	**95% CI**	**PR**	**95% CI**
SEI (low = ref)	5.06	(-0.99, 11.12)	2.52	(-4.06, 9.10)	0.85	(0.54, 1.32)	1.07	(0.66, 1.75)	-0.61	(-3.18, 2.00)	-0.40	(-3.13, 2.32)	0.96	(0.68, 1.35)	0.98	(0.67, 1.42)
Educational attainment (low = ref)	0.66	(-3.95, 5.28)	0.86	(-4.09, 5.81)	1.02	(0.71, 1.45)	0.95	(0.64, 1.41)	-0.10	(-2.11, 1.92)	0.52	(-1.58, 2.61)	0.95	(0.73, 1.25)	0.96	(0.72, 1.28)

Note: Model 1 is unadjusted, Model 2 is adjusted for covariates (age, gender, race, BMI, and perceived stress)
Abbreviations: PR, prevalence ratio; CI, confidence interval; BMI, body mass index; PSS, Perceived Stress Scale; ref, reference group

Table 4. Associations of WASO or Sleep Efficiency and Hypertension or Systolic and Diastolic Blood Pressures, Midlife in the United States Study (MIDUS II) 2004-2006

	SEI												**Educational attainment**											
	Hypertension				**Systolic BP**				**Diastolic BP**				**Hypertension**				**Systolic BP**				**Diastolic BP**			
	Model 1		**Model 2**		**Model 1**		**Model 2**		**Model 1**		**Model 2**		**Model 1**		**Model 2**		**Model 1**		**Model 2**		**Model 1**		**Model 2**	
	PR	**95% CI**	**PR**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95 % CI**	**Beta**	**95 % CI**	**PR**	**95% CI**	**PR**	**95% CI**	**Beta**	**95 % CI**	**Beta**	**95 % CI**	**Beta**	**95 % CI**	**Beta**	**95 % CI**
WASO (high = ref)	0.80	(0.61, 1.05)	0.83	(0.62, 1.11)	**-5.19***	(-10.25, -0.14)	-4.87	(-10.45, 0.71)	-2.68	(-5.73, 0.36)	-2.61	(-6.06, 0.84)	0.87	(0.71, 1.06)	0.87	(0.70, 1.08)	-3.34	(-7.45, 0.76)	-3.31	(-7.65, 1.04)	-2.30	(-4.71, 0.12)	-2.07	(-4.72, 0.58)
Sleep Efficiency (low = ref)	0.94	(0.76, 1.16)	0.90	(0.70, 1.16)	-0.95	(-5.48, 3.59)	-2.58	(-7.77, 2.62)	-2.52	(-5.24, 0.19)	-2.66	(-5.85, 0.53)	0.88	(0.74, 1.04)	**0.81***	(0.66, 0.98)	-2.77	(-6.41, 0.87)	**-4.69***	(-8.63, -0.75)	-1.98	(-4.12, 0.16)	**-2.59***	(-5.00, -0.18)

*P-values <0.05

Note: Model 1 is unadjusted, Model 2 is adjusted for covariates (age, gender, race, BMI, and perceived stress)

Abbreviations: PR, prevalence ratio; CI, confidence interval; BMI, body mass index; PSS, Perceived Stress Scale; ref, reference group

Table 5a. Prevalence ratios of Hypertension with SEI and WASO or Sleep Efficiency, Midlife in the United States Study (MIDUS II) 2004-2006

	Model 1		**Model 2**		**Model 3**		**Model 4**	
	PR	**95% CI**	**PR**	**95% CI**	**PR**	**95% CI**	**PR**	**95% CI**
SEI (low = ref)	**1.24***	(1.02, 1.51)	1.22	(0.98, 1.53)	1.23	(0.98, 1.54)	1.22	(0.98, 1.53)
Age	--	--	1.01	(1.00, 1.02)	1.01	(1.00, 1.02)	1.01	(1.00, 1.02)
Gender (female = ref)	--	--	1.17	(0.95, 1.44)	1.16	(0.94, 1.43)	1.16	(0.94, 1.43)
Race (mixed = ref)	--	--	1.01	(0.79, 1.31)	1.01	(0.78, 1.30)	1.01	(0.79, 1.31)
BMI	--	--	1.00	(0.98, 1.02)	1.00	(0.98, 1.02)	1.00	(0.98, 1.02)
PSS	--	--	1.00	(0.99, 1.02)	1.00	(0.99, 1.02)	1.00	(0.99, 1.02)
WASO (high = ref)	--	--	--	--	0.82	(0.62, 1.10)	--	--
Sleep Efficiency (low = ref)	--	--	--	--	--	--	0.90	(0.71, 1.15)

