SLEEPJ, 2019, 1–9

doi: 10.1093/sleep/zsz116
Advance Access Publication Date: 13 May 2019
Original Article

ORIGINAL ARTICLE

Empirical derivation of cutoff values for the sleep health metric and its relationship to cardiometabolic morbidity: results from the Midlife in the United States (MIDUS) study

Ryan C. Brindle[1,2,*], Lan Yu[1], Daniel J. Buysse[1] and Martica H. Hall[1]

[1]Department of Psychiatry, University of Pittsburgh School of Medicine, Pittsburgh, PA and [2]Department of Psychology and Neuroscience Program, Washington and Lee University, Lexington, VA

*Corresponding author. Ryan C. Brindle, Department of Psychology and Neuroscience Program, Washington and Lee University, 204 West Washington Street, Lexington, VA 24450. Email: rbrindle@wlu.edu.

Abstract

Study Objectives: Emerging evidence supports a multidimensional perspective of sleep in the context of health. The sleep health model, and composite sleep health score, are increasingly used in research. However, specific cutoff values that differentiate "good" from "poor" sleep, have not been empirically derived and its relationship to cardiometabolic health is less-well understood. We empirically derived cutoff values for sleep health dimensions and examined the relationship between sleep health and cardiometabolic morbidity.

Methods: Participants from two independent Biomarker Studies in the MIDUS II (N = 432, 39.8% male, age = 56.92 ± 11.45) and MIDUS Refresher (N = 268, 43.7% male, age = 51.68 ± 12.70) cohorts completed a 1-week study where sleep was assessed with daily diaries and wrist actigraphy. Self-reported physician diagnoses, medication use, and blood values were used to calculate total cardiometabolic morbidity. Receiver operating characteristic (ROC) curves were generated in the MIDUS II cohort for each sleep health dimension to determine cutoff values. Using derived cutoff values, logistic regression was used to examine the relationship between sleep health scores and cardiometabolic morbidity in the MIDUS Refresher cohort, controlling for traditional risk factors.

Results: Empirically derived sleep health cutoff values aligned reasonably well to cutoff values previously published in the sleep health literature and remained robust across physical and mental health outcomes. Better sleep health was significantly associated with a lower odds of cardiometabolic morbidity (OR [95% CI] = 0.901 [0.814–0.997], p = .044).

Conclusions: These results contribute to the ongoing development of the sleep health model and add to the emerging research supporting a multidimensional perspective of sleep and health.

Statement of Significance

Despite the increasing use of the sleep health model in research, cutoff values that differentiate "good" from "poor" sleep have not been empirically derived and its relationship to cardiometabolic health is less-well understood. We empirically derived cutoff values for each sleep health dimension and examined whether sleep health related to cardiometabolic morbidity. Empirically derived sleep health cutoff values reasonably aligned to cutoff values previously published in the sleep health literature and were robust across mental and physical health outcomes. Poor sleep health was significantly associated with a greater prevalence of cardiometabolic morbidity. These results contribute to the development of the sleep health model and add supportive evidence for a multidimensional perspective of sleep.

Key words: sleep health; hypertension; diabetes; heart disease; alertness; sleep quality; sleep efficiency; sleep duration; sleeping timing; sleep regularity

Submitted: 3 October, 2018; **Revised:** 15 March, 2019

Introduction

Evidence is emerging to support the simultaneous consideration of multiple sleep parameters in the context of physical and mental health outcomes. For example, individuals with both insomnia *and* objectively assessed short sleep duration have greater disease risk than individuals suffering from insomnia or short sleep duration alone [1]. In another study, individuals with sleep apnea symptoms *and* short sleep duration or a high number of daytime naps had increased cardiovascular risk, operationalized using a variant of the Framingham risk score [2]. A multidimensional approach to analyzing the relationship between sleep and health may provide complimentary information to that gleaned from consideration of individual dimensions of sleep, alone.

The sleep health model has been described as a multidimensional sleep/wake profile that promotes physical and mental well-being [3]. Consideration of sleep as a multidimensional construct also makes intuitive sense, as individual dimensions of sleep do not occur in isolation. Based on this model, a single sleep health score is derived from measures of daytime alertness and sleep duration, timing, regularity, efficiency, and quality. These six sleep dimensions have been shown to load onto a single sleep health factor and demonstrate good construct validity [4]. Sleep health scores outperformed a single measure of sleep duration in predicting poor self-reported health in ROC analysis and are associated with physical and mental health risk in a linear, graded fashion [5, 6]. Sleep health scores also appear to be sensitive to factors, such as sex, race, education, and stress [5–7].

Despite its increasing use in research, the optimal cutoff values used to differentiate "good" and "poor" across each sleep health dimension remain to be determined. For example, "good" sleep duration has been defined as 6–8 h [3], 7–8 h [7], and 7–9 h [6] across different studies, whereas "good" sleep efficiency has been defined as a sleep latency of <30 min [6], <30 min of wake time at night (including latency and wake after sleep onset) [3–5], or a sleep efficiency of ≥85% [7] in independent reports. Whereas all published cutoff values are well-wrought and justified using a mixture of published findings and data from meta-analyses and large representative cohort studies, no study, to our knowledge, has attempted to empirically derive cutoff values for each sleep health dimension.

