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Actigraphy-based sleep analysis in sedentary and overweight/obese adults with primary hypertension: data from the EXERDIET-HTA study

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Abstract

Purpose The aim of this study was to analyze actigraphy-based sleep quantity and quality in sedentary and overweight/obese adults with primary hypertension (HTN) divided by sex and cardiorespiratory fitness (CRF) and to assess the association of sleep parameters with body composition, blood pressure (BP), and CRF.

Methods This is a cross-sectional design utilizing data from the EXERDIET-HTA study conducted in 154 non-physically, obese adults with HTN (53.3 ± 7.8 years). Sleep parameters (total bedtime; total sleep time, TST; and sleep efficiency = (TST/total bedtime) × 100)) were calculated from raw accelerometer data (ActiGraph GT3X+). Peak oxygen uptake ($\dot{V}O_{2peak}$) determined the CRF. Blood pressure was assessed with the 24-h ambulatory BP monitoring. The distributions of $\dot{V}O_{2peak}$ were divided into tertiles (low, medium, and high CRF) in each sex. Series of linear regression analyses were conducted between sleep, fitness, and health-related variables.

Results Short sleep duration (6.2 h) both on weekdays and weekends, poor sleep quality (< 85% of efficiency), and no significant differences in sleep variables between women and men, nor among CRF groups, were observed. The short sleeping pattern was negatively associated ($P < 0.05$) with mean and night systolic BP (mmHg, $\beta = -0.2$), and sleep efficiency with waist circumference (cm, $\beta = -0.08$, $P = 0.05$).

Conclusions Actigraphy-based sleep analysis reinforces that sleep disorders, such as short sleep duration and poor sleep quality, are associated with high BP and abdominal obesity in sedentary adults with overweight/obesity and HTN. Sleep pattern did not appear to be related with CRF level in this population.

Keywords Actigraphy · Ambulatory blood pressure · Sleep quantity · Sleep quality · Cardiorespiratory fitness

Introduction

Sleep plays an essential role in our physical health, and some consider sleep extension a non-pharmacological intervention for the prevention, management, and treatment of overweight/obesity and primary hypertension (HTN) [1]. During sleep time, important changes occur in autonomic nervous system functions and other physiologic events, which influence blood pressure (BP) responses [1]. Mean nocturnal BP should decrease by approximately 10% to 20% during nighttime sleep (a phenomenon known as nocturnal dipping) compared to mean daytime BP values [1].

Current sleep quantity and quality patterns are likely due to changes in the socioeconomic environment and lifestyle [2]. In fact, the population, in general, sleeps around 1.5 to 2 h/day less than people did a century ago [1]. Sleep efficiency, a good indicator of sleep quality, is obtained from [(total sleep time/total bedtime) × 100] [3], and values below 85% are usually considered indicative of clinically significant reduced sleep efficiency [3]. There is evidence to suggest that an inadequate sleep pattern has adverse effects on cardiovascular, endocrine, and immune function and to increase total mortality risk [2]. Numerous studies have shown that 7–8 h sleepers have the

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lowest risk of all-cause mortality [4]. However, many studies show the presence of a U-shaped association, those with shorter and longer sleep durations having a significantly higher risk of mortality [2], cardiovascular disease, HTN [5], and obesity [6].

On the other hand, aerobic exercise capacity referred to as cardiorespiratory fitness (CRF) (i.e., peak amount of oxygen that can be taken in, transported to and utilized by the working tissue during exercise) and objectively measured by peak oxygen uptake ($\dot{V}O_{2peak}$) correlates positively with quality of life, whereas it is negatively associated with fatal and nonfatal cardiovascular events, independent of other risk factors [7]. Sleep and CRF influence each other through complex and bilateral interactions. Indeed, beneficial effects of high CRF on sleep may be explained by interactions in the circadian rhythm, metabolic, immune, thermoregulatory, vascular, mood, and endocrine systems. On the other hand, sleep disorders may difficult the person's capacity to exercise and also increase the risk of exercise-induced injuries [8]. Hence, it could be hypothesized that individuals with low CRF would present sleep disturbances or worse sleep quality, along with other cardiovascular risk (CVR) factors. Ideally, sleep duration and efficiency should be measured using objective techniques and appliances for a representative period in free-living conditions and regular life activity [9]. Although polysomnography is regarded as the gold standard in the assessment of sleep in medicine, this measurement presents limitations due to its intricacy and costs. Accelerometers and actigraphy are a more economical and easily available alternative: they allow easy recording of multiple nights, have limited influence on natural sleep [10], and have been validated for the assessment of sleep measures in previous studies [11].

