Original Article

Sleep irregularity and the association with hypertension and blood pressure levels: the ELSA-Brasil study

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Objective: To evaluate the associations of sleep irregularity with hypertension (HTN) and blood pressure (BP) levels.

Methods: Adult participants from the ELSA-Brasil performed a clinical evaluation including objective sleep duration (actigraphy), insomnia, and a sleep study for defining obstructive sleep apnoea (OSA). To quantify sleep irregularity, we used two parameters obtained through actigraphy: 7-day standard deviation (SD) of sleep duration and 7-day SD of sleep-onset timing. A multivariate analysis was used to determine the independent associations of sleep irregularity with HTN and SBP/DBP values.

Results: We studied 1720 participants (age 49 ± 8 years; 43.4% men) and 27% fulfilled the HTN diagnosis. After adjustments for age, gender, race, BMI, excessive alcohol consumption, physical activity intensity, urinary sodium excretion, insomnia, objective sleep duration and OSA (apnoea–hypopnoea index ≥15 events/h), we found that the continuous analysis of 7-day SD of sleep duration was modestly associated with prevalent HTN. However, 7-day SD of sleep duration more than 90 min was independently associated with SBP [β: 1.55; 95% confidence interval (CI) 0.23–2.88] and DBP (β: 1.07; 95% CI 0.12–2.01). Stratification analysis excluding participants with OSA revealed that a 7-day SD of sleep duration greater than 90 min was associated with a 48% higher chance of having HTN (OR: 1.48; 95% CI: 1.05–2.07). No significant associations were observed for the SD of sleep-onset timing.

Conclusion: Objective measurement of sleep irregularity, evaluated by SD of sleep duration for 1 week, was associated with HTN and higher BP levels, especially in participants without OSA.

Keywords: circadian rhythms, hypertension, obstructive sleep apnea, sleep duration, sleep irregularity

Abbreviations: β, beta value coefficient; ∼, nearly; AASM, American Academy of Sleep Medicine; ACR, albumin-to-creatinine ratio; AHI, apnoea–hypopnoea index; BP, blood pressure; CI, confidence interval; CIS-R, Clinical Interview Scheduled Revised; EDS, excessive daytime sleepiness; ELSA-Brasil, The Brazilian Longitudinal Study of Adult Health; HTN, hypertension; OR, odds ratio; OSA, obstructive sleep apnoea; SD, standard deviation; WASO, wake time after sleep onset

INTRODUCTION

Sleep disturbances constitute a myriad of heterogenic conditions that impair the homeostasis of this essential behavioural and physiological phase, accounting for one-third/one-fourth of the human lifespan [1]. Among the more than 80 reported sleep disorders, obstructive sleep apnoea (OSA) is, by far, the most studied sleep-related condition associated with hypertension (HTN). Indeed, OSA has been consistently associated with prevalent and incident HTN [2–6]. The potential implications of sleep disturbances for HTN also gained interest for short sleep duration (frequently associated with sleep deprivation) and insomnia [7–11].

Recent evidence suggests that the irregularity of sleep patterns, rather than sleep duration *per se*, may serve as a new cardiovascular risk factor [12]. Using two measurements of sleep irregularity based on wrist actigraphy [7-day standard deviation (SD) of sleep duration and 7-day SD of sleep onset timing], Huang *et al*. [12] found that irregular sleep duration and timing contribute to incident cardiovascular diseases, independent of OSA, sleep quantity and/or quality. Whether sleep irregularity is associated with HTN is unclear. Previous evidence from Korea using questionnaires found that sleeping more (at least 1 h) on the

Journal of Hypertension 2023, 41:670–677

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Received 1 July 2022 **Revised** 29 November 2022 **Accepted** 5 January 2023

DOI:10.1097/HJH.0000000000003392

weekend to compensate for weekday sleep deficit (weekend catch-up sleep) was associated with a lower prevalence of HTN [13].

In this study, we explore the associations of objective measurements of sleep irregularity with HTN (primary aim) and blood pressure (BP) levels (secondary aim) in a large sample of adults. We hypothesized that markers of sleep irregularity are independently associated with prevalent HTN and higher BP levels regardless of other sleep conditions, such as OSA, sleep duration and insomnia.

METHODS

This is a substudy of the ongoing ELSA-Brasil study [14]. The sleep approach in the ELSA-Brasil was conducted in the participants of the Sao Paulo site [3]. The local ethical committee approved the study (number 65903817.8.0000.0076), and all the participants provided informed consent. We performed the following evaluations.