Aims of the current study were several-fold. First was to empirically derive cutoff values for each sleep health dimension in the sleep health model and to assess the degree to which cutoff values are robust across physical and mental health outcomes. Cutoff values are valuable for future research and clinical application only to the extent that they change little as a function of outcome. Using a second, independent sample, our second goal was to examine the degree to which sleep health, constructed using empirically derived cutoff values, associated with cardiometabolic morbidity, hypertension and diabetes. Cardiovascular diseases and metabolic disorders are among the leading causes of morbidity and mortality worldwide and carry enormous economic burden [8, 9]. As such, identification of risk factors for adverse cardiometabolic outcomes is crucial to prevention. Based on emerging evidence supporting a multidimensional sleep perspective and robust univariate associations between cardiometabolic outcomes and sleep duration [10–12], timing [13, 14], efficiency [15–17], and quality [12, 18], we hypothesized that good sleep health would be associated with a lower prevalence of cardiometabolic conditions, even after accounting for traditional cardiometabolic risk factors. Finally, we aimed to determine if the sleep health metric outperformed other more commonly used epidemiological measures such as self-reported sleep duration and latency. If sleep health is found to outperform other metrics, this would provide impetus for inclusion of sleep health measures in future studies.

Methods

Participants

Data for the current report came from participants in the MIDUS II (N = 1255) and MIDUS Refresher (N = 863) Biomarker studies. The overarching goal of the MIDUS Study is to explore behavioral and psychosocial factors that impact age-related health and illness in a representative sample of Americans. The Biomarker studies focused on the collection of physiological measures and health behaviors. The MIDUS II Biomarker study was conducted from 2004 to 2009 and recruited participants from the original MIDUS I cohort, recruited in 1994. The MIDUS Refresher study (2011–2014) recruited a national probability sample of adults with the goal of replenishing the original MIDUS I cohort. The MIDUS Refresher Biomarker Study (2012–2016) was conducted using the same methods as the MIDUS II Biomarker study so that data could be combined to strengthen cross-sectional and future longitudinal analyses. Additional details regarding the methods and samples have been published elsewhere [19, 20]. In both studies, concurrent measurement of sleep using daily diary and wrist actigraphy was undertaken at the University of Wisconsin—Madison study site only, limiting the size of each sample. Only participants having concurrent daily diary and actigraphy data were analyzed. After exclusions for other missing data, the final analytical sample was 700 (Figure 1). Approval from appropriate Institutional Review Boards was granted for this study and all participants gave informed consent before participation.

Procedure

After completion of the Biomarker Study visit, participants were invited to participate in a 7-day sleep study. Following consent, participants were sent home with instructions and study materials and given a reminder phone call by trained research staff the day before the study was to begin. Participants were instructed to wear a wrist actigraph and complete a daily sleep diary for seven consecutive days. The diaries were prelabelled with the dates upon which each participant was supposed to complete each diary entry. All studies began on a Tuesday at 0700 h and ended at the same time the following Tuesday. Participants then mailed the study materials back to the investigators in prepaid envelopes.

Daily Sleep Diary and Pittsburgh Sleep Quality Index

Participants completed the Pittsburgh Sleep Quality Index (PSQI) [21] on day 1 of the laboratory visit as part of a large questionnaire battery. For the purposes of this study, the two items concerning sleep duration and sleep latency were analyzed. The

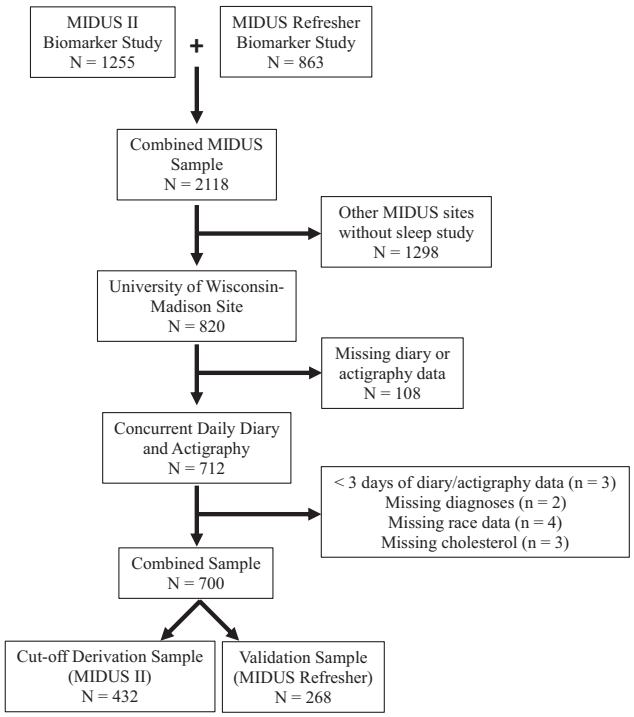

Figure 1. Participant flow chart.

daily sleep diary comprised a morning and evening section. In the morning, participants reported on their previous night's sleep. Questions included, among others, items related to timing of sleep, nighttime awakenings, sleep difficulties, and sleep quality. In the evening before going to bed, participants reported on that day's activities including daytime alertness. Diary data were entered into SPSS using a blind double entry verification system and discrepancies were adjudicated using standardized procedures.

