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Original Contribution

Associations of Sleep Characteristics With Cognitive Function and Decline Among Older Adults

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Sleep laboratory studies find that restricted sleep duration leads to worse short-term cognition, especially memory. Observational studies find associations between self-reported sleep duration or quality and cognitive function. However self-reported sleep characteristics might not be highly accurate, and misreporting could relate to cognition. In the Sleep Study of the National Social Life, Health, and Aging Project (NSHAP), a nationally representative cohort of older US adults (2010–2015), we examined whether self-reported and actigraph-measured sleep are associated with cross-sectional cognitive function and 5-year cognitive decline. Cognition was measured with the survey adaptation of the multidimensional Montreal Cognitive Assessment (MoCA-SA). At baseline ($n = 759$), average MoCA-SA score was 14.1 (standard deviation, 3.6) points of a possible 20. In cross-sectional models, actigraphic sleep-disruption measures (wake after sleep onset, fragmentation, percentage sleep, and wake bouts) were associated with worse cognition. Sleep disruption measures were standardized, and estimates of association were similar (range, −0.37 to −0.59 MoCA-SA point per standard deviation of disruption). Actigraphic sleep-disruption measures were also associated with odds of 5-year cognitive decline (4 or more points), with wake after sleep onset having the strongest association (odds ratio = 1.43, 95% confidence interval: 1.04, 1.98). Longitudinal associations were generally stronger for men than for women. Self-reported sleep showed little association with cognitive function.

actigraphy; cognition; cohort study; sleep

Abbreviations: MCI, mild cognitive impairment; MMSE, Mini-Mental State Examination; MoCA-SA, Montreal Cognitive Assessment survey adaptation; MrOS, Osteoporotic Fractures in Men; NSHAP, National Social Life, Health, and Aging Project; SOF, Study of Osteoporotic Fractures; TST, total sleep time; WASO, wake after sleep onset.

Observational and experimental studies have found associations between sleep and cognitive function. The strongest and most consistent findings have been in experimental studies demonstrating a clear relationship between restricted sleep in a laboratory setting and next-day short-term performance in memory-related tasks (1–5). Laboratory-based studies have also demonstrated relationships between sleep deprivation and other cognitive domains, including language, visuospatial ability, and decision-making, although findings for these have been less consistent (Alhola and Polo-Kantola (6), review).

Sleep manipulated in laboratory environments differs by design from home sleep patterns, and there might be systematic differences between laboratory study volunteers—who are generally healthy and young—and paid adults in the community. Observational studies also demonstrate relationships between self-reported sleep characteristics among community-dwelling individuals and cognitive function (7–10). However, survey responses on sleep duration have low to moderate correlation with sleep characteristics objectively estimated by either polysomnography or wrist actigraphy (11–13). Reporting sleep duration could be cognitively challenging, because accurate answers require determining usual bedtime and waking time, which can have daily variation, and performing mental arithmetic, often around midnight. Additionally, inaccurate reporting has been linked to health determinants, including socioeconomic indicators, raising the possibility that associations between self-reported sleep and health outcomes could be biased (12).

To address limitations of self-reported sleep characteristics, a few cohorts have added objective measures. For research about

cognitive function, older adults are the population of greatest interest because they have the highest risk of cognitive decline and report worse sleep (14). Two cohorts objectively measuring sleep have found associations between actigraphic indicators of poor sleep and cognitive impairment: the women-only Study of Osteoporotic Fractures (SOF) study and the men-only Osteoporotic Fractures in Men (MrOS) study (15–18). Findings differed somewhat between the studies, suggestive of possible sex differences in the sleep-cognition relationship. Both cohorts used the Mini-Mental State Examination (MMSE) and the Trail Making Test Part B as measures of cognitive function. The Trail Making Test Part B is a test of executive function, and the MMSE is a widely used screening test for severe cognitive impairment and dementia, but has it has low sensitivity for mild cognitive impairment (MCI) or tracking moderate changes (19–21).

In this study, we used data from a nationally representative cohort study of older adults that included a multidomain cognitive assessment sensitive to MCI to assess: 1) cross-sectional associations of actigraph-measured and self-reported sleep characteristics with cognitive function; and 2) longitudinal associations between sleep and 5-year decline in cognitive function. We examined whether associations differed for actigraphy and survey measures and investigated interactions by sex.

