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Original Article

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Sleep as a contributor to socioeconomic disparities in hypertension: The Midlife in the United States (MIDUS II) Study

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Abstract

Study Objectives: Hypertension is highly prevalent and is a major risk factor for cardiovascular disease. There is a higher burden of hypertension among individuals of lower socioeconomic status (SES), yet the role of sleep in understanding socioeconomic disparities in hypertension is unclear. We investigated whether sleep quality is a partial mediator of the association between SES and hypertension.

Methods: We used data from the Midlife in the United States II Study, 2004–2009 (n = 426). Analyses were conducted in 2023. Participants underwent 7-day actigraphy and clinical assessments. Sleep quality measures included actigraphy-defined wakefulness after sleep onset (WASO) and sleep efficiency. Hypertension was measured via three consecutive blood pressure readings, and SES was measured via educational attainment. Models were fit adjusting for age, gender, race, body mass index, and perceived stress.

Results: Participants had a mean age of 53.5 years (SD = 12.4) and 41.0% were African American. The prevalences of poor WASO (>30 minutes), low sleep efficiency (<85%), and hypertension were 77.7%, 67.1%, and 61.0%, respectively. Education was not associated with hypertension. However, individuals with low vs. high sleep efficiency had 24% higher prevalence of hypertension (aPR = 1.24, 95% CI: 1.02 to 1.51), higher systolic blood pressure (aβ = 4.61, 95% CI: 0.69 to 8.53), and higher diastolic blood pressure (aβ = 2.50, 95% CI: 0.10 to 4.89). Education was not significantly associated with sleep after adjustment. There was no evidence of sleep mediating the SES-hypertension relation.

Conclusions: Effective interventions to lower hypertension prevalence should consider targeting sleep quality. Future research should explore the intersectionality of SES and race in hypertension.

Key words: sleep; hypertension; socioeconomic status; health disparities; education; social determinants of health

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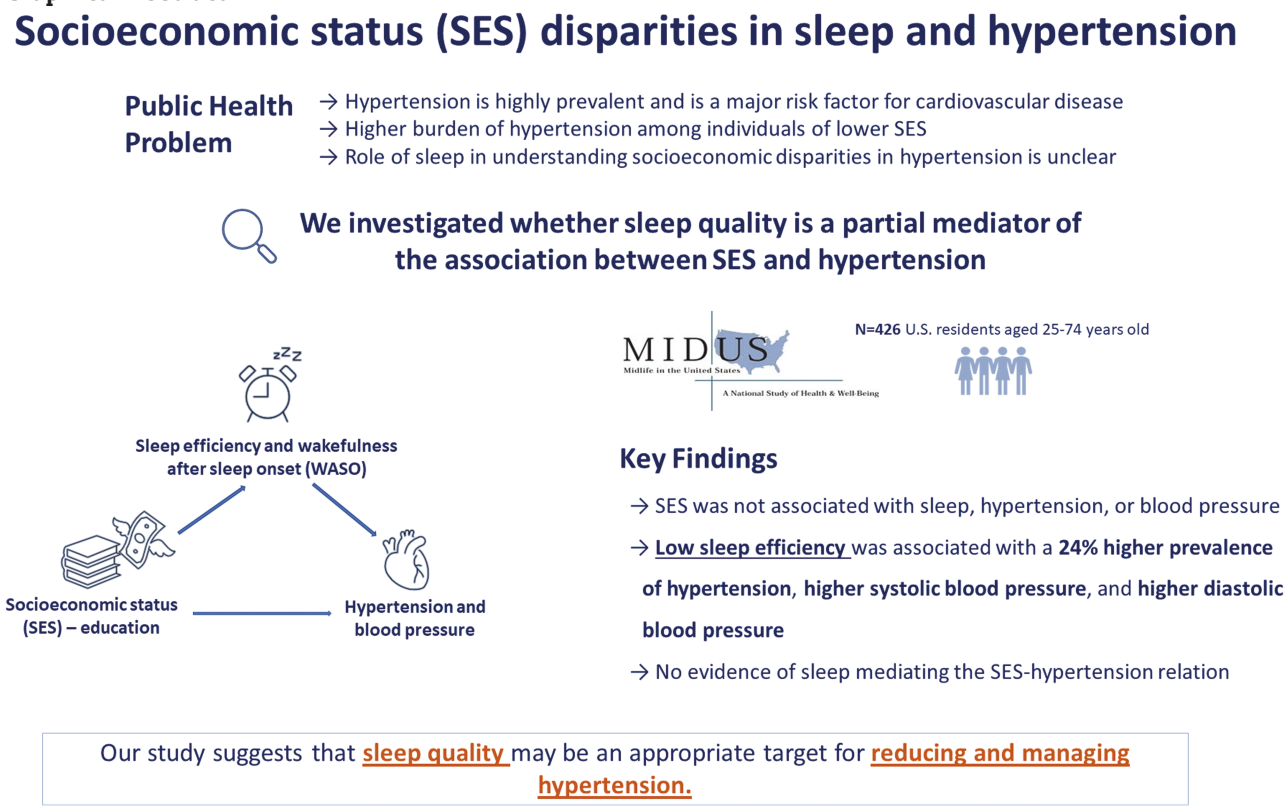
Graphical Abstract



