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Poor Sleep Is Associated With Impaired Cognitive Function in Older Women: The Study of Osteoporotic Fractures

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Background. The association between objectively measured sleep and cognition among community-dwelling elderly persons remains understudied. This observational, cross-sectional analysis examined this association.

Methods. Results are from 2932 women (mean age 83.5 years) in the Study of Osteoporotic Fractures between 2002 and 2004. Cognitive function was measured by Mini-Mental State Examination (MMSE) and Trail Making B Test (Trails B). Cognitive impairment was defined as MMSE < 26 or Trails B > 278 seconds. Sleep parameters measured objectively using actigraphy included total sleep time, sleep efficiency, sleep latency, wake after sleep onset (WASO), and total nap time.

Results. There were 305 women (10.6%) with MMSE < 26 and 257 women (9.3%) with Trails B > 278 seconds. Compared with women with sleep efficiency $\geq$70%, those with $<$70% had a higher risk of cognitive impairment (MMSE < 26 multivariate odds ratio [MOR] = 1.61; 95% confidence interval [CI], 1.20–2.16; Trails B > 278 MOR = 1.96; 95% CI, 1.43–2.67). Higher sleep latency was associated with higher risk of cognitive impairment (per half hour: MMSE < 26 MOR = 1.23; 95% CI, 1.13–1.33; Trails B > 278 MOR = 1.13; 95% CI, 1.04–1.24), as was higher WASO (per half hour: MMSE < 26 MOR = 1.15; 95% CI, 1.06–1.23; Trails B > 278 MOR = 1.24; 95% CI, 1.15–1.34). Women who napped $\geq$2 hours per day had a higher risk (MMSE < 26 MOR = 1.42; 95% CI, 1.05–1.93; Trails B > 278 MOR = 1.74; 95% CI, 1.26–2.40). There was no significant relationship for total sleep time.

Conclusion. Objectively measured disturbed sleep was consistently related to poorer cognition, whereas total sleep time was not. This finding may suggest that it is disturbance of sleep rather than quantity that affects cognition.

SLEEP disorders are extremely common and affect up to as many as 50% of older adults (1,2), yet they are frequently underdiagnosed and untreated, particularly among elderly persons (3,4). In addition, at least 10% of those persons 65 years old or older will develop cognitive impairment, with the rate rising exponentially with age (5,6). With life expectancies rising, this number will increase in future years. Insomnia, or difficulty falling or staying asleep at night, has been associated with complaints of problems with memory and concentration, particularly in older populations (1,7). Yet the association between objectively measured fragmented sleep and decreased cognitive function has not been extensively studied in older community-dwelling adults, where even a modest association could have large public health importance.

Many previous studies that have examined the association between sleep parameters and cognition have focused on specific populations with clinical disorders—such as patients with Alzheimer's disease, insomnia, or sleep-disordered breathing—or on institutionalized elderly persons (8–16). Furthermore, the majority of studies have focused on sleep-disordered breathing, not measures of total sleep time, sleep fragmentation, or napping (9–14,16–21). A large study ($N = 718$) examined the association of polysomographic-measured total sleep time and sleep efficiency to cognitive impairment in older men but, to our knowledge, no results from large studies of older women have been published (17). Other studies that have investigated objective measures of sleep fragmentation have had relatively small sample size ($N = 39$–60) and have limited adjustment for the potential confounders of age, gender, or education (8,16,22).

Actigraphy has been used for over 25 years to assess sleep and wake behavior (23). High correlations for differentiating sleep from wake states were found between actigraphy and polysomnography (PSG), the ''gold standard'' for sleep assessment (24,25). There are many benefits to using actigraphy including assessing daytime activity and re-

cording data continuously for weeks or longer. In addition, actigraphy is less invasive and more cost effective than is PSG, and is useful in populations where PSG would be difficult to record such as in large epidemiologic studies (23).

This study examined data gathered in the Study of Osteoporotic Fractures (SOF), a longitudinal study designed to examine the risk factors of osteoporotic fractures in women and to test the hypothesis that poor sleep measured objectively by actigraphy is associated with lower cognitive function in older women. This study population provides a unique opportunity to study this question in a large community-dwelling cohort, with additional data collected to allow for adjustment of possible confounders.

