

Obstructive sleep apnea negatively impacts objectively measured physical activity

Trent A. Hargens¹  · Ryan A. Martin¹ · Courtney L. Strosnider¹ · Gabrielle Elam Williams Giersch¹ · Christopher J. Womack¹

Received: 26 April 2018 / Revised: 25 June 2018 / Accepted: 12 July 2018 / Published online: 18 July 2018

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Abstract

Purpose Obesity and obstructive sleep apnea (OSA) are frequent comorbid conditions. The impact of OSA on objectively measured physical activity (PA), independent of obesity, is not clear. The purpose of this study is to examine the effect of OSA on PA measured via accelerometer.

Methods Overweight-to-obese individuals were recruited and screened for the presence of OSA via portable diagnostic device and divided into an OSA ($n = 35$) and control group ($n = 24$). Daytime sleepiness was assessed with the Epworth Sleepiness Scale. Body composition was assessed with dual-energy X-ray absorptiometry. Subjects wore an accelerometer (Actigraph GT3X+, Actigraph Corp., Pensacola, FL) for a minimum of 4 and maximum of 7 days, including at least one weekend day.

Results There were no group differences in body mass index (BMI) or daytime sleepiness. Waist and neck circumference were higher in the OSA group. The OSA group was significantly older than the control group. The OSA group had fewer steps, moderate intensity minutes, moderate-to-vigorous minutes, number of PA bouts per day (\geq moderate intensity PA for ≥ 10 consecutive minutes), and total number of PA bouts. When adjusted for age, the PA bout data was no longer significant.

Conclusion Individuals screened as likely possessing OSA were less physically active than individuals without OSA when measured through objective means. We found no group differences in daytime sleepiness, BMI, or percent fat, suggesting other mechanisms than obesity and sleepiness for this difference.

Keywords OSA · Obesity · Physical activity · Accelerometer · Steps

Introduction

Recent estimates on the prevalence of obstructive sleep apnea (OSA) in US adults have increased in the last 2 decades. Previous estimates have suggested the overall OSA prevalence to be approximately 2–4% of middle-aged adults [1]. More recently, estimates have ranged between 3 and 17% for moderate-to-severe OSA, depending on age and gender, with male gender and increasing age resulting in the greater prevalence numbers [2]. Obesity is a common risk factor for OSA, where it has been estimated that 40–60% of obese individuals have OSA [3], and 70% of those with OSA are obese [4]. The

relationship between OSA and obesity has been well established, with both conditions, independent of each other, also increasing the risk for cardiovascular disease (CVD), diabetes mellitus, hypertension (HTN), and other conditions [5, 6].

The physiological mechanisms linking OSA and other chronic conditions are numerous and not fully understood. Mechanisms include hypoxia, sympathetic activation, insulin resistance, endothelial dysfunction, oxidative stress, and systemic inflammation [6–12]. Low levels of physical activity (PA) or sedentary behavior also increase the risk for CVD and diabetes, which could further exacerbate disease risk. In addition, excessive daytime sleepiness is another risk factor for OSA, which may further exacerbate sedentary behavior. Epidemiological research has suggested a relationship between OSA and decreased PA [13–15]. These studies, however, utilized subjective questionnaire data to establish this relationship.

Other studies have examined PA in OSA utilizing subjective means [16], objective means [17–22], or a combination

✉ Trent A. Hargens
hargenta@jmu.edu

¹ Department of Kinesiology, James Madison University, Harrisonburg, VA 22807, USA

[23] and have found OSA to be associated with decreased PA. However, one study found that continuous positive airway treatment (CPAP) did not increase PA post treatment [24]. In addition, many of these studies utilized differing methods in collecting PA data, such as only a single day (or undefined time period) of measurement, recruiting subjects with comorbid conditions that would also impact PA, recruiting only inactive subjects, not including a control group for comparison, or utilizing a now discontinued objective PA device (The SenseWear Body Media device).

As a result, while published research suggests that OSA results in lower PA, the findings are not clear. Advances in accelerometer technology currently allow for a more comprehensive analysis of PA habits, including sedentary time, and are a widely accepted means for objectively assessing PA [25]. Therefore, the purpose of this study was to examine whether objective measures of PA and sedentary behavior are altered in individuals with likely OSA compared to those without OSA.

