






# Multidimensional Sleep Health Problems Across Middle and Older Adulthood Predict Early Mortality

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## Abstract

**Background:** Having multiple sleep problems is common in adulthood. Yet, most studies have assessed single sleep variables at one timepoint, potentially misinterpreting health consequences of co-occurring sleep problems that may change over time. We investigated the relationship between multidimensional sleep health across adulthood and mortality.

**Methods:** Participants from the Midlife in the United States Study reported sleep characteristics in 2004–2006 (MIDUS-2; M2) and in 2013–2014 (MIDUS-3; M3). We calculated a composite score of sleep health problems across 5 dimensions: Regularity, Satisfaction, Alertness, Efficiency, and Duration (higher = more problems). Two separate models for baseline sleep health ( $n = 5\ 140$ ; median follow-up time = 15.3 years) and change in sleep health ( $n = 2\ 991$ ; median follow-up time = 6.4 years) to mortality were conducted. Cox regression models controlled for socio-demographics and key health risk factors (body mass index, smoking, depressive symptoms, diabetes, and hypertension).

**Results:** On average, 88% of the sample reported having one or more sleep health problems at M2. Each additional sleep health problem at M2 was associated with 12% greater risk of all-cause mortality (hazard ratio [HR] = 1.12, 95% confidence interval [CI] = 1.04–1.21), but not heart disease-related mortality (HR = 1.14, 95% CI = 0.99–1.31). An increase in sleep health problems from M2 to M3 was associated with 27% greater risk of all-cause mortality (HR = 1.27, 95% CI = 1.005–1.59), and 153% greater risk of heart disease mortality (HR = 2.53, 95% CI = 1.37–4.68).

**Conclusions:** More sleep health problems may increase the risk of early mortality. Sleep health in middle and older adulthood is a vital sign that can be assessed at medical checkups to identify those at greater risk.

**Keywords:** Aging, Cardiovascular, Hazard ratio, Midlife, Mortality, Sleep health

Sleep is one of the key markers of aging (1,2). Many older adults express concerns about their sleep (3,4) and experience less deep sleep and more wake after sleep onset (2). More fragmented sleep and more daytime napping frequently observed in older adults are closely related to their worsened health conditions, such as cognitive decline, incident Alzheimer's disease, and Parkinson's disease pathology (5–7). Many studies have focused on sleep in late adulthood, with less attention to changes in sleep from middle adulthood. Yet, sleep is often degraded from middle to older adulthood for a variety of reasons. For example, middle-aged men and women begin to experience degraded sleep quality and the onset of sleep disorders (eg, insomnia and sleep apnea) due to stress from work and family pressures, increased body weight, and/or menopause (8–11). Examining multiple sleep characteristics and their changes across middle and older adulthood may

add insight into how sleep is a vital sign and predicts who may have shorter time left in life.

Indeed, in studies of sleep and mortality, both long and short sleep durations are associated with a higher risk of mortality (12–14). However, results are not consistent across studies. Based on a review of 42 studies, Kurina et al. (15) have shown that only 14 studies show a U-shaped relationship between sleep duration and mortality, whereas 23 studies show no association at all, and the remaining 5 show associations for either short or long sleep duration. Some research has also found that the relationship between sleep duration and mortality does not exist in older adults (age >65; (16)). These inconsistent results should not undermine the importance of studying how sleep influences mortality, because sleep duration alone may not comprehensively capture age-related changes in sleep characteristics that may be associated with mortality.

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A sleep health perspective by Buysse (2014) suggests that the importance of sleep in health needs to be understood across multiple sleep dimensions. Specifically, the Ru-SATED—Regularity in sleep, Satisfaction with sleep, Alertness during waking hours, Timing of sleep, Sleep Efficiency, and Sleep Duration—are proposed as key dimensions that support optimal health and functioning. Assessing these multiple sleep dimensions in older adults has shown that time in bed, hours spent napping, and wake-up time are the most important sleep dimensions predictive of all-cause and cardiovascular mortality (17). Another study examining patterns of multidimensional sleep health suggests that the combination of long sleep, high efficiency/satisfaction, and daytime sleepiness may indicate a high-risk sleep phenotype in older adults that is predictive of all-cause mortality (18). Furthermore, the few studies focusing on middle adulthood show that having more sleep health problems (based on a composite score summarizing multidimensional sleep health) is associated with a higher risk of heart disease (19). Taken together, these studies suggest that it is important to use a comprehensive measure of multidimensional sleep health or “co-occurring sleep problems” to thoroughly examine the effect of sleep on mortality.

Another gap in the literature on sleep and mortality is a lack of longitudinal sleep assessments. Most studies have used habitual sleep characteristics measured at one time point, lacking information on whether within-person changes in sleep health over time predict mortality risk. The limited studies that examine longitudinal changes in sleep have focused on single sleep variables such as sleep duration only, reporting that individuals who are consistently sleeping less than 5 hours per night and those who exhibit inconsistent patterns (those who decreased or increased sleep duration by 4.9 to 7.0 hours per night for more than 4 years) have increased mortality risk (20). The aforementioned study shows great variability in sleep duration in normative aging. Given that changes in individual sleep dimensions are likely to be related to one another, changes in multidimensional sleep health may provide better insight into the importance of sleep on the risk of mortality than a sleep dimension measured at a single timepoint.