METHODS

Study population

The National Social Life, Health, and Aging Project (NSHAP) is a nationally representative study of community-dwelling, older US adults born between 1920 and 1947 that has fielded 3 waves: in 2005/2006, 2010/2011, and 2015/2016. In wave 2, spouses and coresident partners of original cohort members were invited to participate. Each wave included in-home interviews and biomeasures. The NSHAP Sleep Study is a substudy that began in wave 2. A randomly selected one-third of wave 2 respondents ($n = 1{,}117$) were asked to participate, wearing a wrist actigraph for 72 hours (3 nights) and answering additional questions in a booklet. Those alive 5 years later were recontacted as part of wave 3. Here, we refer to the wave 2 Sleep Study as "baseline" and wave 3 as "follow-up."

Selected cohort members were asked if they would participate in the Sleep Study during the in-home interview, but the protocol required that they be recontacted to arrange delivery of materials. Of 1,117 respondents who were asked to participate, 897 initially agreed. Among them, 823 were successfully recontacted in the available timeframe, and 801 returned usable sleep data (returning a wrist actigraph with recorded data or a sleep booklet with responses; see Figure 1 for a flow chart).

We included participants who had at least 1 night of actigraphic data. This analysis is limited to those born between 1920 and 1947 ($n = 759$) (Figure 1), the initial birth-year range. Agreement to participate in the Sleep Study and return of actigraphy data were not themselves related to cognitive function (22).

Of those with sleep data, 24.2% were not interviewed again at the 5-year follow-up due to death, poor health, or other reasons (Figure 1). Thus, cross-sectional results include 759 participants and longitudinal results include 555.

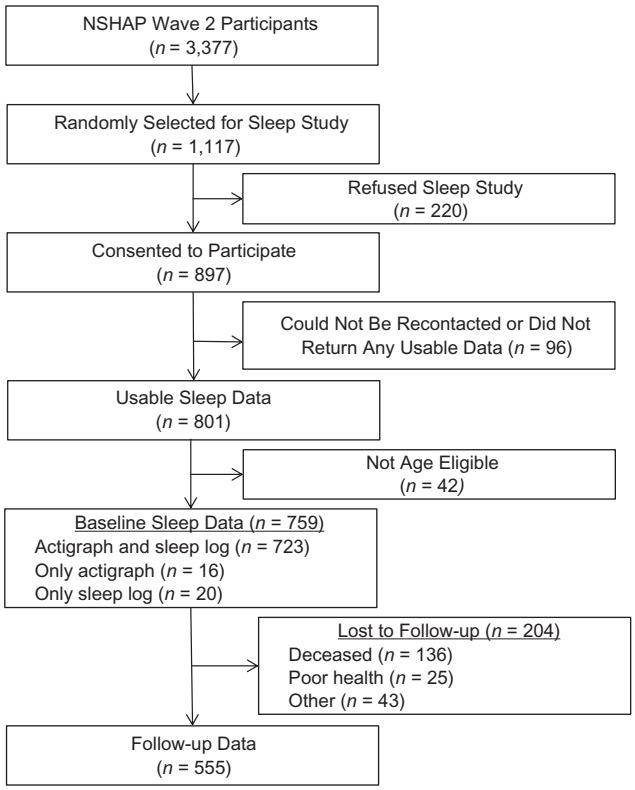

Figure 1. Flow chart of participants in the National Health, Social Life and Aging Project (NSHAP) Sleep Study, United States, 2010–2015. Among those who returned wrist actigraphs with usable data, 93% recorded 3 nights of sleep. Another 6% recorded 2 nights, and 1% recorded 1 night. We included all participants who had any actigraphy data.

Wrist actigraphy

Collection of the sleep data has been fully described elsewhere (23). Participants were instructed to wear the wrist actigraph (Actiwatch Spectrum; Philips Respironics, Murrysville, Pennsylvania) for 72 hours. The Actiwatch was set to record activity data in 15-second epochs. When the device was returned, data were downloaded and analyzed using Philips Respironics software (version 5.59) and the validated settings (23).

A participant's rest intervals were first set by the software, based only on the activity pattern. Then each record was reviewed by the investigators, and rest intervals were revised based on additional information, that the software did not use: the participant-initiated event marker time stamp that they were asked to press at each bedtime and waking time and the light sensor on the actigraph. Overall, the event marker was pressed 84% of the total nights analyzed (23). The software scores each 15-second epoch as sleep or not based on activity counts in that epoch and surrounding epochs. The sleep interval is the period within each rest interval beginning with the first epoch scored as sleep and ending with the last epoch scored as sleep.

