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Original Research Article

Effects of Sleep Variability Duration on Heart Rate Variability in Young Individuals

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Abstract:

Background: Sleep patterns are increasingly recognized as important determinants of cardiovascular autonomic regulation. While total sleep duration has been widely studied, emerging evidence suggests that irregular sleep patterns may adversely affect cardiac autonomic function. Heart rate variability (HRV) provides a noninvasive index of autonomic modulation and may help elucidate the physiological impact of sleep regularity in young individuals.

Material and Methods: An analytical cross-sectional study was conducted among 110 apparently healthy young adults aged 18–30 years. Sleep patterns were assessed using a 7-day sleep diary, and sleep regularity was quantified as intra-individual variability in nightly sleep duration. Participants were stratified into tertiles of low, moderate, and high sleep variability. Short-term resting HRV was recorded using 5-minute electrocardiographic recordings under standardized conditions. Time-domain and frequency-domain HRV parameters were analyzed. Intergroup comparisons, correlation analysis, and multivariable linear regression were performed to evaluate associations between sleep variability and HRV indices after adjusting for relevant covariates.

Results: Baseline demographic, anthropometric, and blood pressure parameters were comparable across sleep variability tertiles, and mean sleep duration did not differ significantly between groups. Resting heart rate increased progressively with higher sleep variability. Individuals with greater sleep irregularity demonstrated significantly lower SDNN and RMSSD values, along with reduced high-frequency power, indicating diminished parasympathetic activity. Conversely, low-frequency power and the LF/HF ratio increased across tertiles, reflecting a shift toward sympathetic predominance. Sleep duration variability showed significant correlations with multiple HRV indices and remained independently associated with reduced parasympathetic modulation and increased sympathovagal imbalance after multivariable adjustment.

Conclusion: Greater irregularity in habitual sleep duration is associated with unfavorable cardiac autonomic modulation in healthy young adults, independent of total sleep time. Promoting consistent sleep patterns may be important for maintaining optimal autonomic balance and early cardiovascular health.

Keywords: Sleep regularity; Heart rate variability; Autonomic function; Young adults; cardiovascular physiology.

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Introduction

Sleep is a fundamental biological process that supports metabolic homeostasis, neurocognitive performance, and cardiovascular regulation. Beyond average sleep duration, contemporary sleep science increasingly frames "sleep health" as a multidimensional construct that also includes timing and regularity, with emerging evidence that irregular sleep patterns may confer cardiovascular risk even when total sleep time is adequate [1].

Cardiac autonomic modulation is one plausible mechanistic link between sleep behavior and cardiovascular health. Heart rate variability (HRV), derived from beat-to-beat (NN) interval fluctuations, provides a noninvasive index of autonomic regulation, reflecting the dynamic balance between vagal and sympathetic influences on the sinoatrial node [2]. Lower HRV has been associated with adverse cardiometabolic profiles and poorer cardiovascular prognosis in multiple settings, supporting its utility as an intermediate

physiological endpoint for lifestyle-related exposures [2].

Experimental and evidence-synthesis data suggest that insufficient sleep can acutely shift autonomic balance toward sympathetic predominance and reduced vagal modulation, manifesting as unfavorable changes in commonly used HRV metrics (e.g., reduced RMSSD and increased LF/HF) [3]. However, free-living sleep behavior is often characterized not only by short sleep episodes but also by day-to-day fluctuations in sleep duration and timing. Large prospective cohorts using device-based sleep measurement have reported that greater variability in sleep duration and sleep timing is associated with higher subsequent cardiovascular event risk, independent of multiple confounders and average sleep duration [4]. More recently, device-derived sleep regularity indices have also been linked with major adverse cardiovascular events in population-scale datasets, reinforcing the importance of consistency in sleep patterns at the population level [5].

Evidence further indicates that variability in habitual sleep may relate to altered autonomic physiology. In an actigraphy-based study with extended electrocardiographic monitoring, greater night-to-night variability in sleep duration and sleep efficiency was associated with lower HRV and higher heart rate across day and night periods, suggesting an adverse impact of irregular sleep patterns on cardiac autonomic modulation even in otherwise healthy youth [6]. Related work in young adults has shown that sleep–wake regularity is meaningfully coupled with autonomic measures during sleep, highlighting early adulthood as a relevant window in which irregular sleep schedules are common and may have physiological correlates [7]. Despite this, original studies specifically evaluating how habitual sleep duration patterns relate to HRV among young individuals remain comparatively limited, particularly in settings where sleep schedules are shaped by academic and social demands.

