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Poor Sleep Quality and Functional Decline in Older Women

Adam P. Spira, Ph.D.[1], **Kenneth Covinsky, MD, MPH**[2], **George W. Rebok, Ph.D.**[1], **Naresh M. Punjabi, MD, Ph.D.**[3], **Katie L. Stone, Ph.D.**[4], **Teresa A. Hillier, MD, MS**[5], **Kristine Ensrud, MD, MPH**[6], and **Kristine Yaffe, MD**[7]

[1]Department of Mental Health, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD

[2]Division of Geriatrics, Department of Medicine, University of California, San Francisco & San Francisco VA Medical Center, San Francisco, CA

[3]Division of Pulmonary and Critical Care Medicine, Department of Medicine, Johns Hopkins School of Medicine & Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD

[4]California Pacific Medical Center Research Institute, San Francisco, CA

[5]Kaiser Permanente Center for Health Research, Portland, OR

[6]Department of Medicine, Minneapolis VAMC & Department of Medicine and Division of Epidemiology and Community Health, University of Minnesota, Minneapolis, MN

Corresponding author: Adam P. Spira, Ph.D., 624 N. Broadway, Hampton House, Rm. 794, Baltimore, MD 21205. Tel: (410) 614-9498; Fax: (410) 614-7469; aspira@jhsph.edu. Alternate corresponding author: Kristine Yaffe, MD, 4150 Clement St., San Francisco, CA 94121. Tel: (415) 221-4810 x3985; Fax: (415) 750-6641.

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Author Contributions:
Dr. Adam Spira: developed study concept and design, analyzed and interpreted data, prepared manuscript.
Dr. Kenneth Covinsky: developed study concept and design, and participated in data analysis and interpretation, and in manuscript preparation.
Dr. George Rebok: participated in data analysis and interpretation, and in manuscript preparation.
Dr. Naresh Punjabi: participated in data analysis and interpretation, and in manuscript preparation.
Dr. Katie Stone: developed study concept and design, participated in data analysis and interpretation, and in manuscript preparation.
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[7]Departments of Psychiatry, Neurology, and Epidemiology and Biostatistics, University of California, San Francisco & San Francisco VA Medical Center

Abstract

OBJECTIVES—To determine whether objectively measured sleep quality predicts five-year incident instrumental activities of daily living (IADL) impairment and decline in grip strength and gait speed in older women.

DESIGN—Prospective cohort

SETTING—Participants' homes, Study of Osteoporotic Fractures sites

PARTICIPANTS—817 women (mean 82.4 years at baseline)

MEASUREMENTS—Participants completed 4.1 ±0.7 nights of wrist actigraphy at baseline, and measures of IADL impairment, grip strength, and gait speed at baseline and five-year follow-up.

RESULTS—After five years of follow-up, approximately 41% of participants had incident impairment in ≥1 IADL. The quartile of women with the shortest total sleep time had a 93% greater odds of incident IADL impairment than the longest sleepers (adjusted odds ratio (AOR) = 1.93, 95% confidence interval (CI) 1.25, 2.97). Similarly, the quartile of women with the lowest sleep efficiency had a 65% greater odds of impairment than those with the highest (AOR = 1.65, 95% CI 1.06, 2.57). Women in the shortest total sleep time quartile had double the odds of declining grip strength, compared to those with the longest total sleep time (AOR = 1.97, 95% CI 1.17, 3.32). Finally, women in the quartiles with the most wake after sleep onset and the lowest sleep efficiency had an approximately 90% greater odds of grip strength decline than those with the least wake after sleep onset (AOR = 1.90, 95% CI 1.11, 3.24) and sleep efficiency (AOR = 1.92, 95% CI 1.12, 3.29).

CONCLUSION—Findings indicate that shorter sleep duration, greater wake after sleep onset, and lower sleep efficiency are risk factors for functional or physical decline in older women.

Keywords

sleep; actigraphy; function; IADLs; women

INTRODUCTION

Poor sleep quality is highly prevalent among older adults. Epidemiologic research suggests that approximately half of elders have a complaint about their sleep.[1, 2] In addition, preliminary evidence from a few studies suggests that suggests that poor sleep is associated with functional impairment in the elderly.[1, 3–8] However, there are several limitations to existing studies of sleep and functioning. First, the cross-sectional designs of most of these studies preclude inferences about direction—whether poor sleep quality precedes and might therefore be a cause of self-reported functional decline, or vice versa. Second, most studies have used self-report measures of sleep, rather than objective measures like polysomnography (PSG), or actigraphy—a method of measuring sleep by recording wrist movement. Objective and subjective sleep measures commonly disagree in older adults,[9] and while self-report of sleep can be influenced by psychopathology or cognitive impairment,[9, 10] objective measures of sleep should be less affected by these biases. Third, most studies have focused on self-reported functioning. Reports of difficulty completing basic activities and performance-based measures, such as grip strength and gait speed, assess different domains of functioning. It is important to examine the impact of sleep in both of these domains.

