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Association between Sleep and Physical Function in Older Men: The MrOS Sleep Study

Thuy-Tien L. Dam, MD[1], **Susan Ewing, MS**[2], **Sonia Ancoli-Israel, PhD**[3], **Kristine Ensrud, MD, MPH**[4], **Susan Redline, MD, MPH**[5], and **Katie Stone, PhD**[6] **for the Osteoporotic Fractures in Men (MrOS) Research Group**

[1]Department of Medicine, University of California San Diego, La Jolla, California

[2]Department of Epidemiology and Biostatistics, University of California San Francisco, San Francisco, California

[3]Department of Psychiatry, University of California San Diego, La Jolla, California

[4]Departments of Epidemiology and Medicine, University of Minnesota and VA Medical Center, Minneapolis, Minnesota

[5]Department of Pediatrics, Case Western Reserve University, Cleveland, Ohio

[6]Research Institute, California Pacific Medical Center, San Francisco, California

Abstract

OBJECTIVES: Determine whether sleep quality is associated with physical function in older men.

DESIGN: Cross-sectional

SETTING: Six U.S. centers

PARTICIPANTS: 2,862 community dwelling men

MEASUREMENTS: Total hours of nighttime sleep [TST], wake after sleep onset [WASO], sleep latency [SL], and sleep efficiency [SE] measured by actigraphy. Sleep stage distribution, respiratory disturbance index (RDI) and hypoxia measured by polysomnography. Measures of physical function including grip strength, walking speed, chair stand and narrow walk were obtained.

RESULTS: In age adjusted models, TST, SE<80%, WASO > 90 min, RDI > 30 and hypoxia were associated with physical function (i.e. mean grip strength was 2.9% and the mean walking speed was 4.3% lower in men with WASO > 90 minutes compared to men with WASO <90 minutes). After adjusting for potential covariates, differences in grip strength and walking speed

Corresponding author: Thuy-Tien L. Dam, MD University of California, San Diego School of Medicine 9500 Gilman Dr. MC-0659 La Jolla, CA Phone: 858-534-8060 Fax: 858-534-4952 Email: tldam@ucsd.edu.

Author Contributions:
Thuy-Tien Dam: lead analysis concept and design, lead interpretation of data and preparation of manuscript
Susan Ewing: completed data analysis, assisted with interpretation of data and preparation of the manuscript
Sonia Ancoli-Israel: assisted with interpretation of the data and preparation of the manuscript
Susan Redline: principal investigator of the Case Western PSG Reading Center assisted with interpretation of the data and preparation of the manuscript
Kris Ensrud: principal investigator, assisted with interpretation of the data and preparation of the manuscript
Katie Stone: lead study concept and design for entire cohort, assisted with interpretation of the data and preparation of the manuscript

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remained significantly associated with WASO > 90 min, SE < 80% and hypoxia, but not with TST or RDI > 30.

CONCLUSION: Increased sleep fragmentation and hypoxia is associated with poorer physical function in older men.

Keywords

sleep; physical function; older men

INTRODUCTION

The proportion of the U.S. population greater than 65 years old is expected to increase from 12% in 2000 to 20% in 2030 (United States Census Bureau), with the largest growth in those 85 years and older. Among those aged 65 years and older, 35 to 40% are disabled or experience some limitation in activities of daily living. Decline in physical function in older adults results in increased falls, fractures, disability, and mortality (1) (2).

In addition, sleep complaints and sleep related problems increase with age. The 2003 National Sleep Foundation survey demonstrated that 67% of adults 65 years old have at least one sleep related complaint (3). Subjective complaint of insomnia is the most frequent sleep disturbance with a prevalence of 10-15% in the United States (4-6). Moreover, increased nighttime awakenings and arousals and decreased “deep” sleep are associated with aging (7). Data from the Sleep Heart Health Study showed that an age-related decline in slow wave sleep was greater in men than women (8). Several factors can contribute to disturbed sleep in elderly, particularly poor sleep hygiene, depression, use of medications, and co-morbidities (9).

The aging U.S. population with an increased prevalence of physical disability and sleep related problems, may place a large burden on health care services and economic resources. Little is known about the relationship between sleep quality and physical function in older adults. Therefore, the aim of this study is to determine whether measures of sleep, including total hours of sleep per night, indices of sleep fragmentation, sleep architecture, and sleep disordered breathing are associated with physical function in a large cohort of elderly men. We hypothesized that impairments in sleep quality, as measured by decreased nighttime sleep, decreased sleep efficiency, increased sleep fragmentation, and increased frequency of sleep related respiratory disturbances, would be associated with poorer physical function as manifested by weaker grip strength, slower walking speed and inability to complete a chair stand or narrow walk course.