Methods

Subjects

Overweight men ($n = 47$) and women ($n = 12$) were recruited from the James Madison University campus and surrounding community through campus-wide emails, word-of-mouth, and campus notices. Subjects were all 18 years or older. All subjects underwent a pre-screening to identify any potential exclusion criterion. All subjects were non-smokers, free from previous diagnoses of cardiovascular disease, congestive heart failure, or pulmonary disease. In addition, any subject with a significant orthopedic issue, which may impact their ability to exercise or perform physical activity, was excluded. To minimize the impact of body composition on physical activity, all subjects recruited were classified as overweight-to-obese according to body mass index (BMI) classifications. All methods and procedures were approved by the Institutional Review Board of James Madison University. All subjects read and signed a written informed consent document after all procedures were explained in full.

OSA risk evaluation

All subjects were previously undiagnosed with OSA. To screen for the likely presence of OSA, subjects underwent an unattended, home sleep evaluation using a type III validated device (ApneaLink™ Plus, ResMed Corp., San Diego, CA) [26]. Sleep data was analyzed by the device software, with an apnea-hypopnea index (AHI; events/hour) determined automatically by the software. AHI values were reviewed by a certified sleep technician and verified by a physician specialized in sleep medicine for accuracy. Apnea and hypopnea

events were scored in accordance with published standards [27, 28]. The OSA group included subjects with an AHI score of ≥ 5 events/h; the control group included subjects with an AHI score of < 5 events/h.

Body composition and anthropometry assessment

Height was measured with a wall-mounted stadiometer to the nearest 0.5 cm. Body weight was measured to the nearest 0.1 kg with a physician's scale. Waist circumference (WC) measurements were taken using a cloth tape measure with a spring-loaded handle, in accordance with the American College of Sports Medicine guidelines [29]. Neck circumference (NC) was measured with the same cloth tape measure at the widest portion of the neck. Percent body fat, fat-free mass, and fat mass were determined via total body dual-energy X-ray absorptiometry (DXA) (Lunar iDXA, GE Healthcare, Little Chalfont, UK).

Physical activity assessment

Subjects were instructed on the proper use and wear of an accelerometer (Actigraph GT3X+, Actigraph, Pensacola, FL) to assess PA based on manufacturer instructions. The Actigraph was worn at waist level in line with the right anterior axillary line. Subjects were instructed to wear the device during all waking hours, with the exception of any water-based activities. A minimum of 4 days, including one weekend day, and a maximum of 7 days were utilized, in accordance with previous investigations and similar to methods used in the National Health and Nutrition Examination Survey (NHANES) [30]. All accelerometer data was downloaded and analyzed with the validated software (Actilife software, Actigraph, Pensacola, FL) using the PA classifications established by Freedson et al. [31]. All days analyzed were > 10 h of wear time. Wear time was determined as specified by Troiano et al. [32].

Briefly, the Actigraph uses activity counts as its measure of acceleration. The higher number of counts assessed per minute, the greater the intensity of the PA. Using the cutpoints established by Freedson et al. [31], intensity of PA is defined by the following: sedentary time is 0–99 activity counts/min, light intensity is 100–1951 counts/min, moderate intensity is 1952–5724 counts/min, vigorous intensity is 5725–9498 counts/min, and very vigorous is > 9499 counts/min. Moderate-to-vigorous intensity physical activity (MVPA) is defined as 1952 or greater counts/min. In addition, a sedentary bout is defined as a minimum length of 10 consecutive minutes of sedentary time, and a PA bout is defined as a minimum length of 10 consecutive minutes of ≥ 1952 activity counts/min.

Statistical analysis

All statistical analyses were performed using IBM SPSS Statistics (version 24.0). Independent sample *t* tests were used to determine group differences in study variables. Pearson correlations were calculated to determine a relationship between age and PA variables. When significant relationships were found, analysis of covariance (ANCOVA) was utilized with age as the covariate to determine group differences. A value of $P < 0.05$ was considered statistically significant.