Actigraphy

The Actiwatch system (Models 64 and 2; Philips Corporation, Andover, MA) was used for both Biomarker studies to measure activity, via motion-sensor, throughout the duration of the sleep study. Actiwatches were programmed by staff to begin collecting data at 0700 h on Tuesday and collected activity data in 30-s epochs until study conclusion. Actiware software (versions 5 or 6) was used for data analysis. A standard stepwise procedure was used to determine the rest interval. First, the rest interval was defined offline based on responses from the daily sleep diary. Second, when diary data were not available, events markers were used to define the rest interval if event markers appeared to be reliable. Third, if data were missing and event markers were found to be unreliable, rest intervals were imputed using the following rules: (1) for a missing weekday, the rest interval was imputed using at least three other adjacent weekdays, (2) for a missing weekend, the rest interval was imputed using the other weekend day [19, 20]. In the combined analytical sample (N = 700), diary data were used in 668 cases (95.4%), event markers were used in 40 cases (5.7%) and some degree of imputation was used in 32 cases (4.6%). Once the rest interval was defined, Actiware software calculated summary statistics using a wake activity threshold of 40 and an epoch length of 30 s.

Cardiometabolic outcomes

Cardiometabolic outcomes were derived from several measures including, self-reported physician diagnoses, current medication use, and blood panel values. Participants self-reported physician diagnoses of heart disease, hypertension, stroke, and diabetes and current medications during a medical history interview. Participants were first asked whether they had any of the outcomes of interest. If participants responded "yes," a follow-up question probed whether the outcome was diagnosed by a physician. For the purposes of this study, participants were only considered to have the diagnosis if it was reported to have been diagnosed by a physician. Diagnoses associated with medication use were coded using ICD9 classifications. Finally, hemoglobin A1c was measured from a fasting blood sample taken in the morning on day 2 of the laboratory visit and analyzed using an immunoturbidometric assay [19, 20]. Dichotomous outcome variables were created for each outcome (1 = diagnosis present, 0 = diagnosis absent). This coding scheme was adopted because we were interested in quantifying the degree to which good sleep health decreased risk for cardiometabolic morbidity. If a participant endorsed a physician diagnosis or reported medication use consistent with a diagnosis the diagnosis was considered present. In the case of diabetes, if a participant also had an HbA1c value greater than 6.5% [22], diabetes was considered present, even if a physician diagnosis and medication use were absent. For omnibus analysis, a dichotomous total cardiometabolic morbidity variable was created that quantified the number of participants who had any of the outcomes of interest. In follow-up analyses, hypertension and diabetes were examined individually. The low prevalence of stroke and heart disease (Table 1) in the MIDUS Refresher sample precluded individual analysis of these outcomes.

Traditional cardiometabolic risk factors

Sociodemographic and behavioral risk factors included age, sex (male/female), body mass index (BMI), race, education, smoking status, alcohol consumption, and physical activity [23, 24]. Height and weight were measured by study staff and BMI calculated as kg/m^2. It should be noted that BMI and BMI-squared were both used as covariates as reports have suggested that high and low BMI carry risk [25]. Race was coded as white and nonwhite, and education was defined along five categories: less than high school, high school diploma, some college, bachelor's degree, and graduate school/terminal degree. Smoking status was determined based upon responses to the question "Have you ever smoked regularly," coded dichotomously. Alcohol consumption over the past month was defined across four categories: never to <1 day/week, 1–2 days/week, 3–4 days/week, and 5+ days/week. Participants were dichotomized (yes/no) on physical activity based on their response to a question asking whether they engaged in at least 20 min of exercise or physical activity three times a week. As depression has been linked with increased cardiometabolic risk [26, 27], depression history, operationalized as a self-reported physician diagnosis of depression was used as a covariate. Depression history was used instead of

Table 1. Sample characteristics

Variable	MIDUS II (*n* = 432)	MIDUS refresher (*n* = 268)	*P*
Age, *M* (*SD*) years	56.92 (11.5)	51.68 (12.7)	<.001*
Age, range years	35–85	26–77	
Sex, *n*, % male	172, 39.8%	117, 43.7%	.316†
Race, *n*, %	White = 299, 69.2%	White = 175, 65.3%	.282†
	Black = 119, 27.5%	Black = 72, 26.9%	
	Other‡ = 14, 3.2%	Other‡ = 21, 7.8%	
Education, *n*, %	<HS = 37, 8.6%	<HS = 19, 7.1%	.025†
	HS graduate= 100, 23.1%	HS graduate = 37, 13.8%	
	Some college = 126, 29.2%	Some college = 87, 32.1%	
	College graduate = 82, 19.0%	College graduate = 66, 24.4%	
	Postgraduate = 87, 20.1%	Postgraduate = 59, 21.8%	
BMI, kg/m^2	30.64 (7.3)	31.32 (8.3)	.252*
Cholesterol, mg/dL	183.6 (39.3)	179.0 (45.0)	.153*
Exercise	310, 71.8%	159, 59.3%	.001†
20 min/3× per week, *n*, %yes			
Depression Dx, *n*, %yes	72, 16.8%	56, 20.9%	.177†
Days consuming alcohol			.400†
Never–<1 day/week	281, 65.0%	152, 56.7%	
1–4 days/week	114, 26.4%	89, 33.2%	
5–7days/week	37, 8.6%	24, 9.0%	
Smoking status, *n*, %yes	214, 49.5%	102, 38.1%	.003†
Cardiometabolic morbidity			
Hypertension, *n*, %yes	177, 41.0%	113, 42.2%	.756†
Heart disease, *n*, %yes	47, 10.9%	21, 7.8%	.186†
Diabetes, *n*, %yes	101, 23.4%	58, 21.6%	.594†
Stroke, *n*, %yes	26, 6.0%	4, 1.5%	.004†
Total morbidity, *n*, %yes	227, 52.5%	131, 48.9%	.346†
Sleep characteristics			
Daytime alertness	2.03 (0.74)	2.05 (0.73)	.777*
Quality	2.41 (0.78)	2.37 (0.79)	.510*
Timing	192.67 (108.9)	192.00 (73.7)	.929*
Regularity	51.26 (48.7)	52.32 (34.6)	.755*
Efficiency	79.31 (10.6)	79.55 (11.1)	.780*
Duration	369.07 (66.9)	371.1 (67.8)	.697*

HS = high school.