METHODS

Participants

From March 2000 to April 2002, the Osteoporotic Fractures in Men Study (MrOS) recruited 5,995 community dwelling men at six clinical centers in the United States (Birmingham, Alabama; Minneapolis, Minnesota; Palo Alto, California; Monongahela Valley near Pittsburgh, Pennsylvania; Portland, Oregon; and San Diego, California). To be eligible to participate, participants must have been community dwelling men at least 65 years of age, without bilateral hip replacements, and able to ambulate without the assistance of another person. Details of the MrOS cohort, design and recruitment have been published elsewhere (10,11).

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During December 2003 to March 2005, participants from the MrOS cohort were invited to participate in the ancillary study titled Outcomes of Sleep Disorders in Older Men (MrOS Sleep Study). To participate in the MrOS Sleep Study, men had to agree to a comprehensive sleep assessment that included validated sleep questionnaires, an in-clinic interview, a series of clinical measures including physical function, a single overnight in-home polysomnography (PSG) study, plus wrist actigraphy. Of the 3135 men who participated in the MrOS Sleep Study, 2862 (91.3%) had both PSG and actigraphy measurements of sufficient quality to be included in the analysis. Of those excluded in the analyses, 179 refused PSG, 45 had PSG studies that did not meet study quality standards, and 49 did not have actigraphy data.

Men were screened for use of mechanical devices during sleep including pressure mask for sleep apnea (CPAP or BiPAP), mouthpiece for snoring or sleep apnea, or oxygen therapy. In general, those who reported nightly use of any of these devices were excluded from the MrOS Sleep Study. However, the study sample includes 17 men who were able to forgo use of their sleep devices during the PSG recording night. The institutional review boards at each clinic site approved the study, and written informed consent was obtained from all participants.

In-home Overnight Polysomnography

Unattended PSG data were collected over one night at the participant's residence using the Sleep Monitoring System (Safiro, Compumedics, Inc). Prior to the participant's bedtime, two trained staff members performed home visits to attach sensors and initiate the PSG data collection, which were modeled after procedures developed for the Sleep Heart Health Study (12). The recording montage consisted of two central electroencephalograms, bilateral electrooculograms; bilateral chin electromyogram, a bipolar ECG, nasal-oral thermistry, nasal flow via pressure transducer and nasal cannula, abdominal and respiratory inductance plethysmography, finger pulse oximetry, bilateral leg movements by piezoelectric sensors, and body position. Data were transmitted to the Case Western PSG Reading Center (Cleveland, OH) for centralized scoring by trained research polysmonolgists.

The research polysomnologists underwent rigorous certification and participated in ongoing quality improvement, with close monitoring of within and between scorer agreement. PSG were graded based on previously described approaches (13). PSG data quality for MrOS was excellent with less than a 4% failure rate; the quality of more than 70% of PSG studies were graded as excellent or outstanding.

The respiratory disturbance index (RDI) was defined as the number of apneas and hypopneas per hour of sleep. Each apnea or hypopnea was associated with at least a 3% oxygen desaturation. Certified scorers blinded to data on the leg movement channels staged sleep from the EEG, EMG and EOG channels using standard criteria. Sleep stage distribution was characterized by the percent of total sleep time spent in each stage of sleep (i.e. % sleep stage 1, 2, 3 and 4, and % REM). Hypoxia was defined as a separate exposure and analyzed as the percentage of sleep time with oxygen saturation <90%.

Wrist Actigraphy

The Octagonal Sleep Watch actigraph (Ambulatory Monitoring, Inc, Ardsley, NY) was used to estimate sleep/wake activity and was given to participants during the clinic visit along with instructions to continuously wear the actigraph on the nondominant wrist for five consecutive 24-hour periods. The actigraph contained a piezoelectric linear accelerometer (sensitive to 0.003g and above), a microprocessor, 32K RAM memory and associated circuitry. Voltages were generated each time the actigraph moved and were gathered

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continuously and summarized over 1-minute epochs. Actigraphy data were collected in 3 modes: zero crossing mode (ZCM), proportional integration mode (PIM), and time above threshold mode (TAT). Data were scored using Action W software, based on an algorithm developed at the University of California, San Diego (14) These algorithms calculate a moving average, which takes into account the activity levels immediately before and after the current minute to determine if the given time point should be coded as sleep or wake. The average of the sleep parameters over all nights was used in all analyses to minimize night-to-night variability.

Actigraphy parameters used for analyses included: total hours of sleep per night (TST); sleep efficiency (SE; the percentage of time the participant spent sleeping while in bed); sleep latency (SL; the number of minutes it took for a participant to fall asleep from the time they reported getting into bed); and wake after sleep onset (WASO; a measure of sleep fragmentation which represents the number of minutes a participant was awake during a typical sleep period, after the initial onset of sleep of at least 20 minutes in duration). Sleep diaries asking when participants went to bed and when lights were turned off were used to calculate sleep latency.

Data recorded concurrently (n=909 men) comparing actigraphy to polysomnography indicated a moderately strong correlation (r=0.6) between total sleep time measured by actigraphy (PIM mode) and total sleep time scored by polysomnography. The intraclass correlation between PSG and actigraphy for SE and WASO were 0.61 and 0.58, respectively. (15) The correlation between subjective measures of sleep (obtained by the Pittsburgh Sleep Quality Index) and objective measures of sleep were weak : TST (r=.31), SE (r=.13) and SL (r=.16).