A cross-sectional study of actigraphically measured sleep and functional outcomes in older women in the Study of Osteoporotic Fractures (SOF) reported that women with the longest total sleep time (≥7.5 hours) had a greater odds of self-reported functional impairment than those who slept between 6.8 and 7.5 hours, and that greater wake after sleep onset was associated with a greater odds of impairment.[4] The authors of that study also found that actigraphic sleep parameters were associated with differences in performance-based measures of physical functioning. For example, women with the shortest sleep duration and those with the greatest wake after sleep onset had the slowest gait speed. Moreover, those with the greatest wake after sleep onset had stronger grips than those with the least.[4]

In the present prospective study, also of older women in the SOF cohort, we determined the association between poor actigraphic sleep quality and both incident IADL impairment and decline in performance-based measures of physical function.

METHODS

Study Sample

Participants were women enrolled in the Study of Osteoporotic Fractures (SOF), a prospective cohort study of 9,704 women aged 65 and older recruited in 1986–1988 from population-based listings in Baltimore, MD; Minneapolis, MN; Monongahela Valley/ Pittsburgh, PA; and Portland, OR. Women were recruited irrespective of bone mineral density and fracture history; those unable to walk without assistance or with bilateral hip replacements were excluded. Since initial recruitment, participants have been followed continuously, with at least biannual contact. Between 1997 and 1998, 662 African-American women were also recruited. The present study uses data from the Year-16 SOF visit (2002 to 2004) and the Year-20 visit (2006 to 2008). A total of 4,727 women participated in the year-16 visit, which we refer to as “baseline,” for the present study. Of these, 1,009 women were from the Baltimore site, which did not participate in the five-year follow-up visit. Thus, these women were excluded from our sample. Of the 3,718 women from other sites, 2,570 had actigraphy data for at least one of the three sleep parameters of interest (see below), and 2,509 of these women also had complete IADL data at baseline. We excluded 1,131 women with any IADL difficulty at baseline, and 45 more reporting a history of Alzheimer’s disease (AD) or using an AD medication at baseline, or missing data for these variables. Of the remaining 1,333, a total of 1,048 participated in the follow-up visit. Of the 285 women who did not participate in the follow-up visit, 233 were confirmed to have died (by death certificate), 2 were believed to have died based on proxy report (but this had not yet been confirmed), 26 had terminated participation, and 24 were unaccounted for.

Of the 1,048 women who participated in follow-up, 221 only completed questionnaires and therefore did not provide follow-up IADL data, and ten more were missing follow-up data. Thus, we studied the remaining 817 with complete IADL data at follow-up. Of these, 782 had complete data on grip strength at baseline, and 762 also had complete grip strength data at follow-up. Similarly, 806 women with complete IADL data at both study visits had gait speed data at baseline, and 776 of these had gait speed data at follow-up. Reasons for missing data included incomplete responses to IADL-related questions or refusal or inability to complete physical measures. All women provided written informed consent, and all study protocols were approved by the IRB at each institution.

Assessment of Sleep Quality

At baseline, participants completed wrist actigraphy, a method of quantifying sleep by recording movement with an actigraph. Participants were instructed to wear actigraphs (SleepWatch-O, Ambulatory Monitoring, Inc., Ardsley, NY) on the non-dominant wrist for

at least three consecutive 24-hour periods. On average, they wore actigraphs for 4.1 ± (standard deviation) 0.7 nights (median = 4.0). Data were recorded in 1-minute epochs in three different modes: zero crossing; time above threshold; and proportional integration mode (PIM). A prior investigation in older women from the SOF cohort who had data from both actigraphy and polysomnography found that data collected in PIM were most highly correlated with those collected via polysomnography.[11] Thus, we analyzed the data collected using PIM. We used three standard sleep parameters, extracted using Action W-2 software (Ambulatory Monitoring, Inc.). To facilitate editing of actigraphic data, participants completed sleep diaries while wearing actigraphs. They reported a range of variables, including time into bed, time that they attempted to go to sleep (referred to here as "lights off"), time out of bed, and times that the actigraph was removed. Each parameter was averaged across nights of actigraphy: total sleep time (TST; total number of minutes in bed spent asleep following "lights off"); wake after sleep onset (WASO; total number of minutes in bed spent awake after the first 20-minute block of continuous sleep that followed "lights off"); and sleep efficiency (SE; percent of time in bed following "lights off" spent asleep). Details on actigraphy scoring in SOF have been published previously.[12]

Measures of Functional Decline

IADLs were measured at baseline and at follow-up using items from, or adapted from, the 1984 National Health Interview Survey's Supplement on Aging.[13] Participants were asked whether they had any difficulty preparing their meals, doing heavy housework, or shopping for groceries or clothes, whether any reported difficulty was due to a physical or health problem, and the degree of difficulty they had. Because the analytical sample consisted of women whose responses to these questions indicated no difficulty in these three domains at baseline, incident IADL impairment was defined as any difficulty in one or more of these domains at five-year follow-up, based on responses to these questions.

