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PROSPECTIVE EVALUATION OF SLEEP REGULARITY AND ITS EFFECT ON HEART RATE VARIABILITY AND RESTING CARDIOMETABOLIC PARAMETERS IN HEALTHY YOUNG ADULTS

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ABSTRACT

Background: Sleep regularity is an important component of sleep health and reflects the consistency of sleep and wake timing across days. Irregular sleep patterns may disturb circadian rhythm, alter autonomic balance, and influence early cardiometabolic risk. Heart rate variability is a useful non-invasive marker of autonomic function, while resting cardiometabolic parameters provide insight into cardiovascular and metabolic health status. Evaluation of these parameters in healthy young adults may help identify early physiological changes associated with irregular sleep behavior. **Aim:** To prospectively evaluate sleep regularity and assess its effect on heart rate variability and resting cardiometabolic parameters in healthy young adults.

Materials and Methods: This prospective observational study was conducted in a tertiary care hospital among 105 healthy young adults. Participants were categorized into a regular sleep group (n = 56) and an irregular sleep group (n = 49) based on sleep timing variability. Sleep parameters including average sleep duration, bedtime variability, wake-up time variability, sleep latency, nocturnal awakenings, sleep efficiency, and social jetlag were assessed. Heart rate variability parameters including resting heart rate, mean RR interval, SDNN, RMSSD, pNN50, LF power, HF power, LF/HF ratio, and total power were recorded under resting conditions. Resting cardiometabolic parameters including blood pressure, fasting blood glucose, lipid profile, body mass index, waist circumference, and waist–hip ratio were measured using standard procedures. Data were analyzed using IBM SPSS Statistics version 27.0, and $p < 0.05$ was considered statistically significant.

Results: Irregular sleepers had significantly higher body weight, BMI, waist circumference, and waist–hip ratio compared with regular sleepers. Regular sleepers had longer average sleep duration, lower bedtime and wake-up time variability, shorter sleep latency, fewer nocturnal awakenings, higher sleep efficiency, and lower social jetlag ($p < 0.001$). HRV parameters were significantly better in the regular sleep group, with higher SDNN, RMSSD, pNN50, HF power, and total power, while irregular sleepers showed higher resting heart rate, LF power, and LF/HF ratio. Irregular sleepers also had significantly higher systolic and diastolic blood pressure, fasting blood glucose, total cholesterol, triglycerides, and LDL cholesterol, with lower HDL cholesterol. Sleep regularity showed significant positive correlations with SDNN, RMSSD, HF power, and HDL cholesterol, and negative correlations

with LF/HF ratio, resting heart rate, BMI, systolic blood pressure, fasting blood glucose, and triglycerides.

Conclusion: Regular sleep patterns were associated with better autonomic function and a healthier cardiometabolic profile in young adults. Sleep regularity may serve as an important early marker of cardiovascular and metabolic health.

Keywords: Sleep regularity; Heart rate variability; Cardiometabolic parameters; Young adults; Circadian rhythm.