Statement of Significance

This study uses data from the Midlife in the United States study to investigate the role of sleep quality in understanding socioeconomic disparities in hypertension. The analysis included 426 adults and found that low sleep efficiency measured via 7-day actigraphy was associated with higher systolic blood pressure, higher diastolic blood pressure, and a higher prevalence of hypertension. This study contributes to a limited understanding of the pathways contributing to socioeconomic gradients in hypertension and suggests that sleep quality may be an appropriate target for reducing and managing hypertension. This article is timely, given the addition of sleep health as a key component of the Life's Essential 8 measures for improving and maintaining cardiovascular health.

Hypertension is the single largest contributor to cardiovascular disease and is a major public health concern [1–3]. Nearly half of all adults in the United States have hypertension, and among these individuals, 76% do not have their hypertension under control [4, 5]. High systolic and diastolic blood pressure are predictive of adverse cardiovascular outcomes, including myocardial infarction, ischemic stroke, and hemorrhagic stroke [6, 7]. Therefore, there is a clear need to control and prevent our hypertension burden, which scientific bodies such as the American Heart Association agree will stem from addressing social determinants of health [8].

Health disparities in hypertension in the United States exist by race, ethnicity, and socioeconomic status (SES)—social determinants that contribute to an individual's ability to achieve optimal health [9, 10]. SES is generally defined as the position of an individual or household within a society based on a measured combination of occupation, education, income, wealth, and residential neighborhood and dictates differential access to and control of material and social resources in a society [11, 12]. Numerous studies and meta-analyses have indicated that different components of SES are associated with hypertension [13–15]. Specifically, research has shown that years of educational schooling, a measure of SES, is independently, inversely associated with blood pressure [16–18]. Further examination of degree attainment has shown that individuals with a graduate degree have significantly lower systolic blood pressure than high school degree holders [19]. Similarly, household income, another measure of SES, has been associated with hypertension [14, 20]. While research has shown that these various components of SES are associated with hypertension, more research is needed to understand the mechanisms through which SES may interact with other social determinants of health to shape hypertension disparities.

In addition to SES, longstanding research indicates that race and ethnicity are important social determinants of disparities in hypertension. Historically minoritized racial and ethnic groups in the United States have a greater burden of hypertension compared to their non-Hispanic White counterparts [10]. For example, African American adults have the highest rates of hypertension in the United States [10]. However, little is known about the pathways contributing to socioeconomic gradients in hypertension,

i.e. racial and ethnic differences among varying levels of SES [21]. Studies have shown that high rates of hypertension persist among African Americans with high SES, suggesting that high SES is not always a protective factor against hypertension incidence [22, 23]. Thus, it is important to identify and examine potential pathways contributing to socioeconomic and racial and ethnic disparities in hypertension.

One potential pathway to understand socioeconomic disparities in hypertension is sleep. Sleep health is associated with hypertension and is one component of the Life's Essential 8 (LE8) key measures for improving and maintaining cardiovascular health [24–26]. Sleep health is defined as a multifaceted, biobehavioral process characterized by multiple dimensions including satisfaction, appropriate timing, adequate duration, efficiency, and sustained alertness during waking hours, and is an integral component of physical and mental well-being [27]. Poor sleep quality has also been significantly associated with a greater likelihood of hypertension and coronary heart disease [28, 29]. As sleep continues to be recognized as an important risk factor for hypertension, more research is needed 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 with low income and educational attainment [30, 31]. Studies exploring sleep as a contributor to socioeconomic disparities in hypertension are limited. However, it is plausible that sleep may mediate the SES-hypertension relationship, as other studies have demonstrated a strong inverse association between SES (measured as adult male occupational position) and coronary heart disease risk, of which short sleep duration mediated part of the association [30]. Therefore, to fill this gap in research, we used data from the Midlife in the United States (MIDUS II) study to investigate whether sleep quality was a partial mediator of the association between SES and hypertension. In Supplementary Analyses, we explored whether these associations differed by race and ethnicity.

