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ORIGINAL RESEARCH

Sleep Duration Irregularity is Associated with Elevated Blood Pressure During Submaximal Exercise in Young Adults

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Purpose: Irregularity in nightly sleep duration is reported to associate with elevated blood pressure (BP), but it is unclear whether this association can be observed with BP measured during exercise after controlling for factors known to influence the exercise pressor reflex.

Methods: Twenty-nine young adults (22±4y; 19 men, 10 women) performed cycling exercise until volitional fatigue to assess peak oxygen uptake (VO_2). Actigraphy was used to monitor sleep duration and daily physical activity for seven consecutive days after which participants completed two bouts of moderate-intensity cycling while BP and VO_2 were measured using a Tango+ device and indirect calorimetry, respectively. Systolic BP was averaged from the two bouts of exercise and expressed as a change from seated rest (ΔSBP). Sleep duration regularity was calculated as standard deviation (SD) and coefficient of variation (CV).

Results: Systolic BP at seated rest, during exercise, and ΔSBP was 113±13, 152±21, and 38±13 mmHg, respectively. Sleep duration SD (range 10–146 min) and sleep duration CV (range 2–54%) when excluding weekend nights were significantly correlated with ΔSBP (r = 0.58 and r = 0.62, respectively; both $p<0.01$) after adjusting for age, sex, body mass index, peak VO_2, physical activity, resting systolic BP, chronotype, and the VO_2 response to exercise. Sleep duration regularity analyzed with weekend nights included (across all seven days) was also significantly correlated with ΔSBP ($p\leq0.01$), but had weaker correlation coefficients.

Conclusion: These results indicate that sleep regularity, especially when excluding weekend nights, is associated with the rise in systolic BP during moderate-intensity exercise in young adults. Sleep duration regularity may be a useful tool to capture the impact of intermittent nights of insufficient sleep on BP dysregulation.

Keywords: sleep duration, sleep variability, blood pressure, exercise, VO_2, chronotype

Introduction

Sleep is now recognized as an important lifestyle factor for optimal cardiovascular health. So much so that in 2022, the American Heart Association added sleep health to their "Life's Essential 8" list of modifiable risk factors aimed at reducing the development of heart disease and stroke.[1] The current recommendation for adults is to sleep 7–9 hours per night.[2] This recommendation stems partly from research that finds sleep less than 7 hours per night (insufficient sleep) is associated with higher blood pressure (BP) in both cross-sectional comparisons as well as longitudinal analyses.[3] A weakness, however, of focusing on self-reported or average weekly sleep duration is that it does not capture intermittent nights of insufficient sleep that may be present throughout the week. A single night of insufficient sleep has been shown to elevate systolic BP the subsequent morning.[4] Possible mechanisms that explain this phenomenon include augmented sympathetic activity and decreased baroreflex sensitivity that are reported to occur after sleep loss.[5,6] Thus, analyzing for sleep duration regularity may be a useful tool to capture the impact of intermittent nights of insufficient sleep on BP dysregulation.

The most common method of assessing sleep duration regularity is through the calculation of standard deviation (SD) from nightly sleep duration across the measurement period. Using this approach, Hoopes et al reported no difference in resting BP between young adults with lower sleep duration SD and those with higher sleep duration SD.[7] Culver et al

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also observed no association between resting BP and sleep duration SD in young adults using bivariate correlations, but after adjusting for sex and body mass index (BMI) using partial correlations these researchers observed higher sleep duration SD to correlate with higher resting BP.[8] This latter finding is consistent with research in middle-aged to older adults that report sleep duration SD to associate with risk factors for hypertension[9] as well as contribute to an increased risk of developing cardiovascular disease.[10] Considering the large and often fluctuating demands of young adults as they try to find a balance between college, work, and social life has been linked to higher sleep duration irregularity,[11] more research focused on this age group is needed to understand the influence of sleep variability on cardiovascular health.