Physical Function

During the clinic visit, MrOS participants completed a number of physical function tests. Grip strength (kg) was measured with a Jamar dynamometer. Participants completed two trials for each hand, and the average of the four trials was used for analyses. Sixty-one of the 2862 men in our cohort were excluded from the grip strength analysis (58 men were unable to complete the test, 2 men refused to do it, and 1 had missing data).

Gait speed was measured in meters/second on a standard six meter walking course, using the time to complete two trials. Twenty-nine men were excluded from this analysis due to not being able to attempt the test (n=23), attempting the test but being unable to do it (n=5), and for having missing data (n=1). Participants were instructed to walk at their normal pace. To test balance, participants were asked to complete a narrow walking course. Participants were instructed to walk within a 20-cm path over six meters and were given three attempts to complete two successful trials. A deviation was considered stepping outside the 20-cm path or relying on a wall or the staff to maintain balance. If a participant had two or fewer deviations from the 20-cm path, his trial was considered successful. If a participant had three or more deviations, the trial was considered unsuccessful. If a participant was unable to complete at least one successful narrow walk trial, he was considered to be unable to complete the narrow walk. One participant was excluded from this analysis due to missing data.

Each participant was asked to rise once from a standard chair without using his arms to stand. Participants were considered unable to complete the chair stand exam if they refused, could not stand, required the assistance of another person or used their arms to stand. Two men were excluded from this analysis due to missing data.

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Other Measures

All participants completed a self-administered questionnaire that included questions about race/ethnicity, education level, marital status, medical history, self-reported health and current smoking status. The Epworth Sleepiness Scale (ESS) was used to assess subjective daytime sleepiness. The Physical Activity Score for the Elderly (PASE) was used to assess physical activity level (16). Participants were considered to have co-morbid diseases if they reported a history of a physician diagnosis of one or more of the following: cardiovascular disease, osteoarthritis, diabetes, chronic obstructive pulmonary disease, or Parkinson's disease. Participants were asked to bring in all current medications (prescription, over-the-counter, vitamins) used within the last 30 days and a computerized dictionary was used to categorize the medications (17). All prescription medications recorded by the clinics were stored in an electronic medications inventory database (San Francisco Coordinating Center, San Francisco, CA). Each medication was matched to its ingredient(s) based on the Iowa Drug Information Service (IDIS) Drug Vocabulary (College of Pharmacy, University of Iowa, Iowa City, IA). Participants were considered users of antidepressants if they were taking an ingredient that was categorized by IDIS as “Antidepressants-SSRI”, “Antidepressants-other”, “Antidepressantstri/tetracyclic” or “Antidepressants-MAO inhibitor”. Height (cm) was measured on Harpenden stadiometers, and body weight (kg, in indoor clothing without shoes) was measured with a calibrated balance beam or digital scale using standard protocols. Body mass index (BMI) was calculated as kg/m^2.

Statistical Analyses

Demographic, sleep and physical function characteristics were summarized using means and standard deviations for continuous data and counts and percentages for categorical data. Skewed variables were summarized using medians and interquartile range. Chi-square tests of homogeneity and t-tests were used to compare the demographic and physical function characteristics of men with WASO >90 min compared to men with WASO <90 min.

Each sleep predictor was analyzed both as a continuous variable and as a categorical variable. The associations with physical function were similar; therefore, categorical sleep predictor results were presented for ease of interpretation. The independent main effects variables included total sleep time (TST ≤ 6 hours, 6-8 hours, and ≥ 8 hours), sleep apnea (RDI >30 compared to <30), sleep efficiency (SE<80% compared with SE>80%), sleep latency (SL >30 minutes compared to <30 minutes), wake after sleep onset (WASO >90 minutes compared to <90 minutes), % REM sleep (quartiles) and hypoxia (>1% of sleep time with O_2 saturation <90% vs. <1%). Since measures of sleep averaged over five nights were more likely representative of usual sleep, analyses using data collected by actigraphy for TST, SE, SL and WASO are presented here. PSG data were used for models that modeled sleep architecture, RDI and hypoxia.

Least-squared-means linear regression was used to examine the adjusted association between the various sleep predictors and continuous physical function outcome measures (grip strength and usual walking speed) with results presented as adjusted means and 95% confidence intervals (CI). Logistic regression was used to model the association between the sleep measures and binary physical function outcomes (inability to perform one chair stand and the inability to complete narrow walk test) with results presented as odds ratios (OR) and 95% CI. Known or suspected determinants of sleep and physical function outcome were examined for potential confounding in age-adjusted models. Covariates in age-adjusted models with p<.1 were considered for inclusion in the full multivariate model. Because of the numerous outcomes, one set of confounders was chosen for the multivariate model. Thus, any confounder associated with four or more sleep predictors at p<0.1 was included in

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the final multivariate model. All multivariate models were adjusted for age, BMI, clinic site, antidepressant use, hypertension, comorbid disease, PASE score and smoking.