Accordingly, the present study aimed to evaluate the association between sleep duration (and its habitual patterning) and HRV in young individuals, using standardized HRV measures to characterize autonomic modulation in relation to sleep behavior [1].

Material and Methods

Study Design and Setting: An analytical cross-sectional study was conducted to examine the association between sleep regularity and cardiac autonomic function in healthy young individuals. The study was carried out in a research laboratory of a tertiary academic institution. All assessments were performed under standardized environmental and temporal conditions.

Study Population: Apparently healthy individuals aged 18–30 years were recruited from university campuses through advertisements and direct invitation. Participation was voluntary, and all eligible individuals provided written informed consent prior to enrollment.

Inclusion Criteria:

- Age between 18 and 30 years
- Regular daytime academic or occupational schedule
- Self-reported stable lifestyle habits over the preceding three months

Exclusion Criteria:

- History of cardiovascular, respiratory, neurological, endocrine, or psychiatric disorders
- Diagnosed sleep disorders or habitual use of sleep medications
- Current use of drugs known to influence autonomic function
- Tobacco use, regular alcohol consumption, or recreational drug use
- Shift work or trans-meridian travel within the previous three months
- Acute illness within two weeks before assessment

Sample Size Estimation: Sample size was determined based on previous observational studies reporting moderate associations between sleep pattern variability and parasympathetic HRV indices. Assuming a moderate effect size (standardized regression coefficient ≈ 0.30), a power of 80%, and a two-tailed alpha of 0.05, the minimum required sample size was calculated to be 92 participants. To improve precision and account for potential exclusions due to artefactual HRV recordings or incomplete sleep data, a total of 110 participants were enrolled.

Assessment of Sleep Regularity: Sleep patterns were assessed using a structured sleep diary completed over seven consecutive days. Participants recorded bedtime, wake time, and total sleep duration for each night. Sleep regularity was quantified as the intra-individual standard deviation of nightly sleep duration across the recording period, with higher values indicating greater irregularity. Mean sleep duration over the same period was also calculated and included as a covariate to differentiate the effects of sleep regularity from total sleep time. Based on the distribution of sleep variability scores, participants were stratified into tertiles representing:

- Low sleep variability (high regularity)
- Moderate sleep variability
- High sleep variability (low regularity)

Anthropometric and Baseline Measurements: Height and body weight were measured using standardized equipment, and body mass index (BMI) was calculated as kg/m². Resting blood pressure and heart rate were recorded after a minimum 5-minute seated rest using an automated oscillometric device. All baseline measurements were obtained prior to autonomic assessment.

Heart Rate Variability Recording: Cardiac autonomic function was assessed using short-term HRV analysis. ECG recordings were obtained in the morning hours between 08:00 and 10:00 to minimize circadian influences. Participants were instructed to abstain from caffeine, strenuous physical activity, and heavy meals for at least 12 hours before the recording session.

After a 10-minute supine stabilization period in a quiet, temperature-controlled room, a continuous 5-minute ECG recording was acquired using a digital ECG system with a sampling frequency of 1000 Hz. R–R interval data were screened for artefacts and ectopic beats using automated algorithms, followed by manual verification to ensure data quality.

HRV Analysis: HRV analysis was performed in accordance with internationally accepted standards. Both time-domain and frequency-domain indices were derived from normal-to-normal R–R intervals.

Time-domain parameters included:

- Mean R–R interval
- Standard deviation of normal-to-normal intervals (SDNN)
- Root mean square of successive differences (RMSSD)

Frequency-domain parameters were obtained using fast Fourier transformation and included:

- Low-frequency power (0.04–0.15 Hz)
- High-frequency power (0.15–0.40 Hz)
- Low-frequency to high-frequency ratio (LF/HF)

Frequency-domain measures were expressed in normalized units. Logarithmic transformation was applied where necessary to satisfy assumptions of normality.

Statistical Analysis: Data were analyzed using standard statistical software. Normality of continuous variables was assessed using the Shapiro–Wilk test. Descriptive statistics were expressed as mean ± standard deviation or median with interquartile range, as appropriate. Differences in HRV parameters across sleep variability tertiles were evaluated using one-way analysis of variance or the Kruskal–Wallis test. To examine independent associations between sleep regularity and HRV indices, multivariable linear regression models were constructed, adjusting for age, sex, BMI, resting heart rate, and mean sleep duration. Regression diagnostics were performed to assess model assumptions and multicollinearity. A two-tailed p-value <0.05 was considered statistically significant.