Results

Subject characteristics

Subject characteristics for each group are presented in Table 1. By study design, AHI was significantly higher in the OSA group vs. the control group ($P < 0.001$) and fell within the moderate OSA severity range. The AHI values ranged between 5 and 71 events/h for the OSA group. Of the 35 OSA subjects, 18 had an AHI in the mild OSA category (AHI 5–14.9 events/h) and 17 had an AHI in the moderate-to-severe OSA category (AHI > 15 events/h). The OSA group was significantly older than the control group ($P = 0.002$). There was no difference in BMI or percent body fat between groups, but the OSA group had a greater WC ($P < 0.01$) and NC ($P < 0.001$) compared to the control group. Epworth Sleepiness Scores did not differ between groups.

Physical activity profile

There was no difference between groups in the number of days assessed. The AHI was significantly and negatively correlated with several variables including average total time in PA bouts per day ($r = -0.27, P = 0.04$), total time in PA bouts

($r = -0.27, P = 0.04$), average number of sedentary bouts per day ($r = -0.27, P = 0.04$), average MVPA minutes per day (Fig. 1), average light intensity PA minutes per day ($r = -0.35, P < 0.01$), and average moderate intensity PA minutes per day ($r = -0.29, P = 0.03$). Physical activity measures are presented in Table 2. There was no group difference in number of days assessed. There was no group difference in any sedentary time variables. The OSA group had a significantly lower number of PA bouts for the assessment period as well as fewer number of PA bouts per day compared to the control group. In addition, the OSA group had significantly less moderate intensity PA minutes, total MVPA minutes (Fig. 2), and fewer steps per day (Fig. 3). There was a trend for lower light intensity PA minutes, but it did not reach statistical significance ($P = 0.06$).

Due to the age difference between groups, Pearson correlations were calculated between age and the PA variables. There was a significant negative correlation between age and the total number of PA bouts ($r = -0.26, P = 0.045$) and the average number of PA bouts per day ($r = -0.28, P = 0.03$). No other PA variable was related to age. As a result, ANCOVA was utilized on these variables, with age as a covariate, which resulted in the group differences no longer being significant, but rather trending toward significance ($P = 0.08$ and 0.07 for total number of PA bouts and average number of PA bouts per day, respectively). When we assessed the PA data by comparing the control group to mild OSA and moderate-to-severe OSA, respectively, we found that both OSA groups showed similar differences compared to the control group. As a result, we present the results of the analysis comparing the control group to any severity of OSA.

Discussion

In this study, we implemented a widely used, accepted, and validated method for objectively assessing PA, and examined the impact of OSA on PA without confounding comorbid conditions. We compared individuals with likely OSA to subjects without OSA who were matched for BMI and percent fat and found OSA results in a significantly lower overall PA profile. These findings were independent to the effects of BMI and total body adiposity or daytime sleepiness. To our knowledge, this is the first study to use these objective means for assessing PA in OSA subjects, with results suggesting that other physiological mechanisms may be responsible for this difference in PA.

Previous epidemiological studies have established a relationship between OSA and PA [13–15]. Quan et al. [13] found that vigorous PA ≥ 3 h per week was associated with decreased risk of prevalent sleep disordered breathing (SDB), even after adjusting for obesity, CVD, HTN, and sleepiness. Peppard et al. [14] reported that as exercise hours per week

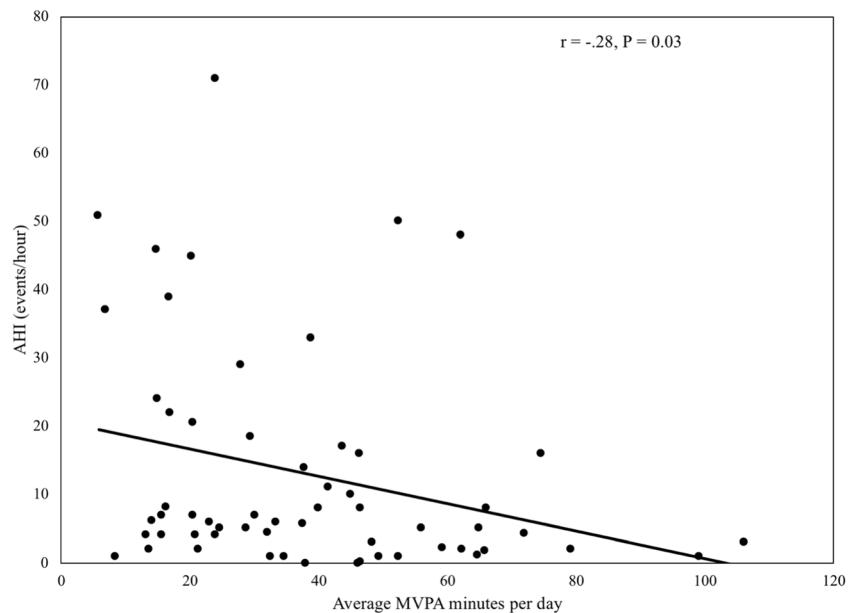
Table 1 Subject characteristics. Data presented \pm SD