ANOVA and chi-square analyses are denoted by * and †, respectively. Chi-square analysis for race was conducted using the dichotomous coding scheme described in the text.

‡Includes participants endorsing Native American, Alaskan/Eskimo, Asian, Hawaiian/Pacific Island, or other ethnic backgrounds.

self-report measures because it was more strongly associated with out outcomes of interest. Finally, total cholesterol from a fasting blood sample was considered a biological risk factor.

Sleep health construction

The sleep health model holds that sleep is multidimensional and comprises six dimensions: daytime alertness and sleep quality, timing, regularity, efficiency, and duration. Dimensions were derived from self-report and actigraphy data. Daytime alertness and sleep quality were self-reported on a 5-point Likert-type scale in daily sleep diaries. Anchor words for alertness and sleep quality were 1 (most alert) to 5 (not at all alert) and 1 (very good) to 5 (very poor), respectively. Sleep timing was operationalized as the actigraphy-derived sleep midpoint, occurring midway between sleep onset and offset. The variability in sleep midpoint across the week, quantified using standard deviation, represented sleep regularity for each participant. Finally, standard measures of sleep efficiency and duration were quantified using actigraphy data.

Analytic strategy

The larger MIDUS II sample was used to derive cutoff values. Receiver operating characteristic (ROC) curves were generated for each sleep health variable to assess sensitivity, specificity, and area under the curve (AUC). For all six sleep health dimensions, average values for the week were calculated from daily values to achieve measures of habitual sleep. The primary outcome measure was total cardiometabolic morbidity and cutoff values from this ROC analysis were carried over to future analyses. However, to assess the degree to which cutoff values varied as a function of outcome, supplementary ROC analyses using hypertension, diabetes, and self-reported physician diagnosis of depression as outcome variables were undertaken. Sensitivity was the probability of classifying a participant with a cardiometabolic disorder correctly, and specificity represented the probability of correctly identifying a participant without a cardiometabolic disorder. Area under the curve represented the overall accuracy of classifying participants, with higher values indicating greater accuracy. The Youden index [28] was calculated for each sleep health dimension to determine optimal

cutoff values. The Youden index aims to maximize combined sensitivity and specificity and has a range of 0 to 1, with greater values indicating better fit. For cases where a U-shaped relationship between predictor and cardiometabolic morbidity was expected (e.g. sleep duration and timing), predictor data were median-centered and squared to achieve linearity with the outcome variable. Nonlinear predictors have been shown to violate the binormal assumption of ROC analysis and produce sigmoidal ROC curves that tend to be biased toward null associations. Transforming the predictor by first median-centering and then squaring has been shown to achieve linearity with outcome variables, conform with the binormal assumption of ROC analysis, produce a typical concave-down ROC curve, and significantly reduce bias [29].

In the MIDUS Refresher sample, sleep health scores were calculated using cutoff values derived from the MIDUS II sample. All sleep health dimensions were first centered using the respective cutoff value, producing a new variable that could be interpreted as "distance" from the cutoff value. Next, all sleep health dimensions were transformed to z-scores to achieve a uniform scale across the dimensions. Z-scores were coded such that greater values indicated better sleep health. Finally, all six of the individual sleep health dimensions z-scores were summed, creating a total sleep health score.

Correlation analysis and analysis of variance (ANOVA) were used to test the relationship between sleep health scores and cardiometabolic risk factors. Interrelationships between the continuous measures of the sleep health dimensions were tested using Spearman's correlation. Logistic regression was used to examine the relationship between sleep health and cardiometabolic diagnoses. First, an unadjusted model was tested with sleep health predicting cardiometabolic diagnoses. Next, age, sex, race, education, smoking status, alcohol consumption, BMI, BMI [2], cholesterol, depression history, and physical activity were added as covariates to determine the extent to which sleep health predicted cardiometabolic diagnoses, controlling for traditional risk factors.

To determine whether sleep health outperformed other more commonly used sleep metrics several analyses were undertaken. First, logistic regression was used to examine the relationship between each individual sleep health dimension and cardiometabolic diagnoses, controlling for traditional risk factors. Second, logistic regression was used to determine if measures of sleep duration or latency (in 10 min bins) from the PSQI were associated with cardiometabolic morbidity. Finally, area under the curve from ROC analyses using cardiometabolic morbidity as the outcome and sleep health, PSQI sleep duration, or PSQI sleep latency as predictors were compared using the method outlined by DeLong *et al.* [30]. Analyses were carried out using SPSS version 25 (IBM Analytics, New York, NY).

Results

Participants

Participant characteristics for both samples are shown in Table 1. Overall, samples were comparable, with the exception of age, education, exercise, and number of smoking participants. The MIDUS Refresher sample was significantly younger, tended to be more highly educated, exercise less, and had fewer smokers. There was no significant difference between the groups in cardiometabolic disease prevalence or sleep health dimensions.

