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ORIGINAL ARTICLE

The bidirectional association between physical activity and sleep in middle-aged and older adults: a prospective study based on polysomnography

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Abstract

Study Objectives: To examine the bidirectional association between physical activity (PA) and polysomnographically (PSG)-assessed sleep parameters characterized by total sleep time (TST), sleep onset latency (SOL), wake time after sleep onset (WASO), sleep efficiency and percentage of TST in N1, N2, and N3 stages and rapid eye movement (REM) sleep in middle-aged and older adults.

Methods: Longitudinal study based on a subsample of the Wisconsin Sleep Cohort. Self-reported PA information was used to estimate the metabolic equivalents of task (MET-minutes/week) activity and in-laboratory PSG exams provided information on sleep parameters at baseline and after 3–11 years of follow-up between 2004 and 2015. Poisson and linear regression models controlling for confounders estimated associations of sleep outcomes with changes in PA.

Results: A total of 424 participants (45.8% female; mean ± *SD* age 60.1 ± 7.5 years) were followed over an average of 5.0 ± 1.6 years. Compared to baseline PA of <500 MET-minutes/week (reference category), 500 to 1500 MET-minutes/week of PA was associated with lower incidences of TST <6 hours (relative risk, RR: 0.49; 95% confidence interval, CI: 0.27; 0.88), WASO >60 minutes (RR: 0.58; 95% CI: 0.41; 0.82) and sleep efficiency <80% (RR: 0.61; 95% CI: 0.39; 0.94), adjusting for sociodemographic, health behaviors and medical conditions. No significant associations were observed between baseline sleep characteristics and changes in PA through the follow-up.

Conclusion: In this prospective study, an intermediate level of PA at baseline predicted lower risk of incident short sleep time, higher WASO and lower sleep efficiency measured with PSG.

Statement of Significance

Most epidemiological evidence regarding the association between physical activity (PA) and sleep in adults has been based on cross-sectional studies or self-reported sleep parameters. In this longitudinal study based on polysomnography, PA was associated with a lower risk of short sleep duration, wake time after sleep onset and lower sleep efficiency. In addition to the already-established beneficial effects of PA for health, this behavior might be considered as a protective factor for sleep disturbances in middle-aged and older adults.

Key words: physical activity; sleep; polysomnography; cohort study

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Introduction

Low levels of physical activity (PA) and poor sleep are known risk factors for cardiovascular and all-cause mortality [1–3]. Systematic reviews based on intervention studies observed that PA is associated with improved subjective sleep quality and objective sleep parameters including sleep onset latency (SOL) and sleep efficiency [4–6]. Several observational studies also examined the association between PA and sleep in adults, but most were cross-sectional [7–9] or based on subjective sleep information [10–14]. Although self-reported sleep data are useful, in middle-aged and older adults they are only poorly-to-moderately correlated with objective measurements from accelerometry [15] and polysomnography (PSG) [16] and, thus, could lead to inaccurate results.

Among the cross-sectional studies that have addressed the relationship between PA and objective sleep, findings are inconsistent. In one study, Mitchell et al. [7] evaluated sleep parameters with wrist accelerometers over seven consecutive days and found no associations between moderate to vigorous PA and nighttime total sleep time (TST) or sleep efficiency in adult women. PSG has also been used in some cross-sectional studies, with conflicting results. For example, in midlife women, greater amounts of sports/exercise activity were associated with higher in-home PSG-assessed sleep efficiency [8]. In contrast, in another study of community-dwelling adults, the authors did not find associations between PSG-assessed sleep measures and sedentary time or moderate to vigorous PA [9].

Although PSG is the gold standard measure to evaluate sleep, we could not find prospective studies which evaluated PA as a predictor of PSG-assessed sleep outcomes. However, several cohort studies have investigated changes in self-reported sleep as outcomes with varied findings. While some found that the higher levels of PA are related to low incidence of insomnia [10–12], others found that PA did not predict sleep quality [13] or changes in sleep duration [14].

Thus, this study aims to examine whether PA at baseline predicts sleep disturbances including TST, SOL, wake time after sleep onset (WASO), sleep efficiency and percentage of TST in N3 (stages 3 and 4) and rapid eye movement (REM) sleep, evaluated with PSG at baseline and an average of 5 years of follow-up. Based on previous evidence derived from intervention studies, we hypothesized that higher levels of PA, independent of confounding factors, are associated with lower risks of short TST, longer SOL and WASO, lower sleep efficiency and lower percentages of TST in N3 and in REM sleep. In addition, as some evidence suggests an inverse relationship in which sleep conditions could be associated with reduced PA [13, 17, 18], we also explored associations of baseline sleep conditions with change in PA over the follow-up period.