Sleep measures used in this study are all calculated within the sleep interval: total sleep time (TST: summed duration of all epochs scored as sleep), wake after sleep onset (WASO:

summed duration of all epochs scored as wake), sleep fragmentation (the sum of the percentage of epochs with any motion, which might or might not be scored as sleep, and the percentage of immobile periods less than 1 minute long), percentage sleep (TST divided by the sleep interval), and number of wake bouts (distinct series of contiguous epochs scored as wake). Sleep measures were calculated as an average over the number of nights (mean = 2.84 (standard deviation, 0.56) nights). Additionally, we categorized individuals into diurnal types (24, 25) using the average midpoint of their sleep intervals, divided into 3 categories (8:00 PM to 1:59 AM, 2:00 AM to 2:59 AM, and 3:00 AM to 8:59 AM), which included all participants.

Survey sleep data

The Sleep Study booklet included the question, "How many hours do you usually sleep at night?" and questions about the frequency of 3 insomnia symptoms: trouble falling asleep, waking up during the night, and waking up too early and not being able to fall asleep again. Responses were "rarely or never," "sometimes," or "most of the time." These responses (coded 0, 1, or 2) were combined with an NSHAP core question on frequency of feeling rested upon waking in the morning (reverse coded) to create a scale of insomnia symptoms ranging from 0 to 8, with a higher score indicating more insomnia symptoms.

Cognition

Cognitive function was measured using the Montreal Cognitive Assessment (26) adapted for survey administration (MoCA-SA) (19, 27). The MoCA was developed as a cognitive screening tool for use in clinical practice to assess MCI across key cognitive domains and has a 90% sensitivity in detecting clinically diagnosed MCI (26). The MoCA-SA is highly correlated ($r = 0.97$) with the full MoCA (19, 27). The MoCA-SA includes 11 items measuring 8 domains: orientation, naming, visuoconstruction, executive function, attention, abstraction, memory, and language. The score on the MoCA-SA ranges from 0 to 20. It was administered in each wave, in English or Spanish.

Other measures

Demographics included age (continuous), sex, race/ethnicity, and education.

Other risk factors for cognitive impairment included frailty, depression, a comorbidity index, medications, alcohol use, body mass index, napping, frequent physical activity, and sleep apnea (at follow-up only). The frailty scale includes 4 of the 5 physical criteria proposed by Fried et al. (28): weak grip strength, slow gait, exhaustion, and low physical activity. We omitted unintentional weight loss, the fifth criterion, because we did not have prior measured weight for spouses and partners enrolled for the first time in wave 2 (29). This generated a 4-point scale, with higher points indicating greater frailty. Depression was measured using an 11-item short form of the Center for Epidemiologic Studies Depression Scale, which generated a score with a range of 0–22, with higher scores indicating greater depressive symptomatology (30). We used a modification of the Charlson Comorbidity Index developed for NSHAP, which includes 10 of the 19 conditions in the full index. The modified index creates a scale with a range of 0–16 and is highly correlated with the full index ($r = 0.89$) (31). Indicators for current medication use include prescription antidepressants and both prescription and nonprescription sleep aids, including anxiolytics, sedatives, and hypnotics. For the follow-up wave, a question was added asking if participants had ever been diagnosed with sleep apnea.

In the sleep booklet, participants were asked about napping. They were asked to record the total time they spent napping each of the 3 days in the following categories: no nap, less than 15 minutes, 15 minutes to 1 hour, or more than 1 hour. We used the duration midpoints from the responses to estimate nap length to 4 categories (in minutes: 0, 7.5, 37.5, 90) and averaged over the 3 days.

Statistical analysis

We present demographic, risk factor, and sleep characteristics at baseline for all Sleep Study participants and those in the subgroup with 5-year follow-up.

We standardized the actigraph-measured disruption measures (WASO, fragmentation, percentage sleep, and number of wake bouts) to facilitate comparisons. For the cross-sectional analyses, we used linear regression to assess the association between each sleep parameter and MoCA-SA at baseline, adjusted for demographics, and then added risk factors for cognitive impairment.

To examine 5-year cognitive change, we focused on those participants with a clear decline. At follow-up, 43% had a score that was the same or within 1 point of their baseline score, and 20% increased by 2 or more points, suggestive of a learning effect. We dichotomized cognition scores at the cutpoint that most closely represented the lowest quintile, a decline of 4 or more points. We used logistic regression to examine associations between sleep characteristics and cognitive decline, first adjusting for demographics and then adding risk factors for decline. We included sleep apnea diagnosis as a risk factor in the decline models but not cross-sectional models, because it was assessed only at follow-up. We also tested for interactions of sex with sleep measures.