Sleep Health Dimension Cutoff Values

Table 2 displays the ROC results and cutoff scores for each sleep health dimension derived from the MIDUS II sample. Table 2 also shows the percentage of participants falling above/below each cutoff value in the MIDUS Refresher sample. "Good" self-reported daytime alertness and sleep quality were indicated by scores of less than 2.2 and 2.8, respectively, on a 5-point Likert-type scale where smaller numbers indicated increasingly positive impressions. A time window between 2:24 and 3:30 am

Table 2. Sleep health cutoff values

Component	Source	Cutoff value (derived from MIDUS II)	MIDUS refresher sample				
			Good (% *n*)	Poor (% *n*)	Area (*SE*)	Sensitivity	Specificity
Daytime alertness	Daily sleep diary question 1 "How alert were you today" 1 (most alert) to 5 (not at all alert)	<2.2 = good ≥2.2 = poor	61.9	38.1	0.55 (0.03)	0.42	0.67
Quality	Daily sleep diary question 20 "Overall quality of your sleep last night" 1 (very good) to 5 (very poor)	<2.8 = good ≥2.8 = poor	70.5	29.5	0.54 (0.03)	0.35	0.74
Timing	Average actigraphy derived sleep midpoint	2:24 am – 3:30 am = good <2:24 am = poor >3:30 am = poor	42.5	57.5	0.57 (0.03)	0.66	0.50
Regularity	Standard deviation of actigraphy derived sleep midpoint	<1 h 5 min = good ≥1 h 5 min = poor	76.5	23.5	0.52 (0.03)	0.24	0.84
Efficiency	Average actigraphy calculated sleep efficiency	>83.0 = good ≤83.0% = poor	45.5	54.5	0.64 (0.03)	0.69	0.56
Duration	Average actigraphy calculated sleep duration	5 h 20 min to 7 h 6 min = good <5 h 20 min = poor >7 h 6 min = poor	60.4	39.6	0.55 (0.03)	0.48	0.67

emerged as an indicator of "good" sleep timing and a sleep regularity score of approximately 1 h (or less) indicated "good" sleep regularity. A score of greater than 83% differentiated "good" from "poor" sleepers on the sleep efficiency dimension and sleep durations between 5 h 20 min and 7 h 6 min per night was considered "good." Whereas an increasing positive total sleep health score indicates better sleep health, and an increasing negative score, poor sleep health, the average sleep health score was 0.20 (3.37) with a range of 5.52 to –14.82. Approximately 60% of the sample obtained sleep health scores above zero, indicating a net balance of "good" sleep health.

Supplementary analyses using hypertension, diabetes, and depression as outcome variables, in general, showed the cutoff values to be relatively robust to variations in outcome (Supplementary Table 1). The cutoff value for sleep quality and sleep efficiency varied little across outcomes, between 2.6 and 2.8 and 78%–83% for quality and efficiency, respectively. Across outcomes, sleep regularity ranged from approximately 40 to 65 min, while time window reflecting "good" sleep timing was consistently between 2:30 and 3:30, with the exception of diabetes, where the window was wider. The cutoff values for daytime alertness ranged from 1.8 to 3.1 and the duration constituting "good" sleep duration varied the most from its widest at 4 h 23 min–7 h 58 min for diabetes to 5 h 20 min–7 h 6 min at its narrowest for total cardiometabolic morbidity.

Strong correlations emerged between the self-reported sleep health dimensions of alertness and sleep satisfaction (r = .66, p < .001). All other interdimension relationships were smaller in magnitude (r-range: .31 to –.001; Supplementary Table 2). Sleep health was significantly related to age (r = .16, p = .008), as age increased, sleep health scores increased (i.e. better sleep health). Importantly, sleep health cutoff scores were similar when compared across "younger" (n = 209; <56 years) and "older" (n = 223; ≥56 years) subgroups using a median split (Supplementary Table 3). Sleep health scores were significantly higher (i.e. better sleep health) in white participants, $F(1, 266) = 26.05$, $p < .001$, $\eta^2 = .089$, nonsmokers, $F(1, 266) = 7.10$, $p = .008$, $\eta^2 = .026$, consumers of alcohol at least 1 day a week, $F(2, 265) = 4.73$, $p = .01$, $\eta^2 = .034$, those without a diagnosis of depression, $F(1, 266) = 5.99$, $p = .015$, $\eta^2 = .022$, and those with higher education, $F(4, 263) = 3.46$, $p = .009$, $\eta^2 = .050$. Sleep health did not significantly vary as a function of sex (p = .46), exercise frequency (p = .23), BMI (r = –.08, p = .20), or cholesterol (r = .03, p = .67).

Sleep health and cardiometabolic morbidity

In unadjusted analyses, better sleep health was significantly associated with lower odds of total cardiometabolic morbidity or hypertension alone (Table 3). In analyses adjusted for sex, age, education, race, BMI, smoking status, cholesterol, depression, alcohol consumption, and physical activity, better sleep health remained significantly associated with lower odds of cardiometabolic morbidity, OR [95% CI] = 0.901 [0.814–0.997], p = .043 and hypertension, OR [95% CI] = 0.903 [0.817–0.997], p = .044. No significant association between sleep health and diabetes emerged.

Analyses concerned with determining if sleep health outperformed other commonly used sleep measures were, on the whole, inconclusive. In general, individual sleep health dimensions did not relate to cardiometabolic morbidity, with the exceptions of sleep duration and sleep regularity which related to cardiometabolic morbidity (p = .041) and hypertension (p = .010; Figure 2), respectively. The PSQI measure of sleep duration and latency were not significantly associated with any cardiometabolic outcome (all $p \geq .20$; Figure 2). Comparison of ROC-derived AUC values from models using sleep health, PSQI sleep duration, and PSQI sleep latency as predictors also failed to reveal any significant differences (all $p \geq .20$).