Fewer studies have assessed the association between sleep duration regularity and exercise BP. Meth et al found the change in systolic BP from rest to 1-minute after the YMCA step test to have a positive correlation with sleep duration irregularity (based on interquartile range) after adjusting for average sleep duration, age, sex, BMI, and physical activity the week prior to testing.[12] Shortcomings of this study, however, is that BP was not measured during exercise, intensity of exercise was not consistent between participants, and BP was measured 1-minute into recovery from exercise. In young adults, systolic BP decreases rapidly (~10–15 mm Hg) within the first minute of exercise and can vary based on cardiorespiratory fitness level.[13] Therefore, to address these limitations, the purpose of this study was to examine for a relationship between sleep duration regularity and BP measured during exercise at a set relative intensity and after controlling for variance explained by confounding variables including cardiorespiratory fitness level. We hypothesized that higher sleep duration irregularity would associate with an elevated BP response to exercise.

Methods

Participants

College students, both men (n = 20) and women (n = 10), between the ages of 18–39 years completed this study. One man was removed from the final analysis because BP was not measured during exercise due to poor electrode placement. In the end, data from 29 participants were included in the final analysis. Inclusion criteria included no recent history (<6 months) of sleep problems, physically inactive as defined by less than three days per week of planned exercise, and non-obese as defined by a BMI < 30 kg/m^2. Exclusion criteria included taking medications or supplements to improve sleep, being a smoker and/or vaper, and a previous diagnosis of having diabetes mellitus, pulmonary disease, or coronary heart disease. Menstrual cycle phase was not controlled for in this study as past research reports no difference in heart rate or BP responses to moderate-intensity cycling between low and high hormone phases of the menstrual cycle in young women.[14,15] Methods of participant recruitment for this study included weekly advertisements through the university Email announcement system, study fliers posted throughout the university campus, and announcements in undergraduate courses.

Study Design

This study was reviewed and approved by the Human Research Protection Program at Texas Tech University (approval #IRB2021-1039), and its procedures conformed to standards set by the Declaration of Helsinki. Participants reported to the laboratory for two study visits across an eight-day period. All visits took place in the morning, and time for each visit was kept constant for each participant. Instructions prior to each visit were to avoid strenuous exercise for 24-hours and to abstain from caffeine and food intake for 12-hours and 4-hours, respectively. The first visit consisted of reviewing the consent form, completing a medical history questionnaire, screening, and a maximal cycling exercise test. No study measurements were taken before obtaining written informed consent from each participant. At the end of the first visit, participants were instructed to maintain their normal sleep behavior for seven consecutive days while sleep and physical activity was monitored using actigraphy. On the eighth day, participants reported to the laboratory for the second visit which involved heart rate variability testing and submaximal cycling exercise.

Study Measurements

Maximal Exercise Test

A ramp protocol on a cycle ergometer (Lode Corival, The Netherlands) was used to assess cardiorespiratory fitness. The exercise test consisted of a 3-minute warmup of cycling at 0W followed by a ramp increase in work rate at 30 W/min for

men and 20 W/min for women until participants were unable to maintain their self-selected pedal rate (drop of ≥10 rpm). Breath-by-breath pulmonary gas exchange was measured continuously during exercise using indirect calorimetry (Ultima CardiO$_2$, MedGraphics, St. Paul, MN, USA). Peak VO_2 was recorded as the highest 30-second average obtained at the end of exercise, and was considered a proxy of cardiorespiratory fitness level. All but one participant reached an exercise heart rate within 10 beats of their age-predicted maximum (range −13 to +7 bpm; 93–103% of predicted), and all participants reported a rating of perceived exertion ≥16 on the Borg 6–20 scale (range 16–20, median = 19).