*P-values <.05

Note: Model 1 is unadjusted, Model 2 is adjusted for covariates (age, gender, race, BMI, and perceived stress), Model 3 is adjusted for covariates and WASO, Model 4 is adjusted for covariates and Sleep Efficiency

Abbreviations: PR, prevalence ratio; CI, confidence interval; BMI, body mass index; PSS, Perceived Stress Scale; ref, reference group

Table 5b. Prevalence ratios of Hypertension with Educational Attainment and WASO or Sleep Efficiency, Midlife in the United States Study (MIDUS II) 2004-2006

	Model 1		**Model 2**		**Model 3**		**Model 4**	
	PR	**95% CI**	**PR**	**95% CI**	**PR**	**95% CI**	**PR**	**95% CI**
Educational attainment (low = ref)	1.06	(0.91, 1.23)	1.04	(0.88, 1.23)	1.04	(0.88, 1.23)	1.04	(0.88, 1.23)
Age	--	--	1.01	(1.00, 1.02)	1.01	(1.00, 1.02)	**1.01****	(1.00, 1.02)
Gender (female = ref)	--	--	1.06	(0.89, 1.25)	1.05	(0.89, 1.24)	1.04	(0.88, 1.22)
Race (mixed = ref)	--	--	1.02	(0.85, 1.22)	1.02	(0.85, 1.22)	1.03	(0.86, 1.23)
BMI	--	--	1.00	(0.99, 1.01)	1.00	(0.99, 1.01)	1.00	(0.99, 1.01)
PSS	--	--	1.00	(0.99, 1.02)	1.00	(0.99, 1.02)	1.00	(0.99, 1.01)
WASO (high = ref)	--	--	--	--	0.87	(0.70, 1.08)	--	--
Sleep Efficiency (low = ref)	--	--	--	--	--	--	**0.81***	**(0.66, 0.98)**

*P-values <.05, ** <.01

Note: Model 1 is unadjusted, Model 2 is adjusted for covariates (age, gender, race, BMI, and perceived stress), Model 3 is adjusted for covariates and WASO, Model 4 is adjusted for covariates and Sleep Efficiency

Abbreviations: PR, prevalence ratio; CI, confidence interval; BMI, body mass index; PSS, Perceived Stress Scale; ref, reference group

Table 6a. Beta coefficients of Systolic and Diastolic blood pressure with SEI and WASO or Sleep Efficiency, Midlife in the United States Study (MIDUS II) 2004-2006

	Systolic								**Diastolic**							
	Model 1		**Model 2**		**Model 3**		**Model 4**		**Model 1**		**Model 2**		**Model 3**		**Model 4**	
	Beta	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**
SEI (low = ref)	4.44	(0.23, 8.64)	4.72	(-0.13, 9.57)	4.80	(-0.028, 9.63)	4.70	(-0.15, 9.56)	0.88	(-1.66, 3.43)	0.60	(-2.41, 3.62)	0.64	(-2.37, 3.65)	0.58	(-2.42, 3.59)
Age	--	--	**0.29***	(0.086, 0.50)	**0.28***	(0.071, 0.49)	**0.30***	(0.090, 0.51)	--	--	-0.10	(-0.23, 0.027)	-0.11	(-0.24, 0.019)	-0.10	(-0.23, 0.031)
Gender (female = ref)	--	--	1.31	(-3.47, 6.10)	1.05	(-3.72, 5.83)	0.99	(-3.84, 5.83)	--	--	2.40	(-0.58, 5.37)	2.26	(-0.72, 5.23)	2.06	(-0.93, 5.06)
Race (mixed = ref)	--	--	0.48	(-4.83, 5.79)	0.32	(-4.96 5.61)	0.56	(-4.76, 5.87)	--	--	2.12	(-1.18, 5.42)	2.04	(-1.26, 5.33)	2.20	(-1.09, 5.49)
BMI	--	--	0.21	(-0.16, 0.59)	0.19	(-0.18, 0.57)	0.21	(-0.17, 0.59)	--	--	0.10	(-0.13, 0.34)	0.093	(-0.14, 0.33)	0.10	(-0.13, 0.34)
PSS	--	--	0.094	(-0.25, 0.44)	-0.07	(-0.28, 0.42)	0.062	(-0.29, 0.42)	--	--	0.14	(-0.073, 0.36)	0.13	(-0.086, 0.35)	0.11	(-0.11, 0.33)
WASO (high = ref)	--	--	--	--	-4.97	(-10.51, 0.58)	--	--	--	--	--	--	-2.63	(-6.08, 0.83)	--	--