Methods

Participants

Community-dwelling women 65 years old or older were recruited from population-based listings in four U.S. areas: Baltimore, Maryland; Minneapolis, Minnesota; Portland, Oregon; and the Monongahela Valley, Pennsylvania. At the baseline visit, women were excluded if they were unable to walk without help or had a previous bilateral hip replacement. At all subsequent visits, no exclusion criteria were used. The SOF enrolled 9704 Caucasian women from September 1986 through October 1988 (26). Initially, African American women were excluded from the study due to their low incidence of hip fractures, but from February 1997 through February 1998 662 African American women were enrolled (27).

The focus of this analysis is data gathered at the most recent biannual visit, which took place between January 2002 and February 2004. Of the 4727 women at this visit, 1795 are not included in this analysis. Both actigraphy and cognitive function data were collected on only those participants with clinic or home visits, so those 1051 women (22.2%) who only had self-administered questionnaire data gathered were not eligible for this analysis. Of the 3676 participants with clinic or home visits, 511 did not have actigraphy measured, 120 did not have cognitive function measured, and 113 did not have either measure performed. Of those women without actigraphy, 92 (14.7%) had the measurement done but there was a malfunction with the actigraph data file. Therefore, there were 2932 women (79.8% of total eligible population) with both actigraphic data and cognitive function measurements who are the focus of our analysis. The institutional review boards at each clinic site approved the study, and written informed consent was obtained from all participants.

Cognition

Two cognitive function tests, the Mini-Mental State Examination (MMSE) and the Trail Making B Test (Trails B), were administered by trained clinic staff. The MMSE is a global measurement of cognitive function, with components for orientation, concentration, language, praxis, and immediate and delayed memory. The MMSE score ranges from 0 to 30, with higher scores representing better cognitive functioning (28). The Trails B is a timed test that measures attention, sequencing, visual scanning, and executive function. A faster time for completion (in seconds) represents better cognitive functioning (29). To examine clinically meaningful differences in cognitive function, cognitive impairment was defined in two ways: as an MMSE score less than 26 (30), and more than 1.5 standard deviations (*SD*s) above the mean for completion of the Trails B (>278 seconds).

Sleep Parameters

Sleep parameters were measured using an actigraph, which is a small device used to detect movement. The actigraph, SleepWatch-O (Ambulatory Monitoring, Inc., Ardsley, NY), looks like a wristwatch and was placed on the nondominant wrist. Movement is measured by a piezoelectric biomorph-ceramic cantilevered beam, which generates a voltage each time the actigraph is moved. These voltages are gathered continuously and summarized over 1-minute epochs. Data were collected in the three modes of zero crossings (ZCM), digital integration (DIM, also known as proportional integration mode), and time above threshold (TAT; 31).

ActionW-2 software (Ambulatory Monitoring, Inc.) was used to analyze the data and score the sleep and wake periods (32). The Cole–Kripke algorithm was used for data collected in the ZCM mode, and the UCSD scoring algorithm was used for data collected in the DIM and TAT modes (33,34). These algorithms calculate a moving average, which takes into account the activity levels immediately before and after the current minute to determine if the timepoint should be coded as sleep or wake. The time participants got in and out of bed were obtained from sleep diaries kept by the participants while wearing the actigraph.

Women were to wear the actigraphs continuously for a minimum of three 24-hour periods. The average of the sleep parameters over all nights was used in this analysis to minimize night-to-night variability. The actigraphy parameters used were: total hours slept per night while in bed; sleep efficiency (the percentage of time the participant is sleeping while in bed); sleep latency, the amount of time until the onset of sleep (onset defined as completion of 20 continuous minutes of sleep after getting into bed); minutes of wake after sleep onset (WASO), a measure of sleep fragmentation, defined as the total minutes of time scored as awake from the onset of sleep to the end of the sleep interval; and napping time, defined as the amount of time in minutes the participant was scored as sleeping during the time she was not in bed. Total nap time was categorized as less than 2 hours and 2 or more hours. Sleep efficiency was categorized as <70% compared with ≥70%.

Other Measurements

All participants completed a questionnaire which included questions about medical history, self-reported health, physical activity (walking for exercise), smoking, and alcohol use. Caffeine intake was estimated based on self-report of the average daily number of cups of caffeinated coffee, tea, or cans of caffeinated soda (35). The Geriatric Depression Scale (GDS) was used to assess depressive symptoms (36). Functional status was measured with information on six independent activities of daily living (IADL) (37,38). Participants were asked to bring in all current medi-

cations used within the last 2 weeks, and a computerized medication coding dictionary was used to categorize the medications (39). Body weight and height were measured, and body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters.