The current study examined the association between multidimensional sleep health over time in relation to time to death (shorter time to death = a higher mortality risk) in a national sample of adults. To assess “how many” sleep health problems co-occur within an average adult, we created a sleep health composite score, using 5 sleep health dimensions as suggested by Buysse (17). For mortality outcomes, we used 2 indicators—all-cause and heart disease-related mortality, given that heart disease is a leading cause of death in the United States (21). We hypothesized that having more sleep health problems at baseline and/or experiencing an increase in sleep health problems over time would be associated with higher risk for all-cause mortality and heart disease-related mortality.

## Method

### Participants

Data were obtained from the Midlife in the United States (MIDUS) Study, which recruited a sample of community-living, English-speaking adults. Participants completed telephone interviews and mailed self-administered questionnaires. Comprehensive assessments of sleep measures using

self-administered questionnaires were first captured in the second wave (MIDUS 2, collected between 2004 and 2006;  $n = 5,555$ ) and then repeated in the third wave of data collection that occurred approximately 10 years later (MIDUS 3, collected between 2013 and 2014). To minimize attrition at follow-up, participants were also contacted between 2018 and 2019 for the MIDUS 3 data collection (referred to as the MIDUS 3 Retention Study). Of these participants, 4,363 provided sleep data in MIDUS 2 (M2) and 3,768 participants provided sleep data at MIDUS 3 (M3).

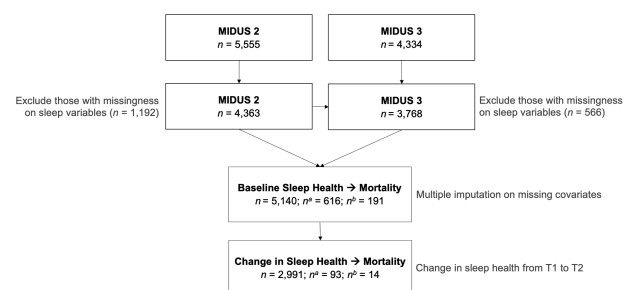
This study used baseline sleep health and change in sleep health as predictors of mortality. For the baseline sleep health to mortality analyses, 4,363 participants who provided valid sleep data at M2 were combined with 777 participants who only provided sleep data at M3, resulting in a total of 5,140 participants. The median follow-up time was 15.3 years for the baseline sample (Range = 0.08–16 years). For the change in sleep health to mortality analyses, 2,991 participants provided 2 waves of sleep data at M2 and M3, and the median follow-up time from M3 was 6.4 years (Range = 0.08–6.58 years). Figure 1 displays the sample flowchart.

Comprehensive details of the MIDUS study can be found elsewhere (22–24). The MIDUS study was approved by all appropriate Institutional Review Boards and all MIDUS participants provided informed consent. The current study was exempt from the IRB review process because it used publicly available, deidentifiable data.

## Measures

### Multidimensional sleep health problems

We created a sleep health composite score capturing regularity, satisfaction, alertness, efficiency, and duration dimensions (17) using survey data. Sleep timing was included in the original conception of sleep health, but not assessed in the MIDUS survey data. We operationalized each sleep health dimension based on valid items used in previous studies (19,25,26). Consistent with previous literature and guided by previously used empirical cut points (19,27–32) (also see Table 1), each sleep dimension was binary coded, such that 1 indicated having a sleep health problem (eg, irregularity, inefficiency, short/long sleep duration). The sum of all sleep dimension indicators yielded the number of sleep health problems. Higher values indicated a greater number of sleep health problems (Range = 0–5). Although the original conceptualization of sleep health highlights a positive framing (17), we chose to operationalize it in a reversed way to provide more intuitive inference in relation to our outcome of interest (mortality)



**Figure 1.** Participant flowchart. <sup>a</sup>Number of all-cause mortality. <sup>b</sup>Number of heart disease mortality.

and compare its effect size to those of other well-known risk factors.

### Mortality

Mortality data were obtained from the National Death Index reports and included relevant information on deceased status, cause of death, and year of death. All living respondents were right censored (=0) at the latest time point available. Any person who died was coded as 1 for all-cause mortality. ICD-10 codes I00-I99 were used to determine heart disease-related mortality (eg, hypertensive heart disease, acute myocardial infarction). Consistent with past work, ICD-10 codes R96-R99, which are characterized as ill-defined conditions of death, were not considered heart disease related ( $n = 1$ ; (33)).

Using the data available as of April 2022, the mortality data set available from MIDUS included all participants who died since participating in the study until December 2018. If the participant was not included in the mortality data set, then they were censored as being alive in December 2018. There were a number of participants who completed the MIDUS 3 Retention Study, which collected data from participants between 2018 and 2019 ( $n = 651$ ). If they participated in MIDUS 3 Retention Study, they were right censored after December 2019 (eg, someone who provided data in December 2019 would receive a follow-up time one month longer than December 2019).