We have provided 5 sensitivity analyses in Web Appendix 1 (available at https://academic.oup.com/aje): 1) assessing selection bias due to loss to follow-up using the inverse Mills ratio (32); 2) using a threshold for cognitive decline of 3 points instead of 4; 3) excluding individuals reporting Alzheimer's or dementia diagnoses at baseline; and 4) a cross-sectional model including only those with follow-up data in order to adjust for sleep apnea. We also considered a fifth analysis, a model using just 1 insomnia item—trouble waking during the night—as a more direct comparison with WASO than the 4-item scale.

We evaluated spousal correlation using multilevel models clustering on household identification, but we did not find any correlation after adjustment for demographics in our baseline model. As such, all analyses took into account the study design and sampling weights to account for the complex survey design and nonresponse (33). All data were analyzed using Stata, version 15.1 (StataCorp LLC, College Station, Texas).

Table 1. Baseline Characteristics for All Participants and for Those Included in the 5-Year Follow-up, National Health, Social Life and Aging Project Sleep Study, United States, 2010–2015

Characteristic	All (n = 759)		With 5-Year Follow-up (n = 555)	
	%	Mean (SD)	%	Mean (SD)
Age		71.9 (7.3)		70.5 (6.6)
Female sex	54.0		57.7	
Race/ethnicity				
White	82.3		82.6	
Black	7.5		7.7	
Hispanic	6.8		6.5	
Other	3.4		3.3	
Education				
Less than high school	14.5		13.2	
High school	24.4		25.3	
Some college	37.2		36.7	
College degree or higher	24.8		24.8	
Modified Charlson comorbidity score[a]		1.1 (1.4)		0.9 (1.2)
0	44.9		49.5	
1	27.2		26.7	
2	15.3		15.1	
3	7.1		5.2	
>4	5.5		3.4	
Frailty (range, 0–4)[b]		1.1 (1.2)		1.1 (1.2)
Depression (range, 0–22)[c]		7.6 (3.4)		7.5 (3.4)
Medication use				
Antidepressants	17.2		16.7	
Sleep aids	8.9		8.9	
Alcohol use >4 days/week	12.4		12.3	
BMI category[d]				
Underweight	<1.0		<1.0	
Normal	24.7		22.9	
Overweight	35.3		36.4	
Obese	39.6		40.3	
Physical activity ≥1/week	43.4		45.0	
Daytime naps (minute)		15.0 (20.9)		12.8 (18.5)
Sleep apnea	Not assessed		14.6	

Abbreviations: BMI, body mass index; SD, standard deviation.

[a] Modified Charlson Comorbidity Index included heart condition, stroke, cancer, diabetes, hypertension, arthritis, bone fractures/osteoarthritis, chronic obstructive pulmonary disease/asthma, Alzheimer's disease/dementia, and incontinence.

[b] Frailty scale: weak grip strength, slow gait, exhaustion, and low physical activity.

[c] Depression: 11-item short from of the Center for Epidemiologic Studies Depression Scale.

[d] BMI was calculated as weight (kg)/height $(m)^2$ and classified as underweight, <18.5; normal 18.5–24.9; overweight, 25.0–29.9; or obese, ≥30.0.

RESULTS

Table 1 shows baseline characteristics for all participants and for those included in the follow-up assessment. Both younger and female participants were more likely to survive to follow-up. Race/ethnicity and education were similar for the full baseline cohort and those with follow-up.

Table 2 shows actigraph-measured and self-reported sleep characteristics and MoCA-SA scores at baseline and at baseline for those with follow-up. At baseline, the average MoCA-SA score was 14.1 (standard deviation, 3.6); those with follow-up had a slightly higher baseline average (14.7; standard deviation, 3.3).

Table 2. Sleep Characteristics and Montreal Cognitive Assessment Scores at Baseline for All Participants and for Those With 5-Year Follow-up, National Health, Social Life and Aging Project Sleep Study, United States, 2010–2015

Sleep Characteristic	All (n = 759)		With 5-Year Follow-up (n = 555)	
	%	Mean (SD)	%	Mean (SD)
Actigraph-Measured Sleep Characteristics				
WASO (minutes)		38.7 (22.5)		36.5 (19.6)
Fragmentation		14.4 (6.0)		13.8 (5.5)
Total sleep time, hours				
<6.00	13.8		13.1	
6.00–6.99	27.5		28.3	
7.00–7.99	34.6		35.2	
8.00–8.99	17.1		17.1	
≥9.00	6.9		6.3	
Percentage sleep		91.8 (0.04)		92.2 (0.04)
Wake bouts (number)		45.8 (21.6)		44.9 (20.2)
Diurnal phase				
8:00 PM to 1:59 AM	21.6		20.8	
2:00 AM to 2:59 AM	36.0		37.4	
3:00 AM to 8:59 AM	42.5		41.8	
Self-Reported Sleep Characteristics				
Self-reported, hours				
<6.00	7.6		7.9	
6.00–6.99	16.0		15.5	
7.00–7.99	25.3		27.5	
8.00–8.99	32.2		32.5	
≥9.00	19.0		16.7	
Insomnia symptom score[a]		2.8 (2.1)		2.8 (2.1)
Cognitive Score				
MoCA-SA baseline		14.1 (3.6)		14.7 (3.3)