Materials and Methods

Study population

The MIDUS study is a national, longitudinal study on health and aging that began in 1995 and includes over 7000 U.S. residents aged 25–74 years old [32]. Approximately 75% of surviving respondents participated in MIDUS II ($n = 5895$), the second wave of the MIDUS study, 9–10 years later in 2004 [33]. The goal of the study 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. Data for the present study were derived from MIDUS II Project 4 ($n = 1255$), which included comprehensive bioindicator and health assessment data [33]. Respondents participated in overnight, clinic-based biomarker data collection at sites located in Los Angeles, CA; Madison, WI; and Washington, D.C. To increase the representation of African American adults in Project 4, an additional site was added in Milwaukee, WI, and African American participants were oversampled ($n = 592$). Our study population is restricted to participants at the University of Wisconsin-Madison study site ($n = 533$), where participants could elect to take part in a week-long sleep substudy in which actigraphy data were collected to characterize sleep patterns. MIDUS data collection is reviewed and approved by the Education and Social/Behavioral Sciences and the Health Sciences IRBs at the University of Wisconsin-Madison.

Socioeconomic status measures

Educational attainment was measured on a 12-point scale (e.g. 1 = no school/some grade school; 5 = high school degree; 9 = 4-year college degree/B.A., 12 = advanced graduate/professional degree) and was available for all participants. Consistent with prior publications, low educational attainment was considered high school completion or less, while high educational attainment was considered some college or more [34].

A subset of participants had information on Duncan's socioeconomic index score (SEI), which was used as our measure of socioeconomic status (SES) in sensitivity analyses. SEI is a composite measure of SES defined by averaging standardized Z-scores for education, household income, and occupational prestige, and transforming the final score to a range from 0 to 100 [35]. A higher SEI score indicates a higher socioeconomic advantage. SEI was dichotomized into low versus high SEI at the median, which is consistent with prior publications [36].

Hypertension and blood pressure measures

Resting blood pressure was measured during an initial physical exam by General Clinical Research Center nurses. Participants sat quietly and rested for 5 minutes. While seated, three consecutive blood pressure readings, with a 30-second interval between each assessment, were recorded [37]. 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 stages 1 and 2 hypertension [38]. Systolic and diastolic blood pressure readings were also independently analyzed as continuous measures.

Sleep quality measures

Sleep quality was measured via wrist actigraphy [37]. The sleep study protocol required that participants continuously wear the Mini Mitter Actiwatch-64 activity monitor (Respironics, Inc.) for seven consecutive days and nights, in addition to completing a daily sleep diary for the same time period [37]. Actiwatches were set to collect activity data in 30-second epochs, which were then used for analysis with Actiware software. 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.

Wakefulness after sleep onset (WASO) and sleep efficiency were analyzed separately as our sleep quality measures. WASO refers to the total amount of time spent awake after sleep onset, and sleep efficiency refers to the percentage of total time in bed spent asleep [39]. Although WASO and sleep efficiency are both measures of sleep continuity and are highly correlated, they were both included in our analyses (in separate models) as they are measured differently and have been shown to have different strengths of associations with the same health outcomes [39, 40]. 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 [41]. Thus, low WASO was defined as ≤30 minutes and high WASO was defined as >30 minutes. In accordance with empirically derived cutoff values for sleep health dimensions, high sleep efficiency was defined as having an actigraphy score ≥85% and low sleep efficiency was defined as score <85% [42]. WASO and sleep efficiency were additionally analyzed continuously in sensitivity analyses.

Covariates

Age, gender, race and ethnicity, body mass index (BMI), and perceived stress have been shown to be associated with hypertension or SES in previous studies and were therefore considered as covariates for all analyses [43–46]. Age and gender were self-reported using self-administered questionnaires [33]. Race and ethnicity groups included non-Hispanic Black and/or African American, non-Hispanic White, and Multiethnic (due to small sample sizes, a group of participants who responded with Native American or Alaska Native Aleutian Islander/Eskimo, Native Hawaiian or Pacific Islander, Hispanic, other, or don't know). 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 [33]. Perceived stress was measured via the Perceived Stress Scale (PSS), a widely used psychological instrument for measuring the perception of stress [47]. 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). The prompts assessed the degree to which situations in their life were appraised as stressful. PSS scores were then obtained by reversing responses to the four positively stated items and summing scores across all scale items.