Several sensitivity analyses were performed for the actigraphy sleep predictors. First, the age-adjusted and multivariate models were re-run using the other two actigraphy modes (TAT and ZCM). Results were materially unchanged with no consistent differences between the three modes. The correlation between total sleep time as measured by actigraphy from the PIM mode and total sleep time as scored with polysomnography was stronger than the correlation with the other two modes (PIM r=0.61, TAT r=0.54, ZCM r=0.39). Therefore, results from the PIM mode are presented. Second, we restricted our models to men with at least 5 nights of actigraphy data to determine if the exclusion of men with fewer nights of actigraphy altered the observed associations between sleep and physical function. We also repeated our analyses restricted to the men with better quality scores on their sleep diary and excluded men whose self-reported in-bed or out-of-bed times did not correspond well with their actigraphy data. We adjusted for hand tremor in the multivariate models to determine if this had any effect on the observed associations. Finally, because baseline (waketime) oxygen saturation could be a factor in determining the nocturnal hypoxia, we completed a sensitivity analysis that excluded men with resting waketime oxygen saturation ≤92%. All analyses were performed using SAS software, version 9.1 (SAS Institute, Cary, NC).

RESULTS

Characteristics of the Study Population

Characteristics of the 2862 MrOS Sleep participants with polysomnography and actigraphy data are presented in Table 1. Participants on average were 76.4 + 5.5 years with a BMI of 27.2 + 3.8 kg/m^2; 91% of the men were Caucasian. Approximately 50% reported a physician diagnosis of hypertension and at least one comorbid disease, but only 2% were currently smoking. About one third of the participants slept less than six hours a night, took at least 30 minutes to fall asleep, had a SE <80%, and/or had > 90 minutes of arousals at night. About 13% of the men had excessive daytime sleepiness (ESS >10), 52% had > 1% of their sleep time with oxygen saturation <90% and 18% of participants had sleep apnea (RDI >30).

Compared to men with fewer nighttime arousals (WASO <90 minutes), men with WASO > 90 minutes were, on average, older, had higher BMI and had more co-morbid diseases. These men were also less physically active, and had weaker grip strengths, slower walking speed and more difficulty with the chair stand and narrow walk tests (Table 2).

Actigraphy Measures of Sleep and Physical Function

Actigraphy data were collected for an average of 5.2 + 0.8 nights. Actigraphic measures of sleep fragmentation, such as lower sleep efficiency (defined by SE <80%) and increased nocturnal awakenings (defined by WASO > 90 min), were significantly associated with poorer performance on all physical performance measures in both age-adjusted and multivariate-adjusted analyses (Tables 3 and 4). For example, after adjustment for age, mean grip strength was 2.9% lower and the mean walking speed was 4.3% lower in men with WASO > 90 minutes compared to men with WASO <90 minutes (Table 3). These differences in grip strength and walking speed were slightly attenuated but remained significant after adjusting for potential covariates.

There were 2030 (70.9%) men who had Periodic Limb Movement Disorder (≥5 periodic limb movements per hour of sleep) on overnight PSG. A higher percentage of men with WASO ≥ 90 min had PLMD compared to men with WASO <90 min (77.8% vs. 67.7%, p<. 001). A higher percentage of men with WASO ≥ 90 min had SDB (RDI >30) compared to

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men with WASO <90 min (25.3% vs. 13.8%, p<.001). The associations between WASO and all physical function outcome except for inability to perform the narrow walk, were strengthened by excluding the men with PLMD, suggesting that PLMD may partly mediate the association between WASO and physical function. At the same time, however, adjustment for PLMD in the models had very little effect on the point estimates for WASO. Therefore, PLMD may be a mediator, but not a confounder of the association between WASO and physical function. However, additional adjustment for SDB did not alter the association between WASO and grip strength, walking speed, inability to perform chair stand or inability to perform narrow walk test in either the age-adjusted or multivariate models. Therefore, the relationship between increased WASO and decreased physical function may not be explained by sleep disordered breathing.

Similar findings for sleep efficiency with grip strength and walking speed were found. Additionally, men with increased nighttime awakenings had a 70% higher odds of not completing one chair stand (OR 1.7, 95% CI 1.2, 2.5) and 30% higher odds of not completing the narrow walk test (OR 1.3, 95% CI 1.1, 1.6) (Table 4). Similar odds ratios were seen in men with decreased sleep efficiency and increased sleep latency.

Decreased nighttime sleep was associated with decreased grip strength and walking speed, and increased odds of not completing the chair stand and the narrow walk test in age-adjusted analyses. However, after multivariate adjustment, only grip strength remained significant. The association appeared u-shaped; men who slept 6-8 hours per night had the strongest grip strength while men who slept ≤ 6 hours or ≥ 8 hours had the weakest grip (Table 3). Additional adjustment for excessive daytime sleepiness did not change the association between total nighttime sleep and the mean grip strength, walking speed and inability to complete chair stand or inability to complete narrow walk test.