At baseline and at follow-up, participants also completed performance-based measures of physical functioning, including grip strength (kg, measured by hand dynamometer) and gait speed (time to walk 6 meters).[14] Grip strength was calculated by computing the mean strength over two trials in the left hand, then in the right hand, and then computing the mean of those two means. Gait speed was calculated by averaging over two trials at each visit.

Additional Measures

Upon enrollment in SOF, demographic data were collected from each participant. At Year-16 participants completed questionnaires regarding history of medical comorbidities and health behaviors (e.g., smoking, alcohol and caffeine use, exercise). Height was measured by stadiometer with participants barefoot (or in thin socks); weight was measured by balance beam scale; body mass index (BMI; kg/m^2) was calculated from these measures. Participants were asked to bring all medications and dietary supplements taken over the preceding 30 days to the Year-16 study visit, and medications were classified by brand or generic name using a computerized dictionary.[15] General cognitive performance was assessed by the Mini-Mental State Examination[16] (MMSE). Depressive symptoms were measured by the 15-item Geriatric Depression Scale[17] and anxiety symptoms were measured by the Goldberg Anxiety Scale.[18]

Statistical Analyses

To determine the association between sleep parameters and incident IADL impairment, we conducted bivariate and multivariable-adjusted logistic regression analyses. Each model contained quartiles of one actigraphic sleep parameter as the primary predictor. Reference quartiles were those with the longest TST, least WASO, and greatest SE. Incident IADL impairment was the outcome. To account for the non-linear association between age and

incident IADL impairment, we added a quadratic term (age^2) to multivariable models with this outcome.

To determine the association between sleep parameters and decline in physical performance, we conducted additional analyses, with quartiles of sleep parameters as the primary predictors and substantial decline in either grip strength or gait speed as the outcome. To identify decliners, we subtracted performance at follow-up from performance at baseline, and considered women in the highest quartile of change to have declined on that measure.

To account for physical performance at baseline, models for decline in grip strength and gait speed were adjusted for baseline performance on these measures. Additional potential confounders were selected for inclusion in fully adjusted models if they were associated with quartiles of any sleep parameter and any of our three dichotomous outcomes at the $p \leq 0.10$ level, according to Kruskal-Wallis tests for continuous variables and χ^2 or Fisher's exact tests for categorical variables. Specifically, multivariable models included age, obesity (BMI <30, ≥30),[19] MMSE score, 15-item Geriatric Depression Scale score, Goldberg Anxiety Scale score, antidepressant use, history of diabetes, and history of coronary artery disease (angina or myocardial infarction). We conducted tests for linear trend across quartiles of sleep parameters.[20] Continuous predictors were centered at the mean in all regression models. We excluded two participants who did not complete all GDS items from analyses involving fully adjusted models.

To explore the possibility of a U-shaped association between TST and our outcomes, we fitted models with TST entered as a continuous predictor, and compared these to models with TST entered as both a continuous predictor and as a quadratic term (TST^2) using likelihood ratio χ^2 tests. Based on non-significant results, and on visual inspection of LOWESS plots of continuously measured TST against the log odds of our outcomes, we determined that there was little support for a U-shaped association.

An $\alpha < 0.05$ was selected for statistical significance. Analyses were performed using Stata 10.1 (StataCorp, College Station, TX).

RESULTS

The mean age ± standard deviation (SD) of the study sample was 82.4 ±3.3 years (range 70 – 94) at baseline, and 87.3 ±3.3 years (range 75 – 99) at 5-year follow-up. Of the 817 women, 103 (12.6%) were non-white and 325 women (39.8%) had education beyond high school. On average, they slept for a total of 409.2±66.0 minutes, spent 65.9 ±40.4 minutes awake after initial sleep onset, and had a sleep efficiency of 79.9% ±9.9% (Table 1). Across TST quartiles, women differed significantly by age, race, BMI, and antidepressant use (Table 2). In addition, women in different WASO quartiles differed by race, BMI, and depressive symptoms, and those in different sleep efficiency quartiles differed by race, BMI, MMSE score, and depressive symptoms (all $p < 0.05$). The mean interval between baseline and follow-up visits was 4.9 ±0.6 years in duration (range 3.5 – 6.3).

When we compared the women in our analysis sample to those who participated in SOF Visit 8 but were excluded from our sample, the excluded women were older, more likely to be white, consumed less alcohol and less caffeine, had more depressive symptoms and anxiety symptoms, were less likely to walk for exercise, were more likely to take an antidepressant or benzodiazepine medication, and to have a history of diabetes, coronary artery disease, congestive heart failure, COPD, and osteoarthritis (all $p < 0.05$). They did not differ by educational attainment (≤ high school vs. > high school), smoking status, obesity status, or history of hypertension.