Considering all of the above, and the lack of sleep assessment studies with actigraphy in hypertensive populations, this study analyzed actigraphy-based sleep quantity and quality in sedentary and overweight/obese adults with HTN before starting a non-pharmacological therapeutic strategy divided by sex and CRF. A secondary aim was to assess the association of sleep parameters with body composition, BP, and CRF. We hypothesized that hypertensive and overweight/obese participants would have short sleep duration and poor sleep efficiency, which could be associated with their CVR factors.

Methods

This observational study was included in the EXERDIET-HTA study. The ethics committee of the University of the Basque Country (UPV/EHU, CEISH/279/2014) and the Ethics Committee of Clinical Investigation of Araba University Hospital (2015-030) approved the study design, study protocols, and informed consent procedure (ClinicalTrials.gov identifier, NCT02283047). All participants provided written informed consent.

Study population

A group of 186 adults took part in the study. All participants were sedentary, overweight/obese, and had the diagnosis of HTN [i.e., mean systolic blood pressure (SBP) ≥ 140 mmHg and/or diastolic blood pressure (DBP) ≥ 90 mmHg or used antihypertensive medications], 86.9% of participants were taking medication). Participants were recruited from the medical services and local media. Participants with no diagnosis of HTN were assessed with ambulatory blood pressure monitoring (ABPM) to confirm the HTN status by a cardiologist. All other inclusion and exclusion criteria were specified in the study protocol [12].

Data were retained from those participants who had measurements in actigraphy, anthropometry, ambulatory BP monitoring (ABPM), and CRF using the metabolic gas analysis system. Thirty-two participants did not meet the inclusion criteria and were excluded from the analysis. Finally, a sample of 154 individuals aged between 24 and 69 years (53.3 ± 7.8 years) was obtained, 101 men and 53 women.

Measurements

A full description of the study protocol was previously presented elsewhere [12]. A brief explanation of the main measures is presented below.

Anthropometry measurements for the assessment of body composition included stature (SECA 213, Hamburg, Germany), total body mass (BM) (SECA 869, Hamburg, Germany), BMI, and waist and hip circumferences (SECA 200, Hamburg, Germany) to calculate the waist-to-hip ratio (WHR).

Ambulatory BP monitoring was measured with an oscillometric ABPM 6100 recorder (Welch Allyn, New York, USA). The device measured BP an entire day, at 30-min intervals during the daytime and at 60-min intervals during nighttime, and no other device was implemented during this period (e.g., the actigraph). The variables registered from the ABPM were mean values of SBP and DBP during the day and night periods and mean heart rate (HR).

Physical fitness was determined by performing a symptom-limited cardiopulmonary exercise test (CPET). The CPET was performed on an electronically braked Lode Excalibur Sport cycle ergometer (Groningen, Netherlands), starting at 40 W with a gradual increment of 10 W every minute applied until volitional exhaustion. The expired gas was analyzed using a commercially available metabolic cart (Ergo Card, Medi-soft S.S, Belgium Ref. USM001 V1.0).

The $\dot{V}O_{2peak}$ values were divided into tertiles (low, medium, and high CRF) in each sex. The details regarding the range in each group were as follows: the lowest tertile (low-CRF group), $\dot{V}O_{2peak} \leq 21$ mL kg^{-1} min^{-1} in men and $\dot{V}O_{2peak} \leq 16$ mL kg^{-1} min^{-1} in women; the medium tertile (medium-CRF group), $22 < \dot{V}O_{2peak} \leq 26$ mL kg^{-1} min^{-1} in men and $17 < \dot{V}O_{2peak} \leq 20$ mL kg^{-1} min^{-1} in women; the highest tertile (high-CRF group), $\dot{V}O_{2peak} > 27$ mL kg^{-1} min^{-1} in men and $\dot{V}O_{2peak} > 21$ mL kg^{-1} min^{-1} in women.