Clinical evaluation

Each participant was interviewed in the workplace and visited the research centre for clinical examination following standard protocols. Interviews and examinations at each site were conducted by trained personnel with strict quality control [15,16]. The procedures, measurements and definitions for chronic conditions were previously described [17,18], and are also reported in the online supplement.

Hypertension diagnosis and blood pressure measurement

BP was measured with the validated Omron HEM 705CPINT oscillometric device (Omron Co, Kyoto, Japan) and an appropriate cuff size after a 5 min rest with the patient in a sitting position with feet on the floor and an empty bladder. Three measurements were taken at 1 min intervals. The mean of the second and third measurements was used in the analyses. HTN was defined by SBP at least 140 mmHg and/or DBP at least 90 mmHg and/or the current use of medications to control HTN [18,19].

Sodium and potassium excretion

Following the ELSA-Brasil's routine (previously reported by our group) [20], urine samples for measuring sodium excretion were collected between 1900 and 0700 h. The participants were asked to record the exact start and end times of the sample collection and any losses. This 12-h analysis was chosen to minimize the inaccuracy of the urine volume collected and stored during working periods and to avoid the influence of significant sodium loss in sweat. Previous validation of 12-h urine collected at night as a reliable tool to estimate 24-h intake/excretion of sodium and potassium was performed observing the following premises: volume at least 250 ml, collection time between 600 and 840 min (10–14 h), and a 12-h creatinine excretion at least 7.2 and less than 16.8 mg/kg in men and at least 5.6 and less than 12.6 mg/kg in women. Urine sodium and potassium were measured by the selective ion electrode method) [18].

Albumin–creatinine ratio

The albumin-creatinine ratio (ACR) was calculated from the 12-h urine samples obtained from concentrations of albumin and creatinine in the urine. The Jaffé kinetics methods (Advia 1200; Siemens) were used to measure urinary creatinine and the immunochemical assay (BN II Nephelometer; Siemens Dade Behring) was used to measure urinary albumin [20,21]. An abnormal ACR value was defined as greater than 30 mg/g of creatinine.

Sleep assessment

Wrist actigraphy

The objective sleep duration was assessed using the Actiwatch 2 wrist actigraph (Philips Respironics, Murrysville, Pennsylvania, USA) as previously described [3,22,23]. The participants were instructed to wear the actigraph continuously over a period of seven consecutive days and nights on the nondominant wrist. This evaluation was carried out in a typical week. Participants were asked to press the event marker button on the actigraph each night when they began trying to fall asleep and again when they got out of bed each morning. As previously described [12], we considered two measures to quantify sleep regularity: 7-day SD of sleep duration and [12] 7-day SD of sleep onset timing [12]. As described in detail in the statistical analysis section, these sleep irregularity markers presented low collinearity by the variance inflation factor analysis ($\sim$1), allowing independent evaluations for HTN and BP levels. Moreover, the actigraphy has an integrated light sensor to allow for the recording of photopic light. Sleep efficiency was defined as the percentage of time spent asleep in the sleep interval ('lights off' to 'lights on'). The wake after sleep onset (WASO) was defined as the minutes awake during the sleep period after sleep onset.

Overnight home sleep study

As previously described [3], sleep studies were performed using the Embletta Gold polygraph (Natus Medical Inc., Ontario, Canada), a standardized level-3 portable diagnostic device [24,25]. All the studies were manually scored according to the American Academy of Sleep Medicine (AASM) 2012 criteria [26] by an expert in sleep medicine. The sum of the number of apnoea and hypopnoea events per hour determined the apnoea–hypopnoea index (AHI). We excluded participants with predominantly (>50%) central sleep apnoea. An internal pragmatic validation suggested a good performance of this device in our population [27]. Considering the evidence suggesting that mild OSA may not be associated with increased cardiovascular risk, we used a more conservative AHI cut-off of at least 15 events/h of sleep [28].

Insomnia

We used an adapted Brazilian version of the Clinical Interview Scheduled Revised (CIS-R) [29], a validated instrument to evaluate insomnia and psychiatric morbidity [30,31]. Insomnia was defined by any complaint of difficulty initiating or maintaining sleep, and significantly reported daytime fatigue [CIS-R fatigue subscale (B) score of 2 or more] for at least 30 days [29].