Incident IADL Impairment

Of the 817 participants, 333 (40.8%) reported incident impairment in ≥1 IADL at follow-up. Specifically, 285 (34.9%) had incident impairment in their ability to do heavy housework, 177 (21.7%) in their ability to shop, and 60 (7.3%) in their ability to prepare meals..

In unadjusted logistic regression analyses, shorter TST was associated with a greater risk of incident IADL impairment, compared to longer TST (Table 3). Those in the quartile with the shortest TST had an 86% increase in the odds of incident impairment (odds ratio (OR) = 1.86, 95% confidence interval (CI) 1.25, 2.77), compared to those in the quartile with the longest TST. In addition, there was a significant linear association between shorter sleep duration and odds of incident IADL impairment across TST quartiles (p for trend = 0.002). These associations remained after adjustment for age, age^2, obesity, MMSE score, 15-item Geriatric Depression Scale score, Goldberg Anxiety Scale score, antidepressant use, history of diabetes, and history of coronary artery disease (OR = 1.93, 95% CI 1.25, 2.97; p for trend = 0.004).

In unadjusted analyses, we observed a similar association between WASO and incident IADL impairment (Table 3). Women in the highest quartile of WASO had a 74% higher odds of incident impairment than those with the least WASO (OR = 1.74, 95% CI 1.17, 2.59). In addition, there was a positive linear association between the amount of WASO and the odds of incident IADL impairment (p for trend = 0.004). This association was no longer significant after multivariable adjustment (OR = 1.46, 95% CI 0.94, 2.26; p for trend = 0.096).

Lower sleep efficiency also was associated with an increased risk of incident IADL impairment (Table 3). In unadjusted analyses, compared to women with the highest sleep efficiency, those in the quartile with the lowest sleep efficiency had almost a 90% increase in the odds of incident impairment (OR = 1.88, 95% CI 1.26, 2.81), and those in the quartile with the second-lowest sleep efficiency had a 69% higher odds of incident disability (OR = 1.75, 95% CI 1.17, 2.62). There was an inverse, linear association between level of sleep efficiency and odds of impairment (p for trend < 0.001). These associations decreased slightly after adjustment but remained statistically significant.

Decline in Performance-Based Measures of Physical Functioning

On average, participants' grip strength weakened by 2.4 ± 3.6 kg from baseline to follow-up, and their gait speed slowed by 0.2 ± 0.2 m/second. Based on the quartile definition described above, 182 (23.9%) of women had substantial decline (>4.75 kg of weakening) in grip strength, and 194 (25.0%) of women had substantial decline (>0.269 m/second of slowing) in gait speed.

In analyses adjusted for baseline grip strength, women with the shortest TST at baseline had an 87% greater odds of substantial weakening in grip strength between baseline and follow-up, compared to women with the longest TST (OR = 1.87, 95% CI 1.13, 3.08) (Table 4). There was a linear association across TST quartiles, such that shorter TST was associated with a greater odds of weakened grip strength (p for trend = 0.034). These associations remained after further adjustment for age, obesity, MMSE score, 15-item Geriatric Depression Scale score, Goldberg Anxiety Scale score, antidepressant use, history of diabetes, and history of coronary artery disease (OR = 1.97, 95% 1.17, 3.32; p for trend = 0.029).

Greater WASO also was associated with an increased odds of substantial decline in grip strength. After adjusting for baseline grip strength, women in the third and fourth quartiles of WASO had a 76% (OR = 1.76, 95% CI 1.05, 2.94) and 80% (OR = 1.80, 95% CI 1.08,

3.00) greater odds of grip strength weakening, respectively, compared to women in the lowest WASO quartile. There was a linear association between greater WASO and greater odds of substantial weakening in grip strength (p for trend = 0.014). These associations remained in the fully adjusted model (third quartile OR = 1.86, 95% CI 1.09, 3.16; fourth quartile OR = 1.90, 1.11, 3.24; p for trend = 0.015).

Lower sleep efficiency also was associated with an increased risk of weakened grip strength. In analyses adjusted for baseline grip strength, women in the quartile with the lowest sleep efficiency had a 90% greater odds of weakening than those with the highest sleep efficiency (OR = 1.90, 95% CI 1.14, 3.17). Similarly, women in the quartile with the second-lowest sleep efficiency had a 67% greater odds of substantial weakening, compared to those with the highest sleep efficiency, although this association did not reach significance (OR = 1.67, 95% CI 0.997, 2.80). The association between lower sleep efficiency and decline in grip strength increased in a linear fashion across quartiles of sleep efficiency (p for trend = 0.010). These associations remained after adjustment (OR = 1.92, 95% CI 1.12, 3.29; p for trend = 0.016).