Polysomnography Measures of Sleep and Physical Function

Men with the least amount of REM sleep (<14.8%) had the weakest grip strength and slowest walking speed (Table 3). No consistent associations were seen with stage 1, stage 2 or stage 3/4 sleep and physical function (data not shown).

Men with sleep apnea (RDI > 30) had 2.2% lower grip strength and 5.2% slower walking speed than men without sleep apnea after adjustment for age (Table 3). Men with RDI > 30 had approximately twice the odds of not completing one chair stand (OR 1.9, 95% CI 1.3, 2.8) and approximately 1.5 times the odds of not completing the narrow walk test (OR 1.5, 95% CI 1.1, 1.8) (Table 4). However, after adjusting for covariates, sleep apnea was not significantly associated with any of the four physical function measures.

Compared to men who had <1% sleep time with hypoxia (O_2 saturation <90%), men with > 1% sleep time with hypoxia (O2 saturation <90%) had approximately a 2.2% lower grip strength and a 5.1% slower walking speed in age adjusted models. These differences were attenuated, and were clinically significant, after adjustment for age, BMI, clinic site, antidepressant use, hypertension, comorbid disease, PASE, and smoking (Table 3). Men who had >1% sleep time with hypoxia had higher odds of being unable to perform the narrow walk in both age-adjusted (OR 1.7, 95% CI 1.4, 2.1) and multivariate-adjusted (OR 1.3, 95% CI 1.1, 1.7) analyses (Table 4).

Analyses substituting PSG measures for TST, SE, SL and WASO were consistent with the actigraphy results for the walking speed and inability to do narrow walk outcomes, but not for the grip strength or inability to do chair stand. Analyses using PSG measures for WASO and SE instead of actigraphy showed that the mean grip strength was the same regardless of

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whether men had fragmented sleep and the odds ratio for chair stand was much weaker with PSG.

Actigraphy Sensitivity Analyses

Most of the actigraphy associations were slightly stronger when the actigraphy analyses were restricted to men with at least five nights of actigraphy compared to when the analyses included all men. However, the associations were slightly weaker when the analyses were redone only with men whose self-reported in-bed and out-of-bed times corresponded well with their actigraphy data. For example, men with sleep efficiency <80% had a multivariate adjusted OR of 1.5 (95% CI 1.0, 2.3) for being unable to perform the chair stand when the actigraphy analyses were restricted to men with better out-of-bed quality scores compared to an OR of 1.7 (95% CI 1.2, 2.5) when all men with actigraphy were included. The addition of hand tremor to the multivariate models did not affect the findings. Sensitivity analyses excluding the men with resting waketime oxygen saturation ≤92%, did not affect the associations between hypoxia and physical function. Overall, the actigraphy sensitivity analyses demonstrated that qualitatively the associations did not change.

DISCUSSION

In this cohort of 2862 community dwelling older men, objective measures of sleep fragmentation, including nighttime awakening and multiple long wake episodes, were independently associated with weaker grip strength, slower walking speed and inability to complete a single chair stand or narrow walk test. Fragmented sleep occurs chronically in older adults and has been correlated with daytime sleepiness, impaired mood and decreased mental flexibility and attention (18-20). Sleep fragmentation was not a marker of poorer health, and/or medication use.

Increased sleep latency was independently associated with inability to perform a single chair stand. These results are consistent with findings found in older women. Women who had a similar amount of nighttime arousals (WASO 1.1-1.6 hours) were 54% less likely to complete five chair stands and their gait speeds were 3.4% slower compared to women in the lowest quartile. There appears to be an association between poorer physical function with men who had prolonged sleep latency and increased sleep fragmentation. These findings suggest a possible association between chronic insomnia and decreased function in older men since increased sleep latency and sleep fragmentation could indicate problems with sleep initiation and sleep maintenance insomnia.

Previous studies have shown that sleep durations <6 hours or >8 hours per night and complaints of daytime sleepiness were associated with poor health and reduced physical function (21,22). In the Study of Osteoporotic Fractures, older women with short and long sleep durations had slower gait speed and required more time to complete five chair stands (23). Except for grip strength, there were no associations between nighttime sleep duration and physical function in our cohort of older men. Men who had short (≤ 6 hours) and long (≥ 8 hours) sleep durations had weaker grip strength compared to men who slept between 6-8 hours. Although measures of NREM sleep were not associated with physical function, decreasing amounts of REM sleep appeared to be associated with weaker grip strength and slower walking speed in older men.