Abbreviations: MoCA-SA, Montreal Cognitive Assessment survey adaptation; SD, standard deviation; WASO, wake after sleep onset.

[a] Range, 0–8; a combined metric (0 = never/rarely, 1 = sometimes, 2 = most of the time) from 4 questions: feeling rested in the morning, trouble falling asleep, trouble waking during the night, and trouble waking too early.

Cross-sectional associations between sleep and cognition

All 4 actigraphic measures of sleep disruption were significantly associated with cognition after adjustment for demographics and for additional risk factors (Table 3). More disrupted sleep was associated with a lower MoCA-SA score. WASO was the most strongly associated with cognition, with a 1-standard-deviation (22.5 minutes) increase in WASO associated with a 0.59 point lower MoCA-SA score (95% confidence interval: −0.85, −0.33). Diurnal phase was not associated with cognition. There was no evidence of a linear association between TST and MoCA-SA score or suggestion of a U-shaped association.

There was no evidence of a linear association between self-reported sleep duration and MoCA-SA, although those reporting shortest sleep (<6 hours) had higher average MOCA-SA scores. There was no evidence of an association for insomnia symptoms.

Sleep and 5-year decline in cognitive function

In the demographic-adjusted models (Table 4), WASO and percentage sleep were significantly associated with odds of cognitive decline, and fragmentation trended in the same direction. Number of wake bouts was not associated with decline. Further adjustment for risk factors had little effect on the odds ratio estimates for WASO, fragmentation, and sleep percentage. There was also some evidence that earlier circadian timing (sleep midpoint between 8:00 PM and 1:59 AM) was associated with higher risk of decline, compared with those with later circadian timing (odds ratio = 2.22, 95% confidence interval: 0.80, 6.23). Those with low actigraph-measured TST (<6 hours) had increased odds of cognitive decline, compared with those sleeping 7–8 hours (odds ratio = 3.41, 95% confidence interval: 1.19, 9.76). Self-reported short sleepers trended in the same pattern. Insomnia was not associated with odds of decline.

In the interaction-by-sex models, there was no evidence for different cross-sectional associations (data not shown), but there was evidence that the associations between sleep disruption and cognitive decline were stronger among men than among women (Table 5). Interaction terms were significant for sleep fragmentation and number of wake bouts, and there was a pattern of sex difference for WASO and percentage sleep. There was qualitative interaction for wake bouts, such that more wake bouts increased the odds of cognitive decline for men but decreased the odds of decline for women.

In sensitivity analyses for selection, the inverse Mills ratio was not significant in any of the selection models. It slightly attenuated our observed measures of association but did not change the pattern of results (Web Table 1). None of our sensitivity analyses substantively changed measures of association (Web Tables 2–5).

DISCUSSION

In a nationally representative cohort of older men and women, we found that actigraphic measures of sleep and self-reported measures of sleep have different cross-sectional and longitudinal associations with cognitive function and decline. We found that actigraphic measures of sleep disruption and quality were negatively associated with cognition measured concurrently and were associated with 5-year cognitive decline. In the cross-sectional analysis, this association did not differ between men and women, but actigraphic measures of sleep disruption were more strongly associated with 5-year cognitive decline among men compared with women. Short actigraph-measured TST was associated with higher odds of cognitive decline, but there was no evidence of a linear or U-shaped association across the full range of TST. We did not find that self-reported sleep quality (insomnia symptomology) was associated with worse cognition or cognitive decline. Perceptions of sleep duration were also unrelated to cognitive function and decline. We found some evidence that earlier diurnal phase might be associated with greater 5-year

Table 3. Associations Between Baseline Sleep Characteristics and Baseline Montreal Cognitive Assessment, From Ordinary Least Squares Regression Models, National Health, Social Life and Aging Project Sleep Study, United States, 2010–2015