	Systolic								**Diastolic**							
	Model 1		**Model 2**		**Model 3**		**Model 4**		**Model 1**		**Model 2**		**Model 3**		**Model 4**	
	Beta	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**
Sleep Efficiency (low = ref)	--	--	--	--	--	--	-2.53	(-7.70, 2.63)	--	--	--	--	--	--	-2.66	(-5.85, 0.54)

P-values <.001

Note: Model 1 is unadjusted, Model 2 is adjusted for covariates (age, gender, race, BMI, and perceived stress), Model 3 is adjusted for covariates and WASO, Model 4 is adjusted for covariates and Sleep Efficiency

Abbreviations: CI, confidence interval; BMI, body mass index; PSS, Perceived Stress Scale; ref, reference group

Table 6b. Beta coefficients of Systolic and Diastolic blood pressure with Educational Attainment and WASO or Sleep Efficiency, Midlife in the United States Study (MIDUS II) 2004-2006

	Systolic								**Diastolic**							
	Model 1		**Model 2**		**Model 3**		**Model 4**		**Model 1**		**Model 2**		**Model 3**		**Model 4**	
	Beta	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**
Educational attainment (low = ref)	1.29	(-2.14, 4.73)	1.10	(-2.66, 4.87)	1.06	(-2.69, 4.82)	1.04	(-2.70, 4.78)	-0.95	(-2.97, 1.07)	-0.93	(-3.22, 1.37)	-0.95	(-3.24, 1.34)	-0.96	(-3.24, 1.33)
Age	--	--	**0.30****	(0.15, 0.45)	**0.30****	(0.15, 0.45)	**0.31****	(0.16, 0.46)	--	--	-0.063	(-0.15, 0.028)	-0.063	(-0.15, 0.028)	-0.059	(-0.15, 0.032)
Gender (female = ref)	--	--	0.15	(-3.63, 3.94)	0.087	(-3.69, 3.87)	-0.23	(-4.01, 3.55)	--	--	1.18	(-1.13, 3.49)	1.14	(-1.17, 3.45)	0.97	(-1.34, 3.28)
Race (mixed = ref)	--	--	0.81	(-3.14, 4.75)	0.87	(-3.06, 4.81)	1.06	(-2.86, 4.98)	--	--	0.33	(-2.08, 2.73)	0.37	(-2.77, 3.45)	0.47	(-1.93, 2.87)
BMI	--	--	-0.014	(-0.18, 0.21)	0.010	(-0.19, 0.21)	-0.018	(-0.18, 0.21)	--	--	-0.036	(-0.16, 0.083)	-0.038	(-0.16, 0.081)	-0.034	(-0.15, 0.090)
PSS	--	--	-0.039	(-0.24, 0.32)	0.032	(-0.25, 0.31)	-0.010	(-0.29, 0.27)	--	--	0.17	(-0.005, 0.34)	0.16	(-0.0091, 0.33)	0.14	(-0.033, 0.31)

	Systolic								**Diastolic**							
	Model 1		**Model 2**		**Model 3**		**Model 4**		**Model 1**		**Model 2**		**Model 3**		**Model 4**	
	Beta	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**	**Beta**	**95% CI**
WASO (high = ref)	--	--	--	--	-3.29	(-7.63, 1.06)	--	--	--	--	--	--	-2.08	(-4.74, 0.57)	--	--
Sleep Efficiency (low = ref)	--	--	--	--	--	--	**-4.68***	(-8.62, -0.73)	--	--	--	--	--	--	**-2.60***	(-5.01, -0.19)