Statistical analysis

Data are expressed as the means and standard deviations or as medians and interquartile intervals according to the distribution of the continuous variables. Categorical variables are presented as frequencies and percentages. We estimated the odds ratio (OR) and the 95% confidence interval (95% CI) in the different logistic regression models. Linear regression analysis was used to estimate beta values (β) and 95% confidence intervals (CI) to test associations of sleep variables and BP levels. We built three models of adjustments for both multivariate logistic and linear regression analyses. Model 1: unadjusted; model 2 includes traditional risk factors for HTN: age, gender, race, BMI, diabetes, excessive alcohol consumption, physical activity intensity, urinary sodium, potassium excretion, albumin–creatinine ratio (ACR), and antihypertensive medications (the latter used only for the BP endpoint); model 3: model 2 plus the presence of insomnia, objective sleep duration, and OSA. The multicollinearity was obtained through tolerance, as described by Myers [32]. A variance inflation factor lower than 5–10 indicates low multicollinearity among the predictor variables [33]. All statistical analyses were performed using IBM SPSS Statistics for Windows, version 28.0 (IBM Corp., Armonk, New York, USA). A *P* value less than 0.05 was considered statistically significant.

RESULTS

Demographic and clinical characteristics

We initially invited 2463 participants who fulfilled the inclusion criteria during the 2-year recruitment period. The final sample constituted 1720 patients as 743 individuals met one or more exclusion criteria (Fig. 1).

Our sample consisted of predominantly overweight and middle-aged women. Insomnia was observed in 19.5% of the participants (Table 1). Compared with participants without HTN, those with HTN (27%) were older, overweight, predominantly male, and had a higher proportion of blacks. The frequency of antihypertensive medications in the HTN group was 78.5%. In addition, they had a higher 7-day SD of sleep duration, sleep onset timing, WASO, and lower sleep efficiency (Table 2). The frequency of OSA was also higher in the HTN group. On the other hand, objective sleep duration was not different between groups (Table 2).

Figure 2 depicts two typical examples of 7-day sleep duration measured by actigraphy in our population showing significant differences in the parameters of sleep regularity, despite having similar objective sleep durations.

Regression models testing the associations between sleep irregularity and hypertension and blood pressure levels

Logistic regression analyses were performed to test the associations among the sleep parameters with HTN. An