Discussion

Emerging evidence supports the use of multidimensional sleep measures in the context of health and disease. Sleep health, a multidimensional sleep/wake profile from which a multidimensional sleep health score can be derived, has been used [3]. However, appropriate cutoff values to differentiate "good" and "poor" sleep, have yet to be empirically derived. As such, the primary goal of the present study was to empirically derive cutoff values for each of the six dimensions comprising the sleep health score. In a second, independent sample, we aimed to examine the cross-sectional relationship between sleep health and cardiometabolic morbidity using the empirically derived cutoff values. Cutoff values emerged that reasonably aligned to cutoff values already published in the sleep health literature. Moreover, these cutoff values were robust to changes in outcome, suggesting they represent reliable boundaries across which dysregulated sleep confers risk. Better sleep

Table 3. Sleep health predicting self-reported physician DXs (MIDUS refresher; N= 268)

Outcome	OR	95% CI	P
Unadjusted models			
Total cardiometabolic morbidity	0.909	0.844–0.980	.014
Hypertension	0.926	0.860–0.996	.038
Diabetes	0.919	0.847–0.997	.041
Adjusted model: Sex, age, education, race, BMI, smoking status, cholesterol, depression, alcohol, and physical activity			
Total cardiometabolic morbidity	0.901	0.814–0.997	.043
Hypertension	0.903	0.817–0.997	.044
Diabetes	0.968	0.869–1.078	.548

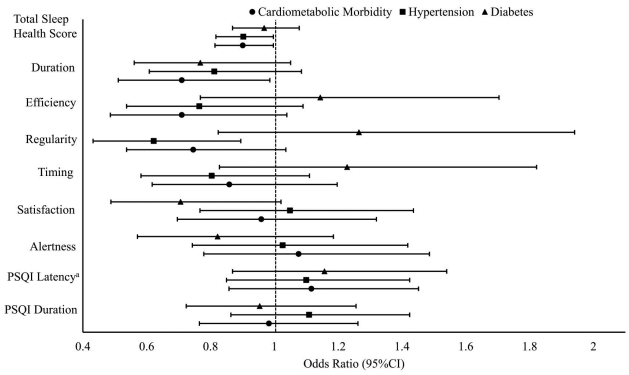

Figure 2. Logistic regression results in MIDUS Refresher cohort (N = 268). Scores for sleep health and sleep health components were computed using empirically derived cutoff values from the MIDUS II cohort. Individual regression models were run for each sleep health component. Odds ratios (95% CI) are adjusted for sex, age, BMI, BMI^2, education, race, smoking status, alcohol consumption, physical activity, cholesterol, and depression history. [a]Sleep latency in 10 min bins.

health was significantly associated with reduced odds of reporting cardiometabolic morbidity and hypertension, even after extensively controlling for traditional sociodemographic and biological risk factors. These results bolster published data examining the sleep health model and add to an emerging body of research considering the association of multidimensional sleep profiles with health.

The cutoff values derived in the present study reasonably aligned to already published cutoff values. This finding is not surprising, given that previously published cutoff scores have most often been based on distributions of previously published cohort data, meta-analyses, and reviews. Our finding that 2:24–3:30 am defines "good" sleep timing aligns closely with the originally published cutoff of 2–4 am [3–5] and the subsequently published cutoffs of 2:27–3:38 am and 2:41–3:54 am for males and females [7], respectively. If anything, our results suggest that a smaller, 1.5–1 h, time window may be most appropriate for defining "good" sleep timing. Despite the small time window, sleep timing was the sleep health dimension with the greatest percentage of participants falling in the "poor" category. Previous studies have used 30 min as a cutoff value to define sleep regularity [7]. In other words, sleep regularity can be considered "good" if sleep midpoint varies by less than 30 min, on average, across a given number of nights. Our results suggest that 65 min is an appropriate cutoff value for differentiating "good" and "poor" sleep regularity. Although this window is wider than previously published values, 65 min is approximately the width of the time window we found appropriate to define sleep timing. We found that a sleep efficiency of 83% corresponded most closely with a previously published study of childhood trauma and sleep health, where a value of 85% was used, based on the clinical goal of achieving 85% sleep efficiency in those receiving treatment for insomnia [7]. Other studies have used sleep latency and wake after sleep onset to operationalize the sleep efficiency dimension of sleep health. In these studies, less than 30 min of sleep latency [6] or combined sleep latency and wake after sleep onset [3–5] is considered to be consistent with "good" sleep continuity.

The cutoff values for sleep duration were notably shorter than previous reports. Our results indicated that 5 h 20 min–7 h 6 min represented "good" sleep duration whereas other studies have used values of 7–8 h [7], 6–8 h [3–5], and 7–9 h [6]. However, actigraphy was used to measure sleep in the present study, while sleep durations were self-reported in prior studies of sleep health. Thus, methodological differences may account for these somewhat discrepant results. Methods used to quantify subjective reports of alertness and satisfaction vary widely across studies, making comparisons difficult. Whereas the current study and one other [6] used a 5-point Likert type scale, some studies have used a 0–2 Likert type scale [3–5] or a 100-point visual analog scale [7]. Still others have used standardized questionnaires such as the Epworth Sleepiness Scale and its cutoff value of 10 to operationalize alertness.