The gas exchange threshold (GET) was determined using the V-slope method[16] in conjunction with the ventilatory equivalent method.[17] The VO_2 at the GET was adjusted for the delayed rise in VO_2 at the onset of exercise.[18] This was done by shifting the linear VO_2 versus work rate (and time) relationship to the left, by subtracting two-thirds of the ramp work rate (20W for men and 13W for women) from the work rate initially identified at the GET.[19] The adjusted work rate for 95% of the GET was then used to prescribe moderate-intensity exercise for all participants at the second visit.

Sleep Monitoring

A triaxial accelerometer (GT9X, ActiGraph, Pensacola, USA) sampling at 30Hz was used to measure sleep duration. Participants wore the accelerometer at their wrist, and were instructed to only remove it when bathing or swimming. Actigraphy is reported to provide similar estimates of sleep duration than polysomnography,[20,21] which is considered the gold-standard method for sleep monitoring. Sleep data was reduced to 60-second epochs and processed using a combination of Cole-Kripke and Tudor-Locke algorithms in ActiLife software (version 6.13.3, ActiGraph). The Cole-Kripke algorithm determined epochs as "sleep" or "wake". The Tudor-Locke method determined sleep periods by identifying bedtime as five consecutive minutes of sleep epochs and wake as ten consecutive minutes of wake epochs at the end of a sleeping period. To complete a more accurate sleep analysis, participants were asked to keep record of their bed and wake times during the observation period. The self-reported clock times were used to refine sleep periods identified by ActiLife software to more accurately determine sleep duration. The standard deviation (SD) and coefficient of variation (CV) of nightly sleep duration was used to estimate sleep duration regularity. The CV was included as it has been frequently used in the literature to assess intraindividual night-to-night variability in sleep.[22] Lastly, the midpoint of sleep on weekend days (MSF) corrected for sleep debt on weekdays (MSFsc) was calculated as an index of chronotype.[23] This variable was included in this study since internal circadian rhythm influence hemodynamic (eg, heart rate) responses to isometric[24] and aerobic exercise.[25]

Daily Physical Activity

Daily physical activity was objectively measured using a second triaxial accelerometer (GT3X+, ActiGraph, Pensacola, Florida) sampling at 30Hz. This accelerometer was worn at the left hip aligned with the foot, and participants were instructed to only remove it when bathing, swimming, or before bed-time. Counts per minute of physical activity data (using vector magnitude) were obtained by summing 1-second epoch data for each 60-second interval. Raw counts were processed using wear time criteria of ≥60 consecutive zeros and ≥10h per day of data using automated software (ActiLife v6, Pensacola, USA). Steps per day was recorded as an index of daily physical activity level.

Heart Rate Variability

Electrocardiography (ECG) was recorded using a 3-lead electrocardiogram (PowerLab, ADInstruments, Colorado Springs, USA). Electrode sites on the body were wiped down with alcohol to ensure good contact with skin before electrodes were placed on the right arm, left arm, and left leg. Participants rested in the seated position for 10-minutes while ECG readings were collected. Instructions during the measurement were to breathe normally, remain relaxed by keeping eyes closed, feet flat on the ground, and hands resting on the legs.[26] Raw ECG readings from lead II were analyzed in the frequency domain using heart rate variability software (HRVanalysis v.1.2, ANS Lab Tools, Saint-Etienne, France) from the last 5-minutes of the ECG recording. Low frequency (LF, 0.04–0.15 Hz) and high-frequency power (HF, 0.15–0.40 Hz) were derived by the Fast Fourier Transform method. Natural logarithms of LF and HF power in milliseconds squared (ms^2) are reported as these values were normally distributed. It is thought that parasympathetic modulation of cardiac rhythm is a major contributor to HF while LF reflects both parasympathetic and sympathetic activity at the heart. Consequently, the LF/HF ratio is considered by some investigators to reflect sympathetic modulations of cardiac rhythm.[27]