Methods

Design and participants

The present study examined longitudinal data from the Wisconsin Sleep Cohort (WSC) study, a prospective population-based study designed to investigate the natural history, risk factors for and outcomes of common sleep disorders and behaviors in adults. The initial population was comprised of 1521 randomly-selected adults who were 30- to 60-year-old employees of state agencies in 1988. The WSC started recruiting subjects for baseline sleep studies in 1989. Subjects were invited for repeat studies at approximately 4-year intervals over up to 25 years of follow-up. Participation rates for 4-year follow-up waves have been an average of ~79%. Collection of detailed data on PA was initiated in October 2004 and is thus not available on all study participants. For approximately one-quarter of the sample, PA assessment and PSG occurred on different occasions (average difference = 1.9 ± 1.1 year). For the other three-quarters of observations, PA was assessed by technician-attended questionnaire administration during the overnight study visits. More detailed descriptions of the design and data collection of the WSC can be found elsewhere [19, 20]. For the present study, we identified the subsample of WSC participants with both PSG and complete information for PA from at least two study visits. This comprised individuals followed between 2004 and 2015. Study protocols and informed consent documents were approved by the Health Sciences Institutional Review Board of the University of Wisconsin-Madison.

Study variables

Sleep

Participants completed study visits at the University of Wisconsin-Madison Clinical Research Unit where they were monitored by PSG overnight during which participants were encouraged to sleep from their usual bedtime to usual wake-time, if possible. A detailed description of PSG procedures can be found elsewhere [21]. Briefly, an 18-channel polysomnographic recording system (model 78, Grass Instruments, Quincy, MA) was used to evaluate sleep stage, respiration, body movements, and additional physiologic parameters. The recording montage included electroencephalography, electrooculography and submental electromyography to determine sleep state; continuous pulse oximetry (model 3740, Ohmeda, Englewood, CO) to assess arterial oxygen saturation; nasal-oral thermocouple (ProTec, Hendersonville, TN) to assess nasal and oral airflow; nasal pressure transducer (Validyne Engineering, Northridge, CA) for nasal airflow; respiratory inductance plethysmography (Respitrace, Ambulatory Monitoring, Ardsley, NY) to detect body abdominal and chest wall excursion. Sleep studies were staged, and respiratory events scored by trained sleep technicians and then reviewed by an expert polysomnographer.

For the present investigation, the following variables were obtained from the PSG: TST (hours), SOL (time from lights out to the first epoch of EEG-evidenced sleep in minutes), WASO (time awake between first and last epochs of EEG-assessed sleep in minutes), sleep efficiency (percent of time in bed spent asleep) and percentage of TST in stage N3 and REM sleep. The average number of apnea plus hypopnea events per hour of sleep defined the apnea-hypopnea index (AHI), used as a covariate in analyses as it can plausibly act as a confounding factor by suppressing PA via mechanisms such as general fatigue and depressed mood, and can also directly impact outcomes (examined sleep characteristics) [20, 22].

Short sleep was defined as TST < 6 hours, which is associated with increased mortality [2]. For each of the other sleep parameters, as we could not find a consensus regarding the cutoff to discriminate those with better and worse sleep indicators, we opted to use the medians at baseline, and rounded the values to simplify the interpretation of the results. For example, the baseline median of WASO was 60.25 minutes and we analyzed the risk of WASO > 60 minutes; similarly, sleep efficiency had a baseline median of 82.8% and the cutoff used to define incident lower sleep efficiency was 80%. To evaluate the robustness of our results, we repeated all analyses using the exact value as the cutoff for the outcomes and the results remained essentially identical.

Physical activity

A modified version of the Paffenbarger Physical Activity Questionnaire [23] was administered by trained personnel to assess the participants' usual frequency, intensity, duration, and type of regular and recreational PA and walking. Responses were used to estimate weekly energy expenditure in these activities in metabolic equivalents of task (MET-minutes/week). PA was divided into three groups according to approximate tertiles of MET-minutes/week: <500 (reference category, 500 MET-minutes/week is approximately equivalent to 25 minutes per day walking at a slow-moderate pace of ~2.5 miles per hour), 500 to 1500 MET-minutes/week, and >1500 MET-minutes/week (1500 MET-minutes/week is approximately equivalent to 40–45 minutes per day of brisk walking at ~4 miles per hour).

Covariates

Sociodemographic variables were obtained to control for potential confounding factors including age (years), sex, educational level (up to high-school vs. higher level) and marital status. Information about smoking (current smoker vs. former or never smoker) and alcohol consumption (number of drinks/week) were also self-reported. Laboratory-assessed weight (kg) and height (m) were used to calculate body mass index in kg/m^2. Participants also were queried "In general, would you say your health is" and chose one of the following options: excellent or very good (categorized as optimal self-rated health, the reference category), or good, fair or poor (suboptimal self-rated health). Depression, defined by symptoms or antidepressant medication use at baseline, was also obtained.

Statistical analysis

Sociodemographic and lifestyle characteristics, self-reported general health, PA and sleep parameters of the original WSC, baseline and followed study populations were described with frequencies, medians and interquartile ranges.

To calculate the risk of developing each sleep disturbance, we considered the number of new cases among those without each specific sleep problem at baseline (Note that sample sizes for those followed for new onset of specific sleep problems — i.e. that had baseline levels that were "healthier"— are indicated in the top row of Table 2 for each sleep parameter).