We observed no associations between sleep parameters and substantial decline in gait speed. For example, in the fully adjusted model, women with the shortest sleep showed no significant increase in the odds of slowing, compared to those with the longest sleep (OR = 1.18, 95% CI 0.71, 1.95) and there was no linear trend across quartiles of TST (p for trend = 0.840). Similarly, in fully adjusted analyses, women with the greatest WASO had no greater odds of slowing than women in the lowest WASO quartile (OR = 1.05, 95% CI 0.63, 1.76); there was no significant linear trend across WASO quartiles (p for trend = 0.633). Further, in fully adjusted analyses, women with the lowest SE showed no increase in the odds of slowing, compared to those with the highest (OR = 1.21, 95% CI 0.72, 2.03), and there was no linear trend across quartiles of SE (p for trend = 0.404).

DISCUSSION

In this prospective study of older women without IADL impairment at baseline, we determined the association between objective sleep quality, measured by actigraphy, and both incident self-reported IADL impairment and decline in performance-based measures of physical function over five years. After adjustment for several potential confounders, we found that shorter TST and lower SE each were associated with an increase in the odds of incident IADL impairment. In addition, we observed independent associations between all three sleep parameters—TST, WASO, and SE—and decline in grip strength. In contrast, we observed no association between these sleep parameters and decline in gait speed.

Our finding that poor sleep is associated with incident IADL impairment is generally consistent with results of other studies in this domain. In addition to the prior study in the SOF cohort described above,[4] a cross-sectional study of more than 9,000 older adults found that elevated scores on a self-report measure of poor nighttime sleep and related daytime complaints were associated with self-reported impairment in ADLs and related tasks.[1] Cross-sectional findings from the Cardiovascular Health Study indicated that self-reported IADL impairment was independently associated with complaints of frequent awakenings and with daytime sleepiness among older women.[5] In addition, a recent study of Finnish adults found that short self-reported sleep duration was associated with impairments in women aged ≥65 years, and among men aged 55 to 64 years.[8]

With respect to our findings regarding sleep disturbance and performance-based functional measures, the cross-sectional study in SOF mentioned above found that short actigraphic sleep duration was associated with slower gait speed, but found no association between

sleep parameters and grip strength.[4] In addition, a cross-sectional study in a cohort of almost 2,900 older men in the Osteoporotic Fractures in Men Study (MrOS) found that elevated levels of actigraphic sleep fragmentation (i.e., SE <80%, WASO ≥90 minutes) were associated with decreased grip strength and gait speed, and that a U-shaped association existed between TST and grip strength; older men with short or long sleep had weaker grip strength than men who obtained six to eight hours of sleep.[7] These cross-sectional associations contrast with our null findings for the prospective association between sleep duration and gait speed, and with the robust association we observed between sleep disturbance and subsequent weakening of grip strength in the same cohort. Thus, short sleep duration might be a consequence of slowing gait speed, rather than a cause, and short sleep duration, greater sleep fragmentation, and lower sleep efficiency predict, and might cause a weakening of grip strength.

As one of the first prospective studies to link objectively measured sleep disturbance to incident IADL impairment and decline in performance-based physical function in a large sample of older adults, our findings have important implications for older adults' quality of life, and potentially for their healthcare. First, our results suggest that short sleep and sleep fragmentation may be risk factors for functional decline and disability among functionally intact older women. Although further research is needed to more firmly establish this longitudinal association, additional consistent findings could prompt greater attention by clinicians to older adults' sleep. In addition, others have suggested that interventions aimed at maintaining or improving sleep quality might promote health-related quality of life in older adults,[21] and our findings raise the possibility that such interventions might help delay or prevent disability. Replication of our findings would pave the way for randomized controlled trials of interventions for poor sleep quality, with the goal of preventing functional decline.

A range of variables might explain the associations we observed between poor sleep and functional decline in our simpler statistical models, including particular medical co-morbidities, cognitive status, symptoms of depression or anxiety, and certain medications. However, we adjusted for these factors in our multivariable analyses when indicated, and were able to demonstrate independent effects. Although residual confounding by these variables could account for our results, another factor that might explain the associations we observed is inflammation. Poor sleep has been linked to inflammatory processes,[22, 23] which are associated with muscle loss and functional decline.[24, 25] Studies are needed that investigate inflammation as a mediator of the association between objectively measured sleep and functional decline.

The present study has multiple strengths, including a prospective design, actigraphic sleep assessment, and both subjective and objective measures of functioning. However, it also has limitations. First, the sample consisted entirely of women, the majority of whom were white. In addition, because these women are the surviving volunteers from a cohort study that began in 1986, they are likely to be healthier on average, than the general population of older women. Thus, further research is needed to determine whether our results apply to men, to women from ethnically diverse backgrounds, and to the general population of older women. Finally, the cutoffs we used for decline in grip strength and gait speed were based on the distribution of these variables in our sample (highest quartile of change in each variable), rather than another objectively measured criterion variable. Therefore, our definition of decline captured women with the greatest change on these measures, but the precise clinical significance of our definition of decline cannot be determined.