Submaximal Cycling Exercise

The exercise protocol consisted of 5-minuntes of low resistance cycling at 20W followed by two 4-minute bouts of moderate-intensity exercise at 95% of the GET separated by 5-minutes of cycling at 20W. Cycling was done on an upright Lode Corival cycle ergometer. Pulmonary gas exchange was measured using indirect calorimetry (Ultima CardiO_2, MedGraphics, St. Paul, MN, USA). Oxygen uptake was averaged at rest and during the last 60-seconds of the initial 20W cycling bout as well as each bout of moderate-intensity exercise. The 60-second average VO_2 for each bout of moderate-intensity exercise was averaged together for analysis. Heart rate and BP were measured during exercise using a Tango+ automated device (SunTech Medical Inc., Morrisville, NC, USA). This device has been shown to provide accurate and reliable measures of systolic BP at rest and during maximal exercise testing as compared to an indwelling catheter.[28] Three EKG electrodes were placed on the torso for measurement of heart rate, and a BP cuff was placed on the left arm. Heart rate and BP were measured at rest while the participant sat on the cycle ergometer, and during the last minute of each bout of moderate-intensity exercise. Participants were instructed to relax their left arm, fully extended at their side while maintaining their cycling cadence during BP measurements. As noted above, one participant was excluded from the blood pressure analysis as the device gave an error during measurement likely due to poor EKG placement or adherence to the skin. Heart rate and BP from each moderate-intensity bout were averaged together for analysis.

Statistical Analysis

Two-tailed paired t-tests were used to compare hemodynamics between rest and exercise conditions as the data were normally distributed. Pearson correlation was used to assess the relationship between sleep duration regularity and BP. Variables that were not normally distributed were transformed to follow a normal distribution using Johnson transformation. Partial correlations were used to assess the association between sleep duration regularity and BP after adjusting for age, sex, BMI, physical activity (daily steps), cardiorespiratory fitness (peak VO_2), chronotype (MSFsc), resting systolic BP, and metabolic rate during exercise (change in VO_2 from baseline cycling at 20W to 95% of the GET). These covariates were selected to be consistent with past research[8,12] and control for variance by variables conceived to influence BP responses to exercise. Correlation coefficients and associated 95% confidence intervals are reported and can be interpreted as small (r = 0.1–0.3), medium (r = 0.3–0.5), and large (r = >0.5).[29] Statistical significance was considered a priori at $p<0.05$.

Results

Descriptive Data

Average age, log LF, log HF, LF/HF ratio, and chronotype of participants were 22 ± 4y, 7.38 ± 0.90 ms^2, 6.29 ± 1.01 ms^2, 1.19 ± 0.15, and 4:55 a.m. (range 3:26–7:49 a.m.), respectively. By design, participants were non-obese with an average BMI of 22.9 ± 2.7 kg/m^2, and were physically inactive as indicated by their daily step count of 7100 ± 2442 steps per day and low average peak VO_2 of 35 ± 4 mL/kg/min. Average sleep duration across all seven days was 361 ± 48 min (range 276–439 min), sleep duration SD was 62 ± 30 min (range 11–143 min), and sleep duration CV was 18 ± 10% (range 3–50%). After excluding weekend nights (Friday and Saturday), average sleep duration was 361 ± 50 min (range 270–449 min), sleep duration SD was 61 ± 33 min (range 10–146 min), and sleep duration CV was 17 ± 11% (range 2–54%).

Response to Submaximal Exercise

Table 1 presents heart rate and BP data at rest and during moderate-intensity exercise. Moderate-intensity exercise raised heart rate to 74 ± 7% of maximum, and in terms of metabolic demand, the change in VO_2 from baseline (20W) to moderate-intensity cycling was 9.84 ± 2.84 mL/kg/min. Systolic BP, but not diastolic BP, increased significantly during exercise ($p<0.001$; Table 1). The change in systolic BP (ΔSBP) from rest to exercise was 38 ± 13 mm Hg (range 22–75 mm Hg).