To analyze the relationship between PA levels and the risk of developing sleep disturbances, we used Poisson regression models with robust variance estimates, as described in Zou [24], to estimate relative risks in each category of PA (main predictor) for each sleep outcome. We first examined unadjusted models and then added covariates to adjust the analyses for the potentially confounding effects of age, sex, educational level, marital status, smoking, alcohol intake, follow-up time, and the difference in time between PA and sleep information at baseline (model 1). Finally, we examined a fully adjusted model that additionally included baseline body mass index (BMI), change in BMI over the follow-up, self-rated health, depression, baseline apnea-hypopnea index, and continuous positive airway pressure (CPAP) use at baseline PSG (model 2). Additional sensitivity analyses repeated these analyses including only those observations for which PA and PSG were assessed at the same study visit.

We also explored whether the PA category at baseline predicted continuous changes in each of the sleep parameters. For this purpose, linear mixed-models evaluated the continuous change in the sleep parameter as the outcome and the PA category as the exposure. These models were adjusted for the same covariates as the fully-adjusted models described above. In addition, in supplemental analyses we tested whether continuous PA at baseline and continuous changes in PA over the follow-up period predicted either the discrete sleep outcomes or changes in each of the sleep parameters.

Lastly, "reverse associations" were examined, i.e. whether baseline categorical sleep parameters predict continuous and percent changes in PA (outcomes). In addition to the already-mentioned covariates, these analyses were also adjusted for change in the corresponding sleep parameter over the follow-up period. Additional supplemental analyses examined continuous baseline sleep parameters as well as continuous change in sleep parameters as the predictors in separate models.

The statistical significance level was set at $p < 0.05$. All analyses were performed with SAS statistical software (version 9.4, SAS Institute Inc., Cary, NC).

Results

Among the 1538 WSC participants, 834 had at least one sleep study since October 2004 (when PA assessment was initiated), and 14 were excluded due to missing information for education (n = 7), marital status (n = 1), depression (n = 3) and CPAP use (n = 3). Of these, 424 (52% of 820) were followed and provided information for all key study variables over a follow-up interval of a mean (±*SD*) 5.0 ± 1.6 years (minimum 3.3, maximum 11.2 years). Note that many of the 820 did not have an opportunity to be invited for follow-up sleep assessments before sleep studies ceased in 2015. Table 1 presents the characteristics of the baseline and the followed participants. Among followed individuals, the age range varied from 42 to 77 years. PSG data showed a baseline median of 6.2 hours of TST, 7 minutes of SOL, 60 minutes of WASO, 83% of sleep efficiency, 9% of TST in N1, 63% in N2, 9% in N3 sleep, and 17% in REM sleep (Table 1).

PA categories predicting binary sleep outcomes

A schematic representation of the study design and the number of participants followed in each group of PA level is presented in Figure 1. Relative risks for developing polysomnographically-assessed sleep problems according to baseline PA level are presented in Table 2. The results for unadjusted Poisson regression models showed no associations between baseline PA category and binary sleep outcomes. However, after the adjustment by sociodemographic, lifestyle and health conditions (model 2), we observed lower risks for short sleep time, higher WASO and

Table 1. Characteristics of participants included in the original Wisconsin Sleep Cohort (original sample), of those considered as the baseline for this study and of those finally followed

Baseline characteristics	Original WSC	Baseline for the present study	Followed
Total, *n*	1538	820	424
Sociodemographic, lifestyle and health			
Age (years), median (IQR)	60 (53, 67)	62 (56, 67)	60 (54, 65)
Female, *n* (%)	701 (44.8)	369 (45.0)	194 (45.8)
Lower educational level[a], *n* (%)	386 (25.1)	198 (24.1)	103 (24.3)
Married, *n* (%)	1062 (69.1)	576 (70.2)	300 (70.8)
Current smoker, *n* (%)	187 (12.2)	65 (7.9)	36 (8.5)
Alcohol intake (drinks/week), median (IQR)	2 (0, 6)	2 (0, 5)	2 (0, 5)
BMI (kg/m^2), median (IQR)	30 (26, 35)	30 (26, 35)	30 (26, 35)
Excellent or very good self-rated health, *n* (%)	521 (57.1)[b]	482 (58.8)	269 (63.4)
Depression or antidepressant use, *n* (%)	425 (27.6)	220 (26.8)	111 (26.2)
CPAP use, *n* (%)	93 (6.0)	61 (7.4)	14 (3.3)
Physical activity (MET-minutes/week), *n*	915	820	424
<500	285 (31.1)	255 (31.1)	124 (29.2)
500 to 1500	287 (31.4)	257 (31.3)	144 (34.0)
>1500	343 (37.5)	308 (37.6)	156 (36.8)
Median (IQR)	996 (320, 2165)	1008 (336, 2091)	1041 (420, 1997)
Sleep measures (PSG), *n*	1564	820	424
Apnea-hypopnea index, median (IQR)	7.8 (3.7, 18.0)	8.9 (3.7, 18.2)	9.5 (3.1, 18.9)
TST (hours/night), median (IQR)	6.0 (5.3, 6.8)	6.0 (5.4, 6.7)	6.2 (5.6, 6.8)
SOL (minutes), median (IQR)	10 (5, 20)	8 (4, 17)	7 (3.5, 13)
WASO (minutes), median (IQR)	68 (48, 103)	66 (40, 98)	60 (37, 91)
Sleep efficiency (%), median (IQR)	81 (72, 88)	81 (74, 88)	83 (76, 89)
% of TST in N1, median (IQR)	9 (6, 14)	9 (6, 14)	9 (6, 14)
% of TST in N2, median (IQR)	65 (58, 71)	63 (57, 70)	63 (56, 69)
% of TST in N3 (phases 3 and 4), median (IQR)	7 (2, 14)	9 (4, 15)	9 (4, 16)
% of TST in REM, median (IQR)	16 (11, 20)	16 (13, 20)	17 (13, 20)
TST < 6 hours, *n* (%)	728 (47.3)	400 (48.8)	192 (45.3)
SOL >10 minutes, *n* (%)	756 (49.2)	339 (41.3)	139 (32.8)
WASO > 60 minutes, *n* (%)	880 (57.2)	455 (55.5)	212 (50.0)
Sleep efficiency <80%, *n* (%)	730 (47.5)	368 (44.9)	166 (39.2)
Percentage of TST in N1 <10%, *n* (%)	837 (54.4)	446 (54.4)	240 (56.6)
Percentage of TST in N2 <65%, *n* (%)	750 (48.8)	464 (46.6)	254 (59.9)
Percentage of TST in N3 <10%, *n* (%)	971 (63.1)	467 (57.0)	238 (56.1)
Percentage of TST in REM <15%, *n* (%)	702 (45.6)	347 (42.3)	158 (37.3)