Several factors must be considered when considering the validity of these cutoff values. First, these cutoff values were also derived using actigraphy (except subjective alertness and quality). As actigraphy and subjective measures of sleep capture somewhat different bands of variance [31] it is possible that different cutoff values will arise as a function of methodology. Second, the sample was comprised of midlife adults from a limited region of the United States. The degree to which these values will generalize across diverse samples in unknown. The values reported here represent the first attempt to *empirically* derive a set of clinically relevant sleep health cutoff values. The finding that cutoff values were relatively robust across physical and mental health outcomes is encouraging and suggests that these values may represent a reliable boundary across which dysregulated sleep may confer sleep. Future research will determine the degree to which these values will remain robust across sleep recording methodologies and populations.

The finding that greater sleep health was associated with lower odds of reporting cardiometabolic morbidity adds to the emerging findings relating poor sleep health to clinically significant symptoms of depression [6] and poor self-reported health [4, 5]. Our results showing that sleep health was associated with cardiometabolic morbidity and hypertension in a second sample, independent from that used to derive the cutoff values demonstrates that the cutoff values generalize across samples and, to some extent, across cardiometabolic disorders. The composite measure of sleep health appears to represent a much more precise measure of sleep than individual dimensions, vis-à-vis cardiometabolic health, as indicated by the notably smaller confidence intervals for sleep health in Figure 2, compared with those surrounding individual dimensions of sleep health. Such increased precision provides reason to continue pursuing a multidimensional approach to evaluating relationships among sleep and health.

Several limitations deserve mention. First, we were unable to demonstrate, conclusively, that sleep health outperformed other measures of sleep commonly used in epidemiological research. Whereas comparisons of AUC yielded no significant differences among sleep health and PSQI measure of sleep duration and latency, sleep health was the only measure that significantly related to cardiometabolic risk, even after controlling for sociodemographic and biological risk factors. Given that sleep health has been previously shown to outperform single dimension measures [5], it may be the case that with further development sleep health will outperform traditional measures. For example, individual sleep health dimensions were weighted equally in the present study. It is reasonable to speculate that a sleep health score constructed using differentially weighted sleep health dimensions might achieve greater precision. Second, due to the cross-sectional structure of the study design, conclusions cannot be drawn regarding the causal role of sleep health in the pathophysiology or clinical course of cardiometabolic disorders. Experimental sleep restriction and deprivation studies have reported changes in physiological function (e.g. sympathetic activity, glucose metabolism, hormone section) and health behaviors (e.g. eating) that are consistent with disturbed sleep playing a causal role in the development of cardiometabolic disorders [32, 33]. Experimental and longitudinal research is still needed, however, to explore of the role of different sleep dimensions in the pathophysiology and progression of cardiometabolic disease. Third, no significant relationship between sleep health and diabetes was observed in models that adjusted for traditional cardiometabolic disease risk factors. Given previous research linking poor sleep to diabetes [12], it is possible that the present study lacked the statistical power to detect this relationship, especially since diabetes prevalence was low in the MIDUS Refresher sample. Fourth, in contrast with some [4, 5], but not all reports [34], individuals reporting better sleep health were older than their counterparts with poor sleep health. This may be the result of older individuals having

fewer sleep constraints than younger individuals. Alternatively, it is possible that a survivorship-type bias may be responsible for such a discrepant result. If so, this would lead to an underestimation of the true relationship between sleep health and cardiometabolic morbidity.

To conclude, using two independent samples, the present study empirically derived cutoff values for the six individual dimensions of sleep that comprise sleep health and showed that they are relatively stable across physical and mental health outcomes. In a separate sample, good sleep health was cross-sectionally associated with reduced odds of cardiometabolic morbidity and hypertension. These results contribute to the ongoing development of the sleep health model and add to the emerging research supporting a multidimensional perspective of sleep and health.

Supplementary material

Supplementary data are available at *SLEEP* online.

Acknowledgments

The authors would like to thank all the research staff and participants of the MIDUS Study. The authors are also grateful for the opportunity to use these publically available data.

Disclosure Statement

RCB and MHH have no financial relationships relevant to this article to disclose. DJB is a consultant for BeHealth Solutions, CME Institute, and Emmi Solutions.
Drs Brindle, Buysse, and Hall report grants from the National Institutes of Health.

Funding Source

Since 1995 the MIDUS study has been funded by the following: John D. and Catherine T. MacArthur Foundation Research Network, National Institute on Aging (P01-AG020166), National Institute on Aging (U19-AG051426). Biomarker data collection was further supported by the National Institutes of Health National Center for Advancing Translational Sciences (NCATS) Clinical and Translational Science Award (CTSA) program as follows: UL1TR001409 (Georgetown), UL1TR001881 (UCLA), 1UL1RR025011 (UW). Investigator support came from the National Institutes of Health (F32 HL137227 to RCB and R01 AG047139 to MHH and DJB).