CONCLUSION

Findings from the present study suggest that shorter total sleep time and lower sleep efficiency are associated with an increased risk of incident IADL impairment, and that these factors—along with greater wake after sleep onset—predict substantial decline in grip strength over five years in older women. Further research is needed to confirm these findings and identify the mechanisms linking poor sleep to these outcomes.

Given the prevalence of sleep disturbance in older adults, our findings indicate that a substantial proportion of the population of elders is at elevated risk of sleep-related disability. To the extent that sleep disturbance is causally related to functional decline and disability, improvement of older adults' sleep might help maintain their functioning, independence, and quality of life. As the evidence for adverse health consequences of late-life sleep disturbance accumulates, prevention trials will be needed to determine whether improvement of poor sleep protects health and quality of life in the growing population of older adults.

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References

1. Foley DJ, Monjan AA, Brown SL, et al. Sleep complaints among elderly persons: an epidemiologic study of three communities. Sleep. 1995; 18:425–432. [PubMed: 7481413]
2. Ganguli M, Reynolds CF, Gilby JE. Prevalence and persistence of sleep complaints in a rural older community sample: The MoVIES project. J Am Geriatr Soc. 1996; 44:778–784. [PubMed: 8675924]
3. Ohayon MM, Vecchierini MF. Normative sleep data, cognitive function and daily living activities in older adults in the community. Sleep. 2005; 28:981–989. [PubMed: 16218081]
4. Goldman SE, Stone KL, Ancoli-Israel S, et al. Poor sleep is associated with poorer physical performance and greater functional limitations in older women. Sleep. 2007; 30:1317–1324. [PubMed: 17969465]
5. Newman AB, Enright PL, Manolio TA, et al. Sleep disturbance, psychosocial correlates, and cardiovascular disease in 5201 older adults: The Cardiovascular Health Study. J Am Geriatr Soc. 1997; 45:1–7. [PubMed: 8994480]
6. Quan SF, Katz R, Olson J, et al. Factors associated with incidence and persistence of symptoms of disturbed sleep in an elderly cohort: The Cardiovascular Health Study. Am J Med Sci. 2005; 329:163–172. [PubMed: 15832098]
7. Dam TT, Ewing S, Ancoli-Israel S, et al. Association between sleep and physical function in older men: The Osteoporotic Fractures in Men Sleep Study. J Am Geriatr Soc. 2008
8. Stenholm S, Kronholm E, Sainio P, et al. Sleep-related factors and mobility in older men and women. J Gerontol A Biol Sci Med Sci. 2010; 65:649–657. [PubMed: 20159778]
9. Van Den Berg JF, Van Rooij FJ, Vos H, et al. Disagreement between subjective and actigraphic measures of sleep duration in a population-based study of elderly persons. J Sleep Res. 2008; 17:295–302. [PubMed: 18321246]

10. Rotenberg VS, Indursky P, Kayumov L, et al. The relationship between subjective sleep estimation and objective sleep variables in depressed patients. Int J Psychophysiol. 2000; 37:291–297. [PubMed: 10858574]
11. Blackwell T, Redline S, Ancoli-Israel S, et al. Comparison of sleep parameters from actigraphy and polysomnography in older women: the SOF study. Sleep. 2008; 31:283–291. [PubMed: 18274276]
12. Blackwell T, Ancoli-Israel S, Gehrman PR, et al. Actigraphy scoring reliability in the study of osteoporotic fractures. Sleep. 2005; 28:1599–1605. [PubMed: 16408420]
13. Fitti J, Kovar M. The Supplement on Aging to the 1984 National Health Interview Survey. Vital Health Stat 1. 1987; 21:1–115. [PubMed: 3672938]
14. Cummings SR, Nevitt MC, Browner WS, et al. Risk factors for hip fracture in white women. Study of Osteoporotic Fractures Research Group. N Engl J Med. 1995; 332:767–773. [PubMed: 7862179]
15. Pahor M, Chrischilles EA, Guralnik JM, et al. Drug data coding and analysis in epidemiologic studies. Eur J Epidemiol. 1994; 10:405–411. [PubMed: 7843344]
16. Folstein MF, Folstein SE, McHugh PR. Mini-mental state: A practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res. 1975; 12:189–198. [PubMed: 1202204]
17. Sheikh JI, Yesavage JA. Geriatric Depression Scale: Recent evidence and development of a shorter version. Clin Gerontol. 1986; 5:165–173.
18. Goldberg D, Bridges K, Duncan-Jones P, et al. Detecting anxiety and depression in general medical settings. BMJ. 1988; 297:897–899. [PubMed: 3140969]
19. National Institutes of Health. The Practical Guide: Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. 2000.
20. Vittinghoff, E.; Glidden, DV.; Shiboski, SC., et al. Regression Methods in Biostatistics: Linear, Logistic, Survival, and Repeated Measures Models. NY: Springer; 2005.
21. Driscoll HC, Serody L, Patrick S, et al. Sleeping well, aging well: A descriptive and cross-sectional study of sleep in “successful agers” 75 and older. Am J Geriatr Psychiatry. 2008; 16:74–82. [PubMed: 18070833]
22. Irwin MR, Wang M, Campomayor CO, et al. Sleep deprivation and activation of morning levels of cellular and genomic markers of inflammation. Arch Intern Med. 2006; 166:1756–1762. [PubMed: 16983055]
23. Irwin MR, Wang M, Ribeiro D, et al. Sleep loss activates cellular inflammatory signaling. Biol Psychiatry. 2008; 64:538–540. [PubMed: 18561896]
24. Cohen HJ, Harris T, Pieper CF. Coagulation and activation of inflammatory pathways in the development of functional decline and mortality in the elderly. Am J Med. 2003; 114:180–187. [PubMed: 12637131]
25. Lee CE, McArdle A, Griffiths RD. The role of hormones, cytokines and heat shock proteins during age-related muscle loss. Clin Nutr. 2007; 26:524–534. [PubMed: 17590243]