IQR, interquartile range; MET, metabolic equivalent; SF-36, Short-form quality of life instrument.
[a]Up to high school.
[b]Available for 913 participants.

lower sleep efficiency in the group with the middle category of PA (500 to 1500 MET-minutes/week at baseline) compared to the reference group (<500 MET-minutes/week). The additional analyses that included only those with PA and PSG assessed at the same study visit showed similar coefficients.

PA categories predicting continuous changes in sleep parameters

Regarding the relationship between baseline level of PA and changes in sleep during the follow-up, a greater decrease in the percent of TST in N3 was observed for those with intermediate or high PA at baseline when compared to the reference group (Table 3). Figure 2 illustrates the percent change in sleep duration and in each sleep stage between the baseline and the follow-up and according to the level of PA at baseline. Compared with the <500 MET-minutes/week participants, those with an intermediate PA (500 to 1500 MET-minutes/week) at baseline had a greater increase in sleep duration and decrease in N3.

Continuous PA and change in PA predicting binary sleep outcomes and continuous changes in sleep parameters

No association was seen in additional analyses with continuous PA variables as exposure (Supplementary Table S1).

Sleep conditions at baseline predicting continuous and percent changes in PA

Additionally, in general, sleep conditions did not predict changes in PA over the follow-up (Table 4 and Supplementary Table S2).

Discussion

In this prospective cohort of middle-aged and older adults based on in-laboratory PSG-assessed data, compared to lower levels of PA, having PA between 500 and 1500 MET-minutes/week predicted lower risk of new onset of TST < 6 hours/night, WASO > 60 minutes and sleep efficiency <80%. No significant associations