*P-values <.05, <.0001**

Note: Model 1 is unadjusted, Model 2 is adjusted for covariates (age, gender, race, BMI, and perceived stress), Model 3 is adjusted for covariates and WASO, Model 4 is adjusted for covariates and Sleep Efficiency

Abbreviations: CI, confidence interval; BMI, body mass index; PSS, Perceived Stress Scale; ref, reference group

Table 7. Estimates of total, direct, and indirect effects of SEI or Educational Attainment on Hypertension and the percent (%) mediated by WASO or Sleep Efficiency, Midlife in the United States Study (MIDUS II) 2004-2006

Socioeconomic Indicator	**Hypertension**							
	Mediator: WASO (high = ref)				**Mediator: Sleep Efficiency (low = ref)**			
	Total effect PR (95% CI)[a]	**Direct effect PR (95% CI)[a]**	**Indirect effect PR (95% CI)[a]**	**% Percent Mediated**	**Total effect PR (95% CI)[a]**	**Direct effect PR (95% CI)[a]**	**Indirect effect PR (95% CI)[a]**	**% Percent Mediated**
High SEI (low = ref)	1.22 (0.96, 1.54)*	1.23 (0.97, 1.55)*	1.23 (0.97, 1.55)*	-1.57%	1.22 (0.99, 1.55)*	1.22 (0.98, 1.54)*	1.00 (0.99, 1.02)*	0.33%
High educational attainment (low = ref)	1.04 (0.88, 1.24)*	1.04 (0.88, 1.24)*	1.00 (0.99, 1.02)*	3.64%	1.05 (0.89, 1.24)*	1.04 (0.88, 1.23)*	1.00 (0.99, 1.04)	6.38%

*P-values <.0001

Abbreviations: PR, prevalence ratio; CI, confidence interval; ref, reference group

[a]Confidence intervals are 95% bias-corrected bootstrap confidence intervals.