### Covariates

We controlled for sociodemographics and known health risk factors. Sociodemographics included age (in years), sex (male = 1, female = 0), race/ethnicity, education, household total income (\$USD from wages, pension, social security, and other sources), and work status (1 = currently working, 0 = not working). For race/ethnicity, 2 dummy coded variables were created: an indicator for non-Hispanic Black individuals (1 = non-Hispanic Black, 0 = not non-Hispanic Black) and an indicator for Hispanic individuals and all other races (1 = Hispanic or another race, 0 = not Hispanic or another

race). By including these 2 indicators in the model, the reference category was non-Hispanic White individuals. Education was coded into 4 categories: 0 = Some high school/No GED, 1 = High school diploma or GED, 2 = Some college/associate degree, 3 = Bachelor's degree or higher. Household income was log transformed due to the variable being skewed (skewness = 1.58) and leptokurtic (ie, narrow; kurtosis = 2.70). Following standard practices, a constant of 1 was added to household income prior to log transformation to account for the 213 participants who reported 0 for their household income (34).

Health covariates included body mass index (BMI), diabetes, hypertension, smoking status, and depressive symptoms. These health covariates were selected based on their associations with sleep health, heart disease, and mortality (19,35,36) and on consultation with experts to not “over-control” (eg, deciding not to control for stroke, heart-related diseases) and bias results toward Type II error. BMI was calculated by dividing self-reported weight by height ( $\text{kg}/\text{m}^2$ ). For diabetes and hypertension, participants reported if they ever experienced or were treated for diabetes in the past 12 months (1 = yes, 0 = no) and if they were ever diagnosed with hypertension by a physician (1 = yes, 0 = no). For smoking status, 2 dummy coded variables were created using data at the time of data collection: an indicator for former smokers (1 = former smoker, 0 = not former smoker) and an indicator for current smokers (1 = current smoker, 0 = not current smoker). By including these 2 indicators in the model, the reference category was those who are not currently smoking nor previously smoked. Depressive symptoms were measured using the World Mental Health Organization's Composite International Diagnostic Interview—Short Form (37). Responses ranged from 0 to 6, with higher values indicating more depressive symptoms. Sleep-related items were removed from the depressive symptoms questionnaire. We also adjusted for the month data collection occurred since a previous study found that sleep complaints varied across seasons (38). For baseline sleep health models, we controlled for covariates at M2. For the change models, we controlled for covariates measured at M3.

**Table 1.** Dimensions Used to Construct Multidimensional Sleep Health Composite

Sleep Health Composite Indicators		
Dimension	Items	Cut Points
Regularity	Difference between workday sleep duration and nonworkday sleep duration	1: Absolute value >60 min 0: Absolute value ≤60 min
Satisfaction	Have trouble falling asleep. Wake up during the night and have difficulty going back to sleep. Wake up too early in the morning and be unable to get back to sleep. Feel unrested during the day, no matter how many hours of sleep you had.	1: 1 on at least 1 of 4 items 0: 0 for all 4 items
Alertness	During a usual week, how many times do you nap for 5 min or more?	1: >2 naps/wk 0: ≤2 naps/wk
Efficiency	How long does it usually take you to fall asleep at bedtime?	1: >30 min 0: ≤30 min
Duration	How much sleep do you usually get at night (or in your main sleep period) on weekdays or workdays?	1: <6 or >8 h 0: ≥6 and ≤8 h

*Note:* For each sleep dimension, suboptimal sleep was coded as 1 (vs 0 = optimal sleep). These binary indicators were summed, so that possible scores ranged from 0 to 5 for the sleep health composite. Higher numbers indicated a greater number of sleep health problems.

## Statistical Analyses

### Multiple imputation

Considering the limited number of valid sleep observations at baseline and small number of deaths, multiple imputation was used on missing covariates to maximize the available data. BMI ( $n = 1\ 167$ ) and diabetes status ( $n = 922$ ) had the largest amount of missingness, followed by race/ethnicity ( $n = 26$ ), employment status ( $n = 21$ ), hypertension ( $n = 11$ ), education ( $n = 8$ ), and age ( $n = 1$ ). Given the large degree of missingness, chi-square tests were used to examine differences among those with and without missingness on BMI and diabetes. The percentage of deaths was higher among those with missingness on BMI (17.9%) compared to those with information on BMI (11.5%),  $\chi^2(1, N = 5\ 140) = 13.88, p < .001$ . The percentage of deaths was lower among those with missingness on diabetes (7.3%) compared to those with information on diabetes (12.3%),  $\chi^2(1, N = 5\ 140) = 8.13, p = .004$ . To fully utilize all available data, SAS (SAS Institute, Cary, NC) procedure MI using multivariate normal distribution with 10 imputations was used (39).

### Survival analysis and Cox regression

For hypothesis testing, we used SAS procedure PHREG to estimate Cox regressions (40). First, we examined the associations between baseline sleep health and risk of all-cause and heart disease mortality in separate models. Second, we examined the associations between change in sleep health (M3-M2) and risk of all-cause and heart disease mortality, controlling for M2 sleep health. Significance was determined by using the 95% CI; if the CI included 1, the hazard ratio (HR) was considered not statistically significant.