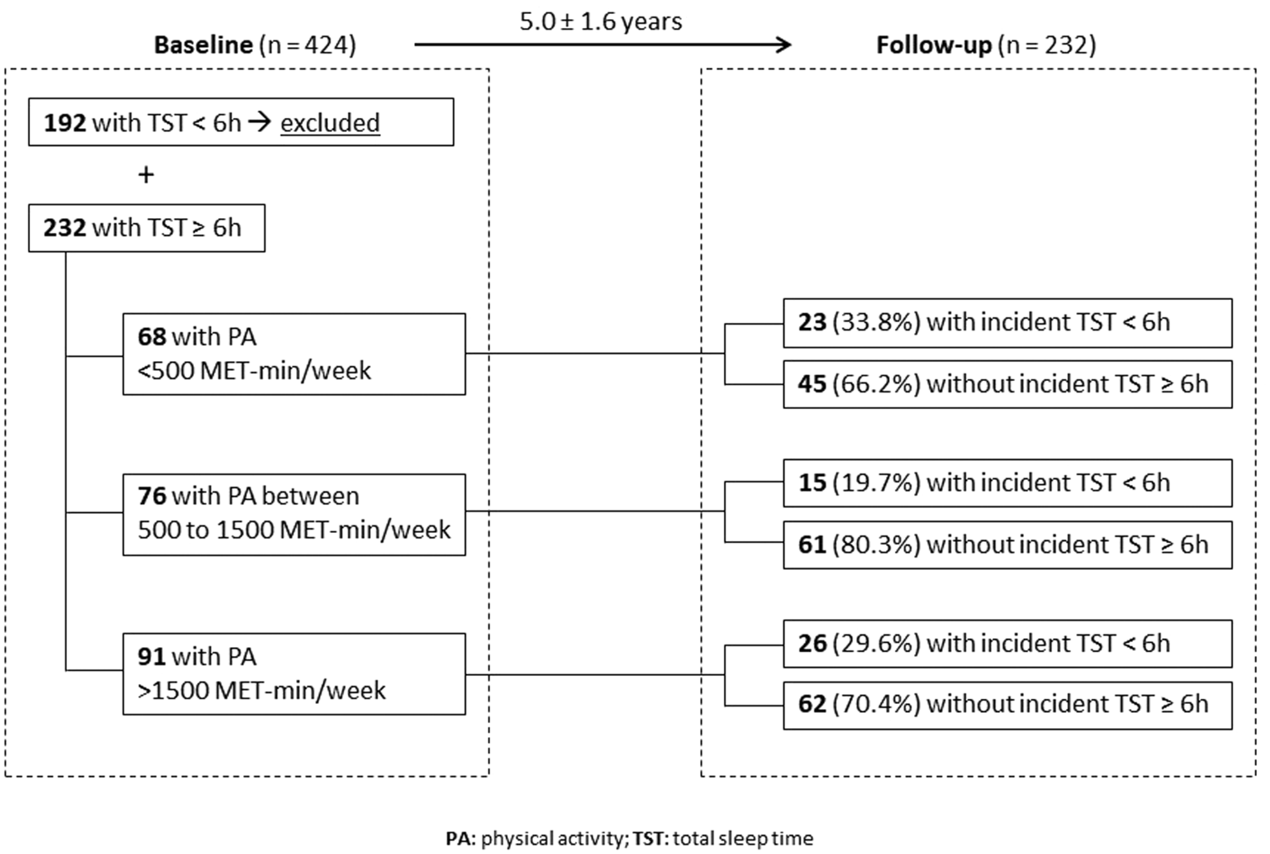

Figure 1. Diagram of the cohort study design used to explore the association between PA level at baseline and incident short sleep time in the follow-up (observation: this same template was applied for the other sleep outcomes).

were found between PA and either SOL or the percentage of TST in N1, N2, N3, or REM sleep, after adjustment for confounders.

The present findings are consistent with most available evidence regarding potential positive effects of PA on sleep. Previous systematic reviews [4–6, 25] have found that interventions based on PA can improve sleep; the degree of influence of PA varied according to individuals' characteristics, exercise programs, and sleep parameters analyzed. For instance, King et al. [26] showed that, compared to controls, older adults who participated in a 12-month exercise program had fewer awakenings during the first third of the sleep period, but no differences were found for other PSG parameters examined. In a study by Passos et al. [27], adults with chronic insomnia with a mean age of 45 years who completed a 6-month exercise training protocol had a significant decrease in SOL and WASO, and a significant increase in sleep efficiency following exercise. In another trial with PSG measurements of adults between 57 and 70 years of age, Melancon et al. [28] observed a decrease in WASO and no change in SOL, TST, and SE after 16 weeks of an exercise program. These findings suggest that sleep parameters are variably influenced by PA. These results are consistent with our study, where we found that PA was significantly associated with lower risk of some but not all sleep disturbances.

To the best of our knowledge, ours is the first study to identify that higher levels of PA predict lower risk of PSG-assessed sleep disturbances in middle-aged and older adults. This is consistent with prospective studies based on subjective sleep information. PA was longitudinally associated with lower risk of subjective indicators of better sleep, including REM disturbance [29] and sleep onset and sleep maintenance problems [30]. Morgan et al. [10] followed older adults for 4–8 years and observed that lower PA levels were associated with a significantly elevated risk of both insomnia persistence and insomnia incidence. Likewise, Chen et al. [12] found that adults aged 65 years and over with a high level of PA at baseline who kept this high level after 10 years of follow-up had lower risk of insomnia than other groups. Similar results were found in another study of adults; after 3 years of follow-up, frequent PA was associated with reduced incidence of insomnia, especially difficulty maintaining sleep [11]. On the other hand, Tsunoda et al. [14] followed older adults for 3–4 years and found a lower incidence of subjective insufficient sleep in those with moderate-to-low PA at baseline, but no relationship was found between PA and incidence of self-reported short TST. In another prospective study, Holfeld and Ruthig [13] followed community-dwelling older adults for 2 years and showed that initial PA did not predict later sleep quality after accounting for prior sleep quality. The divergence between the results of these two last studies in relation to ours and other longitudinal studies might be explained, at least partially, by the limited agreement between subjective and objective sleep duration and quality [15, 16].

In our study, we found that an intermediate but not the highest PA level was significantly associated with lower risk of sleep disturbances, in contrast to some short-term clinical trials that found a dose-response association between

Table 2. Number and percentage of incident sleep outcomes and relative risks[a] (95% confidence interval) of sleep outcomes by physical activity level at baseline