Association Between Sleep Regularity and BP

Simple bivariate correlations between sleep duration regularity and measured variables are presented in Table 2. Only chronotype and the ΔSBP from rest to exercise were found to correlate with sleep duration regularity. Specifically, chronotype had positive correlations with sleep duration SD (7-day: r = 0.61 [0.32, 0.80]; excluding weekend: r = 0.56

Table 1 Hemodynamic Response to Cycling Exercise at 95% of the GET

	Seated Rest	Exercise	t-value	*p*-value
Work rate (W)	0	89 ± 22		
Heart rate (bpm)	74 ± 7	138 ± 14	−25.4	<0.001
Systolic BP (mm Hg)	113 ± 14	152 ± 21	−15.45	<0.001
Diastolic BP (mm Hg)	73 ± 8	72 ± 10	0.29	0.77

Note: Values are mean ± SD.
Abbreviations: GET, gas exchange threshold; BP, blood pressure.

Table 2 Pearson Correlation Coefficients

		Entire Week (7-days)			Excluding Weekend Nights		
	ΔSystolic BP	Sleep Duration	Sleep Duration SD	Sleep Duration CV	Sleep Duration	Sleep Duration SD	Sleep Duration CV
Age	0.42*	−0.08	−0.05	−0.01	−0.08	−0.09	−0.07
Sex	0.10	−0.35	0.23	0.28	−0.36	0.22	0.24
Body mass index	0.23	−0.14	0.07	0.09	−0.14	0.04	0.13
Chronotype	0.24	−0.06	0.61**	0.56**	−0.01	0.69**	0.60**
Daily Steps	−0.27	0.15	−0.14	−0.20	0.13	−0.07	−0.04
Peak VO_2	−0.04	−0.01	−0.13	−0.11	−0.01	−0.17	−0.14
Log LF	0.09	−0.12	−0.06	−0.03	−0.20	−0.14	−0.07
Log HF	0.04	0.31	−0.15	−0.19	0.29	−0.14	−0.20
LF/HF ratio	0.01	−0.49**	0.13	0.20	−0.53**	0.03	0.16
ΔVO2 (exercise)	0.42*	0.17	0.01	−0.05	0.19	0.10	0.12
Resting systolic BP	0.24	−0.36	0.10	0.17	−0.40*	0.13	0.19
Resting diastolic BP	0.20	−0.17	0.35	0.33	−0.23	0.19	0.28
ΔSystolic BP (exercise)	–	−0.36	0.33	0.39*	−0.40*	0.40*	0.49**

Notes: *p<0.05; **p≤0.01.
Abbreviations: SD, standard deviation; CV, coefficient of variation; VO_2, oxygen uptake; LF, low frequency power; HF, high frequency power; BP, blood pressure.

[0.24, 0.77]; both p<0.001) and sleep duration CV (7-day: r = 0.69 [0.44, 0.84]; excluding weekend: r = 0.60 [0.31, 0.79]; both p<0.001) indicating that later chronotypes had higher sleep duration irregularity. The ΔSBP from rest to exercise was positively correlated with sleep duration CV when analyzed across all 7-days (r = 0.39 [0.02, 0.66], p=0.03) and when excluding weekend nights (r = 0.49 [0.15, 0.72], p=0.004). Sleep duration SD was only correlated with the ΔSBP from rest to exercise when excluding weekend nights (r = 0.40 [0.04, 0.67], p=0.02). Adjusting for all potential covariates using partial correlations strengthened the association between sleep duration regularity and the ΔSBP from rest to exercise for sleep duration SD (7-day: adjusted r = 0.50 [0.16, 0.73]; excluding weekend: adjusted r = 0.55 [0.23, 0.76]; both p<0.01) and sleep duration CV (7-day: adjusted r = 0.58 [0.27, 0.78]; excluding weekend: adjusted r = 0.62 [0.34, 0.80]; both p<0.001). Figure 1 shows the relationship between sleep duration CV and the ΔSBP from rest to exercise after adjusting for covariates.