Movements during sleep and wake were objectively and continuously assessed through a triaxial accelerometer (ActiGraph GT3X+, Pensacola, Florida, USA). Participants wore the actigraph on their non-dominant wrist with a velcro strap for eight consecutive days at all times (24-h), except during water-based activities, and no other device was implemented during this period (e.g., ambulatory blood pressure). Each participant received oral instructions on how to wear the actigraph, keeping general recommendations and fulfilling the diary log, indicating the time of reference measurements (i.e., time to: wake up, get up, breakfast, lunch, nap, physical activity, dinner, asleep at night) and also to track all times when the device was removed and replaced. On the eighth day, after the actigraph was returned to the investigators, both actigraph and daily log were collected. The sleep measures were calculated from raw actigraph data for each unit. Actigraph data were downloaded, treated, and analyzed using the manufacturer's software (Actilife 6.11.9) with 60-s epoch length. In-bed and out-bed times were verified to be equal in actigraph and diary log for each day and each participant. If there was any difference, the data were adjusted using diary log information. This discrepancy only took place in "going to sleep time" information. This is because actigraph may count as sleeping time when participant remains laying down motionless. The following sleep variables were derived from actigraph data: bedtime (total time spent in bed), TST (min of sleep between sleep onset and wake time), and sleep efficiency (the ratio between TST and total time spent in bed). Sleep patterns were assessed using a previously validated software algorithm based on the Cole-Kripke scoring method [13] that analyzes the raw actigraph data to calculate sleep time.

Given that obstructive sleep apnea (OSA) could be a confounding factor influencing the relationship between short sleep and BP, we included the STOP-Bang Questionnaire to evaluate the risk of OSA [14]. Thus, participants were classified for OSA risk based on their scores (low risk < 2, moderate range 3–4, and high risk > 5–8). It was considered OSA when the STOP-Bang score showed a high-risk score, together with those that upon inquiry affirmed a medical diagnosis of OSA, including treatment with continuous positive airway pressure or not treatment. Furthermore, the Epworth Sleepiness Scale (ESS) was also added to evaluate the daytime sleepiness of the participants. The ESS is a self-administered questionnaire with eight questions measuring the degree of sleepiness that different situations of daily life produce (from 0 to 3), and differentiating somnolence from fatigue. The ESS results range from 0 to 24 with high scores reflecting high levels of sleepiness and excessive daytime sleepiness is defined as an ESS > 10 [15].

Statistical analysis

Descriptive statistics were calculated for all variables. Data are expressed as the mean ± standard deviation (SD). Baseline characteristics were compared with the use of independent samples *t* test to determine whether there was a significant sex difference for all variables. A related samples *t* test was used to determine differences between weekdays and weekends in each sleep variable. One-way ANOVA was used to examine differences among participants classified by CRF level (low, medium, and high). A Bonferroni post hoc test was used to determine the level of significance when a significant main effect was found.

Linear regression analysis was performed to assess the association between sleep variables (independent variables) with physical and physiological variables (dependent variables) with and without adjustment for covariates. Covariates in model 2 were age, sex, and body mass index (BMI), except when age and BMI were dependent variables. Statistical significance was set at $P < 0.05$. All analyses were conducted using 24.0 version of IBM® SPSS-Statistics© program.

Results

Table 1 displays the participants' physical and physiological characteristics split by sex, along with smoking and medication data. Men presented a higher waist circumference, mean DBP, day DBP, night DBP, and VO_{2peak} compared to women ($P < 0.05$), while women showed higher mean HR values compared to men ($P < 0.05$). These results along with medications data are included and discussed in a previously presented sample [16].

The analysis of sleep variables from actigraphy (Table 2) showed that our population slept around 371 min (6.2 h) per night. Regarding bedtime and TST, men spent more time in bed and slept more minutes on weekends compared to weekdays ($P < 0.001$), but women did not show these differences ($P = 0.2$). There were no statistical differences in sleep efficiency variables between weekdays and weekends or between sexes ($P > 0.05$).

The assessment of OSA indicated that the mean value for OSA score through STOP-Bang questionnaire was 3.7 ± 1.4 (i.e., medium risk) with men showing higher values (4.5 ± 1.3, $P < 0.001$) compared to women (2.8 ± 1.1), and participants stratified as 50% high, 26.3% medium, and 23.7% low risk.