Statistical analysis

We excluded participants from the final sample if they did not have valid actigraphy data for at least 5 days of sleep ($n = 106$, 20%). One participant was excluded for missing education data, and all participants had complete blood pressure readings. Our final analytic sample consisted of $n = 426$ participants. A subset of participants with data on SEI were analyzed in sensitivity analyses ($n = 274$).

Descriptive statistics (Chi-square tests and *t*-tests) were used to compare categorical and continuous variables, respectively, by low versus high education. Spearman's correlation coefficients were used to measure the strength and direction of monotonic associations between educational attainment and our sleep measures (WASO and sleep efficiency). Poisson models with robust standard errors were fit to estimate prevalence ratios (PRs) of the associations between educational attainment and sleep and hypertension. Prevalence ratios (PRs) were estimated rather than odds ratios because the outcome was not rare [48]. Linear regression models were fit to examine associations between educational attainment and systolic and diastolic blood pressure. All models were adjusted for age, gender, race and ethnicity, BMI, and perceived stress. We additionally fit Poisson and linear regression models to estimate the associations between sleep measures and hypertension, systolic blood pressure, and diastolic blood pressure. Our final multivariable Poisson regression examined the association between educational attainment and hypertension, adjusted for all previous demographic, health, and sleep covariates. All analyses were performed in SAS 9.4 (SAS Institute, Cary NC).

For the mediation analysis, we used the SAS PROC CAUSALMED procedure, which uses the estimation of two regression equations as inputs to estimate causal effects for the single mediator model [49–51]. The procedure uses estimated parameters from Equations 1 and 2 below to compute the causal mediation effects using regression-based estimators of the causal indirect, direct, and total effects [49].

$$\text{Equation 1. } M = \text{intercept}(1) + aX + \text{residual} \quad (1)$$

$$\text{Equation 2. } Y = \text{intercept}(2) + cX + bM + hXM + \text{residual} \quad (2)$$

Where X = educational attainment, M = sleep, and Y = hypertension or blood pressure. The procedure first estimates the parameters of Equation 1, the effect of education on sleep, and Equation 2, the effect of sleep on hypertension/blood pressure, adjusted for education and the effect of the education and sleep interaction [49]. Mediation was tested in path models in which each sleep measure was separately modeled as a mediator predicted by education and as a predictor of hypertension or blood pressure. Mediation models were adjusted for age, gender, race and ethnicity, BMI, and perceived stress.

Sensitivity Analyses were conducted among $n = 274$ participants with available data on SEI using the same methods described above.

Results

Participant characteristics overall and by SES (educational attainment) are shown in Table 1. The mean age for the total population was 53.5 years (SD = 12.4), and 59.6% were female. The prevalence of low educational attainment (high school or less) was 46.7%. Age and gender distributions were similar across levels of educational attainment. Overall, 41.0% identified as non-Hispanic Black and/or African American, 50.7% of the population identified as non-Hispanic White, and 8.2% were categorized into a Multiethnic group due to small sample sizes (Native American or Alaska Native Aleutian Islander/Eskimo, Native Hawaiian or Pacific Islander, Hispanic, other, and don't know). In the high education category, 59.0% of participants identified as non-Hispanic White, compared to 32.2% who identified as non-Hispanic Black and/or African American. Overall, 61.0% of the total sample were classified as having hypertension, 77.7% had high WASO (>30 minutes), and 67.1% had low sleep efficiency (<85%). Spearman correlation coefficients showed that the sleep measures were correlated ($\rho = -0.68$) and that as sleep efficiency increased, WASO decreased (Supplementary Table S1).

There were no significant associations between SES (educational attainment) and WASO or sleep efficiency (Table 2). In race-stratified models, there were no associations between educational attainment and sleep measures (Supplementary Table S2).

Educational attainment was not associated with hypertension or blood pressure (Table 3). In Supplementary Analyses stratified by race and ethnicity, there were no significant associations between education and hypertension or blood pressure (Supplementary Table S3). However, the fully adjusted estimates for non-Hispanic Black and/or African American participants and non-Hispanic White participants were on opposite sides of the null, where higher education was protective against hypertension for Black participants, but not for White participants. Estimates were also on opposite sides of the null for diastolic blood pressure, where higher education was associated with a decrease in diastolic blood pressure for Black participants, but an increase for White participants.