To determine whether the correlation between sleep regularity and exercise BP was independent of sleep duration, partial correlations were used to adjust average sleep duration. Table 3 shows that sleep duration CV when excluding weekend nights remained significantly associated with the ΔSBP from rest to exercise after adjusting for average sleep duration (r^2 = 0.13, p=0.04). Interestingly, average sleep duration was no longer significantly correlated (p>0.05) with the ΔSBP from rest to exercise after adjusting for sleep duration regularity (Table 3).

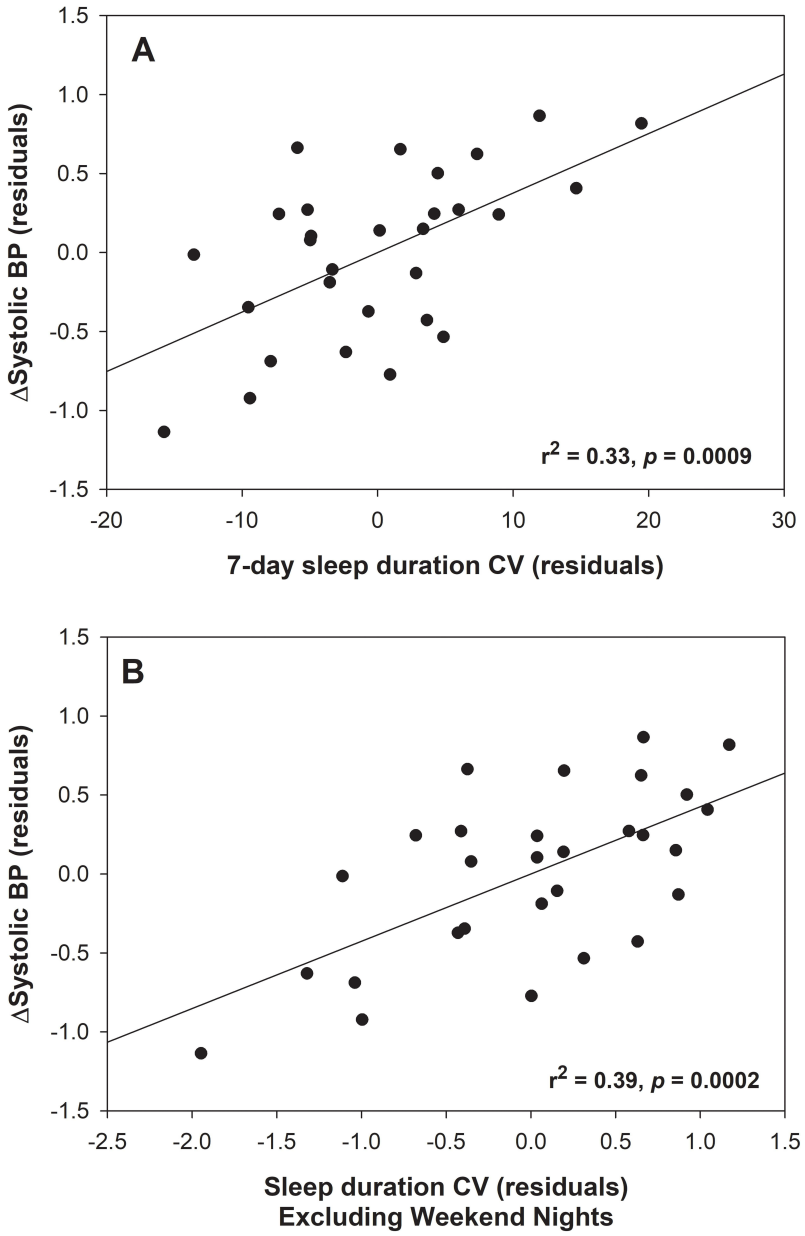

Figure 1 Scatter plot showing the relationship between sleep duration coefficient of variation (CV) and the change (Δ) in systolic BP from rest to moderate-intensity cycling in young adults. Panel A shows sleep duration CV across seven days. Panel B shows sleep duration CV for weekdays excluding Friday and Saturday (weekend) nights. Values are residuals after the adjusting for age, sex, body mass index, daily steps, peak VO_2, chronotype, resting systolic BP, and the change in VO_2 from baseline cycling at 20W to 95% of the gas exchange threshold using partial correlations.