PA at baseline (MET-minutes/week)	TST < 6 hours	SOL > 10 minutes	WASO > 60 minutes	Sleep efficiency < 80%	Percentage of TST in N1 < 10%	Percentage of TST in N2 < 65%	Percentage of TST in N3 < 10%	Percentage of TST in REM < 15%
Total, *n*. of cases/ total (%)	64/232 (27.6)	122/285 (42.8)	107/212 (50.5)	97/258 (37.6)	60/184 (32.6)	47/170 (27.6)	81/186 (43.5)	97/266 (36.5)
PA level at baseline, *n* (%)								
<500	23 (33.8)	36 (47.4)	35 (57.4)	29 (42.0)	23 (36.5)	11 (23.9)	24 (41.4)	28 (38.9)
500 to 1500	15 (19.7)	40 (41.2)	31 (42.5)	25 (28.4)	21 (35.0)	16 (29.1)	29 (45.3)	34 (39.5)
>1500	26 (29.6)	46 (41.1)	41 (52.6)	43 (42.6)	16 (26.2)	20 (29.0)	28 (43.8)	35 (32.4)
Unadjusted model								
<500	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
500 to 1500	0.58 (0.33, 1.02)	0.87 (0.62, 1.21)	0.74 (0.52, 1.04)	0.67 (0.44, 1.04)	0.95 (0.60, 1.54)	1.21 (0.62, 2.35)	1.09 (0.73, 1.65)	1.02 (0.69, 1.50)
>1500	0.87 (0.55, 1.38)	0.87 (0.63, 1.20)	0.92 (0.68, 1.24)	1.01 (0.71, 1.45)	0.72 (0.42, 1.22)	1.21 (0.64, 2.28)	1.06 (0.70, 1.65)	0.83 (0.56, 1.24)
Model 1								
<500	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
500 to 1500	0.60 (0.34, 1.05)	0.93 (0.66, 1.32)	0.70 (0.50, 0.99)*	0.70 (0.45, 1.09)	1.22 (0.75, 2.01)	1.17 (0.58, 2.35)	1.05 (0.69, 1.59)	1.14 (0.78, 1.67)
>1500	0.80 (0.48, 1.32)	0.89 (0.63, 1.26)	0.82 (0.60, 1.12)	1.00 (0.68, 1.47)	0.88 (0.51, 1.53)	1.24 (0.61, 2.53)	0.99 (0.64, 1.54)	1.03 (0.69, 1.54)
Model 2								
<500	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
500 to 1500	0.49 (0.27, 0.88)*	0.93 (0.65, 1.33)	0.58 (0.41, 0.82)**	0.61 (0.39, 0.94)*	1.18 (0.72, 1.92)	1.42 (0.68, 2.98)	1.21 (0.77, 1.89)	1.23 (0.83, 1.83)
>1500	0.63 (0.37, 1.07)	0.91 (0.62, 1.33)	0.66 (0.47, 0.91)*	0.83 (0.55, 1.26)	0.83 (0.47, 1.64)	1.32 (0.61, 2.86)	1.12 (0.71, 1.74)	1.09 (0.72, 1.68)

N1, N2, N3: sleep stages. Model 1: Adjusted by age (years), sex (male vs. female), educational level (through high-school vs. some college), marital status (married vs. not married), current smoker (no vs. yes), alcohol intake (drinks/week), follow-up time (years) and by the difference in time between PA and sleep information (years) at baseline. Model 2: Adjusted by the same variables in Model 1 and by baseline BMI (kg/m^2), change in BMI over the follow-up (kg/m^2), self-rated health (excellent or very good vs. good, fair or poor), depression (no depression symptoms vs. depression symptoms or current use of antidepressants), baseline apnea-hypopnea index and continuous positive airway pressure use at baseline PSG.

[a]Relative risks (95% confidence interval) obtained with Poisson regression models adjusted by the covariates indicated for each model.

*$p < 0.05$; **$p < 0.01$.

exercise and sleep [31]. Indeed, in our study the relative risks were very similar in the two categories of higher PA, although only for the WASO > 60 minutes risk did they reach statistical significance in the group with the highest PA level. This could be due to the low statistical power to detect differences, as the frequency of most sleep disturbances was lower in the highest PA category in relation to the reference category. Moreover, baseline PA level did not predict changes in sleep parameters over ~5 years of follow-up for most of the sleep parameters, except for the percentage of TST in N3, where higher PA levels at baseline were associated with lower percentage of change in percentage of TST in N3, though with a small magnitude of effect. Changes in sleep were quite small and tended to be in the negative direction (e.g. increased SOL and decreased sleep efficiency). All results taken together, our data suggest that the relationship between PA and sleep is nonlinear, although these conclusions should be confirmed in future studies.

Regarding the temporality of the relationship between PA and sleep, in this cohort study we did not find a consistent association of baseline sleep with subsequent changes in PA. However, in the study of Holfeld and Ruthig [13], the authors found that better initial sleep quality predicted higher levels of later PA whereas the inverse association was no longer significant when accounting for prior sleep quality. Their results are also consistent with some evidence of a stronger association between sleep predicting next-day PA rather than PA predicting that night's sleep [17, 18]. Future prospective studies based on objective sleep measurements are still needed to replicate the present findings and to address whether the association between PA and sleep is, in fact, bidirectional over a longer follow-up period.

Several mechanisms may underlie positive associations, if causal, of PA on sleep. These non-exhaustively include: thermoregulatory effects of exercise; sunlight exposure during outdoor PA; exercise-mitigated sleep apnea severity [32]; and, positive effects of PA on mood and stress [33–35]. During PA, body core temperature increases and peripheral temperature decreases [36]; the drop in core temperature subsequent to PA has been hypothesized to promote sleep propensity [37]. Furthermore, when PA occurs in external environments and during the daylight, sunlight exposure could increase melatonin secretion [38], a hormone directly linked to sleep-promoting effects. Also, exercise has been associated with improved mood [39], and depressed mood has been associated with worse future sleep quality in 10 of 13 studies detailed in a systematic review [40].