Statistical Analysis

All analyses were performed using the actigraphy data collected in all three modes (DIM, TAT, ZCM). As results were similar in all three modes, only results based on the DIM mode are shown.

Characteristics known to be related to sleep or cognition (1,2,40) were summarized using means and *SD*s for continuous data and counts and percentages for categorical data. We compared characteristics between this analysis subset and the remaining SOF population to determine if the results based on the subset were generalizable to the entire SOF cohort. For continuous variables, *t* tests were used for those with normal distributions and Wilcoxon rank sum tests were used for those with skewed distributions. Differences in categorical data were examined using chi-squared tests. Fisher's exact tests were used when categorical variables had low expected cell counts.

Linear regression models were used to examine the relationship between sleep parameters and cognitive outcomes. Both outcomes were log-transformed to meet normality assumptions for the models. Data were back-transformed for display of results. To show the magnitude of the relationship of the sleep parameters to cognition, the results from the linear regression models are shown as a percent difference (100 * beta coefficient/mean of cognitive outcome) and 95% confidence intervals (CI) for this percent difference (41–43). Logistic regression models were used to examine the relationship between sleep parameters and cognitive impairment, with results presented as odds ratios (OR) and 95% CI. Known factors associated with cognitive function and characteristics related to sleep parameters were examined for inclusion in multivariate models. Stepwise model selection using entrance and exit criteria of $p = .20$ was used to select the covariates for the models. If the covariate was selected for at least one of the multivariate models, it was included in the final set of covariates. The presence of a nonlinear (quadratic) effect of the sleep predictors was examined but was not apparent, so the linear relationship was modeled.

The analyses were stratified by possible dementia to examine if the association was altered by those women with the worst cognitive functioning. Possible dementia was defined as having an MMSE score less than 20 or reporting ever having been told by a doctor that she had dementia or Alzheimer's disease (30,44). All analyses were performed using SAS software, version 9.1 (SAS Institute, Cary, NC).

RESULTS

Characteristics of the Study Population

The analysis cohort was composed of 2932 women (mean age 83.5 ± 3.7 years, 10.6% African Americans; Table 1). Actigraph data were collected for an average of 4.1 ± 0.8 nights. The average sleep time at night was 6.7 ± 1.3 hours, with 253 (8.6%) of the women sleeping 5 hours or less per night. The mean sleep efficiency was 77.3 ± 11.9%. About one fifth of the participants (20.8%) had sleep efficiency <70%. The participants took 41 minutes to fall asleep on average (*SD* = 40.6). The average WASO was 77.2 ± 48.0 minutes. The average time spent napping per day is 75.0 ± 64.2 minutes, with 18.9% of the women napping 2 hours or more per day (Table 2).

Overall, cognitive impairment as measured by MMSE score less than 26 occurred in 305 (10.6%) women. There were 257 (9.3%) with a Trails B score greater than 1.5 *SD*s above the mean (>278 seconds). The average score for MMSE was 27.8 ± 2.0, and the average time to complete the Trails B was 158.4 ± 79.7 seconds (Table 1). Sixty-five

Table 1. Comparing Characteristics of the Analysis Cohort to the Remaining SOF Cohort