	OSA (n = 35)	Control (n = 24)
Gender (male/female)	30/5	17/7
Age (year)	$45.2 \pm 12.0^*$	35.0 ± 11.7
AHI (events/h)	$20.4 \pm 17.6^*$	2.1 ± 1.4
ESS	7.9 ± 4.0	9.8 ± 5.4
BMI (kg/m^2)	33.0 ± 5.7	30.5 ± 4.3
Total body fat (%)	35.9 ± 7.7	34.1 ± 10.1
Lean mass (kg)	61.4 ± 10.2	57.2 ± 10.6
WC (cm)	$106.4 \pm 11.7^*$	98.6 ± 9.1
NC (cm)	$41.9 \pm 3.3^*$	38.8 ± 2.7

AHI, apnea-hypopnea index; ESS, Epworth Sleepiness Score; BMI, body mass index; WC, waist circumference; NC, neck circumference

* $P < 0.05$ vs. control

Fig. 1 Relationship between apnea-hypopnea index (AHI) and average moderate-to-vigorous intensity physical activity (MVPA) minutes per day



increased, AHI decreased. This relationship also held, similar to Quan et al., after adjusting for body habitus and sleepiness. They found that in individuals who exercised 3–6 h per week, the odds ratio of predicting an $AHI \geq 5$ was 0.67 compared to those who did not exercise at all during the week [14]. Similarly, Simpson et al. [15] reported that in individuals with moderate-to-severe OSA, 48% of male subjects and 42% of female subjects reported ≥ 150 min per week of moderate-to-vigorous intensity exercise, whereas 69% of males and 66% of females without OSA reported reaching that threshold of exercise. Further, they also showed a greater odds ratio of OSA

(moderate-to-severe) for those who exercised less. These studies were well-controlled studies that established a relationship between sleep disordered breathing, OSA, and PA, independent to the effects of obesity and sleepiness. These studies, however, relied on subjective means for assessing PA. Peppard et al. [14] obtained their PA information from a single question asked to subjects, that being “About how many hours per week – if any – do you spend at regular planned exercise (such as jogging, sports, exercise class, workouts at home or a gym)?” Quan et al. and Simpson et al. utilized more extensive questionnaire tools, whereas the current study utilized objective means to collect PA information and used the same measurement tool (ActiGraph accelerometer) and similar methods adopted by NHANES [25, 30]. Results from the current study support the findings of these previous epidemiological studies that demonstrate a relationship between OSA and PA, independent of obesity and sleepiness, and extend them to objectively measured PA.

Another study examined the relationship between SDB or OSA and PA through a much less common method. Suri et al. [17] examined the relationship between walking speed and risk of SDB. They found that a slower walking speed, determined through subjective questionnaire, was associated with an increased risk for SDB [17]. The authors note, however, that their analysis did not fully control for the potential effects of obesity. In their subject sample, the “slow walkers” had a higher BMI than the faster walkers [17].

Several other studies have utilized objective means to assess PA in OSA [18–21, 23]. Bamberga and colleagues found that obese individuals with OSA took significantly fewer steps and expended fewer kilocalories during the day compared to obese subjects without OSA [18]. This assessment, however, was made over a single day only, compared to the 4-day or greater assessment that is recommended and used by