Sleep efficiency was associated with hypertension and blood pressure (Table 3). Individuals with low (<85%) compared to high sleep efficiency had 24% higher prevalence of hypertension (aPR = 1.24, 95% CI: 1.02 to 1.51), higher systolic blood pressure ($a\beta = 4.61$, 95% CI: 0.69 to 8.53), and higher diastolic blood pressure ($a\beta = 2.50$, 95% CI: 0.10 to 4.89), after adjustment for age, gender, race and ethnicity, BMI, and perceived stress. In Supplementary Analyses stratified by race and ethnicity, there were no associations between sleep measures and hypertension or blood pressure (Supplementary Table S3). However, the

Table 1. Participant Characteristics by Level of Educational Attainment Categories, Midlife in the United States II, 2004–2009, (n = 426)

Participant characteristics	Total (n = 426)	Low education (n = 199)	High education (n = 227)
	n (%) or mean (SD)	n (%) or mean (SD)	n (%) or mean (SD)
Demographic			
Age, years	53.5 (12.4)	53.1 (13.4)	53.8 (11.5)
Women	254 (59.6)	117 (58.8)	137 (60.4)
Race and ethnicity			
Non-Hispanic White	216 (50.7)	82 (41.2)	134 (59.0)
Non-Hispanic Black and/or African American	175 (41.0)	102 (51.3)	73 (32.2)
Multiethnic[a]	35 (8.2)	15 (7.5)	20 (8.8)
Socioeconomic status (SES) indicators			
Socioeconomic index score (SEI)[b]	37.7 (13.5)	30.3 (10.6)	42.6 (13.0)
Household income, USD, thousands	34.1 (49.0)	20.4 (37.0)	46.5 (55.0)
Health			
Body mass index (BMI)	29.7 (9.6)	30.1 (11.8)	29.4 (7.1)
Perceived Stress Scale (PSS)	22.7 (6.5)	22.4 (6.3)	22.9 (6.7)
Cardiovascular measures			
Systolic blood pressure, mmHg	132.4 (18.0)	131.7 (16.9)	133.0 (18.9)
Diastolic blood pressure, mmHg	77.3 (10.6)	77.8 (11.4)	76.8 (9.8)
Blood pressure category			
Normal (%)	101 (23.7)	42 (21.1)	59 (26.0)
Elevated (%)	65 (15.3)	39 (19.6)	26 (11.4)
Hypertension (%)	260 (61.0)	118 (59.3)	142 (62.6)
Actigraphy sleep measures			
Wakefulness after sleep onset, minutes	49.0 (24.1)	48.7 (23.0)	49.4 (25.1)
Wakefulness after sleep onset, % > 30 min	331 (77.7)	155 (77.9)	176 (77.5)
Sleep efficiency, percentages	79.4 (10.5)	79.4 (10.9)	79.3 (10.2)
Sleep efficiency, % < 85%	286 (67.1)	132 (66.3)	154 (67.8)

[a]Multiethnic category includes Native American or Alaska Native Aleutian Islander/Eskimo, Native Hawaiian or Pacific Islander, Hispanic, other, and don't know.
[b]SEI data is only available for n = 274 participants.
Abbreviations: mmHg, millimeters of mercury; SD, standard deviation.

Table 2. Associations Between Educational Attainment and Wakefulness After Sleep Onset (WASO) and Sleep Efficiency, (n = 426)

	WASO > 30 min		Sleep efficiency < 85%	
	Model 1[a]	Model 2[b]	Model 1[a]	Model 2[b]
SES measure	PR (95% CI)	PR (95% CI)	PR (95% CI)	PR (95% CI)
Low education (reference = high)	1.00 (0.91, 1.11)	0.99 (0.87, 1.11)	0.98 (0.86, 1.12)	0.98 (0.85, 1.14)

[a]Model 1 is unadjusted.
[b]Model 2 is adjusted for age, gender, race and ethnicity, BMI, and perceived stress.
Abbreviations: BMI, body mass index; CI, confidence interval; min, minutes; SES, socioeconomic status; PR, prevalence ratio; WASO, wakefulness after sleep onset.
≤30 min and ≥85% are ideal for WASO and sleep efficiency, respectively.

magnitude of associations between WASO and sleep efficiency on systolic and diastolic blood pressure appeared to be larger for white participants.

In multivariable Poisson regression models estimating the association between educational attainment and hypertension, there were no observed associations between education and hypertension (Table 4). In the fully adjusted model (model 3), individuals with low (<85%) compared to high sleep efficiency had a 24% higher prevalence of hypertension (aPR = 1.24, 95% CI: 1.02 to 1.51). Low sleep efficiency was also associated with higher systolic (aβ = 4.60, 95% CI: 0.67 to 8.52) and diastolic blood pressure (aβ = 2.51, 95% CI: 0.11 to 4.91; Supplementary Table S4).