Variable	All Participants in Analysis ($N = 2932$)	Remaining SOF Cohort ($N = 1795$*)	p Value†
Cognitive function			
MMSE (range 0–30), mean ± *SD*	27.8 ± 2.0	26.9 ± 2.7	<.0001
MMSE < 26, %	10.6	18.8	<.0001
Trail Making B Test time (s), mean ± *SD*	158.4 ± 79.7	184.8 ± 92.4	<.0001
Trail Making B Test time > 278 seconds, %	9.3	16.5	<.0001
Doctor ever diagnosed dementia/ Alzheimer's disease, %	1.8	12.4	<.0001
Possible dementia (MMSE < 20 or dementia diagnosis), %	2.3	31.4	<.0001
Characteristics			
Age (y), mean ± *SD*	83.5 ± 3.7	85.0 ± 4.5	<.0001
Body mass index (kg/m^2) mean ± *SD*	27.1 ± 5.0	26.5 ± 5.4	.02
African American, %	10.6	8.7	.04
Difficulty with one or more IADLs, %	52.1	64.6	<.0001
History of stroke, %	12.5	18.0	<.0001
History of hypertension, %	60.2	57.7	.10
History of myocardial infarction, %	13.5	13.1	.72
Currently taking antidepressants, %	13.6	18.5	.0007
Currently taking benzodiazepines, %	7.2	8.5	.25
Geriatric Depression Scale score (range 0–15), mean ± *SD*	2.3 ± 2.5	3.5 ± 3.4	<.0001
Education (y), mean ± *SD*	12.9 ± 2.7	12.4 ± 2.8	<.0001
Current smoker, %	2.7	3.5	.11
Average drinks per day in the last 30 days, mean ± *SD*	0.5 ± 0.7	0.4 ± 0.7	<.0001
Daily caffeine intake (mg), mean ± *SD*	151.0 ± 153.2	132.5 ± 139.5	.0003
Takes walks for exercise, %	37.6	31.2	<.0001
Self-reported health, %			
Poor/very poor	2.0	7.8	<.0001
Fair	22.1	28.3	
Good/very good	75.9	63.8	

Notes: *Variables for cognitive measures, body mass index, IADL, and use of medications were only asked of those $N = 744$ who had clinic or home visits.

†Values of p for continuous data are from a t test for normally distributed data and a Wilcoxon rank sum test for skewed data. Values of p for categorical data are from a chi-squared test.

SOF = Study of Osteoporotic Fractures; MMSE = Mini-Mental State Examination; *SD* = standard deviation; IADL = instrumental activities of daily living.

Table 2. Comparing Sleep Measures of the Analysis Cohort to the Remaining SOF Cohort*

Parameter	All Participants in Analysis ($N = 2932$)	Remaining SOF Cohort With Actigraphy Data ($N = 120$)	p Value†
No. of nights of data, mean ± *SD*	4.1 ± 0.8	4.1 ± 1.0	.58
Total sleep time (h), mean ± *SD*	6.7 ± 1.3	6.9 ± 1.5	.15
Total nap time (min), mean ± *SD*	75.0 ± 64.2	108.1 ± 95.6	.0001
Nap time ≥2 hours per day, %	18.9	36.5	<.0001
Sleep efficiency, %, mean ± *SD*	77.3 ± 11.9	74.3 ± 12.4	.003
Sleep efficiency <70%, %	20.8	28.3	.05
Sleep latency (min), mean ± *SD*	41.4 ± 40.6	51.4 ± 59.2	.14
Wake after sleep onset (min), mean ± *SD*	77.2 ± 48.0	98.0 ± 48.7	<.0001

Notes: Sleep efficiency = percentage of time sleeping while in bed; sleep latency = minutes until the onset of sleep; wake after sleep onset = minutes of time scored as awake from the onset of sleep to the end of the sleep interval.

*Averaged over all days/nights data were collected.

†Values of p for continuous data are from a t test for normally distributed data and a Wilcoxon rank sum test for skewed data. Values of p for categorical data are from a chi-squared test.

SOF = Study of Osteoporotic Fractures; *SD* = standard deviation.

women (2.3%) were identified as having possible dementia (MMSE < 20 or ever told by a doctor they had dementia or Alzheimer's disease).

Compared to those 2932 women in the analysis, those 1795 women in the SOF at this visit who did not have cognitive function and actigraphy measured were significantly different for many characteristics (Table 1). Those 1795 women not included in analysis were slightly older on average, had a lower percentage of African Americans, had a worse mean depression score and were more likely to be taking antidepressants, had higher rates of IADL impairment and stroke, were less likely to walk for exercise, and reported worse health status ($p < .05$). There were also small but statistically significant differences in education, BMI, alcohol intake, and caffeine intake ($p < .02$). Most notably, these 1795 women reported being told by their doctor that they had Alzheimer's disease at a rate 6 times higher than this analysis cohort. On average, the 511 participants who were not in the analysis subset but did have cognitive measures performed had lower cognitive functioning than did those women in our analysis subset ($p < .0001$). Those 120 women who are not included in our analysis but did have actigraphy data measured were no different from our analysis subset in total sleep time and sleep latency ($p > .14$), but had more nap time, worse sleep efficiency, and more WASO ($p < .003$) (Table 2).

