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Associations of sedentary time and moderate-vigorous physical activity with sleep-disordered breathing and polysomnographic sleep in community-dwelling adults

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Abstract

Purpose—The purpose of this study was to evaluate the relationship between daytime activity (sedentary time, moderate-to vigorous-intensity physical activity [MVPA]) and indices of polysomnographically (PSG) assessed sleep, including sleep-disordered breathing (SDB).

Methods—One hundred and thirty-six adults (65% female, 59.8 ± 9.1 years, body mass index [BMI] 30.3 ± 6.9 kg m⁻²) provided daily estimates of time spent in light-, moderate-, and vigorous-intensity activity for 6–14 days (mean 9.9 ± 1.8 days) prior to laboratory PSG. Daily sedentary time was calculated as the amount of time spent awake and not in light-, moderate-, or vigorous-intensity activity; time spent in moderate- and vigorous-intensity activity were combined for MVPA. Indices of PSG sleep included timing (sleep midpoint), duration (total sleep time), continuity (sleep efficiency), depth (% slow-wave sleep), and SDB (apnea-hypopnea index [AHI]). Using median splits of sedentary time and MVPA, analyses of covariance examined their relationship with sleep following adjustment for age, sex, race, employment, education, BMI, existing cardiovascular disease, depression history, and mean daily wake time. Binary logistic regression examined the odds of having at least mild-severity SDB (AHI ≥ 5) according to sedentary time, MVPA, and their combination.

Results—Adults with above-median sedentary time (i.e., >841.9 min/day) had significantly greater AHI ($P = .04$) and lower odds of mild SDB ($P = .03$) compared to adults with low sedentary time; adults with high MVPA (>30.5 min/day) had significantly lower AHI compared to adults with low MVPA ($P = .04$). When examined in the same model, adults with high sedentary

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time and low MVPA had significantly higher AHI ($P < .01$) and higher odds of having mild SDB ($P = .03$) than all the other groups. No other sleep measures were related to sedentary time, MVPA, or their combination.

Conclusions—Sedentary time and MVPA were associated with SDB. Whether reducing sedentary time leads to lower SDB severity deserves future exploration.

Keywords

Physical activity; Exercise; Sedentary behavior; Sleep-disordered breathing; Sleep apnea

Introduction

Sleep is a multidimensional behavior, with characteristics such as duration, timing, continuity, depth, and breathing each being relevant to optimal health and functioning [1]. Indeed, poor or inadequate levels of each of these dimensions of sleep have been associated with adverse health outcomes, including cognitive impairment, type 2 diabetes, and cardiovascular disease [1, 2]. Poor or inadequate sleep is common among adults, as up to 30% habitually obtain less than 6 h of sleep per night [3], more than 15% report frequent difficulty initiating or maintaining sleep [4], and up to 10% have clinically significant sleep-disordered breathing (SDB) [5]. Because most adults do not seek professional treatment for their sleep problems [6, 7], lifestyle approaches that optimize sleep may be especially useful.

Physical activity is a behavior that has been commonly associated with better sleep. Epidemiologic studies have found that higher levels of moderate- to vigorous-intensity physical activity (MVPA) are associated with a lower likelihood of sleep disturbance and SDB [8, 9], whereas meta-analyses of experimental studies have documented improvements in sleep continuity, depth, and SDB severity following exercise training [10, 11]. Although some studies have found physical activity to be unrelated to sleep [12, 13], findings have been more consistent for samples in which sleep disturbance was prevalent [14].

Much less is known about the relationship between sedentary behavior and sleep. Sedentary behavior, defined as any waking activity involving low energy expenditure and a sitting or reclining position [15], is more prevalent than any other daytime behavior [16]. Independent of MVPA, high levels of sedentary behavior have been associated with a variety of adverse health consequences [17]. However, reports on the relationship between sedentary time and sleep have been inconsistent. Greater sedentary time has been associated with lower sleep efficiency [18], excessively short or long sleep duration [19], worse sleep quality [20, 21], and a greater likelihood of having sleep problems [22, 23]; however, other studies have found no relationship between sedentary time and sleep [24–29]. In addition to the discrepant findings, no research in this area has assessed sleep with polysomnography (PSG), the gold standard measure, and few studies have examined the combination of MVPA and sedentary time in relation to sleep [20, 21, 24, 27].