Model[a]	Individual Sleep Parameter[b]	Demographic-Adjusted Modeling[c] ($n = 732$)			Risk-Factor Modeling[d] ($n = 575$)		
		Coefficient	95% CI	*P* Value	Coefficient	95% CI	*P* Value
	Actigraph-Measured Sleep Characteristics						
1	WASO	−0.59	−0.85, −0.33	<0.01	−0.55	−0.83, −0.27	<0.01
2	Fragmentation	−0.50	−0.74, −0.26	<0.01	−0.39	−0.66, −0.13	<0.01
3	Percentage sleep	0.46	0.21, 0.71	<0.01	0.37	0.11, 0.63	0.01
4	Wake bouts	−0.37	−0.61, −0.12	<0.01	−0.40	−0.67, −0.13	<0.01
5	Diurnal phase[e]						
	8:00 PM to 1:59 AM	0.06	−0.60, 0.73	0.85	0.30	−0.41, 1.00	0.41
	2:00 AM to 2:59 AM	0	Referent		0	Referent	
	3:00 AM to 8:59 AM	0.01	−0.54, 0.56	0.98	0.19	−0.43, 0.82	0.54
6	Total sleep time, hours						
	<6.00	−0.01	−0.78, 0.77	0.98	0.34	−0.50, 1.17	0.43
	6.00–6.99	−0.19	−0.78, 0.40	0.53	−0.11	−0.76, 0.55	0.75
	7.00–7.99	0	Referent		0	Referent	
	8.00–8.99	−0.09	−0.79, 0.61	0.80	0.00	−0.74, 0.73	0.99
	≥9.00	−0.74	−1.72, 0.25	0.14	−0.71	−1.72, 0.31	0.17
	P for trend		0.61			0.21	
	Self-Reported Sleep Characteristics						
7	Self-reported, hours						
	<6.00	−0.74	−1.39, −0.08	0.03	−0.37	−1.15, 0.40	0.34
	6.00–6.99	−0.56	−1.46, 0.34	0.23	−0.38	−1.43, 0.67	0.48
	7.00–7.99	0	Referent		0	Referent	
	8.00–8.99	−0.11	−0.73, 0.50	0.72	0.24	−0.43, 0.90	0.48
	≥9.00	−0.58	−1.37, 0.22	0.15	−0.14	−0.93, 0.66	0.74
	P for trend		0.32			0.23	
8	Insomnia symptoms[f]	0.01	−0.11, 0.12	0.92	0.05	−0.08, 0.17	0.47

Abbreviations: CI, confidence interval; WASO, wake after sleep onset.

[a] Model numbers indicate separate models.

[b] Continuous sleep parameters are standardized.

[c] Demographic-adjusted models included age, sex, race/ethnicity, and education.

[d] Risk-factor models additionally included frailty, depression, modified Charlson comorbidity score, sleep aids, antidepressants, alcohol use, daytime napping, physical activity, and body mass index.

[e] Calculated from the average midpoint of sleep interval over 3 nights of actigraphy.

[f] Range, 0–8; a combined metric (0 = never/rarely, 1 = sometimes, 2 = most of the time) from 4 questions: feeling rested in the morning, trouble falling asleep, trouble waking during the night, and trouble waking too early. In the risk-adjusted models for the Troubled Sleep Scale, the depression scale, Center for Epidemiologic Studies Depression Scale, does not contain the restless sleep item.

decline. Diurnal phase is a measure of circadian rhythm, and this is often shifted earlier for older adults (25). The observed associations are consistent with sleep disruption and potentially circadian phase shift playing a causal role in cognitive decline or with there being an underlying biological process that predisposes older adults to both. However, in SOF, peak activity occurring later in the day was associated with increased incidence of MCI/dementia (34). This difference could be due to our defining circadian phase using the midpoint of sleep, or it could be due to the age difference between women in NSHAP and in SOF.

Unlike much of the previous sleep literature, we did not find that reported sleep duration or insomnia symptoms were related to cognitive function or decline (7–10). However, our findings are broadly consistent with results from the SOF and MrOS. In both studies, WASO was associated with concurrent cognitive impairment on both the Trail Making Test Part B and the MMSE tests (16, 18). In SOF, low sleep efficiency (TST divided by the rest interval) was also associated with lower cognitive scores on both tests of cognitive function (16). The association between sleep disruption and cognitive decline we observed was also observed in MrOS,

Table 4. Cognitive Decline (4 or More Points on the Montreal Cognitive Assessment) and Baseline Sleep Characteristics, National Health, Social Life and Aging Project Sleep Study, United States, 2010–2015