Discussion

The present study sought to determine whether a relationship exist between sleep duration regularity and the rise in BP during exercise. The main finding in this study was that greater irregularity in nightly sleep duration held a relationship of medium magnitude with higher systolic BP responses to exercise. This finding was independent of the average sleep duration when regularity was calculated as sleep duration CV after excluding weekend nights. More importantly, adjusting for covariates that may influence exercise BP did not reduce the association between sleep regularity and the rise in BP during exercise. These results support the notion that analyzing for sleep duration regularity may be a useful tool to capture the impact of intermittent nights of insufficient sleep on BP dysregulation.

This study analyzed sleep duration regularity across one full week, and also after excluding weekend nights (Friday and Saturday night). This approach was used because past research finds higher reliability in measuring sleep duration using wrist

Table 3 Partial Correlation Coefficients with 95% Confidence Intervals

	Entire Week (7-days)			Excluding Weekend Nights		
	Sleep Duration†	Sleep Duration SD	Sleep Duration CV	Sleep Duration‡	Sleep Duration SD	Sleep Duration CV
adjusted for average sleep duration						
ΔSystolic BP (exercise)		0.22 [−0.15, 0.54]	0.23 [−0.14, 0.55]		0.30 [−0.06, 0.60]	0.36* [0.01, 0.64]
adjusted for sleep duration SD						
ΔSystolic BP (exercise)	−0.26 [−0.57, 0.11]			−0.30 [−0.60, 0.07]		
adjusted for sleep duration CV						
ΔSystolic BP (exercise)	−0.17 [−0.51, 0.20]			−0.20 [−0.53, 0.17]		

Note: †7-day average sleep duration; ‡average sleep duration excluding weekend nights. *$p < 0.05$.
Abbreviations: SD, standard deviation; CV, coefficient of variation; BP, blood pressure.

actigraphy when excluding weekend nights.[30] We included Sunday night in our analysis because Monday mornings would have a similar wake time requirement than other weekdays in our study population that consisted entirely of college students. Interestingly, average sleep duration and sleep duration regularity were similar whether analyzed across all seven days or when excluding weekend nights. However, our results clearly demonstrate that these two approaches to analyze sleep regularity makes a large difference in the strength of association between sleep regularity and exercise BP. First, we observed sleep duration CV to have slightly larger correlation coefficients than sleep duration SD in our bivariate correlation analysis, although both are considered of medium magnitude. Second, we showed that using the approach of excluding weekend nights when analyzing for sleep duration regularity resulted in stronger associations with exercise BP than including weekend nights. In fact, sleep duration CV when excluding weekend nights held an independent relationship with the rise in BP during exercise after adjusting for average sleep duration. These observations add new insight into the investigation of sleep duration regularity on BP, and provides strong support for excluding weekend nights when calculating sleep duration regularity in young adults.