To check the proportionality assumption, we visually assessed the Kaplan–Meier curves (41,42). If the proportionality assumption was violated, extended Cox regressions were performed with (1) time-varying covariates (with covariate  $\times$  time interactions) and (2) using a shorter follow-up time, as suggested by the literature (43–46). The analyses did not incorporate sample weights due to the potential complexity associated with attrition that is common in longitudinal data.

## Results

See Table 2 for the characteristics of the 2 analytic samples (ie, baseline and change models). At baseline, participants were 56.4 years old, on average ( $SD = 12.3$ ). There were slightly more females (55%) than males (45%), and the sample was predominantly non-Hispanic White (79%). The mean of the sleep health problems was 1.7 ( $SD = 1.1$ ), meaning that, on average, our sample of participants had approximately 2 sleep health problems out of a total of 5. The mean of the change in sleep health problems was 0.14 ( $SD = 1.14, Range = -4$  to 4). In total, around 12% of the sample died ( $n = 616$ ); of these, 4% died because of a heart disease-related condition ( $n = 191$ ). Chi-square and one-way analysis of variance (ANOVA) were conducted to examine differences between those who completed M2 only, M3 only, and both M2 and M3 (see Supplementary Appendix Table 1). Results revealed that those who participated in both M2 and M3 were younger, had more education, lower prevalence of diabetes, lower prevalence of high blood pressure, and had fewer sleep health problems at baseline, compared

to those who completed M2 only or M3 only. The mean age of those who died was 67.84 ( $SD = 11.19, Range = 35$ –85 years).

Table 3 shows results from the unadjusted and adjusted Cox regression models predicting all-cause and heart disease mortality. Starting with all-cause mortality as the outcome, the adjusted analyses showed that per additional sleep health problem at baseline, we observed a HR of 1.12 (95% CI = 1.04–1.21) for all-cause mortality but did not reach statistical significance for heart disease mortality (HR = 1.14, 95% CI = 0.99–1.31). See Figure 2, Panel A. The sleep change analyses showed that, per each additional sleep health problem from M2 to M3, the risk of mortality increased by 27% (HR = 1.27, 95% CI = 1.005–1.59) for all-cause mortality and 153% (HR = 2.53, 95% CI = 1.37–4.68) for heart disease mortality. See Figure 2, Panel B.

## Sensitivity Analyses

### Investigating proportionality assumptions

Visual assessment of the Kaplan–Meier curves suggested that the proportionality assumption could be considered questionable (see Supplementary Appendix Figure 1). See Supplementary Appendix Table 2 for the formal proportionality test, showing that the associations of sleep health and some covariates with mortality are time varying. After adjusting for the time-varying associations, results were consistent between the main analyses (without time interactions) and sensitivity analyses (with time interactions). Analyses were further stratified by a follow-up time of  $\leq 4$  years or  $> 4$  years (cutoff selected based on visualization of the Kaplan–Meier curves; see Supplementary Appendix Table 3). Results were consistent with those from the main analyses only when a follow-up time was  $\leq 4$  years (but not when a follow-up time was  $> 4$  years).

### Controlling for family history of heart attack and physical activity

Following previous research (19), analyses were repeated after including family history of heart attack and levels of physical activity as additional covariates (see Supplementary Appendix Table 4). After controlling for family history of heart attack and physical activity, there were no changes in HRs between baseline sleep health and all-cause or heart disease mortality. As for change in sleep health to all-cause mortality, the HR was reduced minimally from 1.27 to 1.25. Similarly, for change in sleep health to heart disease mortality, the HR was reduced from 2.53 to 2.42 after controlling for the additional covariates.

## Secondary Analyses

### Individual sleep indicators and mortality

Additional analyses were conducted to examine the associations of baseline binary indicators of individual sleep dimensions with the risk of mortality. Separate models for all-cause and heart disease mortality were run with all sleep indicators entered in one model (Supplementary Appendix Table 5). In adjusted models, short/long sleep duration and sleep inefficiency (measured by long sleep onset latency) at baseline were associated with increased risk of all-cause mortality. Sleep inefficiency at baseline was associated with increased risk of heart disease mortality.