Table 1 Global characteristics of study population and divided by sex. Values are mean ± SD

	All *n* = 154	Men *n* = 101	Women *n* = 53	P_{M-W}
Age (years)	53.3 ± 7.8	53.5 ± 8.1	52.9 ± 7.2	0.7
BMI (kg/m^2)	32.4 ± 4.1	32.3 ± 4.0	32.8 ± 4.3	0.4
Waist circumference (cm)	103.7 ± 10.9	107.0 ± 9.8	97.4 ± 10.1	*< 0.001**
SBP mean (mmHg)	136.0 ± 12.1	136.6 ± 11.8	134.9 ± 12.7	0.4
DBP mean (mmHg)	78.4 ± 8.0	80.3 ± 7.3	74.8 ± 8.2	*< 0.001**
SBP day (mmHg)	139.4 ± 12.2	140.0 ± 11.8	138.3 ± 13.1	0.4
DBP day (mmHg)	81.1 ± 8.6	83.0 ± 7.8	77.6 ± 9.0	*< 0.001**
SBP night (mmHg)	123.6 ± 15.3	123.7 ± 15.5	123.5 ± 15.0	0.9
DBP night (mmHg)	68.0 ± 8.1	69.2 ± 8.2	65.8 ± 7.6	*0.015**
HR mean (bpm)	72.0 ± 10.3	70.6 ± 10.0	74.6 ± 10.5	*0.019**
$\dot{V}O_{2peak}$ (ml·kg^{-1}·min^{-1})	22.1 ± 5.4	23.8 ± 5.2	18.9 ± 4.3	*< 0.001**
Antihypertensive medication (%)	86.9	85.1	91.0	0.2
Statin (%)	12.9	12.8	13.2	0.9
Hypoglycemic (%)	5.3	6.4	2.9	0.3
ACEI (%)	36.8	34.8	41.2	0.4
ARB (%)	43.6	45.4	39.7	0.4
Diuretic (%)	32.1	31.2	33.8	0.7
CCB (%)	17.7	21.3	10.3	0.05
BB (%)	9.5	8.5	11.8	0.4
Antiplatelet (%)	3.9	4.3	2.9	0.6
Cigarette smoking (%)	11.4	10.6	13.2	0.6

M men, *W* women, *BMI* body mass index, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *HR* heart rate, VO_{2peak} peak oxygen consumption, *ACEI* angiotensin-converting-enzyme inhibitors, *ARB* angiotensin II receptor blockers, *CCB* calcium channel blockers, *BB* beta-blockers

*$p \leq 0.05$

Furthermore, the 96% of those with a previous medical diagnosis of OSA or even high-risk OSA score were under continuous positive airway pressure, and only 4% of them were non-treated OSA. In this sense, participants with treatment and not treatment OSA were divided and separately analyzed. The results showed not significant differences ($P > 0.05$) between groups (treated vs. not-treated OSA) in any of the studied sleep variables; therefore, all participants were all together analyzed. Daytime sleepiness analysis showed that the mean value for ESS in all sample was 7.9 ± 3.9 (men, 7.8 ± 3.5 and women, 8.1 ± 4.3, $P = 0.7$), with the 22.2% of the total sample with values considered as excessive daytime sleepiness.

After dividing our sample into tertiles by CRF values (Table 3), there were statistical differences in BMI, waist circumference, and mean HR. Low-CRF had higher BMI than medium-CRF ($P = 0.02$, mean difference = 2.0, 95% confidence interval (CI) = 0.3–3.7 kg/m^2) or high-CRF ($P < 0.001$, mean difference = 3.9, 95% CI = 2.0–5.8 kg/m^2), and medium-CRF also showed higher BMI than the high-CRF ($P = 0.05$, mean difference = 1.9, 95% CI = 0–3.7 kg/m^2). The high-CRF group had lower values than the low-CRF group on waist circumference ($P < 0.001$, mean difference = − 8.3, 95% CI = 13.5–3.1 cm) and mean HR values ($P = 0.03$, mean difference = − 5.5, 95% CI = 10.5–0.5 bpm). No significant differences were observed in sleep variables among CRF groups.

A series of linear regression analyses were conducted to assess the association of sleep variables (TST, bedtime and sleep efficiency) with physical and physiological variables, adding the dependent variables one by one (Table 4). Waist circumference was negatively associated to TST (β = − 0.2, $P = 0.02$) and bedtime (β = − 0.2, $P = 0.03$) in the unadjusted model; but, after adjustment for covariates, it was only negatively associated with sleep efficiency (β = − 0.08, $P = 0.05$, Fig. 1). Mean SBP was negatively related to TST, both without (β = − 0.2, $P = 0.01$) and with adjustment (β = − 0.2, $P = 0.02$), as well as with bedtime without (β = − 0.2, $P = 0.02$) and with adjustment (β = − 0.2, $P = 0.04$). Day SBP presented an inverse association with TST in the adjusted model (β = − 0.2, $P = 0.03$), but it was not significantly associated with adjustment ($P = 0.06$). Night SBP was negatively associated with TST without (β = − 0.2, $P = 0.01$) and with adjustment (β = − 0.2, $P = 0.01$). Night SBP also had a negative relationship with bedtime without (β = − 0.2, $P = 0.01$) and with adjustment (β = − 0.2, $P = 0.08$). In addition, a significant negative association was found between mean DPB, day DBP, and