Past studies have recognized that average sleep duration and sleep duration regularity are two separate constructs. Indeed, similar average sleep durations have been reported between young adults with higher and lower sleep duration irregularity.[7] Even in the present study we observe heart rate variability (LF/HF ratio) to correlate with average sleep duration, but not sleep duration regularity demonstrating that these sleep health variables carry distinct information. However, one must recognize that a possible negative impact of having irregular nightly sleep durations may be a lower average sleep duration. The Multi-Ethnic Study of Atherosclerosis reported that participants with irregular sleep schedules and insufficient sleep averaged ~70 minutes lower average sleep duration than adults with regular sleep schedules and optimal sleep.[31] Thus, these two measures of sleep health are likely to co-occur, particularly in adults with short sleep durations (<7 hours per night) like those examined in the present study that had an average sleep duration of 6 hours per night. It is therefore theoretically possible that we would remove a large amount of variance in exercise BP explained by sleep duration regularity if we were to add average sleep duration to the full list of covariates in our partial correlational analysis. Instead, we chose to control for variance explained by average sleep duration and sleep duration regularity in a separate analysis to determine which variable held a significant correlation with exercise BP independent of the other. Interestingly, we find that sleep duration CV when excluding weekend nights was the only variable to remain significantly correlated with the rise in BP during exercise. Moreover, the 95% CI for this relationship did not overlap positive and negative values suggesting that sleep duration CV held a clear association with exercise BP independent of average sleep duration.

The physiological mechanisms explaining our results are unclear from the present study. Heart rate variability was not associated with sleep duration regularity suggesting that autonomic dysregulation was not likely a contributing factor in the association between sleep duration irregularity and elevated BP responses to exercise observed in this study. We originally postulated that a person with higher sleep duration irregularity would suffer from insufficient sleep, and as a result, exhibit BP

dysregulation stemming from sympathetic hyperactivity.[4–6] However, our index of sympathetic modulations of the heart (LF/HF ratio) was not associated with sleep duration regularity or the rise in systolic BP during exercise. Other possible mechanisms, unexamined in this study, include baroreceptor sensitivity and peripheral vascular dysfunction. Zhong et al reported decreased baroreceptor sensitivity following acute sleep restriction in young adults,[5] which is relevant to the present study if irregular nights of sleep consisted of short sleep durations. Impaired baroreceptor sensitivity is associated with elevated BP responses to exercise, even in adults with normal resting BP.[32] Young adults with high sleep duration irregularity are also reported to have blunted leg blood flow responses to passive leg movement suggesting impaired microvascular vasodilation.[7] Peripheral vascular dysfunction could impair the regulation of vascular tone during exercise resulting in elevated BP. These possible mechanisms warrant further attention in future work.

Limitations of this study include the population. We recruited young adults, all of which were college students. Sleep health in this population is poor due to academic, financial, and social pressure. Indeed, perceived stress is reported to explain ~25% of the variance in sleep quality in college students.[33] Thus, the generalizability of the present study to older aged populations with different sets of life demands and levels of perceived stress may be minimal. A second limitation of this study is that testing took place in the morning for all participants irrespective of chronotype. It is possible that our morning testing times contributed to an increase in sleep duration irregularity for later chronotypes that typically sleep later than our testing schedule permitted. However, if this did occur, it was only for one night of sleep in our analysis (ie, the night prior to the second study visit) since sleep monitoring started after the first study visit was complete. A third limitation of this work is that we did not account for dietary habits, assess hydration status, or collect measures of psychological stress that are well known to influence BP and BP reactivity. Lastly, we did not measure BP across multiple intensities of exercise in this study. It would be interesting to know whether sleep duration regularity is associated with exercise BP for higher intensities of exercise which are more often performed by athletes or adults undergoing exercise training.

Conclusion

In summary, results from the present study show higher sleep duration irregularity to associate with an elevated BP responses to moderate-intensity cycling exercise in young inactive adults. The observed relationship was independent of confounding variables known to influence exercise BP as well as average sleep duration. These results support the notion that an irregular sleep pattern has the potential to contribute to BP dysregulation during exercise.

Abbreviations

BMI, body mass index; BP, blood pressure; GET, gas exchange threshold; CV, coefficient of variation; HF, high-frequency power; LF, low-frequency power; midpoint of sleep on weekend days corrected for sleep debt on weekdays (MSFsc); SD, standard deviation; SBP, systolic blood pressure; VO2, oxygen uptake.

Data Sharing Statement

Data analyzed for this are available from the corresponding author upon reasonable request.

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Disclosure

The authors report no conflicts of interest in this work.

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