There are a few important limitations of the present study. First, although in-laboratory PSG is the gold-standard method for sleep evaluation, the laboratory context and PSG equipment can influence some sleep parameters so that they are systematically different from the habitual home setting. Also, we used only one night of sleep data at each study visit; this may be a study limitation because of potential night-to-night variability in sleep parameters. However, because we used as baseline sleep studies (for this analysis) PSG evaluations from 2004 and later, all included participants had had previous in-laboratory PSG evaluations (i.e. from WSC inception in 1988 up to 2003), and were thus at least partially habituated to the sleep laboratory environment. Furthermore, we have no reason to believe there would be substantial systematic variations in the difference in sleep parameters assessed in-laboratory vs. naturalistic environments among participants with higher and lower levels of PA. Second, although we used a

Table 3. Median and interquartile range of sleep changes and beta coefficients[a] (standard error) of continuous changes in sleep parameters by PA level at baseline

PA at baseline (MET-minutes/week)	TST change	SOL change	WASO change	Sleep efficiency change	Percentage of TST in N1 change	Percentage of TST in N2 change	Percentage of TST in N3 change	Percentage of TST in REM change
Total, median (IQR)	2.3 (−33.5, 46.0)	2.0 (−3.0, 11.3)	11.5 (−11.0, 40.7)	−1.9 (−8.5, 4.3)	1.1 (−2.6, 4.3)	2.6 (−2.1, 9.6)	−2.5 (−6.6, 0.2)	−1.6 (−5.5, 3.2)
PA at baseline, median (IQR)								
<500	−1.5 (−39.3, 43.0)	2.5 (−3.5, 14.5)	8.5 (−14.5, 42.8)	−2.2 (−9.0, 5.3)	0.3 (−3.4, 3.4)	2.6 (−3.0, 10.7)	−2.3 (−6.6, 1.6)	−2.2 (−6.4, 3.5)
500 to 1500	9.8 (−25.9, 53.5)	1.5 (−3.3, 8.3)	13.5 (−9.6 (37.5)	−0.5 (−8.2, 4.5)	2 (−2.0, 5.0)	2.5 (−1.5, 10.7)	−2.6 (−6.9, −0.1)	−1.4 (−4.9, 2.6)
>1500	0.5 (−36.3, 41.3)	1.8 (−2.5, 9.3)	16.0 (−9.3, 41.2)	−2.5 (−8.5, 3.2)	1.2 (−2.3, 4.4)	2.9 (−2.1, 9.2)	−2.4 (−6.1, 0.1)	−1.2 (−5.1, 3.5)
Unadjusted models								
<500	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
500 to 1500	0.14 (0.12)	−3.86 (2.51)	1.40 (5.27)	0.39 (1.29)	1.26 (0.78)	0.41 (1.16)	−1.49 (0.82)	−0.15 (0.82)
>1500	−0.01 (0.12)	−3.22 (2.46)	6.34 (5.18)	−1.08 (1.26)	1.18 (0.76)	−0.28 (1.14)	−0.99 (0.81)	0.07 (0.81)
Model 1								
<500	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
500 to 1500	0.16 (0.13)	−3.03 (2.56)	1.11 (5.39)	0.41 (1.31)	1.17 (0.79)	0.94 (1.14)	−1.76 (0.78)*	−0.34 (0.83)
>1500	0.01 (0.13)	−2.63 (2.60)	4.45 (5.48)	−0.66 (1.33)	1.14 (0.81)	0.93 (1.15)	−1.66 (0.79)*	−0.42 (0.84)
Model 2								
<500	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
500 to 1500	0.24 (0.13)	−1.99 (2.62)	−2.71 (5.49)	1.26 (1.33)	1.05 (0.81)	0.89 (1.17)	−1.83 (0.80)*	−0.11 (0.85)
>1500	0.12 (0.13)	−0.56 (2.72)	−0.69 (5.71)	0.29 (1.39)	0.75 (0.84)	0.93 (1.21)	−1.82 (0.83)*	0.13 (0.88)

N1, N2, N3: sleep stages. Model 1: Adjusted by age (years), sex (male vs. female), educational level (through high school vs. some college), marital status (married vs. not married), current smoker (no vs. yes), alcohol intake (drinks/week), follow-up time (years) and by the difference in time between PA and sleep information (years) at baseline. Model 2: Adjusted by the same variables in Model 1 and by baseline BMI (kg/m^2), change in BMI over the follow-up (kg/m^2), self-rated health (excellent or very good vs. good, fair or poor), depression (no depression symptoms vs. depression symptoms or current use of antidepressants), baseline apnea-hypopnea index and continuous positive airway pressure use at baseline PSG.

[a]Beta coefficients (standard error) obtained with mixed-effects models adjusted by the covariates indicated for each model.

*$p < 0.05$.