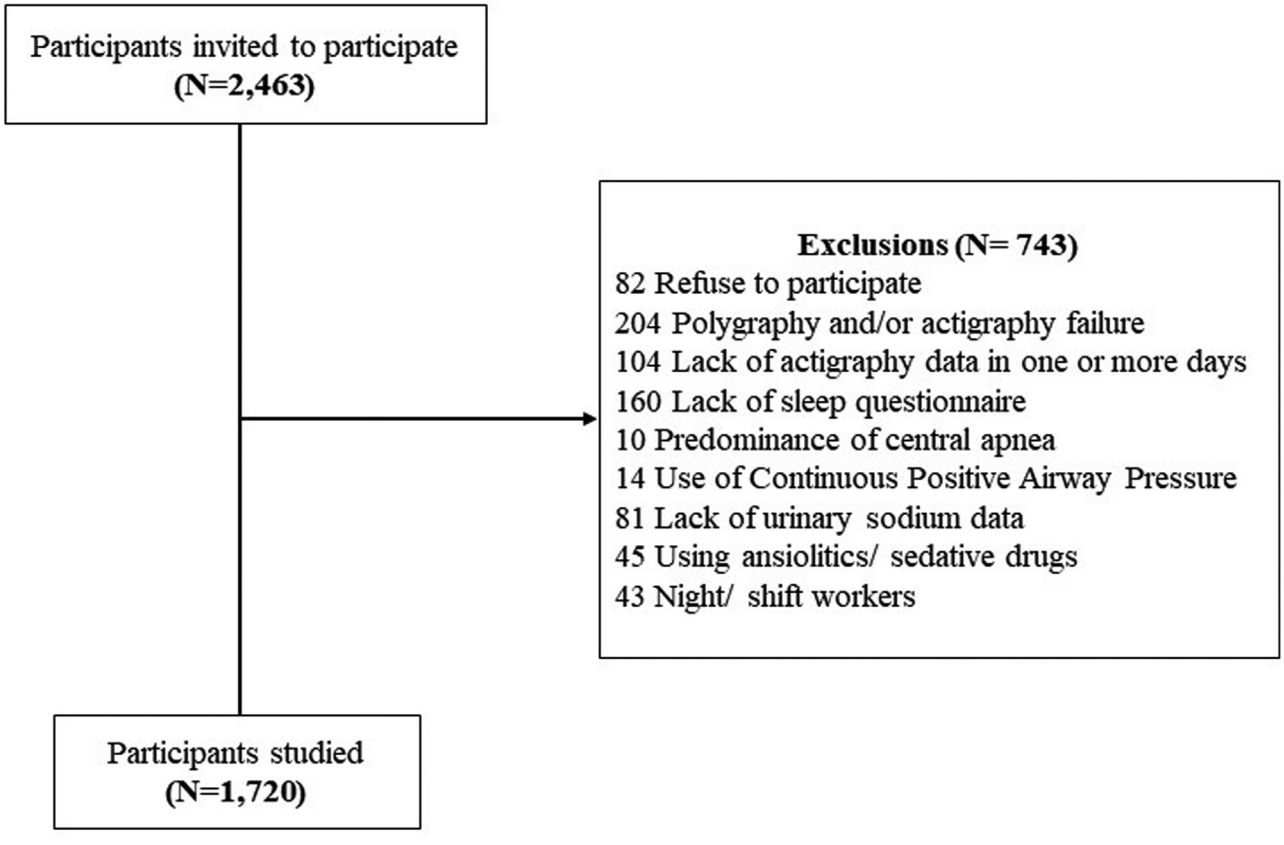

FIGURE 1 Flow chart.

TABLE 1. Demographic, anthropometric and sleep characteristics of all participants according to hypertension status

Characteristics	Overall ($N = 1720$)	HTN		
		No ($N = 1264$) (73%)	Yes ($N = 456$) (27%)	*P* value
Age (years) [mean (SD)]	49 ± 8	48 ± 8	52 ± 8	**<0.001**
Male, sex [*n* (%)]	746 (43.4)	512 (40.5)	234 (51.3)	**<0.001**
Self-reported race [*n* (%)]				**0.001**
Whites	1059 (61.6)	812 (64.2)	247 (54.2)[a]	
Mixed	344 (20.0)	244 (19.3)	100 (21.9)	
Black	211 (12.3)	135 (10.7)	76 (16.7)[a]	
Asian/others	106 (6.2)	73 (5.8)	33 (7.2)	
BMI (kg/m^2) [mean (SD)]	27.0 ± 4.8	26.2 ± 4.3	29.2 ± 5.2	**<0.001**
Obesity [*n* (%)]	392 (22.8)	211 (17.5)	171 (37.5)	**<0.001**
Neck circumference (cm) [mean (SD)]	35.8 ± 3.6	35.3 ± 3.4	37.4 ± 3.8	**<0.001**
Waist circumference (cm) [mean (SD)]	88.8 ± 12.2	86.4 ± 11.2	95.5 ± 12.4	**<0.001**
SBP (mmHg)	118 ± 15	113 ± 11	131 ± 16	**<0.001**
DBP (mmHg)	74 ± 10	72 ± 8	83 ± 11	**<0.001**
Heart rate (bpm)	71 ± 10	71 ± 8	72 ± 11	**0.43**
Diabetes [*n* (%)]	273 (15.9)	126 (10.0)	147 (32.2)	**<0.001**
Urinary sodium excretion (g) [mean (SD)]	1.9 ± 0.9	1.9 ± 0.9	2.2 ± 1.1	**<0.001**
Urinary potassium excretion (g) [mean (SD)]	0.9 ± 0.4	0.9 ± 0.4	0.9 ± 0.4	**0.03**
Albumin-to-creatinine ratio (mg/g) (SD)	12.0 ± 71.1	7.9 ± 9.1	23.5 ± 136.8	**<0.001**
Previous cardiovascular events (stroke and myocardial infarction) [*n* (%)]	37 (2.2)	17 (1.3)	20 (4.4)	**<0.001**
Physical activity intensity (%)				
Insufficient	1325 (77.0)	959 (75.9)	366 (80.3)	0.13
Moderate	243 (14.1)	190 (15.0)	53 (11.6)	
Vigorous	152 (8.8)	115 (9.1)	37 (8.1)	
Current smokers [*n* (%)]	240 (14.0)	186 (14.7)	54 (11.8)	0.07
Excessive drinking [*n* (%)]	158 (9.2)	115 (9.1)	43 (9.4)	0.44
Insomnia [*n* (%)]	336 (19.5)	248 (19.6)	88 (19.3)	0.47
Epworth sleepiness scale [mean (SD)]	9 ± 5	9 ± 5	10 ± 5	0.13
Excessive daytime sleepiness[b] [*n* (%)]	656 (38.1)	467 (36.9)	189 (41.4)	0.05

Continuous variables were compared using one-way ANOVA or the Kruskal–Wallis test as appropriate after assessing the normality assumptions. BP, blood pressure; EDS, excessive daytime sleepiness; HTN, hypertension. Bold indicates values that are statistically significant (set as $P < 0.05$).
[a]Yes vs. No.
[b]Epworth Sleepiness Scale greater than 10.

independent association between continuous 7-day SD of sleep duration and HTN (OR 1.004; 95% CI 1.0002–1.007) was found when we progressively adjusted for factors related to HTN (models 2 and 3) but not when we used the 7-day SD of sleep duration more than 90 (Fig. 3a) or sleep onset timing more than 30 min (further details are shown in Table S1, Supplemental Material, http://links.lww.com/HJH/C139)

Regarding BP levels, the association between the 7-day SD of sleep duration more than 90 min was the only parameter of sleep irregularity independently associated with higher SBP and DBP (Table 3). Based on our model, this marker of sleep irregularity remained significant even considering the presence of antihypertensive medications.