Model[a]	Individual Sleep Parameter[b]	Demographic-Adjusted Modeling[c] ($n = 535$)			Risk-Factor Modeling[d] ($n = 402$)		
		OR	95% CI	*P* Value	OR	95% CI	*P* Value
	Actigraph-Measured Sleep Characteristics						
1	WASO	1.43	1.04, 1.98	0.03	1.50	1.00, 2.24	0.05
2	Fragmentation	1.30	0.96, 1.75	0.09	1.50	1.07, 2.10	0.02
3	Percentage sleep	0.72	0.55, 0.94	0.02	0.63	0.44, 0.90	0.01
4	Wake bouts	1.00	0.77, 1.31	0.99	1.11	0.82, 1.49	0.50
5	Diurnal phase[e]						
	8:00 PM to 1:59 AM	2.22	0.80, 6.23	0.13	3.03	0.82, 11.21	0.10
	2:00 AM to 2:59 AM	1.00	Referent		1.00	Referent	
	3:00 AM to 8:59 AM	1.13	0.62, 2.06	0.68	0.84	0.40, 1.76	0.64
	Total sleep time, hours						
6	<6.00	1.73	0.67, 4.50	0.26	3.41	1.19, 9.76	0.02
	6.00–6.99	1.12	0.55, 2.27	0.75	1.47	0.66, 3.25	0.35
	7.00–7.99	1.00	Referent		1.00	Referent	
	8.00–8.99	1.14	0.47, 2.73	0.78	0.78	0.26, 2.35	0.66
	≥9.00	0.63	0.15, 2.60	0.52	0.50	0.11, 2.31	0.37
	P for trend		0.21			<0.01	
	Self-Reported Sleep Characteristics						
7	Self-reported, hours						
	<6.00	1.54	0.68, 3.48	0.30	1.82	0.70, 4.73	0.22
	6.00–6.99	0.98	0.31, 3.09	0.97	0.90	0.28, 2.95	0.86
	7.00–7.99	1.00	Referent		1.00	Referent	
	8.00–8.99	0.77	0.34, 1.71	0.52	0.76	0.28, 2.08	0.59
	≥9.00	0.79	0.30, 2.12	0.64	0.99	0.32, 3.04	0.98
	P for trend		0.10			0.17	
8	Insomnia symptoms[f]	1.08	0.93, 1.26	0.29	1.04	0.87, 1.24	0.69

Abbreviation: CI, confidence interval; MoCA-SA, Montreal Cognitive Assessment survey adaptation; OR, odds ratio; WASO, wake after sleep onset.

[a] Model numbers indicate separate models.

[b] Continuous sleep parameters are standardized.

[c] Logistic regression models adjusted for age, sex, race/ethnicity, education, and baseline MoCA-SA score.

[d] Risk-factor models additionally included frailty, depression, modified Charlson comorbidity score, sleep aids, antidepressants, alcohol use, daytime napping, physical activity, body mass index, and sleep apnea.

[e] Calculated from the average midpoint of sleep interval over 3 nights of actigraphy.

[f] Range, 0–8; a combined metric (0 = never/rarely, 1 = sometimes, 2 = most of the time) from 4 questions: feeling rested in the morning, trouble falling asleep, trouble waking during the night, and trouble waking too early. In the risk-adjusted models for the Troubled Sleep Scale, the depression scale, Center for Epidemiologic Studies Depression Scale, does not contain the restless sleep item.

where WASO, low sleep efficiency, and the number of long wake episodes were associated with greater cognitive decline over an average of 3.4 years on the MMSE and the Trail Making Test Part B (17). A comparable association was not seen in SOF for either the Trail Making Test Part B or the MMSE (K. Stone, University of California, San Francisco, personal communication, March 2018). However, in a follow-up study among a subsample of SOF participants who were given an extensive battery of clinician-reviewed cognitive tests, low sleep efficiency and longer sleep latency (time between the beginnings of the rest interval and the sleep interval) were associated with incident MCI/dementia (15). Of note, the average age of participants in SOF (87.4 years, and 82.6 years in the substudy) and MrOS (76 years) was older than the participants in the present study (71.9 years) (15–18).