References

1. Vgontzas AN, *et al*. Insomnia with objective short sleep duration: the most biologically severe phenotype of the disorder. *Sleep Med Rev*. 2013;**17**(4):241–254.
2. Buxton OM, *et al*. Sleep health and predicted cardiometabolic risk scores in employed adults from two industries. *J Clin Sleep Med*. 2018;**14**(3):371–383.
3. Buysse DJ. Sleep health: can we define it? Does it matter? *Sleep*. 2014;**37**(1):9–17.
4. Becker NB, *et al*. Sleep health assessment: a scale validation. *Psychiatry Res*. 2018;**259**:51–55.
5. Dalmases M, *et al*. Assessing sleep health in a European population: results of the Catalan Health Survey 2015. *PLoS One*. 2018;**13**:e0194495.
6. Furihata R, *et al*. An aggregate measure of sleep health is associated with prevalent and incident clinically significant depression symptoms among community-dwelling older women. *Sleep*. 2017;**40**:zsw075.
7. Brindle RC, *et al*. The relationship between childhood trauma and poor sleep health in adulthood. *Psychosom Med*. 2018;**80**:200–207.
8. Roth GA, *et al*. Global and regional patterns of cardiovascular disease mortality from 1990–2013. *Circulation*. 2015;**132**:1667–1678.
9. World Health Organization. Global Report on Diabetes. http://www.who.int/diabetes/global-report/en/. Published April, 2016. Accessed June 11, 2018.
10. Guo X, *et al*. Epidemiological evidence for the link between sleep duration and high blood pressure: a systematic review and meta-analysis. *Sleep Med*. 2013;**14**:324–332.
11. Cappuccio FP, *et al*. Sleep duration predicts cardiovascular outcomes: a systematic review and meta-analysis of prospective studies. *Eur Heart J*. 2011;**32**(12):1484–1492.
12. Cappuccio FP, *et al*. Quantity and quality of sleep and incidence of type 2 diabetes: a systematic review and meta-analysis. *Diabetes Care*. 2010;**33**:414–420.
13. Knutson KL, *et al*. Association between sleep timing, obesity, diabetes: the Hispanic Community Health Study/Study of Latinos (HCHS/SOL) Cohort Study. *Sleep*. 2017;**40**. doi:10.1093/sleep/zsx014
14. Bøggild H, *et al*. Shift work, risk factors and cardiovascular disease. *Scand J Work Environ Health*. 1999;**25**(2):85–99.
15. Hoevenaar-Blom MP, *et al*. Sleep duration and sleep quality in relation to 12-year cardiovascular disease incidence: the MORGEN study. *Sleep*. 2011;**34**(11):1487–1492.
16. Knutson KL, *et al*. Cross-sectional associations between measures of sleep and markers of glucose metabolism among subjects with and without diabetes: the coronary artery risk development in Young Adults (CARDIA) Sleep Study. *Diabetes Care*. 2011;**34**(5):1171–1176.
17. Trento M, *et al*. Sleep abnormalities in type 2 diabetes may be associated with glycemic control. *Acta Diabetol*. 2008;**45**(4):225–229.
18. Javaheri S, *et al*. Sleep quality and elevated blood pressure in adolescents. *Circulation*. 2008;**118**(10):1034–1040.
19. Ryff CD, *et al*. *Midlife in the United States (MIDUS 2): Biomarker Project, 2004–2009*. Ann Arbor, MI: Inter-university Consortium for Political and Social Research [distributor]; 2017. https://www.icpsr.umich.edu/icpsrweb/ICPSR/studies/29282. Accessed June 6, 2016.
20. Weinstein M, *et al*. *Midlife in the United States (MIDUS Refresher): Biomarker Project, 2012–2016*. Ann Arbor, MI: Inter-university Consortium for Political and Social Research [distributor]; 2018. https://www.icpsr.umich.edu/icpsrweb/ICPSR/studies/36901. Accessed June 6, 2016.
21. Buysse DJ, *et al*. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res*. 1989;**28**(2):193–213.
22. World Health Organization. Use of glycated haemoglobin (HbA1c) in the diagnosis of diabetes mellitus; 2011. https://www.who.int/diabetes/publications/report-hba1c_2011.pdf?ua=1. Accessed February 21, 2018.

23. US Burden of Disease Collaborators. The state of US health, 1990–2010: burden of disease, injuries, and risk factors. *JAMA*. 2013;**310**:591–608.
24. Yusuf S, *et al*. Global burden of cardiovascular diseases: part I: general considerations, the epidemiologic transition, risk factors, and impact of urbanization. *Circulation*. 2001;**104**(22):2746–2753.
25. Park D, *et al*. Underweight: another risk factor for cardiovascular disease? A cross-sectional 2013 Behavioral Risk Factor Surveillance System (BRFSS) study of 491,733 individuals in the USA. *Medicine*. 2017;**96**:e8769.
26. Andreoulakis E, *et al*. Depression in diabetes mellitus: a comprehensive review. *Hippokratia*. 2012;**16**(3):205–214.
27. Hare DL, *et al*. Depression and cardiovascular disease: a clinical review. *Eur Heart J*. 2014;**35**(21):1365–1372.
28. Youden WJ. Index for rating diagnostic tests. *Cancer*. 1950;**3**(1):32–35.
29. Ho KM. Effect of non-linearity of a predictor on the shape and magnitude of its receiver-operating-characteristic curve in predicting a binary outcome. *Sci Rep*. 2017;**7**(1):10155.
30. DeLong ER, *et al*. Comparing the areas under two or more correlated receiver operating characteristic curves: a nonparametric approach. *Biometrics*. 1988;**44**(3):837–845.
31. Lauderdale DS, *et al*. Self-reported and measured sleep duration: how similar are they? *Epidemiology*. 2008;**19**(6):838–845.
32. Tobaldini E, *et al*. Sleep, sleep deprivation, autonomic nervous system and cardiovascular diseases. *Neurosci Biobehav Rev*. 2017;**74**(Pt B):321–329.
33. Morselli LL, *et al*. Sleep and metabolic function. *Pflugers Arch*. 2012;**463**(1):139–160.
34. Grandner MA, *et al*. Age and sleep disturbances among American men and women: data from the U.S. Behavioral Risk Factor Surveillance System. *Sleep*. 2012;**35**(3):395–406.