**Table 2.** Sample Characteristics

	Baseline Sleep Health ( <i>n</i> = 5 140)	Change in Sleep Health ( <i>n</i> = 2 991)
<i>Sociodemographics and health covariates</i>		
Age (y), <i>M</i> ( <i>SD</i> )	56.4 (12.3)	63.4 (11.0)
Equal to or less than 40 y, <i>n</i> (%)	557 (10.8%)	1 (0.03%)
Between 40 and 60 y, <i>n</i> (%)	2 560 (49.8%)	1 180 (39.5%)
Equal to or greater than 60 y, <i>n</i> (%)	2 023 (39.4%)	1 810 (60.5%)
Sex		
Female, <i>n</i> (%)	2 824 (54.9%)	1 700 (56.8%)
Male, <i>n</i> (%)	2 316 (45.1%)	1 291 (43.2%)
Race/ethnicity <sup>a,b</sup>		
Non-Hispanic White, <i>n</i> (%)	4 034 (78.8%)	2 330 (78.4%)
Non-Hispanic Black, <i>n</i> (%)	726 (14.2%)	370 (12.5%)
Hispanic and all other races, <i>n</i> (%)	359 (7.0%)	272 (9.1%)
Hispanic, <i>n</i> (%)	160 (3.1%)	84 (2.8%)
All Other races <sup>c</sup> , <i>n</i> (%)	199 (3.9%)	188 (6.3%)
Education, <sup>a,d</sup> <i>M</i> ( <i>SD</i> )	2.0 (1.0)	2.0 (0.9)
No high school diploma or GED, <i>n</i> (%)	371 (7.2%)	163 (5.5%)
High school diploma or GED, <i>n</i> (%)	1 383 (26.9%)	751 (25.1%)
Some college or associate degree, <i>n</i> (%)	1 540 (30.0%)	903 (30.2%)
Bachelor's degree or higher, <i>n</i> (%)	1 846 (35.9%)	1 174 (39.3%)
Household income, <i>M</i> ( <i>SD</i> ) (\$USD) <sup>a,e</sup>	72 963.2 (64 249.5)	72 986.8 (60 242.1)
Currently employed/self-employed <sup>a</sup> , <i>n</i> (%)	2 641 (51.4%)	1 377 (46.0%)
BMI <sup>a</sup> (kg/m <sup>2</sup> ), <i>M</i> ( <i>SD</i> )	28.5 (6.3)	28.8 (6.5)
Has diabetes <sup>a</sup> , <i>n</i> (%)	606 (11.8%)	466 (15.6%)
Has hypertension <sup>a</sup> , <i>n</i> (%)	2 124 (41.3%)	1 554 (52.0%)
Smoking <sup>f</sup>		
Never smoked	2 610 (50.8%)	1 612 (53.9%)
Current smoker	1 714 (33.4%)	1 063 (35.5%)
Former smoker	816 (15.9%)	316 (10.6%)
Depressive symptoms (Range = 0–6: higher), <i>M</i> ( <i>SD</i> )	0.6 (1.5)	0.49 (1.5)
Follow-up time (in years),	13.3 (4.5)	5.6 (1.7)
<i>Main variables</i>		
Sleep health composite (Range = 0–5: higher means poorer),	1.7 (1.1)	1.8 (1.1)
0	617 (12.0%)	326 (10.9%)
1	1 699 (33.1%)	962 (32.2%)
2	1 662 (32.3%)	993 (33.2%)
3	857 (16.7%)	522 (17.5%)
4	273 (5.3%)	164 (5.5%)
5	32 (0.6%)	24 (0.8%)
All-cause mortality	616 (12.0%)	93 (3.1%)
Heart disease mortality	191 (3.7%)	14 (0.5%)

Notes: <sup>a</sup>Multiple imputation was used to impute missing values on sociodemographics and health covariates.

<sup>b</sup>Race/ethnicity was dummy coded, so that an indicator for non-Hispanic Black individuals (1 = non-Hispanic Black, 0 = not non-Hispanic Black) and an indicator for Hispanic individuals and all other races (1 = Hispanic or another race, 0 = not Hispanic or another race) were created.

<sup>c</sup>Those in the other race category included Native American/Alaska Native Aleutian Islander/Eskimo, Asian, Native Hawaiian or Pacific Islander, and those who responded "Other."

<sup>d</sup>The mean value of education indicates that on average, those in the baseline and change in sleep health sample had some college education or associate degree (=2).

<sup>e</sup>Household income was log transformed due to the variable being skewed (skewness = 1.58) and leptokurtic (ie, narrow; kurtosis = 2.70).

<sup>f</sup>Smoking status was dummy coded, so that an indicator for former smokers (1 = Former smoker, 0 = Not a former smoker) and an indicator for current smokers (1 = Current smoker, 0 = Not currently smoking) were created. GED = general educational development; *M* = mean; *n* = number; *SD* = standard deviation; *y* = years

## Discussion

The study results show that multidimensional sleep health across middle and older adulthood is a significant correlate of time to death. We found that having a greater number

of sleep health problems at baseline was associated with a higher risk of all-cause mortality. Moreover, those who reported an increase in the number of sleep health problems at M3 compared to M2 had heightened risks of all-cause