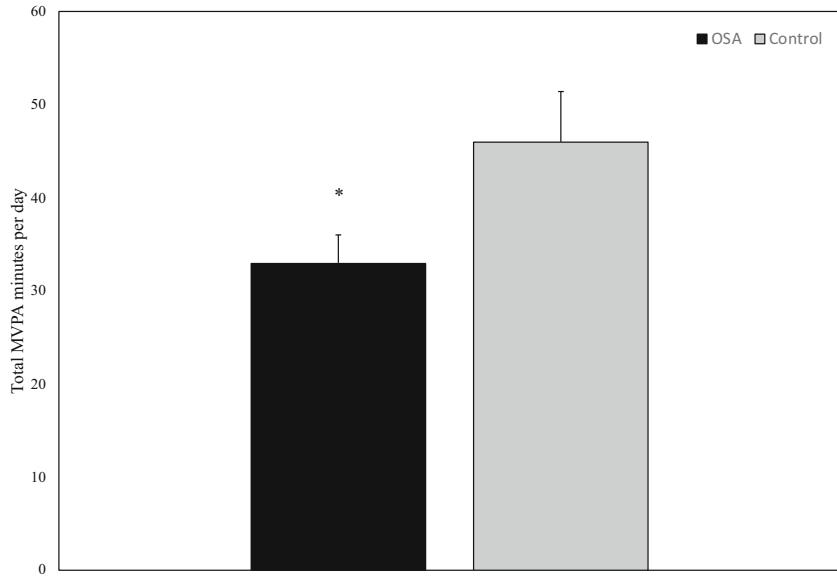
Table 2 Physical activity data. Data presented \pm SD

	OSA (n = 35)	Control (n = 24)
# of PA days assessed	$6.2 \pm .75$	$6.0 \pm .81$
Sedentary variables		
# Sed. bouts/day	20.0 ± 6.6	22.7 ± 6.2
Time per sedentary bout (min)	22.8 ± 2.7	22.5 ± 2.4
Sedentary minutes per day	652.5 ± 184.3	721 ± 167.6
Active variables		
Total # PA bouts	$3.2 \pm 3.4^*$	5.9 ± 5.0
# PA bouts/day	$0.52 \pm 0.5^*$	0.96 ± 0.8
Time/PA bout (min)	15.3 ± 13.6	14.5 ± 7.9
Total time in PA bouts/day	10.5 ± 11.3	16.4 ± 14.9
Total time in PA bouts (min)	64.4 ± 67.7	100.1 ± 91.2
Light intensity PA/day (min)	212.2 ± 89.8	253.9 ± 76.2
Vigorous intensity PA/day (min)	3.4 ± 6.9	1.7 ± 2.6
Very vigorous intensity PA/day (min)	0.005 ± 0.03	0.20 ± 0.58

PA, physical activity

* $P < 0.05$ vs. control

Fig. 2 Moderate-to-vigorous intensity physical activity (MVPA) in individuals with likely obstructive sleep apnea (OSA) compared to a non-OSA control group. $*P < 0.05$ compared to control