The purpose of this study was to examine the relationship between sedentary time, physical activity, and sleep parameters essential to sleep health and quantifiable with PSG, including indices of timing, duration, continuity, depth, and SDB. We hypothesized that high sedentary

time and low MVPA, individually and considered together, would be associated with later sleep timing, shorter sleep duration, worse sleep continuity, lower sleep depth, and greater SDB severity.

Methods

Sample

Participants for these analyses were from a sample of midlife and older adults enrolled in a study on sleep, depression, and cardiovascular risk. The study protocol was approved by the University of Pittsburgh Institutional Review Board. Informed consent was obtained from all individual participants included in the study. The primary eligibility criterion for this study was prior involvement in at least one research study conducted at the University of Pittsburgh from 1982 to 1999 that was focused on sleep and depression.

Of the 177 adults who consented to participate, participants were excluded from the present analyses due to bipolar disorder, psychotic depression or suicidal ideation ($n = 20$), or missing PSG or covariate data ($n = 21$). Compared to those included in the analyses ($N = 136$), excluded participants had a significantly higher apnea-hypopnea index (AHI) (21.0 ± 23.7 vs. 8.1 ± 11.2 ; $P < .001$) and later sleep midpoint (3.40 ± 1.15 vs. 2.91 ± 0.87 h past midnight; $P = .03$). Otherwise, no differences were observed between included versus excluded participants.

Procedures

The participants completed diary-based assessments of daytime and sleep behaviors for 6–14 days prior to two nights of PSG.

Daytime activity assessment—Participants used daily diaries to record daytime and sleep behaviors (mean 9.9 ± 1.8 days). At bedtime each day, participants indicated the time spent in three different intensities of physical activity, with descriptions provided to participants noted in parentheses: light- (i.e., causes small increases in breathing and heart rate, such as vacuuming), medium- (i.e., causes moderate increases in breathing and heart rate, such as brisk walking, dancing, or swimming), or heavy- (i.e., causes large increases in breathing and heart rate, such as jogging) intensity activity. Participants also indicated the number of minutes they napped. Daily sedentary time was calculated as the number of minutes spent awake and not engaged in light-, moderate-, or heavy-intensity activity. The number of minutes of medium- and heavy-intensity activity were combined to provide a daily estimate of MVPA.

Polysomnographic sleep assessment—Polysomnography was conducted in the laboratory over two consecutive nights, with evening bedtime and morning out-of-bed time based upon the participants' self-reported habitual sleep/wake schedules. A standard PSG montage was utilized on both nights, including bilateral frontal, central, and occipital electroencephalogram leads referenced to linked mastoid leads (A1+A2), left and right electrooculogram leads referenced to A1+A2, and bipolar submental is electromyogram. Additional monitors, included for determination of SDB on the first PSG night only,

included a nasal pressure cannula, oronasal thermistor, thoracic and abdominal respiratory inductance plethysmography, and fingertip oximetry. Although SDB was assessed from the first night, other sleep parameters were derived from the second night of recording due to the commonly noted “first-night effect” [30] that was also observed in this sample. Two participants did not complete a second PSG night; for these individuals, we used their first PSG night for assessment of all their sleep parameters. Sensitivity analyses verified that results were unchanged when excluding these two participants.

Visual sleep stage scoring was performed in 30-s epochs according to standard criteria [31]. As a measure of sleep timing, sleep midpoint was calculated as the time midway between sleep onset and time of final awakening. As a measure of sleep duration, total sleep time (TST) was calculated as the total minutes of any sleep stage following sleep onset. As a measure of sleep continuity, sleep efficiency (SE) was calculated as the percentage of TST relative to time in bed. As a measure of sleep depth, slow-wave sleep (SWS) was calculated as the percentage of visually scored N3 sleep relative to TST.