INTRODUCTION

Sleep is a fundamental biological process essential for restoration, neuroendocrine regulation, metabolic homeostasis, and cardiovascular stability. In recent years, sleep has been increasingly recognized not merely as a passive state of rest but as an active physiological condition that influences multiple systems involved in long-term health. Traditionally, sleep research and clinical assessment have emphasized sleep duration and sleep quality; however, growing attention is now being directed toward sleep regularity, which refers to the consistency of sleep and wake timing from day to day. Regular sleep timing helps maintain circadian alignment, supports stable autonomic regulation, and contributes to synchronized metabolic and cardiovascular function. In young adults, who often experience academic stress, social commitments, digital exposure, variable meal timing, and inconsistent daily routines, sleep regularity may be especially vulnerable to disruption. The American Heart Association has also recognized sleep health as an important component of cardiovascular health, highlighting its relevance along with body weight, blood pressure, glucose, lipids, diet, physical activity, and nicotine exposure.[1] Sleep regularity is different from sleep duration. A person may obtain an apparently adequate number of sleep hours but still have irregular bedtimes and wake-up times. Such variability can disturb the circadian system, which regulates hormonal secretion, autonomic tone, body temperature, blood pressure rhythm, glucose metabolism, and lipid handling. Irregular sleep timing may create a mismatch between internal biological rhythms and external behavioral schedules. This misalignment can affect sympathetic and parasympathetic balance, thereby influencing cardiovascular and metabolic parameters even in individuals without diagnosed disease. The National Sleep Foundation has emphasized the importance of consistent sleep timing as a meaningful dimension of sleep health and has identified sleep timing variability as relevant to health, performance, and daily functioning.[2] Sleep health is now considered a multidimensional construct that includes duration, continuity, timing, regularity, satisfaction, alertness, and absence of sleep disorders. Among these dimensions, sleep regularity has clinical importance because it reflects the stability of daily behavioral rhythms. Young adults often appear healthy during routine evaluation, but early physiological changes may occur before overt cardiometabolic disease develops. Therefore, examining sleep regularity in this age group provides an opportunity to identify subtle alterations in autonomic and cardiometabolic function at an early stage. A multidimensional approach to sleep health is particularly relevant because cardiometabolic outcomes are influenced not only by how long a person sleeps, but also by when sleep occurs and how consistently sleep is maintained across days.[3] Heart rate variability is a non-invasive marker of cardiac autonomic modulation and reflects the dynamic interaction between sympathetic and parasympathetic influences on the heart. Higher HRV generally indicates better autonomic flexibility and stronger vagal modulation, whereas reduced HRV suggests impaired autonomic adaptability and increased cardiovascular vulnerability. Time-domain parameters such as SDNN, RMSSD, and pNN50 provide information about overall variability and parasympathetic activity, while frequency-domain measures such as LF power, HF power, and LF/HF ratio help evaluate sympathovagal balance. Since sleep and circadian rhythm strongly influence autonomic function, HRV provides a useful physiological tool for studying the effect of sleep regularity on cardiovascular regulation.[4] Resting cardiometabolic parameters, including blood pressure, resting heart rate, body mass index, waist circumference, fasting blood glucose, and lipid profile, are commonly used indicators of early cardiovascular and metabolic health. Even small unfavorable shifts in these parameters during young adulthood may contribute to future risk if associated lifestyle patterns persist. Sleep irregularity can influence cardiometabolic health through several pathways, including increased sympathetic activity, altered cortisol rhythm, impaired insulin sensitivity, appetite dysregulation, low-grade inflammation, and changes in physical activity and eating behavior. Assessment of these parameters in healthy young adults can therefore help understand whether irregular sleep patterns are associated with early deviations from optimal physiological status.[5] The relationship between sleep and HRV is biologically plausible because sleep plays an important role in overnight autonomic recovery. During healthy sleep, parasympathetic activity generally increases, heart rate declines, and cardiovascular workload is reduced. Disturbed or irregular sleep may blunt this recovery process and contribute to higher resting heart rate, reduced vagal tone, and altered blood pressure regulation. In young adults, this is particularly important because lifestyle-related

autonomic imbalance may remain clinically silent. Studying HRV alongside sleep regularity may therefore provide a sensitive method to detect early functional changes before structural or symptomatic cardiovascular disease becomes apparent.[6]

MATERIALS AND METHODS

This prospective observational study was conducted in a tertiary care hospital to evaluate sleep regularity and its association with heart rate variability and resting cardiometabolic parameters among healthy young adults. The study was designed to assess sleep pattern consistency in day-to-day life and to examine its relationship with autonomic function and selected cardiometabolic health indicators measured under resting conditions. A total of 105 healthy young adult participants were enrolled in the study. Participants were recruited from the tertiary care hospital setting after screening for eligibility. Only apparently healthy individuals within the young adult age group were included. Participants of both sexes were considered eligible, provided they fulfilled the inclusion criteria and gave written informed consent for participation.

Inclusion Criteria

Healthy young adults willing to participate in the study were included. Participants with no known history of cardiovascular, respiratory, endocrine, neurological, psychiatric, or sleep-related disorders were considered eligible. Individuals with stable daily routines and those able to provide reliable sleep-related information were included. Participants were required to refrain from caffeine, nicotine, alcohol, strenuous exercise, and heavy meals before physiological assessment to minimize acute influences on heart rate variability and cardiometabolic parameters.

Exclusion Criteria

Participants with known hypertension, diabetes mellitus, obesity, thyroid disorders, chronic kidney disease, chronic liver disease, diagnosed sleep disorders, psychiatric illness, or any cardiovascular disease were excluded. Individuals taking medications known to affect sleep, autonomic function, heart rate, blood pressure, or metabolism were not included. Participants with acute illness, fever, recent hospitalization, night-shift work, irregular medication use, smoking, alcohol dependence, or incomplete sleep or physiological data were also excluded from the final analysis.

Methodology

Assessment of Sleep Regularity: Sleep regularity was assessed prospectively using sleep-related records maintained by the participants. Participants were instructed to record their daily bedtime, wake-up time, total sleep duration, sleep latency, and number of nocturnal awakenings. Sleep regularity was evaluated based on the consistency of sleep timing across days, particularly variation in bedtime and wake-up time. Additional sleep parameters such as average sleep duration, sleep efficiency, mid-sleep time, social jetlag, and variability in sleep duration were also assessed. Based on sleep timing variability, participants were categorized into more regular and less regular sleep groups for comparison of physiological and cardiometabolic outcomes.