There was no evidence that WASO or sleep efficiency mediated the relationship between SES (educational attainment) and hypertension (Supplementary Figure S1).

Sensitivity analyses

We conducted sensitivity analyses for the subset of our participants with available data on SEI (n = 274). Demographics among this subset were similar to the full sample (Supplementary Table S5), but the two SES measures were not associated with one another (χ^2 = 0.0584). The overall mean SEI score was 37.7 (SD = 13.5).

As with the education measure, there were no significant associations between SEI and WASO or sleep efficiency (Supplementary Table S6), including in race-stratified models (Supplementary Table S2). However, unlike the education measure, in unadjusted models, lower SEI was significantly associated with a decreased prevalence of hypertension (PR = 0.80, 95% CI: 0.66 to 0.98; Supplementary Table S7) and a decrease in mean systolic blood pressure (β = –4.44, 95% CI: –8.64 to –0.23; Supplementary Table S8). These associations were no longer present in the fully adjusted models. In race-stratified models, similar to the education measure, there were no significant associations between SEI and hypertension or blood pressure. However, estimates were also on opposite sides of the null for diastolic blood pressure, where higher SEI was associated with a decrease in diastolic blood pressure for Black participants, but an increase for White participants (Supplementary Table S3).

Discussion

In a cross-sectional analysis of the MIDUS study among African American, Native American or Alaska Native Aleutian Islander/Eskimo, Native Hawaiian or Pacific Islanders, Hispanic, and White adults, we examined associations of SES, sleep quality, and hypertension. We found that (1) low sleep efficiency was associated with blood pressure and hypertension, (2) the association between SEI and hypertension may differ by race and ethnicity, and (3) there was no evidence of sleep mediating the SES-hypertension relation. In our sample of middle-aged adults, there was a high prevalence of hypertension (61.0%) and low sleep efficiency (67.1%). Our results suggest sleep efficiency may be an appropriate target for reducing the burden of hypertension.

The present study found an unadjusted association between SEI and hypertension but was attenuated and no longer significant

Table 3. Associations Between Educational Attainment, Sleep Measures, and Hypertension, Systolic Blood Pressure, and Diastolic Blood Pressure, (*n* = 426)

	Hypertension		Systolic BP, mmHg		Diastolic BP, mmHg	
	Model 1[a]	Model 2[b]	Model 1[a]	Model 2[b]	Model 1[a]	Model 2[b]
	PR (95% CI)	PR (95% CI)	β (95% CI)	β (95% CI)	β (95% CI)	β (95% CI)
SES measure						
Low education (reference = high)	0.95 (0.81, 1.10)	0.96 (0.81, 1.13)	–1.29 (–4.73, 2.14)	–1.04 (–4.78, 2.71)	0.95 (–1.07, 2.97)	0.98 (–1.30, 3.27)
Sleep measures						
WASO > 30 min (reference= ≤ 30 min)	1.15 (0.94, 1.40)	1.15 (0.93, 1.42)	3.34 (–0.76, 7.45)	3.39 (–0.94, 7.72)	2.30 (–0.12, 4.71)	2.13 (–0.51, 4.77)
Sleep efficiency <85% (reference= ≥ 85%)	1.14 (0.96, 1.36)	1.24* (1.02, 1.51)	2.77 (–0.87, 6.41)	4.61* (0.69, 8.53)	1.98 (–0.16, 4.12)	2.50* (0.10, 4.89)

*p-values < .05.
[a]Model 1 is unadjusted.
[b]Model 2 is adjusted for age, gender, race and ethnicity, BMI, and perceived stress.
Abbreviations: BMI, body mass index; BP, blood pressure; CI, confidence interval; min, minutes; mmHg, millimeters of mercury; PR, prevalence ratio; WASO, wakefulness after sleep onset.
≤30 min and ≥85% are ideal for WASO and sleep efficiency, respectively; education and sleep measures were assessed in separate models.