Respiratory events were scored according to standard criteria at the time of study initiation [31]: an apnea was defined as 90% airflow reduction for 10 s and a hypopnea was defined as 30% reduction in airflow accompanied by 4% decrease in oxyhemoglobin saturation. The apnea-hypopnea index served as the index of SDB severity and was calculated as the sum of apneas and hypopneas divided by TST in hours. Participants were categorized as having at least mild-severity SDB if they had an AHI ≥ 5 .

Covariates—Sociodemographic factors and indices of mental and physical health were included as covariates due to their possible relationship with activity and/or sleep. Age, sex, race, and education were self-reported. Race was dichotomized into “white” and “black/other” due to a limited number of nonwhite participants. Education was dichotomized into “less than a college degree” and “college degree or more.” Employment status was dichotomized into “currently employed” and “retired/unemployed.” Participants’ height and weight were measured for calculation for body mass index (BMI; kilograms per square meter). Existing cardiovascular disease (CVD) was based on self-report of any of the following conditions: angina, angioplasty, myocardial infarction, bypass surgery, coronary artery disease, heart failure, heart surgery, stroke, or transient ischemic attack. Depression history was based on a clinician-defined diagnosis of major depressive disorder at the time of participation in an earlier research study (1982–1999) or the current study. Mean diary-based daily wake time was calculated as the interval between each morning’s out-of-bed time to each night’s bedtime minus daytime napping duration, averaged across all days of recording.

Statistical analyses

Skewed sleep variables were transformed prior to analyses. Slow-wave sleep was transformed by natural logarithm (ln), sleep efficiency was reverse-scored (i.e., 100-SE) and ln-transformed, and AHI (plus a constant of 1) was ln-transformed.

Analysis of covariance (ANCOVA) was used to examine differences in AHI and PSG sleep according to sedentary time and MVPA following categorization of sedentary time and

MVPA into two groups each according to median values. Sedentary time and MVPA were examined individually and in combined models; in models that included both sedentary time and MVPA, an interaction term was added. Each model included age, sex, race, education, employment, BMI, existing CVD, depression history, and mean daily wake time as covariates. We also evaluated models that expressed sedentary time and MVPA as percentages of total daily wake time using the above-named covariates (aside from mean daily wake time) in the models; results were unchanged from the models described above.

Using binary logistic regression, we evaluated the odds of having at least mild-severity SDB (i.e., AHI ≥ 5) according to categorizations of sedentary time, MVPA, and their combination as performed in the ANCOVA models. Logistic regression models included the same covariates as noted above.

Analyses were conducted with SAS software (v. 9.4). All tests were two-tailed, with statistical significance set at $P < .05$.

Results

Participant characteristics

Participant characteristics are summarized in Table 1. The majority were female (65.4%), white (93.4%), and had a lifetime history of major depressive disorder (59.6%). Mean daily sedentary (SED) time was 818.7 ± 136.2 min/day; median SED time was 841.9 min/day. Participants reported 46.0 ± 51.6 min/day of MVPA; median MVPA time was 30.5 min/day.

When characterized based upon the combination of categorized sedentary (SED) time and MVPA, groups differed by sex ($P = .01$), depression history ($P = .05$), current employment status ($P = .04$), total wake time ($P < .001$), SED time ($P < .001$), and MVPA time ($P < .001$). See Table 1 for specific between-group differences.

Sedentary time, MVPA, and SDB

Mean AHI for the sample was 8.1 ± 11.2 and 43% ($n = 58$) had at least mild-severity SDB (i.e., AHI ≥ 5). When examined independently of MVPA, adults with high SED time (>841.9 min/day) had significantly higher AHI values ($F_{1,125} = 4.41$, $P = .038$; Fig. 1a) and greater odds of having at least mild SDB (odds ratio [OR] = 3.22 [95% confidence interval 1.14, 9.08]; $P = .027$) compared to adults with low SED time. When examined independently of SED time, adults with high MVPA (>30.5 min/day) had significantly lower AHI values ($F_{1,125} = 4.28$, $P = .041$; Fig. 1b) compared to those with low MVPA. Those with high MVPA also had a statistically nonsignificant lower odds of having at least mild SDB compared to those with low MVPA (OR = 0.52 [0.23, 1.20]; $P = .12$).