Heart Rate Variability Assessment: Heart rate variability was recorded under resting conditions in a quiet, temperature-controlled room. Participants were asked to rest comfortably in the supine or seated position before recording. HRV was measured using a standard electrocardiographic or validated heart rate recording system after ensuring minimal external disturbance. Time-domain and frequency-domain HRV parameters were analyzed. Time-domain parameters included mean heart rate, mean RR interval, standard deviation of normal-to-normal intervals, root mean square of successive differences, and percentage of successive RR intervals differing by more than 50 ms. Frequency-domain parameters included low-frequency power, high-frequency power, total power, and the LF/HF ratio. These parameters were used to assess sympathetic and parasympathetic modulation of cardiac autonomic activity.

Resting Cardiometabolic Parameters: Resting cardiometabolic parameters were measured under standardized conditions. Blood pressure was recorded using a calibrated sphygmomanometer or automated blood pressure monitor after adequate rest. Systolic blood pressure, diastolic blood pressure, pulse pressure, mean arterial pressure, and resting heart rate were documented. Anthropometric parameters including height, weight, body mass index, waist circumference, hip circumference, and waist–hip ratio were measured using standard procedures. Biochemical parameters, where applicable, included fasting blood glucose, fasting insulin, lipid profile, total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and very-low-density lipoprotein cholesterol. These parameters were used to assess resting cardiometabolic status in relation to sleep regularity.

Anthropometric Measurements: Height was measured using a stadiometer with the participant standing erect without footwear. Weight was recorded using a calibrated weighing scale with the participant wearing light clothing. Body mass index was calculated as weight in kilograms divided by height in meters squared. Waist circumference was measured midway between the lower margin of the last rib and the iliac crest, while hip circumference was measured at the widest point over the buttocks. Waist–hip ratio was calculated by dividing waist circumference by hip circumference.

Blood Pressure and Resting Heart Rate Measurement: Blood pressure and resting heart rate were measured after the participant had rested quietly. Measurements were taken in a comfortable position with the arm supported at heart level. At least two readings were recorded, and the average

value was used for analysis. Participants were advised to avoid caffeine, exercise, and emotional stress before measurement to reduce variability in resting cardiovascular parameters.

Biochemical Assessment: Venous blood samples were collected under fasting conditions for assessment of metabolic parameters. Fasting blood glucose and lipid profile were estimated using standard laboratory methods. The lipid profile included total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and very-low-density lipoprotein cholesterol. Fasting insulin and insulin resistance indices such as HOMA-IR may also be calculated, where available, to provide additional information regarding cardiometabolic risk.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 27.0. Continuous variables were expressed as mean and standard deviation for normally distributed data, and as median and interquartile range for non-normally distributed data. Categorical variables were expressed as frequency and percentage. Normality of data was assessed using the Shapiro–Wilk test. Independent samples t-test or Mann–Whitney U test was used to compare continuous variables between groups, depending on data distribution. Chi-square test or Fisher's exact test was used for categorical variables. Correlation between sleep regularity indices, HRV parameters, and cardiometabolic variables was assessed using Pearson's or Spearman's correlation coefficient as appropriate. Multiple linear regression analysis was performed to determine the independent association of sleep regularity with HRV and cardiometabolic parameters after adjusting for potential confounders such as age, sex, body mass index, physical activity, and average sleep duration. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 105 healthy young adults were included in the final analysis. Based on sleep timing variability, participants were categorized into Regular Sleep Group (n = 56) and Irregular Sleep Group (n = 49).

Table 1 shows the baseline demographic and anthropometric characteristics of the participants. The mean age of the total study population was 22.84 ± 2.31 years. The regular sleep group had a mean age of 22.61 ± 2.18 years, while the irregular sleep group had a mean age of 23.10 ± 2.45 years. This difference was not statistically significant (p = 0.284), indicating that both groups were comparable with respect to age. Similarly, sex distribution was also comparable between the groups, with males comprising 53.57% of the regular sleep group and 57.14% of the irregular sleep group (p = 0.712). Height was also not significantly different between the two groups (p = 0.503). However, significant differences were observed in several anthropometric parameters. Participants in the irregular sleep group had significantly higher body weight compared with those in the regular sleep group (66.76 ± 10.54 kg vs. 62.11 ± 9.42 kg, p = 0.019). Body mass index was also significantly higher among irregular sleepers (23.91 ± 3.19 kg/m²) compared with regular sleepers (22.04 ± 2.74 kg/m²), with a p-value of 0.002. Waist circumference was significantly greater in the irregular sleep group (84.36 ± 8.52 cm) than in the regular sleep group (78.94 ± 7.86 cm), suggesting greater central adiposity among participants with irregular sleep patterns (p = 0.001). Waist–hip ratio was also significantly higher in the irregular sleep group (0.88 ± 0.06) compared with the regular sleep group (0.85 ± 0.05, p = 0.006). Hip circumference was higher in the irregular sleep group, but the difference did not reach statistical significance (p = 0.054).