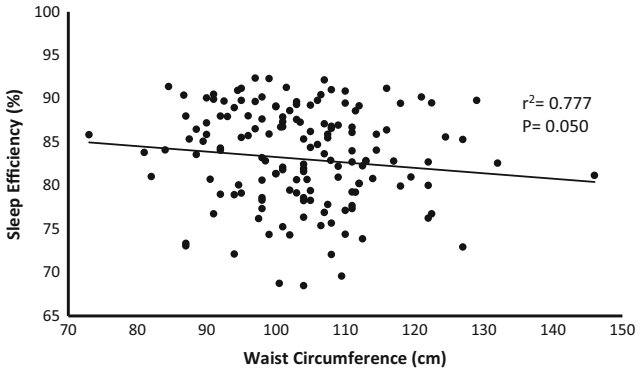

Fig. 1 Association between sleep efficiency (%) and waist circumference (cm)

night DBP with TST (β = − 0.2, β = − 0.2, and β = − 0.2, respectively) and with bedtime (β = − 0.2, β = − 0.1, and β = − 0.2, respectively) when the regressions were conducted without adjustment; however, no significant associations were found following adjustment ($P > 0.05$). Focusing on sleep variables, TST was significantly associated with bedtime and sleep efficiency in unadjusted (β = 0.9 and β = 0.7, respectively) and adjusted model (β = 0.9 and β = 0.7, respectively). When the regression was conducted with adjustment, bedtime was related to TST (β = 0.9, $P < 0.001$) and sleep efficiency (β = 0.4, $P < 0.001$).

Discussion

To our knowledge, the current research represents the first study applying robust and objective measures analyzing sleep (actigraphy), CRF ($\dot{V}O_{2peak}$), and BP (24-h ambulatory BP monitoring). The main findings of the present cross-sectional study were that sedentary and overweight/obese people with HTN (1) had shorter sleep duration (6.2 h) than recommended by guidelines (7–9 h), both on weekdays and weekends; (2) showed poor sleep quality (< 85% of efficiency) with no differences between women and men; (3) sleep pattern did not appear to be related with CRF level in the study population; and (4) the short-sleeping pattern, when examined alone (unadjusted), was negatively associated with waist circumference, SBP, and DBP. These relationships remained significant in mean and night SBP after adjustment. In addition, after adjustment, a significant association between sleep efficiency and waist circumference emerged. Lastly, individuals with longer bedtime at night also showed higher TST and sleep efficiency.

Consensus-based guidelines recommend that adults should sleep seven or more hours per night, with sleep efficiency > 85% on a regular basis to promote optimal health [3]. Thus, the population of the present study, with a mean of 6.2 h of TST, refers shorter than guidelines recommended mean sleep duration (Table 2). Even moderate or short-term sleep deprivation is associated with increased BP and HR through the activation of the sympathetic system and an increase in plasma norepinephrine levels, along with reduced magnesium concentration increasing vascular tone [17]. In fact, previous studies stated that each hour of reduced sleep was associated with a 37% increase in the incidence of HTN [1]. Closely linked with this observation and consistent with our prediction, in our general analysis of sleep duration and HTN, the short-sleeping pattern (i.e., reduced TST and bedtime) was negatively associated with BP (i.e., increased mean SBP and night SBP), even after adjustment (Table 4). Although it could be stated that the low R-squared values, in the present study, are not enough for clinical significance, graded associations between higher SBP and DBP and increase cardiovascular disease risk have been presented (i.e., even with small increases of BP) [18]. Hence, previous prospective studies have demonstrated that nocturnal BP is a better predictor of CVR than daytime BP, and 10 mmHg increases in nocturnal SBP were associated with a 21% increase in cardiovascular death [19]. Other studies showed an inverse association between TST and BP in mean and nocturnal values [5]. Therefore, the present objective assessment of sleep duration from raw actigraph data in hypertensive participants reinforces a high prevalence of short sleep duration (i.e., < 7 h per day) in this population [4]. A possible confounder for poor sleep could be the occurrence of OSA in the study population; however, only 4% of participants suffering from OSA were under no treatment. Linked to this, there is no consideration of excessive daytime sleepiness in the general sample, as the ESS questionnaire revealed. Likewise, participants' sleep quality, measured as 82.8% of efficiency, was poor [3], with no significant differences ($P = 0.4$) between men (82.5%) and women (83.4%). Previous studies have shown that poor sleep efficiency measured by nocturnal polysomnography was associated with a mean increase of 4 mmHg of SBP, and even in non-hypertensive population, the risk of developing HTN was multiplied by 3.5 [1]. Further, while previous investigations consistently report that women spent more time in bed, went to bed earlier, and got up later than men [20], sleep parameters in our study seemed not to be influenced by sex. Differences in the study design and methods, smaller sample of women compared to men, and variability in the population's characteristics may have contributed to these discrepant results. Indeed, the present study participants were mainly middle-aged adults (53.3 years), and changes in women's ovarian steroid production, such as those occurring during the menopausal transition (in the present study, 48.5% of women were on post-menopausal state), are markedly associated with poor sleep [20]. In addition, there were significant differences ($P < 0.001$) between weekday and weekend bedtime and TST among men in our study. In this sense, although an increased weekend catch-up bedtime and sleep duration was found, it was still insufficient (6.4 h of TST) to comply with