TABLE 2. Detailed actigraphy and polygraphy characteristics according to hypertension status

Characteristics	Overall ($N = 1720$)	HTN		
		No ($N = 1264$) (73%)	Yes ($N = 456$) (27%)	*P* value
Wrist actigraphy				
Objective sleep duration (h) [mean (SD)]	6.3 ± 56.4	6.3 ± 54.0	6.3 ± 1.03	0.42
7-day SD of sleep duration, min, mean (SD)	76.6 ± 35.2	75.4 ± 33.5	79.8 ± 39.6	**0.02**
7-day SD of sleep duration >90 min (%)	491 (28.5)	347 (27.5)	144 (31.6)	0.05
Sleep onset timing, min, mean (SD)	23.1 ± 18.3	22.5 ± 18.0	24.8 ± 18.4	**0.01**
7-day SD of sleep onset timing, min, mean (SD)	23.3 ± 20.9	22.7 ± 20.8	25.0 ± 21.2	0.05
7-day SD of sleep onset timing >30 min (%)	284 (22.3)	271 (21.5)	112 (24.6)	0.10
Sleep efficiency (%)	82.7 ± 6.3	83.1 ± 6.0	81.5 ± 5.7	**<0.001**
WASO (min) [mean (SD)]	45.1 ± 18.6	43.7 ± 17.9	48.9 ± 19.8	**<0.001**
Sleep study				
AHI (ev/h)	14.7 ± 14.9	12.8 ± 13.7	20.0 ± 17.0	**<0.001**
OSA (%)	568 (33.0)	346 (27.4)	222 (48.7)	**<0.001**
Lowest SpO_2, (mean, SD)	85 ± 6	86 ± 6	83 ± 6	**<0.001**
Total time with SpO_2 <90% (%)	4 ± 11	3 ± 9	7 ± 15	**<0.001**

Continuous variables were compared using one-way ANOVA or the Kruskal–Wallis test as appropriate after assessing the normality assumptions. AHI, apnoea–hypopnea index; OSA, obstructive sleep apnoea; SD mean, standard deviation mean; SpO_2 less than 90%, percentage oxygen saturation below 90%; WASO, wake time after sleep onset. Bold indicates values that are statistically significant (set as $P < 0.05$).