Table 4. Poisson Regression Models Examining Association Between Educational Attainment and Hypertension, Adjusted for Demographic, Health, and Sleep Covariates, (*n* = 426)

	Model 1	Model 2	Model 3
	PR (95% CI)	PR (95% CI)	PR (95% CI)
Low education (reference = high)	0.96 (0.81, 1.13)	0.96 (0.81, 1.13)	0.96 (0.81, 1.13)
Age, years	1.01* (1.00, 1.02)	1.01* (1.00, 1.02)	1.01* (1.00, 1.02)
Gender (reference = male)	0.96 (0.81, 1.13)	0.96 (0.81, 0.13)	0.97 (0.82, 1.14)
Non-Hispanic Black and/or African American (reference = NHW)	0.96 (0.80, 1.16)	0.96 (0.80, 1.16)	0.96 (0.79, 1.15)
Multiethnic[a] (reference = NHW)	1.28* (1.02, 1.61)	1.29* (1.03, 1.61)	1.24 (0.99, 1.55)
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 > 30 min (reference= ≤ 30 min)	—	1.15 (0.93, 1.43)	—
Sleep efficiency < 85% (reference= ≥ 85%)	—	—	1.24* (1.02, 1.51)

*p-values < .05.
[a]Multiethnic category includes Native American or Alaska Native Aleutian Islander/Eskimo, Native Hawaiian or Pacific Islander, Hispanic, other, and don't know.
Abbreviations: BMI, body mass index; BP, blood pressure; CI, confidence interval; min, minutes; mmHg, millimeters of mercury; NHW, non-Hispanic White; PR, prevalence ratio; PSS, Perceived Stress Scale; WASO, wakefulness after sleep onset.
≤30 min and ≥85% are ideal for WASO and sleep efficiency, respectively.

in the fully adjusted model. Multiple studies have indicated that lower 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 [15]. Our mean systolic and diastolic blood pressures were consistent with other MIDUS studies with larger sample sizes that were not limited to participants with complete sleep actigraphy data [52]. While there were differences in hypertension prevalence by SEI in our sample, our sample size ($n = 274$) may have been too small to detect a meaningful association between SEI and hypertension. Additionally, the present study found no direct association between educational attainment and hypertension, which is not consistent with prior studies conducted within the United States [13, 15, 19, 20]. However, our results could be due to differences in the education-hypertension gradient by race and ethnicity. In our Supplementary Analyses where we explored whether the associations between education and blood pressure varied by race, we found that estimates for education and both hypertension and diastolic blood pressure were in opposite directions of the null for non-Hispanic White versus non-Hispanic Black and/or African American participants. For Black participants, lower education was associated with a higher prevalence of hypertension and a higher mean diastolic blood pressure, while for White participants, associations were in the opposite direction. Similarly, in a study of a large, multicenter Family Blood Pressure Program, Non and colleagues found that the association between education and blood pressure was stronger among the African American participants than the White participants [53]. These results suggest that higher education may be more beneficial for hypertension for African American individuals than for White individuals, who are more likely to benefit from other socioeconomic advantages. Although there was no significant interaction between race and SES in any of the models looking at education or SEI with hypertension, systolic blood pressure, and diastolic blood pressure, our results indicated that the association between SEI and hypertension may differ by race and ethnicity. Future studies should further explore the intersection of race and SES on hypertension in a larger sample, as growing research demonstrates that SES and race and ethnicity can function jointly and independently to affect health.

The current study found no direct association between SES and sleep quality, which is not consistent with prior studies conducted within the United States [25, 31]. Among a sample of predominately White (67.1%), Hispanic/Latino (17.4%), and Black/African American (8.9%) participants, Grandner et al., found that lower SES was associated with higher rates of self-reported sleep complaints [31]. Our results may differ due to a difference in racial composition. Our study oversampled Black/African American participants, which may have masked effects due to potential opposite SES-sleep gradients by race. Racial and ethnic disparities in reports of sleepiness and sleep complaints have been found to be inconsistent, further complicated by the added role of SES [54–56]. Jackson et al. found that shorter sleep duration increases with professional responsibility for Black individuals, while the opposite effect occurs for White individuals [55]. An additional study among Black adults also found that the association between stress and short sleep duration was more pronounced among those of higher educational attainment [57]. This sleep-SES gradient for Black and White individuals 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 [54]. As the sleep-SES association may vary by race, this should be further explored in future analyses. Additionally, while Native American or Alaska Native Aleutian Islander/Eskimo, Native Hawaiian or Pacific Islanders, and Hispanic individuals were included in the present study, the sample size was low, therefore, more studies are needed to evaluate these populations in regard to sleep and the added dimensionality of SES. As these populations, and Black and/or African American populations, are heterogenous groups, within-group studies should also be further explored.