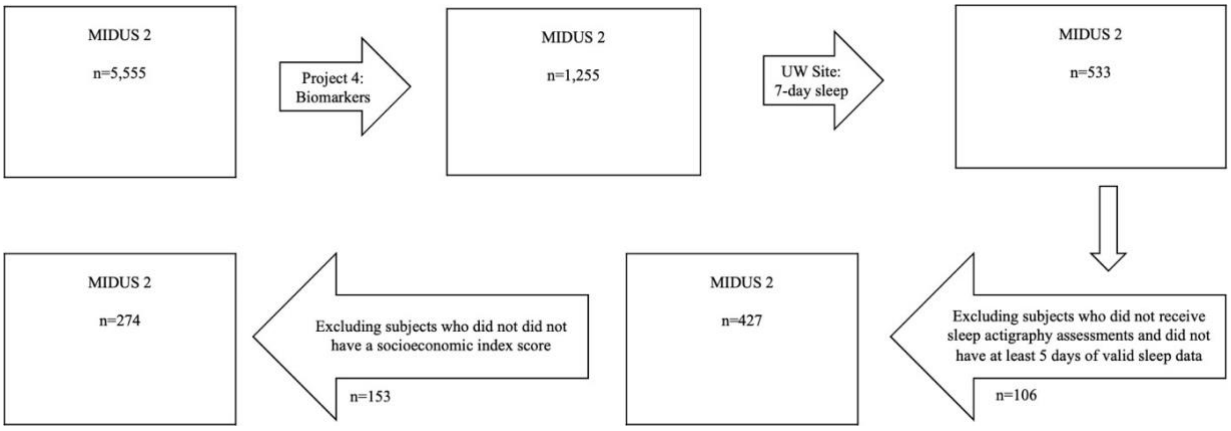

Figure 1. Socioeconomic index (SEI) sampling strategy

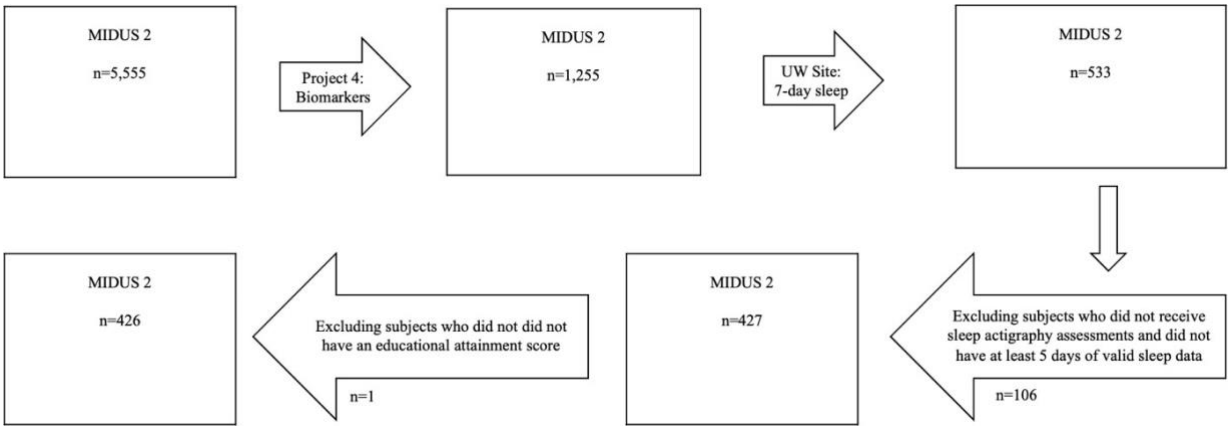

Figure 2. Educational attainment sampling strategy

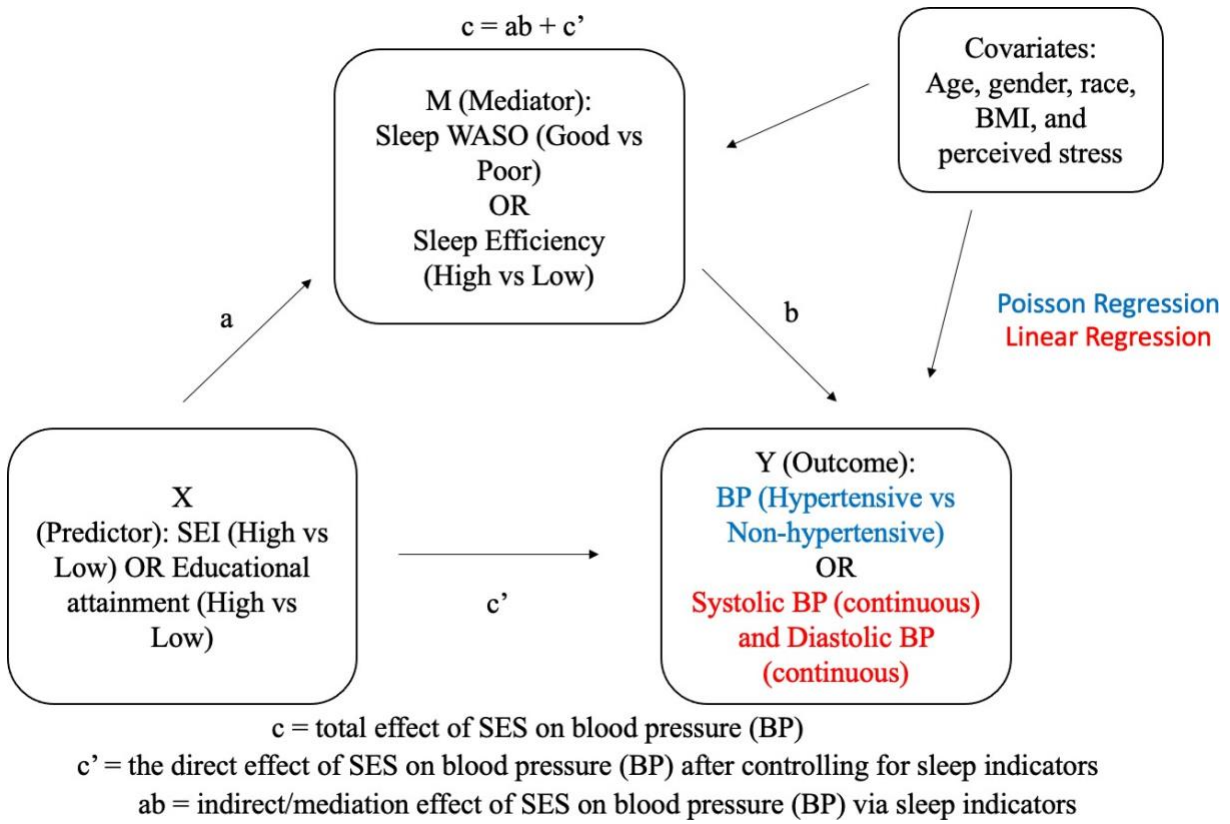