**Table 3.** Results Displaying Mortality Regressed on Sleep Health Composite

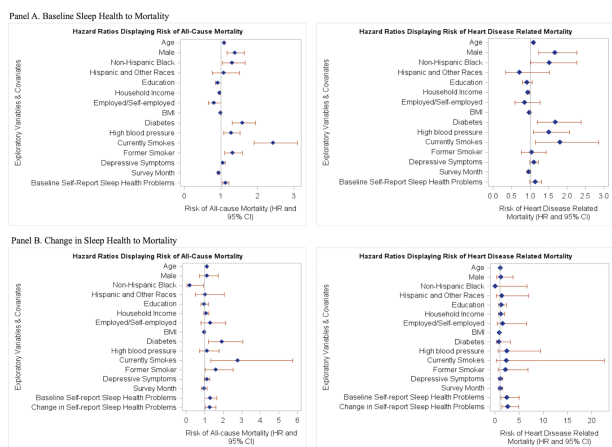
	All-Cause Mortality			Heart Disease Mortality		
	HR	95% CI	<i>p</i> Value	HR	95% CI	<i>p</i> Value
<i>Baseline Sleep Health (n = 5 140)</i>		<i>n</i> <sub>deceased</sub> = 616			<i>n</i> <sub>deceased</sub> = 191	
Unadjusted						
Baseline sleep health	<b>1.21</b>	<b>1.13–1.30</b>	<b>&lt;.001</b>	<b>1.24</b>	<b>1.09–1.40</b>	<b>.001</b>
Adjusted						
Age	<b>1.09</b>	<b>1.08–1.10</b>	<b>&lt;.001</b>	<b>1.10</b>	<b>1.08–1.12</b>	<b>&lt;.001</b>
Male (ref: Female)	<b>1.38</b>	<b>1.17–1.63</b>	<b>&lt;.001</b>	<b>1.66</b>	<b>1.23–2.25</b>	<b>.001</b>
Black (ref: non-Hispanic Whites)	<b>1.29</b>	<b>1.02–1.64</b>	<b>.034</b>	1.49	0.996–2.24	.053
Hispanic/Other (ref: non-Hispanic Whites)	1.06	0.75–1.50	.728	0.69	0.32–1.50	.354
Education	<b>0.92</b>	<b>0.85–0.99</b>	<b>.046</b>	0.92	0.79–1.06	.253
Household income	<b>0.96</b>	<b>0.93–0.99</b>	<b>.003</b>	<b>0.95</b>	<b>0.91–0.997</b>	<b>.036</b>
Currently employed (ref: unemployed)	<b>0.80</b>	<b>0.64–0.99</b>	<b>.049</b>	0.86	0.58–1.27	.439
BMI	1.00	0.98–1.01	.633	0.99	0.96–1.01	.340
Diabetes (ref: no diabetes)	<b>1.58</b>	<b>1.29–1.92</b>	<b>&lt;.001</b>	<b>1.67</b>	<b>1.18–2.37</b>	<b>.004</b>
Hypertension (ref: no Hypertension)	<b>1.28</b>	<b>1.07–1.52</b>	<b>.006</b>	<b>1.49</b>	<b>1.08–2.04</b>	<b>.014</b>
Current smoker (ref: nonsmoker)	<b>2.44</b>	<b>1.91–3.11</b>	<b>&lt;.001</b>	<b>1.82</b>	<b>1.15–2.87</b>	<b>.011</b>
Former smoker (ref: nonsmoker)	<b>1.33</b>	<b>1.11–1.59</b>	<b>.002</b>	1.05	0.76–1.44	.770
Depressive symptoms	1.05	0.996–1.12	.070	1.10	0.997–1.22	.057
Survey month	<b>0.93</b>	<b>0.90–0.96</b>	<b>&lt;.001</b>	0.96	0.91–1.01	.149
Baseline sleep health	<b>1.12</b>	<b>1.04–1.21</b>	<b>.003</b>	1.14	0.99–1.31	.071
<i>Change in Sleep Health (n = 2 991)</i>		<i>n</i> <sub>deceased</sub> = 93			<i>n</i> <sub>deceased</sub> = 14	
Unadjusted						
Baseline sleep health	<b>1.36</b>	<b>1.09–1.68</b>	<b>.006</b>	<b>1.91</b>	<b>1.11–3.29</b>	<b>.021</b>
Change in sleep health	<b>1.35</b>	<b>1.10–1.65</b>	<b>.004</b>	<b>2.10</b>	<b>1.27–3.49</b>	<b>.004</b>
Adjusted						
Age	<b>1.13</b>	<b>1.10–1.16</b>	<b>&lt;.001</b>	<b>1.14</b>	<b>1.06–1.22</b>	<b>.0004</b>
Male (ref: Female)	1.12	0.73–1.73	.604	1.40	0.42–4.60	.585
Black (ref: non-Hispanic Whites)	0.30	0.08–1.19	.087	<i>Not estimable due to the small cell size</i>		
Hispanic/Other (ref: non-Hispanic Whites)	0.99	0.47–2.07	.969	1.36	0.27–6.75	.710
Education	0.97	0.78–1.21	.797	1.24	0.69–2.23	.474
Household income	1.10	0.96–1.28	.180	1.19	0.76–1.87	.435
Currently employed (ref: unemployed)	1.31	0.77–2.22	.319	1.49	0.30–7.41	.622
BMI	0.98	0.94–1.03	.507	0.86	0.73–1.02	.081
Diabetes (ref: no diabetes)	<b>1.84</b>	<b>1.14–2.96</b>	<b>.012</b>	1.04	0.26–4.12	.961
Hypertension (ref: no Hypertension)	1.10	0.70–1.74	.684	2.55	0.67–9.67	.170
Current smoker (ref: nonsmoker)	<b>2.78</b>	<b>1.34–5.77</b>	<b>.006</b>	2.16	0.22–21.30	.510
Former smoker (ref: nonsmoker)	<b>1.60</b>	<b>1.02–2.52</b>	<b>.041</b>	2.06	0.63–6.70	.231
Depressive symptoms	1.12	0.98–1.29	.089	1.12	0.78–1.60	.542
Survey month	0.95	0.81–1.12	.550	1.10	0.72–1.67	.673
Baseline sleep health	<b>1.27</b>	<b>0.98–1.63</b>	<b>.066</b>	<b>2.40</b>	<b>1.20–4.82</b>	<b>.013</b>
Change in sleep health	<b>1.27</b>	<b>1.005–1.59</b>	<b>.046</b>	<b>2.53</b>	<b>1.37–4.68</b>	<b>.003</b>