Table 2 Sleep variables analysis of study population and divided by sex. Values are mean ± SD

	All $n = 154$	P Wd-WEd	Men $n = 101$	P Wd-WEd	Women $n = 53$	P Wd-WEd	$P_{M\text{-}W}$
Sleep efficiency (%)	82.8 ± 7.2		82.5 ± 7.6		83.4 ± 6.2		0.4
Sleep efficiency Wd (%)	82.6 ± 7.1	0.1	82.2 ± 7.5	0.2	83.2 ± 6.4	0.4	0.4
Sleep efficiency WEd (%)	83.2 ± 8.3		83.0 ± 9.0		83.8 ± 7.0		0.5
Bedtime (min)	446.1 ± 61.9		439.7 ± 62.5		458.4 ± 59.3		0.1
Bedtime Wd (min)	439.8 ± 66.5	*< 0.001**	432.4 ± 63.9	*0.001**	453.8 ± 69.5	0.2	0.1
Bedtime WEd (min)	462.0 ± 80.0		457.8 ± 84.5		469.9 ± 70.7		0.3
TST (min)	371.1 ± 68.5		364.3 ± 69.3		384.0 ± 65.4		0.1
TST Wd (min)	365.0 ± 71.4	*< 0.001**	357.3 ± 69.3	*<0.001**	379.8 ± 73.8	0.2	0.1
TST WEd (min)	386.2 ± 83.0		381.9 ± 88.4		394.4 ± 71.7		0.3

Sleep efficiency (%): total sleep time divided by total bedtime multiplied by 100

Wd week day, *WEd* weekend day, *TST* total sleep time at night

*$p \leq 0.05$

sleep guidelines and to get the effect of "recovery sleep" on the activity of the sympathetic nervous system [21].

Contrary to our expectations, sleep pattern did not appear to be related with CRF level (Table 4). Recent studies analyzing the longitudinal change in CRF and odds of incident sleep problems have concluded that a decline in fitness typically accelerated sleep problems [22]. In contrast, a study that measured objectively CRF (i.e., VO_{2peak}) and sleep patterns by accelerometry reported that physical fitness and sleep characteristics were not significantly correlated in young adults and older participants [23]. Therefore, it might be argued that sleep pattern is not related with CRF. However, as we have already shown [16], participants characterized by a low-CRF showed higher BMI ($P = 0.05$) and waist circumference values than medium- and high-CRF ($P < 0.001$), reinforcing the inverse association between CRF and obesity [24]. Related to that result, we found an inverse association between sleep parameters (especially sleep efficiency) and

Table 3 Global characteristics and sleep variables divided by CRF tertiles. Values are mean ± SD