When considered simultaneously, the significant interaction term ($F_{1,123} = 4.93$; $P = .028$) indicated that the association between SED time and AHI differed by MVPA. The combination of MVPA and SED time, when categorized into four separate groups, was significantly associated with AHI ($F_{3,123} = 3.76$; $P = .013$); adults with high SED and low MVPA had significantly higher AHI compared to that of low SED/low MVPA ($P = .015$), high SED/high MVPA ($P = .014$), and low SED/high MVPA ($P = .015$) (Fig. 1c). No other

between-group comparisons were significant. Similarly, when examining the odds of having an AHI ≥ 5 , the significant interaction term ($P = .047$) indicated the odds of mild SDB according to SED time differed by MVPA. When categorized into four groups, adults with high SED and low MVPA had greater odds of having at least mild SDB compared to low SED/low MVPA (OR = 0.15 [0.04, 0.64]; $P = .011$), high SED/high MVPA (OR = 0.29 [0.08, 1.08]; $P = .064$), and low SED/high MVPA (OR = 0.27 [0.09, 0.85]; $P = .025$).

Sedentary time, MVPA, and PSG sleep

Mean sleep midpoint, SE, TST, and SWS for the sample were 2.9 ± 0.9 h past midnight, $84.4 \pm 10.7\%$, 6.5 ± 1.0 h, and $10.0 \pm 9.0\%$, respectively (Table 2). When examined in separate models, none of the sleep variables differed according to SED time ($P = .26$ for each) or MVPA ($P = .32$ for each). When SED time and MVPA were included in the same model, neither SED time ($P = .25$ for each), MVPA ($P = .27$ for each), nor the interaction term ($P = .13$ for each) were significant factors (Table 2).

Discussion

This study evaluated the relationship between sedentary time, physical activity, and PSG-assessed SDB and parameters encompassing sleep timing, duration, continuity, and depth. We found that high sedentary time and low MVPA were associated with higher AHI values and that those with the combination of high sedentary time and low MVPA had higher AHI values and greater odds of having at least mild SDB compared to that of all other sedentary-MVPA combinations. However, we did not observe any significant associations between other sleep parameters and either sedentary time, MVPA, or their combination.

To our knowledge, this is the first report to document that adults with high levels of sedentary time have higher PSG-assessed SDB severity in a community-based sample. Previously, Buman and colleagues reported 15% higher odds of meeting the “high risk” criteria for SDB for every 1-h increase in television use while sitting in a sample of 843 adults [20]. Our results corroborate this prior research and extend these findings to a community-based sample using objectively assessed SDB.

Our finding of low levels of MVPA being associated with higher AHI is consistent with previous epidemiologic and experimental studies. In the Wisconsin Sleep Cohort, greater hours of exercise were associated with lower odds of prevalent and incident SDB [9, 32]. Moreover, a recent meta-analysis of experimental trials found that exercise training significantly reduces AHI [10]. Thus, our results are consistent with previous research and suggest that exercise may have a potential benefit on SDB severity.

Perhaps our most intriguing and novel finding was that the combination of high sedentary time and low MVPA was associated with a significantly higher AHI than any of the other sedentary-MVPA combinations. Buman and colleagues found that regular physical activity was protective against SDB risk associated with sedentary time from television viewing, as the relationship between greater television sitting time and higher odds of meeting the “high risk” SDB criteria was only apparent among adults who were physically inactive [20]. Although these cross-sectional data preclude definitive statements regarding causality,

reducing sedentary time or increasing MVPA may both be effective strategies for reducing SDB severity.

Given the high level of sedentary time noted in this and other samples of adults with SDB [33], reduction of sedentary time may be an appealing intervention target. In particular, displacing sedentary time with light-intensity activity (e.g., standing, household activity) may be a practical intervention approach. To our knowledge, only one study has examined whether sedentary behavior changes in adults with SDB following a lifestyle intervention. In a sample of 63 adults with SDB who had just begun treatment with continuous positive airway pressure (CPAP), those who received a 6-month intervention focused on increasing activity did not modify sedentary time or MVPA more than those who only received CPAP [34]. Future interventions with a more explicit focus on reducing sedentary behavior [35] are needed to address the feasibility and efficacy of such an approach on SDB severity.