The report of different longitudinal associations in MrOS (male cohort) and SOF (female cohort) does not necessarily imply a different effect for men and women, because there are

Table 5. Interaction Between Sex and Sleep Characteristics on Cognitive Decline, National Health, Social Life and Aging Project Sleep Study, United States, 2010–2015

Model[a]	Individual Sleep Parameter[b]	Demographic-Adjusted Modeling[c] (n = 535)		Risk-Factor Modeling[d] (n = 402)	
		OR	95% CI	OR	95% CI
1	WASO				
	Men	1.85	1.25, 2.73	2.39	1.39, 4.12
	Women	1.20	0.76, 1.90	1.12	0.64, 1.95
	P for interaction	0.15		0.06	
2	Fragmentation				
	Men	1.83	1.27, 2.63	2.21	1.40, 3.48
	Women	0.95	0.62, 1.44	1.01	0.61, 1.67
	P for interaction	0.02		0.03	
3	Percentage sleep				
	Men	0.60	0.44, 0.84	0.49	0.31, 0.78
	Women	0.87	0.56, 1.35	0.79	0.45, 1.39
	P for interaction	0.19		0.20	
4	Wake bouts				
	Men	1.34	0.98, 1.83	1.45	0.99, 1.32
	Women	0.67	0.44, 1.01	0.76	0.48, 1.21
	P for interaction	<0.01		0.04	

Abbreviations: CI, confidence interval; MoCA-SA, Montreal Cognitive Assessment survey adaptation; OR, odds ratio; WASO, wake after sleep onset.

[a] Model numbers indicate separate models. Each model included parameters for main effects of sex and the sleep parameter and a term for a sex × sleep parameter interaction.

[b] Continuous sleep parameters are standardized.

[c] Logistic regression models adjusted for age, race/ethnicity, education, and baseline score on MoCA-SA.

[d] Risk-factor models additionally included frailty, depression, modified Charlson comorbidity score, sleep aids, antidepressants, alcohol use, daytime napping, physical activity, body mass index, and sleep apnea.

differences besides sex between the 2 cohorts, including the age distributions at the time of analyses. However, we observed significant differences between men and women in the NSHAP Sleep Study. The reasons for these differences are unknown, although a greater prevalence of obstructive sleep apnea in men versus women could play a role, given that apnea has also been associated with cognitive impairment (35, 36). Indeed, in our sample, men had a higher prevalence of apnea at follow-up than women did (21% vs. 10%). However, we did adjust for apnea in the longitudinal model. While a number of studies have reported differences in sleep characteristics between men and women (37–39), there has been limited research on whether the effects of sleep differ by sex. The studies that do assess this difference are often among younger populations (40, 41), rely on self-reported sleep (42, 43), or deal specifically with sleep apnea as opposed to nonclinical variation in sleep (44, 45). We believe our study is the first to report differential associations of sleep on a health outcome according to sex using objective measures of sleep in a community-based sample of older adults.

A key strength of this study is the measure of cognitive function. Unlike cognitive assessments in many omnibus surveys, the MoCA-SA is a validated multidomain assessment developed to assess and track mild cognitive impairment (19). Additionally, NSHAP's national sampling frame allows results to be generalized to the US population of community-dwelling older adults born between 1920 and 1947. Use of wrist actigraphy allows us to compare observed associations with self-perceptions of sleep duration and quality. While many studies have considered the relationship between self-reported sleep characteristics such as insomnia symptoms and sleep duration with cognition, discrepancies between self-reports of sleep and more objective measures might be pronounced in older populations (11, 46–48). Our relatively small sample size is a limitation that must be acknowledged. Findings of marginal statistical significance might reflect, in part, the sample size and insufficient statistical power to detect associations. We did not carry out an analysis of incident MCI or dementia among those with neither at baseline because that would have further reduced the sample size. While actigraphy estimates sleep from arm motion as opposed to direct measurement of brain activity, it is generally considered a valid and useful approach to objective estimation of sleep characteristics without itself affecting sleep behavior (49). More than 3 days of actigraphy are recommended to assess sleep patterns, particularly to capture variation between workdays and weekends. However, we have found little day-of-the-week effect in this cohort of older adults (23).

Prior evidence that sleep characteristics are indicators of future cognitive decline among older adults has relied mainly

on self-reported sleep characteristics. Previous studies focused on sleep duration as an important contributor to cognitive function, with self-reported short and long durations indicative of higher risk of cognitive decline (8, 10). In contrast to previous studies, we did not find evidence that self-reported sleep duration was a significant contributor to cognitive function. These results call into question the usefulness of self-reports of sleep as measures of sleep pertinent to cognitive function. Similarly, there appears to be no evidence that insomnia is a risk factor for cognitive decline, which might be reassuring for the many older adults who report insomnia symptoms. We have shown here that measured sleep disruption is a more important aspect of sleep than duration for predicting cognitive decline. Our findings add to the evidence that sleep disruption and quality measured using wrist actigraphy are salient dimensions of sleep when considering the relationship between sleep and cognitive function at older ages. Further, we have shown that this association might be stronger among men than among women, but that unexplained finding needs replication.

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