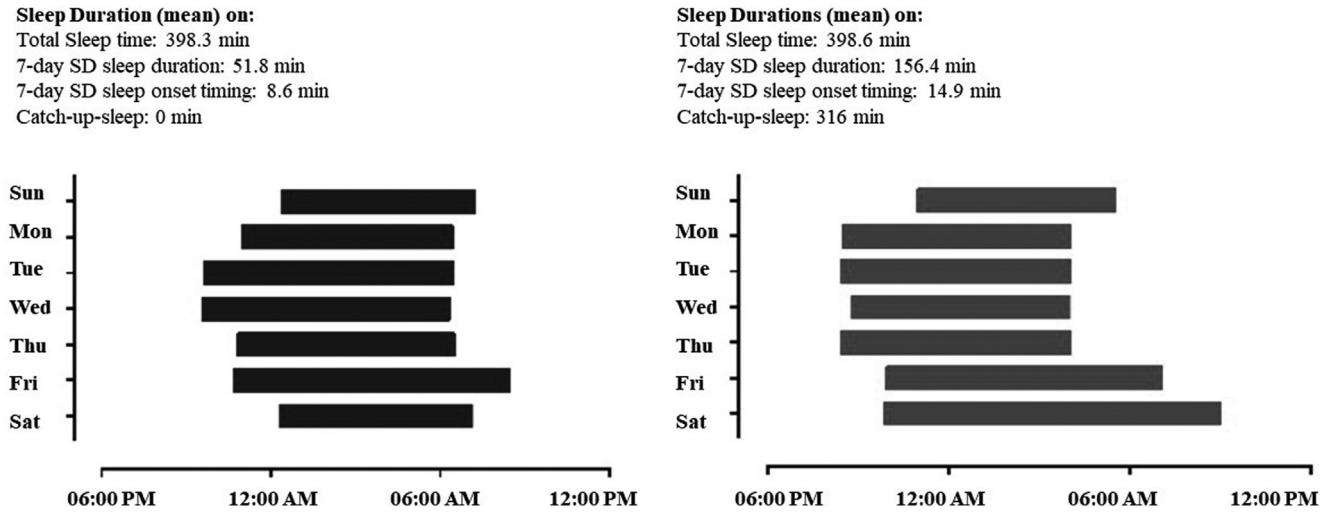

FIGURE 2 Examples of participants presenting a regular versus an irregular sleep pattern. On the left, a normal sleeper with 0 min of weekend catch-up sleep versus on the right an irregular sleeper with a weekend catch-up sleep with 316 min. Please also note significant differences in the 7-day SD of sleep duration and 7-day SD of sleep-onset timing.