A unifying hypothesis for how reducing sedentary time or increasing MVPA may reduce SDB severity is through reduction of the nocturnal rostral fluid shift. The nocturnal rostral fluid shift involves the redistribution of fluid accumulated in the lower extremities during the daytime to the chest/neck region overnight upon reclining to sleep, which increases SDB susceptibility through reducing the upper airway luminal area and increasing upper airway collapsibility [36]. As lower-limb physical activity (via the skeletal-muscle pump) is the primary method to prevent lower-limb fluid accumulation, physical activity may reduce lower-limb fluid accumulation and the magnitude of overnight fluid shift. Previously, Redolfi and colleagues found that daytime sitting duration was correlated with the magnitude of nocturnal rostral fluid shift ($r = -.59$) and AHI ($r = .54$) [37]. Moreover, two experimental trials have demonstrated that 1–4 weeks of moderate-intensity aerobic exercise lowers AHI alongside reduction in the magnitude of the nocturnal rostral fluid shift [38, 39]. However, some studies focused on exercise training and SDB have found post-intervention AHI to be reduced relative to baseline even when no exercise was performed on the day of post-intervention assessment [40]; thus, exercise likely also reduces AHI through other mechanisms, such as more stable respiration through less fragmented and “deeper” sleep and/or increased fatigue resistance of the upper airway dilator muscles. However, these additional mechanisms remain speculative and it is unknown whether reducing sedentary behavior leads to any similar adaptations that reduce AHI.

Contrary to our initial hypothesis, sedentary time and MVPA were not significantly associated with any other PSG sleep parameters (sleep midpoint, sleep efficiency, total sleep time, slow-wave sleep). These findings are in contrast to previous research in which exercise was associated with better sleep continuity, longer sleep duration, and increased sleep depth [11]. However, our lack of significant relationships among MVPA and other indices of sleep may be explained by our use of PSG and the relatively good sleep of this sample, as prior studies have found exercise to be more consistently associated with subjective sleep improvement compared to PSG [11], and exercise seems to be less beneficial for adults without significant sleep disturbance [14].

The majority of our sample consisted of females (65.4%) and adults with a history of depression (59.6%); poor sleep has previously been noted among midlife and older women

[41] and adults with depression [42]. In addition, SDB is commonly associated with higher prevalence of depression [43]. Although we adjusted for sex and depression history in our analyses, these sample characteristics—in addition to being predominantly white and well educated—limit the generalizability of this study. Whether our findings extend to other samples remains unknown.

Additional study limitations include our measurement of activity: physical activity was self-reported and sedentary time was extrapolated as the absence of reported activity during wake time. Self-reported and objectively assessed measures of MVPA and sedentary time have previously been shown to be only modestly correlated [44]. However, the majority of epidemiologic research into the associations between physical activity, sedentary behavior, and health have relied upon self-report [17]. Nevertheless, accelerometry-derived measures of activity and sedentary time would provide more accurate assessments. Finally, the cross-sectional nature of this study precludes any definitive inferences about causality, as the relationship is likely bidirectional (i.e., that SDB may lead to less MVPA and greater sedentary time). Strengths of this study include a community-based sample with diverse patterns of daytime and sleep behaviors and assessment of sleep parameters using PSG.

Overall, we found that high sedentary time and low MVPA, and especially their combination, are associated with higher AHI values. However, sedentary time and MVPA were not associated with any other sleep parameters. These results align with and support recent research documenting the efficacy of exercise training on SDB severity and suggest that reducing sedentary time may also reduce SDB severity.

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Fig. 1.