To our knowledge, no prior study has evaluated the association between sleep apnea and physical function in a population-based sample of older men. In this cohort, physical function measures were not different in men with sleep apnea compared to men without sleep apnea. Our study suggests that sleep related poor physical performance may be affected more by fragmented sleep rather than sleep apnea. Additionally, men who were

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hypoxic (O_2 saturation <90%) for more than 1% of their sleep time demonstrated poorer physical function. In our cohort, men who had lower baseline (waketime) oxygen saturation (O_2saturation ≤92% compared to >92%) proportionately spent more sleep time with oxygen saturation <90%. Men who were hypoxic at rest and had an RDI ≥ 30 proportionately spent the most sleep time with oxygen saturations <90%. However, the mean % sleep time with SaO2<90% for men with RDI≥30 and resting SaO2>92% was 9.43 vs. 12.88 for men with RDI<30 and resting SaO2≤92%. Therefore, it is possible that a person with mild SDB and baseline oxygen saturation in the low 90s may be more likely to spend increased time in oxygen saturation < 90% than a person with moderate or severe SDB with baseline oxygen saturation in mid to upper 90s.

The mechanism underlying the relationship between disturbed sleep and physical function is not well understood. In our analysis we attempted to adjust for factors that might confound any association between sleep disturbances and physical function. However, the associations we observed with sleep fragmentation and hypoxia remained essentially unchanged after adjusting for age, BMI, clinic site, antidepressant use, medical conditions, physical activity level and smoking status. Other factors such as neuroendocrine dysfunction or sub-clinical inflammation may be possible mechanisms. Further research to evaluate mechanisms is warranted.

This large population-based study of nearly 3000 men had several strengths. These men were generally healthy with no preexisting diagnosis of sleep disorders and were not selected based on sleep complaints. Sleep and physical function were measured objectively, thereby decreasing bias from participant subjectivity. In addition, the careful consideration and collection of detailed covariate data and standardized data collection with prospectively designed scoring approaches enhances generalizability and comparison to other samples of older men. Finally, several sensitivity analyses were performed for the actigraphy sleep predictors, which demonstrated that the sleep and physical function associations remained qualitatively unchanged.

However, this study was limited by the cross-sectional ascertainment of sleep and physical function measures, therefore causality cannot be determined. Use of self-reported time in and out of bed may be inaccurate, and could introduce error to those variables that depend on this time as part of the calculation (i.e. sleep latency and sleep efficiency). However, when we restricted our analyses to the men with better in-bed scores and to the men with better out-of-bed scores, the point estimates were virtually unchanged. Since the cohort consisted of white men over the age of 67, these results may not be generalizable to women or younger men.

In conclusion, there is a relationship between sleep quality and physical functioning in older men. Future research should determine the temporality of this association and evaluate whether interventions to improve sleep quality may enhance physical performance in older people.

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Table 1

Characteristics of the 2,862 Men with Polysomnography and Actigraphy Data

Demographics	
Age (yrs, range 67-96)	76.4 ± 5.5
BMI (kg/m^2)	27.2 ± 3.8
Race	
Caucasian (%)	2598 (90.8)
African American (%)	96 (3.4)
Asian (%)	82 (2.9)
Hispanic (%)	55 (1.9)
Other (%)	31 (1.1)
PASE score †	145.6 ± 71.4
Current Smoker (%)	55 (1.9)
Comorbid Disease (%)	1554 (54.3)
Cardiovascular Disease (%)	856 (29.9)
Osteoarthritis (%)	691 (24.2)
Diabetes (%)	383 (13.4)
COPD (%)	148 (5.2)
Parkinson's (%)	35 (1.2)
Hypertension (%)	1435 (50.2)
Antidepressant use (%)	222 (7.8)
Excessive Daytime Sleepiness >10	371 (13.0)
Physical Function Measures	
Grip Strength (kg)	37.9 ± 8.2
Usual walking speed (m/s)	1.1 ± 0.2
Unable to do 1 chair stand (%)	142 (5.0)
Unable to do narrow walk (%)	531 (18.6)
Actigraphy Sleep Measures	
Number of nights of actigraphy	5.2 ± 0.8
Total sleep time (hrs)	6.4 ± 1.2
Total sleep ≤6 hours (%)	900 (31.4)
Total sleep 6-8 hours (%)	1750 (61.2)
Total sleep ≥ 8 hours (%)	212 (7.4)
Sleep efficiency (%)*	85.1 (77.9-89.9)
Sleep efficiency <80% (%)	878 (30.7)
Sleep latency (min)*	21.2 (12.2-37.0)
Sleep latency ≥ 30 min (%)	962 (33.6)
Wake after sleep onset (min)*	68.6 (46-101.0)
Wake after sleep onset ≥ 90 min (%)	916 (32.0)
Polysomnography Sleep Measures	
Stage 1 (%)*	5.9 (4.0-8.6)

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Stage 2 (%)	62.6 ± 9.6
Stage 3/4 (%)*	9.9 (3.8-16.7)
REM (%)	19.3 ± 6.6
RDI*	12.6 (5.9-23.9)
RDI ≥ 30 (%)	500 (17.5)
% Sleep time with O_2 <90% (%)*	1.0 (0.0-3.7)
≥1% sleep time with O_2 saturation <90%	1478 (51.6)
Periodic Limb Movement Disorder > 5	2030 (70.9)

All values expressed as means ± standard deviation or n (%) with the exception of those marked with a

Body Mass Index (BMI), Chronic Obstructive Lung Disease (COPD), Rapid Eye Movement Sleep (REM), Respiratory Disturbance Index (RDI), Oxygen Saturation (O_2)

*which are expressed as medians (interquartile range)

†Physical Activity Scale for the Elderly (PASE) measures reported daily physical activity. The range was 0-592 and higher scores reflect greater physical activity.