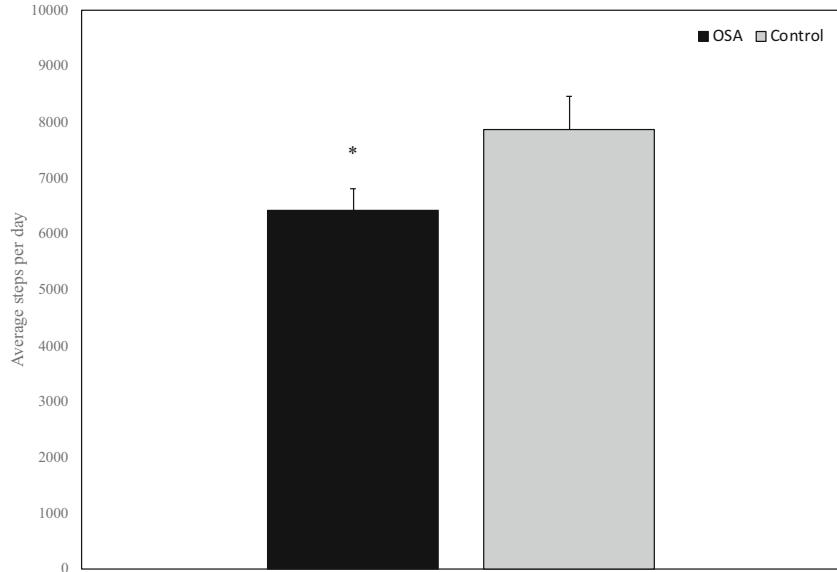


NHANES, and was obtained using the SenseWear Body Media device, which is no longer available and unable to be used in the future [32]. Similarly, Chasens et al. [21] also used the Body Media device to assess PA habits of OSA patients. They reported a mean steps per day value of 6988. Additionally, Verwimp et al. [19] also using the Body Media device to obtain PA data in a sample of 75 OSA patients reported mean steps per day at 6796. A strength of these studies was that 7 days of PA assessment was obtained. These two mean steps per day values reported by Chasens and Verwimp are similar to the results from the current study. Neither study, however, had a non-OSA control group for comparison. In addition, subjects included in their analysis either had several comorbid conditions, including HTN, diabetes, hypercholesterolemia, and smoking, in the case of Verwimp et al., or comorbid conditions could not be completely discounted, as

in the case of Chasens et al. While results from the current study concur with these studies with regard to mean steps per day, we extend those findings by showing that those with OSA took significantly fewer steps than a non-OSA comparison group and did not include comorbid conditions.

It is important to note that the control group in the current study, while not achieving the widely referred to, 10,000 steps per day threshold, did meet a threshold of 7500 steps per day. This threshold has been previously defined as the point at which MVPA recommendations are being met [33]. According the cut points put forth by Tudor-Locke et al., the OSA group in the current study would be classified as “low active” and not meeting the MVPA recommendations [33]. This difference could be a contributory factor in the increased risk for adverse health outcomes seen with OSA, particularly with conditions associated with a more sedentary lifestyle.

Fig. 3 Average steps taken per day in individuals with likely obstructive sleep apnea (OSA) compared to a non-OSA control group. $*P < 0.05$ compared to control



Two other published reports from the same research group also examined objective PA in OSA through use of the now discontinued Body Media device [20, 23]. In the first report, researchers recruited 73 OSA patients who self-reported a sedentary lifestyle, defined as < 30 min of moderate intensity PA 5 days per week. Any potential subject who reported greater than that amount was excluded. Additionally, the included subjects had volunteered to participate in a randomized controlled trial aimed at increasing PA. This may have resulted in these subjects being more motivated to increase their PA during the assessment period [20]. Mean steps per day for their subjects was 7734, with an average MVPA per day of 77 min. The MVPA value is more than double the amount reported with our study. In addition, they report that their subjects spent a mean value of 705 min of sedentary time per day, whereas we report a mean value of 653 min per day. Similar to the previous studies using the Body Media device, this study also did not include a non-OSA comparison group, and they did not report the number of days assessed for PA data. It is also not clear whether comorbid conditions such as HTN, diabetes, or smoking were exclusionary criteria or not. In the second report from this research group, PA was assessed from a minimum of 5 days of data, including one weekend day for 37 individuals diagnosed with OSA [23]. They report a mean MVPA amount of 37 min per day and sedentary time of 540 min per day. Mean steps per day were not reported. For this study, certain comorbid conditions were exclusionary criteria, but the included analysis did include current smokers. Diabetes and HTN were not listed as exclusionary criteria, and it was not addressed in the manuscript as to whether individuals with those conditions were included in the analysis or not. As with the previous studies, no control group was included.

To our knowledge, two other studies have examined PA habits of OSA using accelerometers in a similar fashion to the current study and to NHANES, but utilizing other devices [22, 24]. Kline et al. [22] objectively measured MVPA and steps in an OSA population undergoing an exercise intervention, utilizing a piezoelectric pedometer (NL-1000, New Lifestyles Inc., Lees Summit, MO). They reported a baseline mean steps per day of 5580 and a mean MVPA minutes per day of approximately 13 min. Our mean step value of 6480 steps per day is similar to the findings of Kline et al., but that difference is reflected in the greater number of MVPA minutes seen in the current study. Further, Kline et al. did not employ a non-OSA control group for comparison. Diamanti et al. obtained PA data for 7 consecutive days using the PALLite accelerometer (Pal Technologies Ltd., Glasgow, Scotland) in 24 obese OSA patients. The purpose of this study was to evaluate changes in PA after initiation of continuous positive airway pressure (CPAP) treatment. They reported a mean steps per day value of 3250 prior to treatment. They also reported a mean activity duration per day of 31 min per day,

which is similar to the mean MVPA value reported in the current study for the OSA group. As with the previous studies, Diamanti et al. did not include a non-OSA comparison group. Interestingly, they reported that following CPAP treatment, PA levels did not improve, despite a significant increase in quality of life and decrease in daytime sleepiness. This is consistent with findings of another study that also found that CPAP did not impact PA [16]. This study, in contrast to Diamanti et al., used subjective PA assessment. These findings, taken with the findings of the current study, in which there was no difference in daytime sleepiness, as determined by the Epworth Sleepiness Scale, between the OSA and control group, suggest that OSA-related PA differences are likely due to factors other than sleepiness.