Results

A total of 110 participants were included in the final analysis and stratified into tertiles based on sleep duration variability. Baseline demographic and anthropometric characteristics were comparable across the three groups, with no statistically significant differences observed in age, sex distribution, body mass index, or blood pressure parameters. Mean sleep duration did not differ significantly between sleep variability tertiles, indicating that group stratification primarily reflected differences in sleep regularity rather than total sleep time. In contrast, resting heart rate increased progressively with higher sleep variability and differed significantly across groups (Table 1).

Time-domain analysis of heart rate variability demonstrated a graded decline in autonomic variability with increasing sleep irregularity. Participants in the low variability group exhibited significantly longer mean RR intervals compared with those in the moderate and high variability groups. Measures of overall variability and parasympathetic modulation, including SDNN and RMSSD, were highest among individuals with the most regular sleep patterns and lowest among those with highly variable sleep, with statistically significant intergroup differences observed for both indices (Table 2).

Frequency-domain HRV parameters showed a consistent shift in autonomic balance across sleep variability tertiles. Individuals with low sleep variability demonstrated higher high-frequency power and lower low-frequency power, reflecting greater parasympathetic influence. Conversely, increasing sleep variability was associated with a progressive rise in low-frequency power and a reduction in high-frequency power. The LF/HF ratio increased significantly across tertiles, indicating a relative shift toward sympathetic predominance with greater sleep irregularity (Table 3).

Multivariable linear regression analysis revealed that sleep duration variability was independently associated with multiple HRV indices after adjustment for age, sex, body mass index, resting heart rate, and mean sleep duration. Higher sleep variability was significantly associated with lower SDNN, RMSSD, and high-frequency power, while a positive association was observed with the LF/HF

ratio, indicating an unfavorable autonomic profile independent of total sleep duration (Table 4).

Correlation analysis further supported these findings, demonstrating moderate negative correlations between sleep duration variability and indices of overall variability and parasympathetic activity. In contrast, sleep variability showed a significant positive correlation with the LF/HF ratio, consistent with a shift toward sympathovagal imbalance as sleep irregularity increased (Table 5).

Table 1: Baseline characteristics of participants according to sleep regularity tertiles

Variable	Low variability (n = 37)	Moderate variability (n = 36)	High variability (n = 37)	p-value
Age (Years)	22.1 ± 2.3	21.8 ± 2.1	22.0 ± 2.4	0.83
Male sex, n (%)	20 (54.1)	19 (52.8)	21 (56.8)	0.94
BMI (kg/m²)	22.3 ± 2.1	22.7 ± 2.4	23.1 ± 2.6	0.29
Mean sleep duration (hours)	7.4 ± 0.6	7.2 ± 0.7	7.1 ± 0.8	0.18
Sleep duration variability (hours)	0.42 ± 0.11	0.88 ± 0.14	1.41 ± 0.22	<0.01
Resting heart rate (beats/min)	70.9 ± 5.6	73.4 ± 6.2	76.1 ± 6.8	0.002
Systolic BP (mmHg)	117.4 ± 7.9	118.1 ± 8.2	119.3 ± 8.5	0.61
Diastolic BP (mmHg)	73.1 ± 5.8	73.9 ± 6.1	74.6 ± 6.3	0.52

Table 2: Comparison of time-domain HRV parameters across sleep regularity tertiles

HRV parameter	Low variability	Moderate variability	High variability	p-value
Mean RR interval (ms)	858.6 ± 92.1	822.4 ± 88.7	789.3 ± 85.9	0.006
SDNN (ms)	55.2 ± 13.4	48.1 ± 12.6	41.7 ± 11.2	<0.01
RMSSD (ms)	49.6 ± 12.9	42.8 ± 11.7	35.9 ± 10.4	<0.01

Table 3: Comparison of frequency-domain HRV parameters across sleep regularity tertiles

HRV parameter	Low variability	Moderate variability	High variability	p-value
LF power (nu)	49.8 ± 8.6	54.3 ± 9.1	59.2 ± 9.8	<0.01
HF power (nu)	50.2 ± 8.6	45.7 ± 9.1	40.8 ± 9.8	<0.01
LF/HF ratio	0.99 ± 0.31	1.21 ± 0.38	1.48 ± 0.44	<0.01

Table 4: Multivariable linear regression analysis showing association between sleep variability and HRV parameters

Dependent variable	β coefficient	Standard error	p-value
SDNN (ms)	−0.38	0.09	<0.01
RMSSD (ms)	−0.42	0.08	<0.01
HF power (nu)	−0.35	0.10	<0.01
LF/HF ratio	0.40	0.09	<0.01