Sleep quality and sleep disorders, which can affect sleep quality, such as obstructive sleep apnea and insomnia, have been linked to hypertension prevalence and risk [24, 25, 58]. Per our hypothesis, we found that lower sleep efficiency was associated with hypertension and higher sleep efficiency was associated with lower systolic blood pressure and diastolic blood pressure. Our finding is consistent with a study by Ramos et al., which found that a 10% reduction in sleep efficiency, measured via actigraphy, was significantly associated with a 7.5% greater hypertension prevalence in a study of US Latinos (95% CI: −12.9 to −2.2) [58]. These findings are supported by research demonstrating a significant relationship between sleep efficiency and hypertension. It is hypothesized that sleep disruption or poor sleep quality is associated with the activation of the hypothalamic-pituitary-adrenal axis and the sympathetic nervous system, which may increase hypertension risk [59]. Individuals with poor sleep quality have more interruptions and wakefulness during the sleep period which 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 [60]. Based on these findings, sleep is a viable target for hypertension reduction.

We found no evidence that sleep mediated the association between SES and hypertension. Among the few studies that have considered sleep quality as a mediator between SES and hypertension, Piccolo et al. examined restless sleep as a mediator for SES disparities and multiple outcomes, including hypertension, and also observed no mediation by sleep [61]. Specifically, the authors found that while social disparities in sleep and hypertension incidence were highly significant and mirrored one another, sleep did not have a significant role in mediating SES differences in hypertension. 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 adults, and lower- and middle-class adults were more likely to report restless sleep compared to higher-class adults. Given these findings, while sleep is an acceptable target as an effective intervention for the reduction of hypertension prevalence, further research should consider the potential influence of race-SES disparities on hypertension.

A further understanding of the role of sleep health in shaping socioeconomic disparities in hypertension may help tailor public health interventions in sleep quality among populations with lower socioeconomic status to reduce the burden of hypertension. Poor sleep quality in adults may present as a result of undiagnosed sleep disorders, such as sleep apnea or insomnia, or secondary to comorbidities [62]. 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 [63]. In conjunction, there is a continuing need for further research to examine the treatment of sleep disorders as a form of hypertension management [64]. Apart from disordered sleep, sleep quality has been suggested as a general therapeutic target for the prevention of hypertension, with potential interventions including a

multicomponent sleep intervention consisting of sleep hygiene education, participating in sleep scheduling, and undergoing cognitive behavioral therapy [65]. The high prevalence of poor sleep quality and hypertension in the present study indicates that there is an immediate need to identify strategies to reduce this societal burden. While the Life's Essential 8 (LE8) measures for improving and maintaining cardiovascular health have been updated to include sleep duration, our study highlights the need for an expansion of LE8 to capture other measures of sleep such as sleep quality, as consistent with the multidimensional framework of sleep health [27].

The present study has many strengths. This study contributes to the literature by examining pathways through which social factors are related to hypertension. Although we did not find that sleep mediated the association of interest, we did find that sleep quality was related to hypertension. A large sample of Black individuals were included in the study, thus increasing the diversity of research and yielding a sufficient sample size for Black–White 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 [66]. As self-reported sleep does not corroborate with objective sleep indicators, relying on self-reporting measures may mask different dimensions of sleep-related health outcomes [67]. An additional strength is that this study operationalized SES through a composite measure in sensitivity analyses, which is beneficial in capturing a comprehensive understanding of SES on hypertension.

There are several limitations to this study. This was a cross-sectional study in which SES and hypertension or blood pressure were simultaneously assessed, meaning that we could not establish a temporal relationship between the exposure and outcome [68]. The participants were sampled from urban cities (Madison, WI and Milwaukee, WI), which limits the generalizability. While Black participants were oversampled, allowing race-stratified comparison to non-Hispanic White participants, there were limited participants of other racial and ethnic groups. Educational attainment was used as an individual proxy for SES, which has additional limitations. For example, if participants obtained education outside of the United States, different educational systems may have different implications for educational levels [69]. Additionally, 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 [69].

Overall, low sleep efficiency was associated with higher blood pressure and a higher prevalence of hypertension. Consequently, improvements in sleep quality may be important in the potential reduction and management of hypertension in US adults. 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, and the subsequent impact on hypertension.

Supplementary material

Supplementary material is available at *SLEEP* online.

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Data Availability

The datasets generated and/or analyzed during the current study are available from the Inter-university Consortium for Political and Social Research (ICPSR) online portal, https://www.icpsr.umich.edu. Some data for the MIDUS Milwaukee African American Sample require signing of a data use agreement through ICPSR, and interested parties should email the MIDUS Help Desk (midus_help@aging.wisc.edu).

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