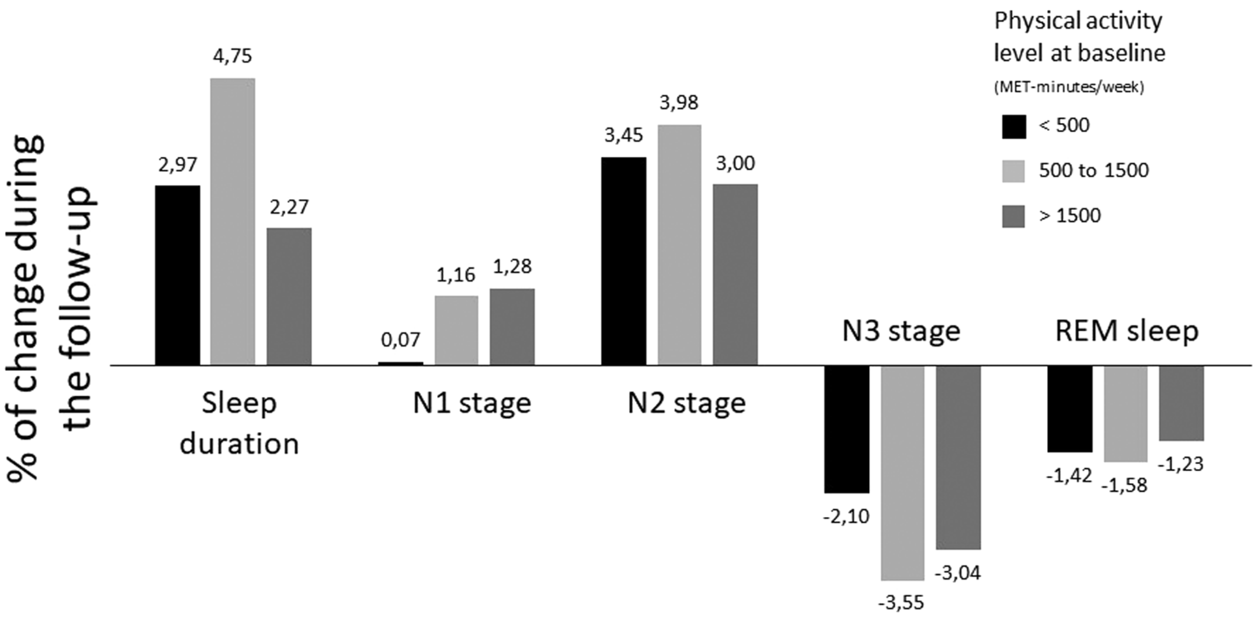

Figure 2. Percentage of change in sleep duration and in sleep stages by PA at baseline.

validated questionnaire for estimation of energy expenditure in MET-minutes/week, self-reported PA is not a gold-standard assessment method and generally overestimates objectively-assessed PA [41]; if the degree of measurement error in PA at baseline was unrelated to risk of developing sleep problems, this would likely result in underestimates of true associations—i.e. a bias to the null between the highest level of baseline PA and sleep problems. Lastly, although residual confounding is possible, the present analyses controlled for several covariates, including sociodemographic, lifestyle and health conditions, as well as changes in PA and in BMI during the follow-up.

This study provides evidence consistent with a beneficial effect of PA on the risk of short sleep time, higher WASO, and lower sleep efficiency. These findings are in accordance with previously published results, supporting the promotion of PA to improve sleep quality and prevent sleep disturbances. In addition to other well-known health-related benefits of PA, PA might also be considered as a potential protective factor for sleep disturbances in middle-aged and older adults.

Table 4. Beta-coefficients[a] (standard error) of continuous and percent changes in PA by sleep conditions at baseline

	Continuous change in PA (MET-minutes/week)	Percent change in PA (%)
Sleep condition (exposure)	Beta-coefficient (standard error)	Beta-coefficient (standard error)
Total		
TST (hours)		
≥6	Reference	Reference
<6	−0.6 (2.5)	27.0 (63.1)
SOL (minutes)		
≤10	Reference	Reference
>10	−2.4 (2.5)	−34.3 (63.9)
WASO (minutes)		
≤60	Reference	Reference
>60	−2.8 (2.5)	46.8 (63.3)
Sleep efficiency (%)		
≥80	Reference	Reference
<80	−1.2 (2.6)	52.6 (65.5)
TST in N1 sleep (%)		
≥10	Reference	Reference
<10	2.5 (2.5)	3.3 (62.4)
TST in N2 sleep (%)		
≥65	Reference	Reference
<65	1.6 (2.5)	6.6 (64.0)
TST in N3 sleep (%)		
≥10	Reference	Reference
<10	−6.1 (2.6)*	−48.0 (66.0)
TST in REM (%)		
≥15	Reference	Reference
<15	0.9 (2.5)	4.4 (63.6)

N1, N2, N3: sleep stages.
[a]Beta-coefficients obtained with mixed-effect models adjusted by age (years), sex (male vs. female), educational level (through high school vs. some college), marital status (married vs. not married), current smoker (no vs. yes), alcohol intake (drinks/week), follow-up time (years), difference in time between PA and sleep information (years) at baseline, baseline BMI (kg/m^2), change in BMI over the follow-up (kg/m^2), self-rated health (excellent or very good vs. good, fair or poor), depression (no depression symptoms vs. depression symptoms or current use of antidepressants), baseline apnea-hypopnea index and continuous positive airway pressure use at baseline PSG.
*$p < 0.05$.

Supplementary material

Supplementary material is available at *SLEEP* online.

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Notes

Conflict of interest statement. None declared.

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