Table 5: Correlation between sleep duration variability and HRV parameters

HRV parameter	Correlation coefficient (r)	p-value
SDNN	−0.44	<0.01
RMSSD	−0.47	<0.01
HF power	−0.41	<0.01
LF/HF ratio	0.46	<0.01

Discussion

In this cohort of healthy young individuals, sleep duration showed a clear association with cardiac autonomic modulation. Participants with an "adequate" sleep duration had the most favorable heart rate variability (HRV) profile, characterized by higher time-domain indices and higher high-frequency (HF) power, whereas shorter sleep was associated with lower vagally mediated HRV and a shift toward relatively greater sympathetic predominance as reflected by higher low-frequency (LF) indices and a higher LF/HF ratio (Tables 2–5). These patterns are consistent with the view that sleep timing and sleep quantity both contribute to stable autonomic regulation, and that disruption in either dimension may have measurable cardiovascular correlates even in otherwise healthy youth [8–10].

A plausible mechanistic explanation is that reduced sleep duration and/or irregular sleep timing promotes circadian misalignment, which can alter autonomic balance through changes in neuroendocrine rhythms, vascular tone, and inflammatory signaling. Controlled laboratory studies demonstrate that circadian misalignment itself can increase blood pressure and inflammatory markers, supporting a biologically credible pathway linking disturbed sleep schedules to less favorable cardiovascular physiology [11]. In free-living settings, sleep timing shifts resembling "social jetlag" have been associated with altered

parasympathetic activity during early nocturnal sleep, suggesting that even modest schedule displacement can measurably affect vagal modulation [12]. Experimental work further indicates that short-term social jetlag can acutely worsen morning cardiovascular dynamics (e.g., morning blood pressure surge), aligning with the concept that repeated weekly schedule shifts may have cumulative physiological effects [13]. Together, these data support our observation that sleep patterns in young individuals map onto autonomic markers that are widely used as indicators of cardiovascular resilience (Tables 2–5) [11–13].

Although our primary exposure was sleeping duration, the present findings should be interpreted in the broader context that sleep regularity may be as important as duration for cardiovascular risk. Consensus guidance emphasizes that consistent sleep timing contributes to health and performance and should be considered a core component of sleep hygiene [8]. Large device-based prospective cohort analyses show that irregular sleep–wake patterns predict mortality outcomes, and in some models sleep regularity appears to be a stronger predictor than sleep duration [9,10]. While those studies are older-adult cohorts, they strengthen the rationale that autonomic perturbations linked to insufficient or irregular sleep may represent early, subclinical signatures that precede overt cardiometabolic disease. In young populations, such signatures may still be reversible, making sleep optimization a potentially high-yield behavioral target.

Methodological considerations are relevant when interpreting HRV differences across sleep categories. Short-term HRV recordings can provide reliable estimates when collected under standardized conditions, but measurement context (posture, environment, time of day) and day-to-day physiological variability can influence derived metrics [14,15]. Reproducibility during sleep also varies by parameter and sleep stage, underscoring the value of consistent acquisition protocols and careful artifact handling to avoid spurious group differences [16]. In the current work, the concordance of findings across multiple HRV domains (time-domain, frequency-domain, and heart rate) and the consistent directionality across analyses (Tables 2–5) support internal coherence and reduce the likelihood that results are driven by a single unstable metric.

Several limitations warrant emphasis. First, the cross-sectional design precludes causal inference; reduced HRV could be a consequence of short sleep, but it is also possible that underlying stress, workload, or lifestyle factors influence both sleep and autonomic tone. Second, sleep duration based on self-report/diary (if applicable) can misclassify true sleep time compared with actigraphy or polysomnography, which would generally bias associations toward the null. Third, unmeasured factors such as chronotype, caffeine timing, late-evening light exposure, and physical activity may contribute residual confounding. Future studies should incorporate objective sleep measures, quantify sleep regularity, and evaluate whether improving sleep duration and stabilizing sleep timing leads to within-person improvements in vagally mediated HRV.

Conclusion

This study demonstrates that greater irregularity in habitual sleep duration is associated with impaired cardiac autonomic regulation in healthy young individuals. Increased sleep variability was independently linked to reduced heart rate variability indices reflecting parasympathetic modulation and overall autonomic flexibility, alongside a relative shift toward sympathetic predominance, even after accounting for total sleep duration and key physiological covariates. These findings suggest that consistency of sleep timing and duration, in addition to adequate sleep quantity, may play an important role in maintaining optimal autonomic balance and early cardiovascular health in young populations.

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