Apnea-hypopnea index values across groups categorized by sedentary time, MVPA, and their combination. **a** AHI according to median split of SED time. **b** AHI according to median split of MVPA. **c** AHI according to combination of SED time and MVPA. Data shown are unadjusted values (mean $\pm$ standard error) prior to analyses. AHI apnea-hypopnea index; MVPA moderate- to vigorous-intensity physical activity; SED sedentary time

Table 1

Participant characteristics

Variable	All (N = 136)	High SED, low MVPA ¹ (n = 48)	Low SED, low MVPA ² (n = 20)	High SED, high MVPA ³ (n = 20)	Low SED, high MVPA ⁴ (n = 48)	Group comparisons	
						P value	Groups
Age, years	59.8 (9.1)	58.4 (10.2)	63.0 (7.9)	59.1 (6.1)	60.2 (9.4)	.29	
Sex, n female (%)	89 (65.4)	34 (70.8)	18 (90.0)	9 (45.0)	28 (58.3)	.01	1, 2 > 3; 2 > 4
Race, n white (%)	127 (93.4)	42 (87.5)	19 (95.0)	20 (100.0)	46 (95.8)	.61	
Education, n with college degree (%)	97 (71.3)	35 (72.9)	11 (55.0)	17 (85.0)	34 (70.8)	.21	
BMI, kg/m ²	30.3 (6.9)	30.7 (8.5)	30.6 (4.9)	28.5 (4.9)	30.5 (6.7)	.65	
Currently employed, n (%)	83 (61.0)	34 (70.8)	8 (40.0)	15 (75.0)	26 (54.2)	.04	1, 3 > 2
Depression history, n (%)	81 (59.6)	26 (54.2)	17 (85.0)	9 (45.0)	29 (60.4)	.05	2 > 1, 3, 4
Existing CVD, n (%)	20 (14.7)	6 (12.5)	1 (5.0)	3 (15.0)	10 (20.8)	.37	
Total wake time, min/day	944.4 (71.4)	977.7 (59.4)	896.8 (45.8)	1003.4 (48.7)	906.3 (65.1)	<.001	1, 3 > 2, 4
SED, min/day	818.7 (136.2)	927.1 (57.8)	758.2 (80.8)	908.3 (41.2)	698.2 (121.8)	<.001	1, 3 > 2 > 4
MVPA, min/day	46.0 (51.6)	9.1 (8.9)	15.8 (10.4)	54.8 (16.3)	91.8 (58.9)	<.001	4 > 3 > 1, 2

Data shown are mean (standard deviation) or n (%), as appropriate. "High" indicates above-median values for SED (841.9 min/day) or MVPA (30.5 min/day); "low" indicates below-median SED and MVPA values. Group comparisons, performed with one-way ANOVA or chi-square analyses, are noted on the far-right column; significant differences between groups are noted according to superscripted numbers next to group label.

BMI body mass index, CVD cardiovascular disease, MVPA moderate- to vigorous-intensity physical activity, SED sedentary time

Table 2

Polysomnographic sleep across groups categorized by sedentary time and MVPA

Variable	All (N = 136)	High SED, low MVPA (n = 48)	Low SED, low MVPA (n = 20)	High SED, high MVPA (n = 20)	Low SED, high MVPA (n = 48)
Sleep midpoint, h ^a	2.91 (0.87)	2.66 (0.83)	3.24 (0.94)	2.84 (0.83)	3.06 (0.85)
SE, % ^b	84.40 (10.70)	85.99 (11.61)	80.69 (12.90)	87.65 (8.22)	82.99 (9.18)
TST, h	6.54 (1.01)	6.51 (1.11)	6.76 (1.29)	6.22 (0.97)	6.61 (0.77)
SWS, % ^c	10.03 (9.00)	10.50 (8.25)	11.33 (9.93)	8.17 (11.56)	9.80 (8.27)

Data presented are unadjusted values [mean (standard deviation)] prior to analyses.

MVPA moderate- to vigorous-intensity physical activity, SE sleep efficiency, SED sedentary time, SWS slow-wave sleep, TST total sleep time

^aExpressed as hours past midnight

^bReverse-scored (i.e., 100-SE) and natural-log (ln) transformed for statistical analyses

^cln transformed for statistical analyses