Notes: Adjusted models controlled for age, sex, race/ethnicity, education, employment status, body mass index (BMI), diabetes, hypertension, smoking status, depressive symptoms, and data collection survey month. Significant associations are bolded. 95% CI = 95% confidence intervals; HR = hazard ratios.

and heart disease mortality. These associations were found after adjusting for an extensive list of sociodemographic characteristics and known health risk factors, improving the validity of our findings. This study contributes to the literature on the health impact of sleep, by using a multidimensional sleep health composite and examining longitudinal changes in sleep health. Overall, findings point to the potential importance of promoting multidimensional sleep

health in adulthood as a novel opportunity to reduce early mortality risk.

The current study is one of the few studies that examined the association between multidimensional sleep health and mortality [but see Refs. 47, 18, and 48 for studies on older adults]. Further, our work based on longitudinal sleep data provides novel insights into how changes in sleep health over the course of aging are associated with mortality. First, we



**Figure 2.** Forest plots of the hazard ratios for mortality regressed on baseline sleep health problems and change in sleep health problems.

examined the associations between baseline sleep health problems and mortality (ie, all-cause and heart disease mortality). In these models, sleep health problems had a meaningful effect size, comparable to the well-known risk factors for mortality. For example, the estimated risk of all-cause mortality associated with one-unit greater number of sleep health problems at baseline (12% higher risk) was larger than being 1 year older (9% higher risk). In other words, the difference between having the least optimal sleep health (5 problems) and having the most optimal sleep health (0 problem) was similar to being nearly 7 years older ( $12\% \div 9\% \times 5$  units). Second, our change models allowed us to gauge the importance of within-person long-term changes in sleep health versus baseline sleep health for mortality. Our results showed that observing changes in sleep health over time may provide more information on one's mortality risk across all causes and for heart disease specific ones, compared to just one-time measurement of baseline sleep health. Each reduction in sleep health problems over time was associated with a 26% decreased risk of all-cause mortality, which can also translate into up to a 130% decrease in mortality risk (if reductions across all 5 sleep health problems). Considering that sleep problems often co-occur, improving sleep health in one dimension may bring a synergistic effect, such that improving regularity in sleep may accompany efficiency and satisfaction in sleep, each of which contributes to reducing mortality risk.

Among the sleep health dimensions, we found that short/long sleep duration and sleep inefficiency (measured by long sleep onset latency) were important sleep health dimensions that determine the risk of all-cause and heart disease-related mortality, respectively, independent of other risk factors (Supplementary Appendix Table 5). Specifically, the results with respect to sleep inefficiency are in line with previous research showing that wake-up time during main sleep is one of the most important sleep dimensions predictive of all-cause and cardiovascular mortality in older adults (47). Note that the sleep satisfaction dimension previously found important for the risk of heart disease (19) was not associated with heart disease (or all-cause) mortality in this study. We measured *dis*-satisfaction in sleep using perceived symptoms of insomnia (ie, trouble falling asleep, nocturnal awakenings, early awakenings, and unrested upon waking). Although these items

were highly loaded to the sleep quality factor in the Sleep Health Index (30), our measure might not be sufficiently sensitive to capture sleep satisfaction/quality among those who do not have insomnia symptoms but still rate their sleep quality as poor. Future research may need to consider measuring overall sleep quality (eg, "how would you rate your sleep quality overall?") or the average of day-to-day sleep quality. Moreover, note that not all the individual sleep dimensions were associated with the mortality outcomes after taking into account the associations of sociodemographic and health covariates. Given that the presence of specific sleep problems may differ across individuals, focusing only on a particular sleep dimension (eg, sleep quality only) may underestimate the effects of sleep on mortality risk and may miss an opportunity to intervene on other sleep problems important for mortality risk (eg, sleep onset latency identified in the current study). Overall, results from this study demonstrate the utility of a multidimensional sleep health approach in more comprehensively capturing its associated risk for early mortality.