	LOW $n = 54$	MEDIUM $n = 58$	HIGH $n = 42$	P
Age (years)	54.5 ± 8.5	54.3 ± 7.4	50.3 ± 6.7	0.01
BMI (kg/m^2)	34.2 ± 4.7^{ab}	32.2 ± 3.4^{b}	30.4 ± 2.9	*< 0.001**
Waist circumference (cm)	107.3 ± 11.5^{b}	103.8 ± 9.7	98.9 ± 9.9	*0.001**
$\dot{V}O_{2peak}$ (ml·kg^{-1}·min^{-1})	17.2 ± 2.8^{ab}	22.7 ± 2.9^{b}	27.7 ± 5.0	*< 0.001**
SBP total (mmHg)	138.1 ± 13.7	134.9 ± 10.2	135.0 ± 12.4	0.3
DBP total (mmHg)	78.4 ± 8.6	77.7 ± 7.7	79.3 ± 7.8	0.6
SBP day (mmHg)	141.0 ± 13.4	138.3 ± 10.6	138.9 ± 12.8	0.5
DBP day (mmHg)	80.9 ± 9.4	80.5 ± 8.2	82.2 ± 8.3	0.6
SBP night (mmHg)	127.8 ± 17.6	121.5 ± 12.7	121.1 ± 14.5	0.05
DBP night (mmHg)	69.3 ± 8.3	66.9 ± 7.8	67.9 ± 8.4	0.3
HR mean (bpm)	74.3 ± 10.8^{b}	72.1 ± 11.2	68.8 ± 7.2	*0.033**
Sleep efficiency (%)	83.6 ± 5.6	81.6 ± 7.1	83.3 ± 8.8	0.3
Sleep efficiency Wd (%)	83.5 ± 5.4	81.1 ± 7.1	83.3 ± 8.7	0.1
Sleep efficiency WEd (%)	83.7 ± 7.1	82.8 ± 8.4	83.3 ± 9.7	0.8
Bedtime (min)	450.2 ± 62.0	442.5 ± 68.3	446.2 ± 52.9	0.8
Bedtime Wd (min)	445.1 ± 62.2	437.0 ± 76.9	436.7 ± 56.6	0.8
Bedtime WEd (min)	462.3 ± 86.5	456.1 ± 80.0	469.9 ± 72.2	0.7
TST (min)	378.1 ± 66.1	363.0 ± 72.9	373.4 ± 65.5	0.5
TST Wd (min)	373.5 ± 65.7	356.8 ± 79.6	365.5 ± 66.9	0.5
TST WEd (min)	389.2 ± 89.5	378.4 ± 78.9	393.3 ± 80.9	0.6

Sleep efficiency (%): total sleep time divided by total bedtime multiplied by 100

BMI body mass index; *SBP* systolic blood pressure; *DBP* diastolic blood pressure; *HR* heart rate; $\dot{V}O_{2peak}$ peak oxygen consumption; *Wd* week day, *WEd* weekend day, *TST* total sleep time at night

*$p \leq 0.05$

[a] Denotes group is significantly different ($p < 0.05$) from MEDIUM CRF group

[b] Denotes group is significantly different ($p < 0.05$) from HIGH CRF group

Table 4 Linear regression models. Association between sleep variables and physical and physiological variables

Independent variables	TST					Bedtime				Sleep efficiency					
Dependent variables	Unadjusted			Adjusted			Unadjusted			Adjusted			Unadjusted			Adjusted		
	β	P	r2	β	P	r2	β	P	r2	β	P	r2	β	P	r2	β	P	r2
Age	0.01	0.5	0.004	0.1	0.4	0.01	0.1	0.4	0.005	−0.08	0.3	0.008	0.01	0.8	< 0.001	0.02	0.8	0.003
$\dot{V}O_{2peak}$	0.1	0.5	0.005	−0.03	0.6	0.3	−0.1	0.4	0.004	−0.02	0.7	0.3	−0.06	0.4	0.004	−0.03	0.7	0.3
BMI	−0.1	0.3	0.006	−0.1	0.3	0.01	−0.1	0.2	0.01	−0.1	0.2	0.02	0.01	0.9	< 0.001	0.01	0.9	0.005
Waist circumference	*−0.2*	*0.02*	*0.03*	−0.1	0.1	0.8	*−0.2*	*0.3*	*0.03*	−0.04	0.3	0.8	−0.1	0.2	0.009	*−0.08*	*0.05*	*0.8*
SBP mean	*−0.2*	*0.01*	*0.04*	*−0.2*	*0.02*	*0.04*	*−0.2*	*0.02*	*0.03*	*−0.8*	*0.04*	*0.04*	−0.1	0.1	0.02	−0.1	0.1	0.03
DBP mean	*−0.2*	*0.02*	*0.04*	−0.1	0.1	0.2	*−0.2*	*0.02*	*0.03*	−0.1	0.1	0.2	−0.1	0.1	0.02	−0.1	0.1	0.1
SBP day	*−0.2*	*0.03*	*0.03*	−0.1	0.1	0.04	−0.1	0.1	0.02	−0.1	0.1	0.04	−0.1	0.1	0.02	−0.1	0.1	0.04
DBPday	*−0.2*	*0.03*	*0.03*	−0.1	0.1	0.2	*−0.1*	*0.05*	*0.02*	−0.1	0.2	0.1	−0.1	0.1	0.02	−0.1	0.1	0.1
SBP night	*−0.2*	*0.01*	*0.04*	*−0.2*	*0.01*	*0.05*	*−0.2*	*0.01*	*0.04*	*−0.2*	*0.02*	*0.05*	−0.1	0.1	0.01	−0.1	0.1	0.03
DBP night	*−0.2*	*0.03*	*0.03*	−0.1	0.1	0.1	*−0.2*	*0.02*	*0.03*	−0.1	0.1	0.06	−0.1	0.2	0.01	−0.1	0.2	0.05
Dipping	0.1	0.1	0.01	0.1	0.1	0.1	0.1	0.1	0.02	0.1	0.1	0.07	0.05	0.6	0.002	0.05	0.5	0.06
TST							0.9	< 0.001	0.8	0.9	< 0.001	0.8	0.7	< 0.001	0.5	0.7	< 0.001	0.5
Bedtime	*0.9*	*< 0.001*	*0.8*	*0.9*	*< 0.001*	*0.8*							*0.4*	*< 0.001*	*0.1*	*0.4*	*< 0.001*	*0.2*
Sleep efficiency	*0.7*	*< 0.001*	*0.5*	*0.7*	*< 0.001*	*0.5*	*0.4*	*< 0.001*	*0.1*	*0.4*	*< 0.001*	*0.1*						