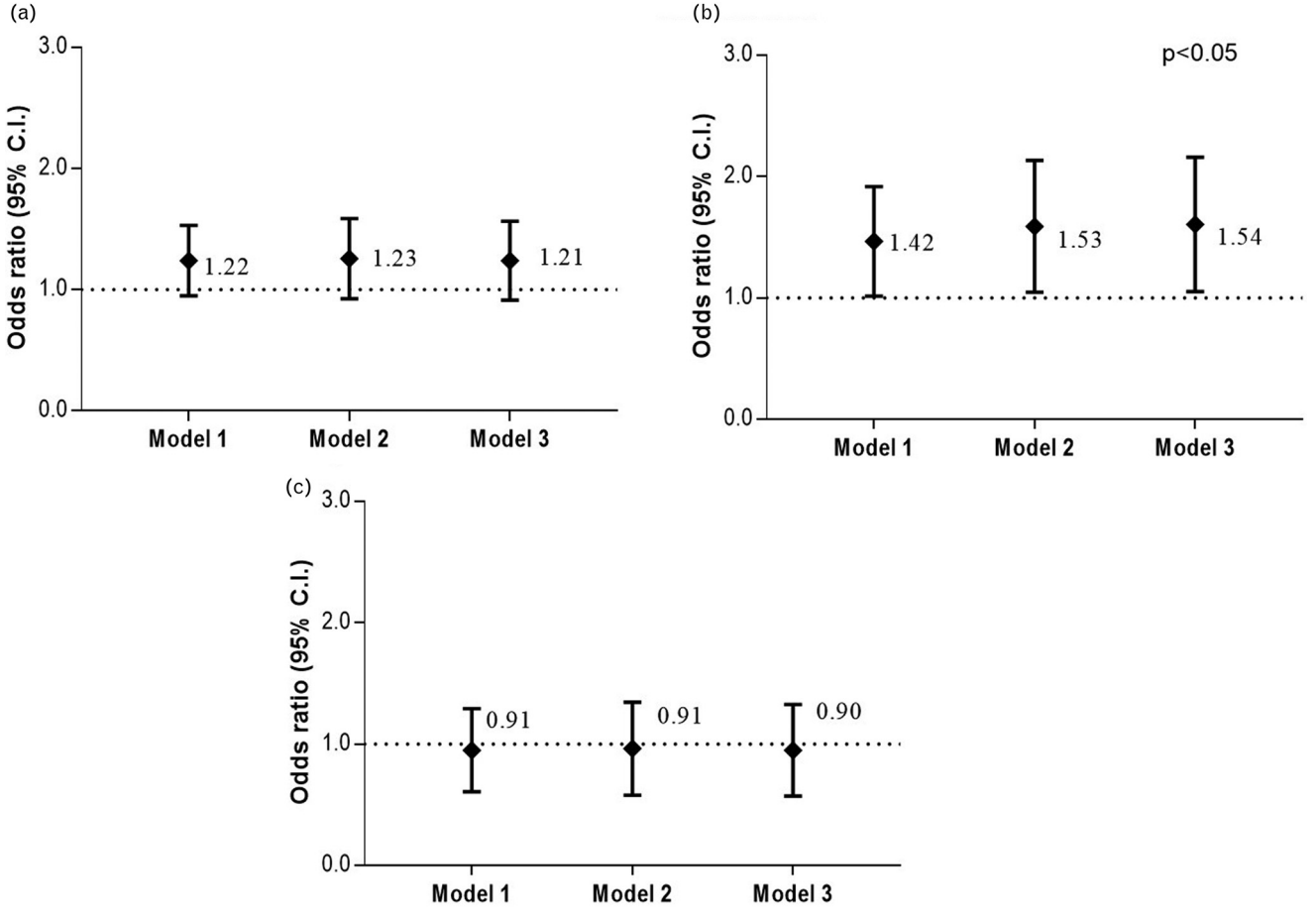

FIGURE 3 (a) Association between 7-day SD of sleep duration more than 90 min and hypertension: model 1: adjustments; model 2: adjusted for age, gender, race, BMI, diabetes, excessive alcohol consumption, physical activity intensity, urinary sodium and potassium excretion, albumin–creatinine ratio (ACR); model 3: adjusted for the model 2 variables plus the presence of insomnia, objective sleep duration and obstructive sleep apnoea (OSA), (b) Association between 7-day SD of sleep duration more than 90 min and hypertension (HTN) without OSA participants and (c) association between 7-day SD of sleep duration more than 90 min and hypertension with only OSA participants: model 1: adjustments; model 2: adjusted for age, gender, race, diabetes, excessive alcohol consumption, physical activity intensity, urinary sodium and potassium excretion, albumin–creatinine ratio (ACR); model 3: adjusted for the model 2 variables plus the presence of insomnia, objective sleep duration.

TABLE 3. Linear regression analysis of sleep parameters and blood pressure levels

Sleep variable	$n = 1720$ (%)	Model 1 β (95% CI)	Model 2	Model 3
SBP				
7-day SD of sleep duration				
Continuous	1720 (100)	0.01 (−0.001 to 0.03)	0.01 (−0.007 to −0.02)	0.009 (−0.008 to 0.02)
Binary				
>90 min	491 (28.5)	**2.21 (0.65–3.76)**†	**1.70 (0.38–3.02)***	**1.64 (0.32–2.96)***
7-day SD of sleep onset timing				
Continuous	1720 (100)	0.01 (−0.02 to 0.04)	−0.01 (−0.04 to 0.01)	−0.01 (−0.04 to 0.01)
Binary				
>30 min	384 (22.3)	0.44 (−1.24 to 2.13)	−0.37 (−1.79 to 1.05)	−0.40 (−1.83 to 1.02)
DBP				
7-day SD of sleep duration				
Continuous	1720 (100)	**0.01 (0.002–0.03)***	0.006 (−0.006 to 0.01)	0.005 (−0.007 to 0.01)
Binary				
>90 min	491 (28.5)	**1.81 (0.74–2.89)**†	**1.20 (0.26–2.15)**	**1.13 (0.19–2.07)***
7-day SD of sleep onset timing				
Continuous	1720 (100)	0.006 (−0.01 to 0.02)	−0.009 (−0.02 to 0.01)	−0.01 (−0.03 to 0.01)
Binary				
>30 min	384 (22.3)	−0.21 (−1.38 to 0.94)	−0.54 (−1.56 to 0.48)	−0.60 (−1.62 to 0.41)

Model 1: unadjusted. Model 2: adjusted for age, gender, race, BMI, diabetes, excessive drinking, physical activity intensity, urinary sodium and potassium excretion, Albumin-to-creatinine ratio (ACR) and antihypertensive medications. Model 3: adjusted for the model 2 variables plus the presence of insomnia, objective sleep duration and the presence of OSA. β, beta coefficient; CI, confidence interval. Bold indicates values that are statistically significant (set as $P < 0.05$).
*$P < 0.05$.
†$P < 0.01$.

Stratified analysis testing the association of sleep irregularity parameters with hypertension and blood pressure levels according to obstructive sleep apnoea status

A supplementary analysis was performed to test the association between sleep parameters and HTN. An independent association was observed between 7-day SD of sleep more than 90 min and HTN in a group without OSA participants (OR 1.54; 95% CI 1.09–2.19) (Table S2, http://links.lww.com/HJH/C139). Consistently, SBP and DBP were independently associated with these sleep parameters (Table S3, http://links.lww.com/HJH/C139). No association was observed in a group with only OSA (Table S2, http://links.lww.com/HJH/C139 and Table S4, http://links.lww.com/HJH/C139).

DISCUSSION

Our study investigated the associations between sleep irregularity and HTN and BP levels in a large sample of adults. The prevalence of HTN in our sample (27%) is consistent with the current literature [19]. After adjusting for the main confounders and OSA, we found that markers of sleep irregularity were independently associated with HTN and BP levels. Specifically, a 7-day SD of sleep duration greater than 90 min was associated with HTN and SBP/DBP levels. A higher chance of having HTN or BP levels once again 7-day SD of sleep duration greater than 90 min was observed when we excluded participants with OSA. Taken together, these results underscore the potential role of sleep patterns (rather than sleep duration per se) in the association with HTN.