Continuous Cognitive Outcomes and Sleep Parameters

All sleep parameters were significantly related to MMSE scores, and all but total sleep time at night were significantly related to the Trails B ($p < .05$) (Table 3). Total sleep time was significantly related to MMSE score, but the association was small. For Trails B score, a positive percent difference means that the cognitive performance is worse for the predictor in question, and a negative percent difference in MMSE score represents worse cognition. For example, those women who took longer to fall asleep had worse cognitive scores (2.5% worse on Trails B, 0.8% worse on MMSE per half-hour increase in sleep latency). Those women with sleep efficiency <70% showed the largest association to cognitive functioning, with a 9.1% worse completion time on the Trails B and a 1.9% worse score on the MMSE, on average. Sleep fragmentation had an inverse relationship to cognitive functioning: For each half-hour increase in WASO, there was 3.5% worse completion time on the Trails B and 0.5% worse on MMSE scores. Those participants who napped 2 or more hours had worse cognition, with an average percent difference of 10.0% for Trails B and 1.2% worse score for the MMSE. These associations were adjusted for the multiple confounders age, race, depression, education, BMI, health status, history of stroke, history of hypertension, IADL impairments, smoking, alcohol use, caffeine intake, antidepressant use, and physical activity.

Table 3. Association Between Sleep and Continuous Cognitive Measures

Predictor	Unit	Percent Difference* (95% Confidence Interval) Age-Adjusted	Multivariate-Adjusted†
Trail Making B Test, s			
Total sleep time	½ hour	−0.42 (−0.63, 0.26)	−0.34 (−0.38, 0.53)
Sleep latency	½ hour	3.89 (1.84, 7.46)	2.45 (0.59, 7.96)
Sleep efficiency, per *SD*	11.9%	−7.13 (−9.21, −5.30)	−5.05 (−8.72, −2.69)
Sleep efficiency <70%	1	15.86 (7.99, 29.45)	9.15 (2.41, 28.73)
Wake after sleep onset	½ hour	4.91 (2.65, 8.71)	3.45 (1.12, 9.78)
Total nap time	½ hour	2.52 (1.22, 4.81)	1.84 (0.51, 5.68)
≥2 h of napping	1	13.41 (6.26, 26.17)	10.04 (2.73, 31.28)
MMSE score			
Total sleep time	½ hour	0.13 (0.02, 0.26)	0.17 (0.05, 0.33)
Sleep latency	½ hour	−0.86 (−1.00, −0.69)	−0.75 (−0.84, −0.61)
Sleep efficiency, per *SD*	11.9%	1.20 (0.83, 1.60)	1.08 (0.67, 1.63)
Sleep efficiency <70%	1	−2.31 (−2.80, −1.73)	−1.91 (−2.26, −1.38)
Wake after sleep onset	½ hour	−0.60 (−0.73, −0.46)	−0.52 (−0.61, −0.39)
Total nap time	½ hour	−0.28 (−0.38, −0.15)	−0.22 (−0.30, −0.09)
≥2 h of napping	1	−1.52 (−2.08, −0.85)	−1.22 (−1.68, −0.56)

Notes: Sleep efficiency = percentage of time sleeping while in bed; sleep latency = minutes until the onset of sleep; wake after sleep onset = minutes of time scored as awake from the onset of sleep to the end of the sleep interval. *SD* = standard deviation; MMSE = Mini-Mental State Examination.

*Percent difference is 100 * (beta coefficient/mean of cognitive outcome).

†Adjusted for age, race, depression, education, body mass index, health status, history of stroke, history of hypertension, functional status, smoking, alcohol use, caffeine, antidepressant use, and physical activity. $p < .05$ for all but total sleep time for Trail Making B Test time.