BMI body mass index, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, $\dot{V}O_{2peak}$ peak oxygen consumption, *TST* total sleep time

Significant values are shown in italics

waist circumference (r = 0.88, $P = 0.05$, after adjustment with age, sex, and BMI, Fig. 1), which is considered a marker for higher metabolic and cardiovascular disease risk [25]. Confounding factors as age, sex, and BMI are associated with the waist circumference and may affect the sleep efficiency. The relationship between sleep and obesity has been previously analyzed [6], and it could be bidirectional [26]. Although not studied in the present investigation, the plausible mechanisms by which reduced sleep may impact body mass homeostasis in our population might be (1) the activation of orexin system hormonal responses regulating appetite, wakefulness, and energy balance, such as leptin and ghrelin, increasing appetite and energy intake and storage in adipocytes [27]; (2) a decreased caloric expenditure led by feelings of fatigue resulting in reductions of exercise [28] or spontaneous physical activity [29]; and (3) altered sympathetic-vagal balance with an increase in sympathetic nervous system along with catecholamine and cortisol levels, which may impact on insulin sensitivity the following morning [30].

It was also observed that participants with longer bedtime at night also showed higher TST and sleep efficiency. Hence, we could naively think that spending more nocturnal bedtime will increase TST and efficiency. However, we should be cautious about this conclusion taking into account the complex etiology of sleep disorders and bedtime distractions that may cause lack of sleep.

Some limitations should be considered. First, actigraphy may overestimate TST and sleep efficiency due to that lack of movement or lying in bed awake motionless is mostly coded as sleep. Second, the present investigation was conducted with an unequal number of women (34.4%) and men (65.6%), which could be the reason for the lack of differences in sleep variables. Third, this was a cross-sectional study, which did not totally describe the cause-effect variables, although we included confounding factors to strengthen the results. Fourth, although the sample size was sufficient as an initial investigation on overweight/obese individuals with HTN, results and statistical power might not be enough and comparable with larger epidemiological studies. Lastly, all the participants of the present study were sedentary, overweight/obese, and suffered from HTN, and a healthy control group was lacking to compare and analyze whether differences exist between groups. Therefore, considering that optimizing sleep seems to be part of the algorithm in the management of hypertension, but it has been difficult to demonstrate the benefit empirically, future studies should consider large-scale investigations and determine the short- and long-term effects of different interventions on sleep patterns.

In conclusion, actigraphy-based sleep analysis reinforces the accumulative evidence of sleep disorders (short sleep duration and poor sleep quality) associated with high BP and abdominal obesity, which may have potential synergistic deleterious effects on CVR in sedentary and overweight/obese

adults with HTN. Further, sleep pattern does not appear to be related with CRF level in the study population.

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Author contribution Conceived and designed the experiment: AMAB and SMM. Data collection and analysis: AMAB, SMM, PC, IGA, GRA. Data interpretation and drafting of the manuscript: AMAB, SMM, PC, IGA, IM.

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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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