Table 1

Descriptive Statistics (Mean ± Standard Deviation) for Actigraphic Sleep Parameters.

	Full Sample	Quartile 1	Quartile 2	Quartile 3	Quartile 4
Total sleep time (minutes)	409.2 ± 66.0	486.4 ± 30.3	432.7 ± 10.2	395.3 ± 13.3	323.3 ± 46.0
n	817	204	202	206	205
Wake after sleep onset (minutes)	65.9 ± 40.4	27.3 ± 7.3	47.1 ± 5.2	67.9 ± 6.5	121.6 ± 38.0
n	816	204	204	204	204
Sleep efficiency (%)	79.9 ± 9.9	89.8 ± 2.4	84.2 ± 1.3	79.0 ± 2.0	66.8 ± 9.7
n	817	204	204	204	205

Table 2

Participant Characteristics (Mean ± Standard Deviation or *n* (%)) across Total Sleep Time Quartiles.

	Total Sleep Time				*p*-value
	Quartile 1 ***n*** **= 204**	**Quartile 2** ***n*** **= 202**	**Quartile 3** ***n*** **= 206**	**Quartile 4** ***n*** **= 205**	
Age	82.5 ±3.3	82.8 ±3.1	82.4 ±2.9	81.8 ±3.8	0.027
Non-white	19 (9.3)	17 (8.4)	20 (9.7)	47 (22.9)	<0.001
Education > high school	82 (40.2)	81 (40.1)	74 (35.9)	88 (42.9)	0.542
BMI ≥30 (kg/m^2)	38 (18.6)	31 (15.4)	49 (24.0)	64 (31.2)	0.001
Smoker	4 (2.0)	6 (3.0)	4 (1.9)	6 (2.9)	0.848
Alcohol use (# drinks/week)	1.1 ±2.4	1.4 ±3.1	1.0 ±2.2	1.2 ±2.9	0.810
Caffeine intake (g/day)	0.1 ±0.1	0.2 ±0.2	0.2 ±0.2	0.2 ±0.1	0.201
Walks for exercise	94 (46.3)	98 (48.8)	102 (50.8)	94 (46.1)	0.756
MMSE score (0–30)	28.5 ±1.3	28.7 ±1.5	28.4 ±1.5	28.4 ±1.7	0.181
Geriatric Depression Scale (0–15)	1.4 ±1.8	1.5 ±2.1	1.2 ±1.6	1.5 ±1.9	0.304
Goldberg Anxiety Scale (0–9)	1.0 ±1.9	1.1 ±2.1	0.9 ±1.7	1.1 ±1.9	0.785
Antidepressant use	24 (11.8)	10 (5.0)	10 (4.9)	13 (6.3)	0.018
Benzodiazepine use	9 (4.4)	10 (5.0)	12 (5.8)	8 (3.9)	0.822
Diabetes	12 (5.9)	14 (6.9)	25 (12.1)	21 (10.2)	0.093
Hypertension	117 (57.4)	112 (55.5)	114 (55.3)	126 (61.5)	0.561
Coronary artery disease	33 (16.2)	26 (12.9)	30 (14.6)	30 (14.6)	0.827
Congestive heart failure	8 (3.9)	5 (2.5)	10 (4.9)	9 (4.4)	0.628
COPD	16 (7.8)	14 (6.9)	19 (9.2)	23 (11.2)	0.449
Osteoarthritis	55 (27.0)	59 (29.2)	52 (25.2)	56 (27.3)	0.845

Note: *N* ranges from 799 to 817. *p*-values are from Kruskal-Wallis tests (with rank ties) for continuous variables and from χ^2 or Fisher's exact tests for categorical variables. BMI = body mass index; COPD = chronic obstructive pulmonary disease; MMSE = Mini-Mental State Examination.