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Table 2

Comparison of Characteristics by Category of Nighttime Awakening

	WASO ≥ 90 min (n=916)	WASO < 90 min (n=1946)	p-value
Demographics			
Age (yrs)	76.9 ± 5.6	76.1 ± 5.4	.001
BMI (kg/m^2)	28.1 ± 4.3	26.8 ± 3.5	<.001
Race			
Caucasian (%)	830 (90.6)	1768 (90.9)	0.73
African American (%)	36 (3.9)	60 (3.1)	
Asian (%)	26 (2.8)	56 (2.9)	
Hispanic (%)	15 (1.6)	40 (2.1)	
Other (%)	9 (1.0)	22 (1.1)	
PASE score	138.6 ± 72.9	148.9 ± 70.5	.001
Current Smoker (%)	23 (2.5)	32 (1.6)	.11
Comorbid Disease (%)	551 (60.2)	1003 (51.6)	<.001
CVD (%)	299 (32.7)	557 (28.6)	.03
Osteoarthritis (%)	238 (26.0)	453 (23.3)	.11
Diabetes (%)	165 (18.0)	218 (11.2)	<.001
COPD (%)	64 (7.0)	84 (4.3)	.003
Parkinson's (%)	13 (1.4)	22 (1.1)	.51
Hypertension (%)	500 (54.6)	935 (48.1)	.001
Antidepressant use (%)	80 (8.7)	142 (7.3)	.18
Physical Function Measures			
Grip Strength (kg)	36.9 ± 8.3	38.4 ± 8.1	<.001
Usual walking speed (m/s)	1.1 ± 0.2	1.2 ± 0.2	<.001
Unable to do 1 chair stand (%)	74 (8.1)	68 (3.5)	<.001
Unable to do narrow walk (%)	224 (24.5)	307 (15.8)	<.001

All values expressed as means ± standard deviation or n (%)

Wake after Sleep Onset (WASO), Body Mass Index (BMI), Physical Activity Scale for the Elderly (PASE), Chronic Obstructive Lung Disease (COPD

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Table 3

Age and Multivariate Adjusted Means (95% Confidence Intervals) for Continuous Physical Function Outcome by Sleep Measures

	Grip Strength (kg)		Usual Walking Speed (m/s)	
	Age adjusted	**Multivariate adjusted**	**Age adjusted**	**Multivariate adjusted**
Total Nighttime Sleep §				
≤6 hours	37.0 (36.5-37.5)	37.0 (36.5-37.5)	1.1 (1.1-1.1)	1.1 (1.1-1.1)
6-8 hours	38.4 (38.0-38.7)	38.4 (38.1-38.8)	1.2 (1.2-1.3)	1.2 (1.1-1.2)
≥8 hours	37.5 (36.5-38.5)†	37.5 (36.5-38.5)†	1.1 (1.1-1.2)†	1.1 (1.1-1.2)
Sleep Efficiency §				
≥80%	38.2 (37.9-38.6)	38.2 (37.8-38.5)	1.2 (1.2-1.2)	1.2 (1.1-1.2)
<80%	37.1 (36.6-37.6)‡	37.3 (36.8-37.8)†	1.1 (1.1-1.1)‡	1.1 (1.1-1.1)*
Sleep Latency §				
<30 min	38.1 (37.7-38.4)	38.0 (37.7-38.3)	1.2 (1.2-1.2)	1.2 (1.1-1.2)
≥30 min	37.5 (37.1-38.0)	37.7 (37.3-38.2)	1.1 (1.1-1.1)‡	1.1 (1.1-1.2)
WASO §				
<90 min	38.2 (37.9-38.6)	38.2 (37.8-38.5)	1.2 (1.2-1.2)	1.2 (1.1-1.2)
≥90 min	37.1 (36.7-37.6)‡	37.3 (36.9-37.8)†	1.1 (1.1-1.1)‡	1.1 (1.1-1.1)†
REM				
Quartile 1 (<14.8%)	37.2 (36.6-37.8)	37.4 (36.8-38.0)	1.1 (1.1-1.1)	1.1 (1.1-1.1)
Quartile 2 (14.8-19.6%)	37.9 (37.3-38.4)	37.8 (37.2-38.3)	1.2 (1.1-1.2)	1.2 (1.1-1.2)
Quartile 3 (19.6-23.7%)	38.6 (38.0-39.2)	38.6 (38.0-39.1)	1.2 (1.2-1.2)	1.2 (1.1-1.2)
Quartile 4 (≥23.7%)	38.2 (37.7-38.8)†	38.2 (37.6-38.7)*	1.2 (1.1-1.2)‡	1.2 (1.1-1.2)*
Respiratory Disturbance Index				
RDI <30	38.0 (37.7-38.3)	38.0 (37.7-38.3)	1.2 (1.1-1.2)	1.2 (1.1-1.2)
RDI ≥30	37.2 (36.5-37.9)*	37.4 (36.7-38.0)	1.1 (1.1-1.1) ‡	1.1 (1.1-1.1)
% Sleep Time with O_2<90%				
<1%	38.3 (37.9, 38.7)	38.2 (37.8, 38.6)	1.2 (1.2, 1.2)	1.2 (1.1, 1.2)
≥1%	37.5 (37.1, 37.9)†	37.6 (37.2, 38.0)*	1.1 (1.1, 1.1)‡	1.1 (1.1, 1.1)†