The main results are consistent with a recent investigation suggesting the potential impact of sleep irregularity on cardiovascular events [12]. In the MESA cohort, Huang *et al.* [12], in a prospective study, found that participants with the most irregular sleep duration or sleep onset timing patterns had a two-fold risk for cardiovascular events compared with participants with the most regular ones after a median follow-up of 4.9 years. Of note, these associations were independent of several traditional cardiovascular risk factors and sleep parameters (including OSA). The potential mechanisms underlying the association between sleep irregularities and cardiovascular disease regardless of OSA are poorly explored, and several hypotheses have been proposed. Circadian clock genes, such as *clock*, *Per2*, and *Bmal1*, were shown experimentally to control a broad range of cardiovascular rhythms and functions, including BP and endothelial function [12,34,35]. Therefore, beyond genetic predispositions to sleep irregularity, circadian disruptions/poor sleep patterns may potentially influence these related genes that may increase vascular tone, the response to stress, and autonomic dysfunction contributing to increased BP [36,37]. In our study, the independent associations of sleep irregularity parameters with HTN and BP levels seem to be modest. However, the clinical relevance of our findings reinforces those of Huang *et al.* [12] and underscores the need for additional studies addressing the potential mechanisms behind the association between sleep irregularity and HTN.

Although the relationship between OSA and HTN is well established, as they share many of the same risk factors (obesity, male gender, and advancing age), pathophysiological mechanisms that link them together (the activation of the renin–angiotensin–aldosterone system, negative intrathoracic pressure putting stress on the heart, nocturnal fluid shift, inflammatory and cytokine-mediated effects of hypoxemia, etc.) [38], and understanding the potential interference of OSA in the parameters of sleep fragmentation (that leads to the exacerbation of potential apnoeic episodes and intensifies the patient's nocturnal sympathetic hyperactivity) [39], our study, similar to Huang *et al.* [12],

suggests that irregular sleep duration in specific settings may be novel and independent risk factors for cardiovascular disease.

Our study has potential strengths and limitations to be addressed. We included a large sample of participants with accurate and standardized BP measurements. The assessment of sleep duration was not only performed by the self-reported method but also obtained through 7 days of actigraphy. All subjective and objective analyses were performed in a blinded fashion. Our regression analysis considered several variables related to HTN such as age, gender, race, BMI, diabetes, alcoholism, physical activity, urinary sodium and potassium, ACR and OSA. The following limitations need appropriate discussion. First, this is a cross-sectional study. Therefore, we cannot claim any causal relationship between sleep irregularity parameters and HTN and BP levels. Second, we performed a portable overnight sleep study rather than polysomnography. This approach had a pragmatic principle because of the limited availability of polysomnography for this large sample of participants not referred for sleep studies. However, sleep monitoring occurred in the participant's usual environment. We published a pragmatic validation of our portable monitor using a subset of 300 participants from the ELSA-Brasil [27]. Due to limited resources, an additional limitation is the lack of 24-h ambulatory BP monitoring in this large cohort. This limitation prevents any inference on the potential impact of sleep regularity on BP control and nocturnal BP. Finally, although our analyses controlled for many potential confounders, residual confounding (e.g. diet quality, antihypertensive class and doses) is still possible. The option for withdrawing medications is not feasible for the ethical committee. On the other hand, the independent associations of markers of sleep irregularity with HTN and BP mimic real-life situations in our epidemiologic study.

In conclusion, this study suggests that sleep irregularity (highlighting the variable 7-day SD of sleep duration >90 min), rather than sleep duration *per se,* is associated with HTN and higher BP levels highlight the importance of circadian patterns of sleep on haemodynamics. Future longitudinal and interventional studies specifically addressing sleep irregularity are necessary to define the role of sleep irregularity in HTN. Additionally, experimental and translational investigations addressing related mechanisms are warranted.

ACKNOWLEDGEMENTS

The authors thank the research team of the ELSA-Brasil, the ELSA-Brasil headquarter in São Paulo and all participants included in this study.

Funding/support: this work was supported by research grants from FAPESP (grants 2012/02953-2 and 2019/23496-8). The Brazilian Longitudinal Study of Adult Health (ELSA-Brasil) was supported by the Brazilian Ministry of Health (Science and Technology Department) and the Brazilian Ministry of Science and Technology (*Financiadora de Estudos e Projetos* and CNPq National Research Council) [grants 01 06 0010.00 RS, 01 06 0212.00 BA, 01 06 0300.00 ES, 01 06 0278.00 MG, 01 06 0115.00 SP, 01.10.0773.00 SP, and 01 06 0071.00 RJ].

Conflicts of interest

There are no conflicts of interest.

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