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Table 3

Associations between Actigraphic Sleep Parameters and Incident IADL Impairment.

Sleep parameters	*n* (%) with incident impairment	Unadjusted OR (95% CI)	MV-adjusted* OR (95% CI)
Total sleep time			
Quartile 1 (longest)	70 (34.3)	(ref)	(ref)
Quartile 2	78 (38.6)	1.20 (0.80, 1.80)	1.26 (0.81, 1.95)
Quartile 3	84 (40.8)	1.32 (0.88, 1.97)	1.34 (0.87, 2.07)
Quartile 4 (shortest)	101 (49.3)	1.86 (1.25, 2.77)	1.93 (1.25, 2.97)
p-value for trend		$p = 0.002$	$p = 0.004$
Wake after sleep onset			
Quartile 1 (least)	69 (33.8)	(ref)	(ref)
Quartile 2	79 (38.7)	1.24 (0.83, 1.85)	1.34 (0.87, 2.06)
Quartile 3	88 (43.1)	1.48 (0.99, 2.22)	1.41 (0.92, 2.17)
Quartile 4 (most)	96 (47.1)	1.74 (1.17, 2.59)	1.46 (0.94, 2.26)
p-value for trend		$p = 0.004$	$p = 0.096$
Sleep efficiency			
Quartile 1 (highest)	66 (32.4)	(ref)	(ref)
Quartile 2	77 (37.8)	1.27 (0.84, 1.91)	1.30 (0.84, 2.00)
Quartile 3	93 (45.6)	1.75 (1.17, 2.62)	1.69 (1.09, 2.60)
Quartile 4 (lowest)	97 (47.3)	1.88 (1.26, 2.81)	1.65 (1.06, 2.57)
p-value for trend		$p < 0.001$	$p = 0.013$

Note: $N = 816–817$ for unadjusted, $N = 794$ for MV-adjusted analyses.

*Adjusted for age, age^2, obesity (body mass index ≥30), Mini-Mental State Examination score, 15-item Geriatric Depression Scale, Goldberg Anxiety Scale, antidepressant use, diabetes, and coronary artery disease. CI = confidence interval; MV = multivariable; OR = odds ratio.

Table 4

Associations between Actigraphic Sleep Parameters and Decline in Grip Strength.*

Sleep parameters	*n* (%) with decline	Model I OR (95% CI)	Model II OR (95% CI)
Total sleep time			
Quartile 1 (longest)	37 (19.4)	(ref)	(ref)
Quartile 2	47 (25.3)	1.54 (0.92, 2.57)	1.59 (0.93, 2.69)
Quartile 3	42 (21.9)	1.32 (0.78, 2.21)	1.32 (0.77, 2.25)
Quartile 4 (shortest)	56 (29.0)	1.87 (1.13, 3.08)	1.97 (1.17, 3.32)
p-value for trend		$p = 0.034$	$p = 0.029$
Wake after sleep onset			
Quartile 1 (least)	35 (18.2)	(ref)	(ref)
Quartile 2	42 (21.8)	1.35 (0.80, 2.29)	1.55 (0.90, 2.66)
Quartile 3	51 (27.4)	1.76 (1.05, 2.94)	1.86 (1.09, 3.16)
Quartile 4 (most)	53 (27.9)	1.80 (1.08, 3.00)	1.90 (1.11, 3.24)
p-value for trend		$p = 0.014$	$p = 0.015$
Sleep efficiency			
Quartile 1 (highest)	35 (18.2)	(ref)	(ref)
Quartile 2	44 (23.0)	1.39 (0.83, 2.35)	1.44 (0.84, 2.46)
Quartile 3	48 (25.8)	1.67 (0.997, 2.80)	1.65 (0.97, 2.82)
Quartile 4 (lowest)	55 (28.5)	1.90 (1.14, 3.17)	1.92 (1.12, 3.29)
p-value for trend		$p = 0.010$	$p = 0.016$

Note $N = 761–762$ for Model I, and $N = 744$ for Model II.

*Decline is defined as >4.75 kg decrease in grip strength from baseline to follow-up. Model I: Adjusted for baseline grip strength. Model II: Adjusted for baseline grip strength, age, obesity (body mass index ≥30), Mini-Mental State Examination score, 15-item Geriatric Depression Scale, Goldberg Anxiety Scale, antidepressant use, diabetes, and coronary artery disease. CI = confidence interval; OR = odds ratio.