Figure 3. Schematic diagram of the mediation model

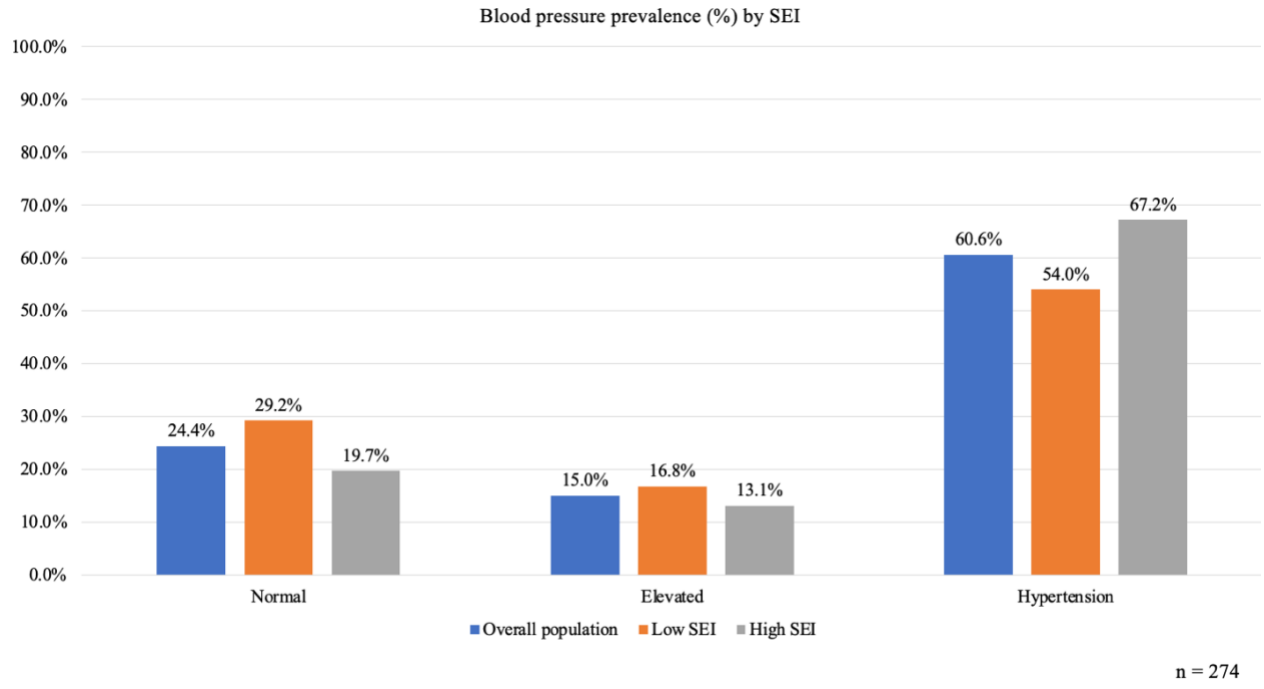

Figure 4. Percentage (%) of blood pressure categories by SEI, Midlife in the United States Study (MIDUS II) 2004-2006