Cognitive Impairment and Sleep Parameters

Cognitive impairment (Trails B > 278 seconds, MMSE < 26) was examined to test whether the associations between sleep parameters and cognition were clinically

significant. For each half-hour increase in sleep latency, women had a 1.13-fold increase in risk of cognitive impairment as measured by the Trails B (OR = 1.13; 95% CI, 1.04–1.24) and a 23% increase in risk as measured by MMSE (OR = 1.23; 95% CI, 1.13–1.33). When compared with women with sleep efficiency ≥70%, those with <70% had a nearly 2-fold increase in risk for those with Trails B > 278 seconds (OR = 1.96; 95% CI, 1.43–2.67) and a 61% higher risk of cognitive impairment using the MMSE criteria (OR = 1.61; 95% CI, 1.20–2.16). For each 30-minute increase in WASO, women had a 24% increased risk of cognitive impairment as measured by the Trails B (OR = 1.24; 95% CI, 1.15–1.34) and a 15% increase in risk as measured by MMSE < 26 (OR = 1.15; 95% CI, 1.06–1.23). Those women who napped 2 or more hours a day had an increased risk of cognitive impairment (Trails B: OR = 1.74, 95% CI, 1.26–2.40; MMSE: OR = 1.42, 95% CI, 1.05–1.93). All models were adjusted for covariates described above.

Subsetting to Women Without Possible Dementia

Results for the association between Trails B and sleep were similar after removing 65 women from the analyses with possible dementia ($p < .05$ except for total sleep time); however, the associations were slightly smaller in magnitude. Results for continuous MMSE scores were also similar after removing these women with possible dementia from the analyses.

Discussion

This cross-sectional analysis of 2932 older community-dwelling women showed a significant association between actigraphic measures of sleep and wake activity and cognition, independent of multiple confounders. In addition, these associations remained significant after removing women with possible dementia. Disturbed sleep (increased WASO, lower sleep efficiency, lower sleep latency) was consistently related to poorer cognition, whereas total sleep time was not. This finding may suggest that it is disturbance of sleep rather than quantity that affects cognition. To our knowledge, this is the first large study on community-dwelling older women to evaluate the relationship between cognitive function and objective sleep parameters gathered by actigraphy.

These results are supported by other studies examining the cross-sectional relationship between cognition and sleep, objectively measured by PSG. Both found no association between total sleep time and cognition, but did find a significant relationship with sleep efficiency (8,17). Other studies did not find similar relationships between sleep quality and cognition. Cognition was not related to WASO or total sleep time in a study of 39 participants with confirmed obstructive sleep apnea (16). This lack of association may be due to small sample size, lack of adjustment for potential confounders, or the fact that the cohort all had confirmed OSA. Two large epidemiologic studies gathered data on sleep disturbances by self-report, and examined the cross-sectional relationship of these in relation to cognitive problems. Both found an inverse relationship: Those participants reporting insomnia had better cognition (2,45). Both studies presented unadjusted results based on self-reported insomnia data.

The mechanism behind this association of sleep parameters and cognitive function was not identified. Multivariate adjustment suggests that these results are not explained by other covariates, which included depression, history of hypertension, and history of stroke. The association is also not explained by women with possible dementia, because results were similar when subset to those women without possible dementia.

This analysis had a number of strengths. There was a large cohort of community-dwelling older women with no inclusion requirements regarding sleep disorders or cognition. There were significant associations with two different tests of cognition, one global test and one test of executive function. Several different sleep parameters were examined, all of which were gathered objectively, and there were extensive data for adjustment of possible confounders. It was examined whether the association found between cognition and sleep quality was being driven by the women with the worst cognitive functioning by subsetting the analysis to those women without possible dementia, and associations remained significant. This result suggests that even moderate cognitive problems are associated with sleep quality.

This analysis also had several limitations. The use of self-reported time in and out of bed may be inaccurate, and could introduce error to those variables that depend on these times as part of the calculation (sleep latency and sleep efficiency). Findings are for older women and may not be generalizable to other populations such as men or younger women. The direction of the relationship, whether poor sleep predicts cognitive decline or lower cognitive functioning predicts worsening of sleep, was unable to be examined because the current analysis was cross-sectional. The 1795 women in the SOF at this visit who did not have cognitive function or actigraphy measured were significantly different than this analysis population for many characteristics, including reporting being told by their doctor that they had Alzheimer's disease at a rate 6 times higher than this analysis cohort. The exclusion of these women from the analysis may bias the results towards the null hypothesis of no association.

Conclusion

These findings suggest there is a cross-sectional association between sleep and cognitive function in older community-dwelling women. Further study is needed to examine if this relationship holds longitudinally for cognitive decline and to examine the direction of the association.

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