Additionally, a wide range of follow-up time in this study (Range = 0.08–16 years) allowed us to identify a critical time gap since the last sleep assessment, within which one's sleep health may be predictive of mortality risk. Using a time point where there is an abrupt change in the survival curve (44,46), we identified 4-years as the change-point to use for the stratified analyses. When we used a follow-up time of 4 years or less, more sleep health problems at baseline and an increase in sleep health problems over time were associated with a higher risk of all-cause mortality. However, when we used a follow-up time greater than 4 years, baseline sleep health was not associated with all-cause mortality and the change model did not run. Further, baseline sleep health became a significant predictor of heart disease mortality when using a follow-up time of 4 years or less, whereas it was not significant when included a longer follow-up in the main analysis. These results may suggest that adults' sleep health may change over a 4-year timeframe and needs to be assessed at medical checkups and refreshed within at least 4 years to use the information as a vital sign for future mortality risk. One possibility that may explain this time window is confounding by premorbid disease. People who have serious illnesses—eg, cancer, heroin addiction, cirrhosis, end stage kidney disease—not captured by our covariates, may have poor sleep health from their major illnesses and also be at high risk of death in the short term. For those who have poor sleep health for other reasons (eg, due to work, bad lifestyle habits, and so on); however, may not have elevated mortality risk in the short term and thus their sleep (relate to nonclinical reasons) may not be predictive of mortality.

This study has limitations that may guide future research. First, our results are based on self-reported sleep variables that may not always agree with objective sleep measures (49,50). The MIDUS also collected actigraphy sleep data at M2, but the sample size and death cases were too small ( $n = 214$ ; 17 deaths, 6 heart disease-related deaths). Future work may benefit from multidimensional sleep health defined using actigraphy (51). Second, the sleep timing dimension was not captured, because the variable was not available in the MIDUS survey data. This might have contributed to underestimating the effect of sleep health on the risk of mortality, if sleep timing is an important dimension for mortality (47). Since the original conceptualization of sleep health includes the timing dimension, future research may want to incorporate sleep

timing variables (eg, bedtime, wake-up time, sleep midpoint). Third, only a small fraction of participants died of a cause related to heart disease ( $n = 191$  for baseline analyses,  $n = 14$  for change analyses). Therefore, the results may be biased due to limited cases. Research with data sets including a greater number of available events is needed to confirm the findings from the current study. Additionally, although we selected health covariates based on literature review and consultation with subject experts, the list may not be comprehensive. For example, we did not control for stroke and heart-related disease to avoid overcontrolling when examining the association of sleep health with heart disease mortality. However, some health conditions could be mediators connecting sleep health to mortality, which requires future research attention. Fourth, we used one of the new approaches to examine multidimensional sleep health—"how many" sleep health problems co-occur within an individual. There are other approaches, such as identifying different groups who have similar types of sleep health problems (18,25,52,53). Future research could explore these diverse approaches to better understand how multidimensional sleep health is associated with mortality and a variety of aging outcomes. Finally, because the MIDUS data composed mainly of non-Hispanic White participants and we did not use sample weights, our findings may not generalize to the population of midlife and older adults.

## Conclusion

This is one of the few studies that links sleep health across middle and older adulthood to mortality. Findings show that multidimensional sleep health from middle to older adulthood is a key factor with respect to time to death. Notably, the effect size of multidimensional sleep health was larger than that of age, which is a well-known risk factor for mortality. As sleep health is malleable, future efforts may need to focus on quantifying the impact of improving sleep health in adulthood on reducing morbidities and early mortality.

## Supplementary Material

Supplementary data are available at *The Journals of Gerontology, Series A: Biological Sciences and Medical Sciences* online.

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## Conflict of Interest

Outside of the current work, O.M.B. received honoraria/travel support for lectures from Boston University, Boston College, Tufts School of Dental Medicine, Eric H. Angle Society of Orthodontists, Harvard Chan School of Public Health, New York University, University of Miami, University of South Florida, University of Utah, University of Arizona, and Allstate, consulting fees from Sleep Number, and an honorarium from the National Sleep Foundation for his role as the Editor-in-Chief of Sleep Health (sleephealthjournal.org). Outside of this work, M.L.W. is a paid statistical consultant for Sleep Number Bed, Noctem Health, and Health Rhythms. M.L.W. also discloses that she receives funds as a statistical consultant for Sleep Number Bed, Health Rhythms, and Noctem Health, outside the current work. The other authors declare no conflict of interest.

## Data Availability

Data and documentation for all MIDUS projects are available to other researchers at the Interuniversity Consortium for Political and Social Research (ICPSR). In addition to the publicly available data at ICPSR, a MIDUS-Colectica Portal (midus.colectica.org) contains rich searchable metadata, links to helpful documentation, and the ability to download customized data sets. Analytic methods specific to the current study are available upon request from the corresponding author. The current study was not preregistered with an analysis plan in an independent, institutional registry.

## Author Contributions

S.L. developed the study concept and drafted the manuscript. C.X.M. analyzed the data. M.L.W. and R.A. reviewed statistical analyses and interpretation of the data. R.A., D.M.A., O.M.B., and S.R.P. contributed to the selection and conceptualization of the study variables. D.M.A. contributed to the MIDUS data collection. All coauthors provided critical revisions to the document for submission.

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