Wake after Sleep Onset (WASO), Rapid Eye Movement Sleep (REM), Oxygen Saturation (O_2)

Multivariate adjustment includes age, BMI, clinic site, antidepressant use, hypertension, comorbid disease (history of at least one medical condition including cardiovascular disease, osteoarthritis, diabetes, COPD and Parkinson's disease), Physical Activity Scale for the Elderly, and smoking

* p-trend <.05

† p-trend <.01

‡ p-trend <.001

§ Measured by actigraphy

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Table 4

Association Between Sleep Measures and Binary Physical Function Outcomes

	Inability to Perform Chair Stand		Inability to Perform Narrow Walk	
	Odds Ratio (95% Confidence Interval)			
	Age adjusted	Multivariate adjusted	Age adjusted	Multivariate adjusted
Total Nighttime Sleep §				
≤6 hours	**1.62 (1.13, 2.32)**	1.29 (0.88, 1.90)	**1.47 (1.19, 1.81)**	**1.25 (1.00, 1.57)**
6-8 hours	1.0 (referent)	1.0 (referent)	1.0 (referent)	1.0 (referent)
≥8 hours	1.36 (0.74, 2.52)	1.25 (0.65, 2.38)	1.18 (0.82, 1.70)	1.11 (0.75, 1.62)
Sleep Efficiency §				
<80%	**2.45 (1.74, 3.45)**	**1.72 (1.19, 2.49)**	**1.72 (1.41, 2.11)**	**1.34 (1.08, 1.66)**
≥80%	1.0 (referent)	1.0 (referent)	1.0 (referent)	1.0 (referent)
Sleep Latency §				
≥30 min	**2.31 (1.64, 3.25)**	**1.67 (1.16, 2.39)**	**1.46 (1.19, 1.79)**	1.21 (0.97, 1.49)
<30 min	1.0 (referent)	1.0 (referent)	1.0 (referent)	1.0 (referent)
WASO §				
≥90 min	**2.29 (1.63, 3.23)**	**1.73 (1.20, 2.49)**	**1.64 (1.34, 2.00)**	**1.33 (1.07, 1.64)**
<90 min	1.0 (referent)	1.0 (referent)	1.0 (referent)	1.0 (referent)
REM				
Quartile 1 (<14.8%)	1.52 (0.92, 2.48)	1.16 (0.69, 1.95)	1.14 (0.86, 1.52)	0.98 (0.73, 1.32)
Quartile 2 (14.8-19.6%)	1.25 (0.74, 2.10)	1.17 (0.68, 2.01)	1.04 (0.77, 1.39)	0.99 (0.73, 1.35)
Quartile 3 (19.6-23.7%)	0.64 (0.35, 1.17)	0.61 (0.33, 1.15)	0.79 (0.58, 1.07)	0.76 (0.55, 1.05)
Quartile 4 (>23.7%)	1.0 (referent)	1.0 (referent)	1.0 (referent)	1.0 (referent)
Respiratory Disturbance Index				
RDI ≥30	**1.93 (1.32, 2.81)**	1.41 (0.93, 2.13)	**1.45 (1.14, 1.84)**	1.11 (0.86, 1.44)
RDI <30	1.0 (referent)	1.0 (referent)	1.0 (referent)	1.0 (referent)
Sleep Time with Oxygen Saturation <90%				
≥1%	**1.56 (1.10, 2.21)**	1.09 (0.74, 1.61)	**1.69 (1.38, 2.06)**	**1.33 (1.07, 1.66)**
<1%	1.0 (referent)	1.0 (referent)	1.0 (referent)	1.0 (referent)

Bolded cells have p value <0.05

Multivariate adjustment includes age, BMI, clinic site, antidepressant use, hypertension, comorbid disease (history of at least one medical condition including cardiovascular disease, osteoarthritis, diabetes, COPD and Parkinson's disease), Physical Activity Scale for the Elderly, and smoking

§Measured by actigraphy