The potential mechanisms linking OSA to a more sedentary behavior profile are not clear. The conventional wisdom was that OSA significantly increases daytime sleepiness, which in turn results in individuals being less likely to be active. Results from the current study, as well as previous studies have shown that sleepiness does not appear to be a primary factor. Despite the fact that the Epworth Sleepiness Scale is widely used in OSA research, there is published research suggesting that obesity may significantly impact excessive daytime sleepiness level derived from the Epworth Sleepiness Scale [34–36]. Given that both subject groups in the current study were obese, this may have been a factor in the lack of difference in reported daytime sleepiness. Healy et al. reported significant associations between accelerometer-derived PA, including MVPA and sedentary time, and WC [37]. This study was conducted on middle-aged individuals without diabetes, but included some with the metabolic syndrome. In the current study, we found that the OSA group had a significantly higher WC than the control group, despite no difference in total percent body fat. It has been well established that obesity, particularly abdominal obesity, significantly increases systemic inflammation. Further, it has also been shown extensively that OSA significantly increases systemic inflammation, independent of other factors, including obesity [6, 38, 39]. Evidence suggests a direct link between adipocyte-derived inflammatory cytokines, including interleukin-6 (IL-6) and tumor necrosis factor alpha (TNF- α) and skeletal muscle dysfunction [40, 41]. It is possible that a greater pro-inflammatory state in the OSA subjects, compared to the non-OSA control subjects, could be altering skeletal muscle function to result in a greater muscle fatigability, and consequently, a lower amount of daily PA. In the current study, we did not obtain measures on systemic inflammation. Future studies assessing PA in OSA patients should include these measures, as well as more direct assessment of skeletal muscle function.

Another potential mechanism linking OSA to lower PA is the effect of hypoxia on skeletal muscle function. Evidence from human and animal studies have suggested that repeated

exposure to hypoxia may alter the skeletal muscle fiber type function, shifting from oxidative type I muscle fibers, to glycolytic type II muscle fibers [42–45]. Type I muscle fibers, due to their slow twitch and oxidative characteristics, are much more fatigue resistant, able to maintain sustained activation longer than type II, or fast twitch muscle fibers. Type II muscle fibers, which rely on glycolytic metabolic pathways, are much more fatigable. It is possible that individuals with OSA, that are therefore chronically exposed to states of hypoxia, may see a shift in muscle fiber type and function to a more enhanced fatigability, and result in less overall PA performed. The current study did not directly assess muscle fiber type.

The current study has some limitations to acknowledge when interpreting our results. These findings are based on a self-referred subject pool. Subjects volunteered for a study whose objective was to examine PA habits, so one must be careful in applying these findings to the broader OSA population. Additionally, the two subject groups were not age matched. Our analysis found that only the PA bout data showed a relationship to age. When age was used as a covariate in the analysis, only the PA bout data did not remain statistically significant. Age did not affect the other PA data. Finally, our study was limited by the relatively small sample size. It should be noted, however, that previously published cross sectional studies examining PA in an OSA population utilized similar sample sizes to the current study.

In conclusion, this is one of the first studies to examine objectively measured PA in OSA compared to a non-OSA comparison group. The current study employed similar methods to the methods in which NHANES measures PA data. Additionally, the current study utilized the same devices that NHANES utilizes and is the first study to do so. Results suggest that OSA, independent to the effects of obesity or sleepiness, results in a more sedentary PA profile. This decrease in PA may be a contributing factor to the increased risk for CVD, HTN, diabetes, and other sedentary-related chronic health conditions seen in individuals with OSA.

Funding information ResMed Foundation, La Jolla, CA provided financial support for this study. The sponsor had no role in the design or conduct of this research.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the study.

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