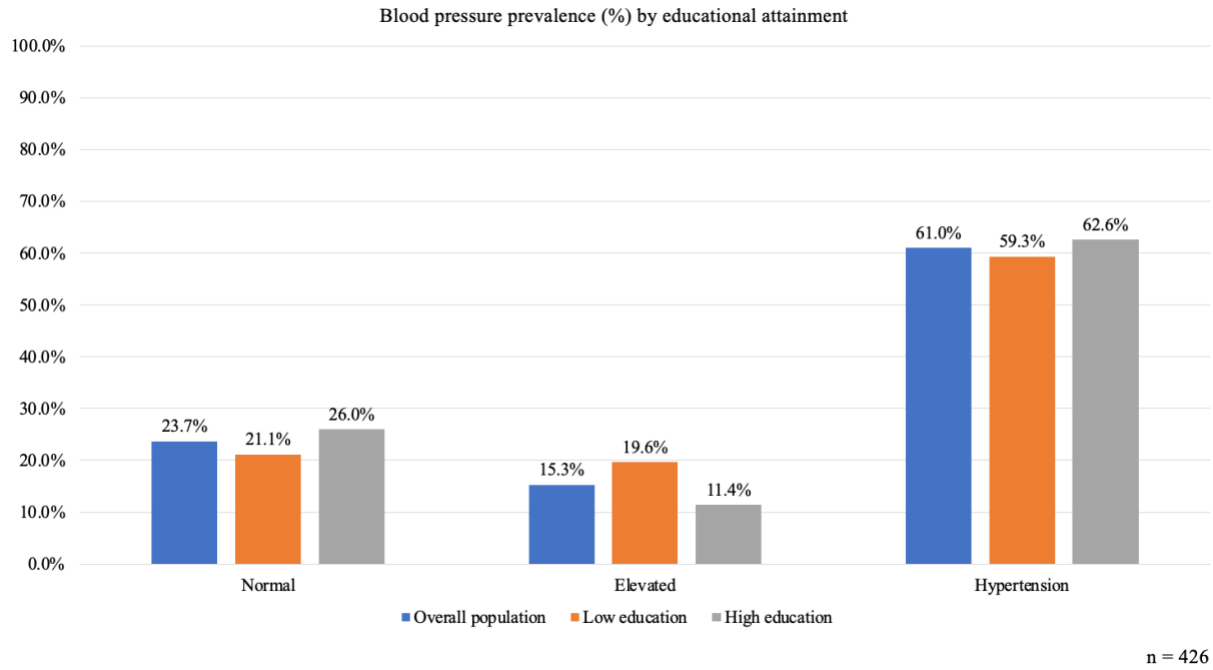

Figure 5. Percentage (%) of blood pressure categories by educational attainment, Midlife in the United States Study (MIDUS II) 2004-2006

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Supplementary Table

Table 1. Prevalence ratios of Hypertension with SEI for separate covariates, Midlife in the United States Study (MIDUS II) 2004-2006

	Model 1		**Model 2**		**Model 3**		**Model 4**		**Model 5**		**Model 6**	
	PR	**95% CI**	**PR**	**95% CI**	**PR**	**95% CI**	**PR**	**95% CI**	**PR**	**95% CI**	**PR**	**95% CI**
SEI (low = ref)	**1.24***	(1.02, 1.51)	1.23	(0.98, 1.54)	1.19	(0.98, 1.45)	**1.24***	(1.00, 1.54)	1.23	(0.98, 1.55)	1.22	(0.97, 1.53)
Age	--	--	1.01	(1.00, 1.02)	1.01	(1.00, 1.02)	1.01	(1.00, 1.02)	1.01	(1.00, 1.02)	--	--
Gender (female = ref)	--	--	1.18	(0.96, 1.46)	1.22	(1.01, 1.47)	1.16	(0.94, 1.43)	--	--	1.17	(0.94, 1.44)
Race (mixed = ref)	--	--	1.01	(0.79, 1.30)	1.00	(0.80, 1.25)	--	--	1.02	(0.80, 1.32)	1.07	(0.84, 1.37)
BMI	--	--	1.00	(0.98, 1.02)	--	--	1.00	(0.98, 1.02)	1.00	(0.98, 1.02)	1.00	(0.98, 1.02)
PSS	--	--	--	--	1.00	(0.99, 1.02)	1.00	(0.99, 1.02)	1.01	(0.99, 1.02)	1.00	(0.99, 1.02)

*P-values <.05

Note: Model 1 is unadjusted, Model 2 is adjusted for PSS, Model 3 is adjusted for BMI, Model 4 is adjusted for race, Model 5 is adjusted for gender, Model 6 is adjusted for age

Abbreviations: PR, prevalence ratio; CI, confidence interval; BMI, body mass index; PSS, Perceived Stress Scale; ref, reference group