Table 2 presents the sleep profile characteristics of the regular and irregular sleep groups. Participants in the regular sleep group had significantly longer average sleep duration compared with those in the irregular sleep group (7.28 ± 0.64 hours vs. 6.54 ± 0.78 hours, p < 0.001). Bedtime variability was markedly lower in the regular sleep group (18.62 ± 6.15 minutes) compared with the irregular sleep group (74.38 ± 18.46 minutes), and this difference was statistically significant (p < 0.001). Similarly, wake-up time variability was significantly lower among regular sleepers (21.44 ± 7.21 minutes) compared with irregular sleepers (69.55 ± 20.12 minutes, p < 0.001). Sleep quality-related parameters were also better among regular sleepers. Sleep latency was significantly shorter in the regular sleep group (14.86 ± 5.32 minutes) compared with the irregular sleep group (24.41 ± 8.56 minutes, p < 0.001), suggesting that regular sleepers were able to fall asleep faster. Nocturnal awakenings were fewer in the regular sleep group (0.84 ± 0.56 per night) than in the irregular sleep group (1.68 ± 0.74 per night, p < 0.001). Sleep efficiency was significantly higher among regular sleepers (91.72 ± 3.64%) compared with irregular sleepers (84.26 ± 5.11%, p < 0.001). Social jetlag was also significantly lower in the regular sleep group (0.48 ± 0.26 hours) compared with the irregular sleep group (1.61 ± 0.58 hours, p < 0.001).

Table 3 compares heart rate variability parameters between the two groups. Resting heart rate was significantly lower in the regular sleep group (68.42 ± 6.14 beats/min) compared with the irregular sleep group (74.58 ± 7.36 beats/min, p < 0.001). This suggests better resting cardiovascular regulation among participants with regular sleep patterns. The mean RR interval was significantly higher in regular sleepers (878.24 ± 74.61 ms) compared with irregular sleepers (806.58 ± 69.84 ms, p < 0.001), which is consistent with the lower resting heart rate observed in the regular sleep group. Time-domain HRV indices were significantly better in the regular sleep group. SDNN, which reflects overall heart rate variability,

was higher among regular sleepers (52.64 ± 10.21 ms) than irregular sleepers (41.32 ± 9.48 ms, p < 0.001). RMSSD, a marker of parasympathetic activity, was also significantly higher in the regular sleep group (44.86 ± 11.18 ms) compared with the irregular sleep group (31.74 ± 8.95 ms, p < 0.001). Similarly, pNN50 was higher among regular sleepers (19.52 ± 6.74%) than irregular sleepers (11.26 ± 5.18%, p < 0.001). Frequency-domain HRV parameters also showed significant differences. LF power was significantly higher in the irregular sleep group (701.22 ± 165.31 ms²) compared with the regular sleep group (612.48 ± 146.84 ms², p = 0.004), suggesting relatively greater sympathetic influence. HF power, which primarily reflects parasympathetic activity, was significantly higher in regular sleepers (548.16 ± 132.54 ms²) than in irregular sleepers (392.48 ± 118.67 ms², p < 0.001). The LF/HF ratio was significantly lower in the regular sleep group (1.24 ± 0.42) compared with the irregular sleep group (1.98 ± 0.56, p < 0.001), indicating a more favorable sympathovagal balance among regular sleepers. Total power was also significantly greater in the regular sleep group (1826.64 ± 384.56 ms²) compared with the irregular sleep group (1498.26 ± 352.81 ms², p < 0.001).

Table 4 shows the comparison of resting cardiometabolic parameters between the regular and irregular sleep groups. Systolic blood pressure was significantly lower in the regular sleep group (114.28 ± 8.46 mmHg) compared with the irregular sleep group (121.54 ± 9.12 mmHg, p < 0.001). Diastolic blood pressure was also significantly lower among regular sleepers (72.18 ± 6.24 mmHg) compared with irregular sleepers (77.62 ± 7.18 mmHg, p < 0.001). Mean arterial pressure followed the same trend, with lower values in the regular sleep group (86.21 ± 5.84 mmHg) compared with the irregular sleep group (92.26 ± 6.36 mmHg, p < 0.001). Metabolic parameters also differed significantly between groups. Fasting blood glucose was significantly lower in the regular sleep group (88.64 ± 7.18 mg/dL) compared with the irregular sleep group (94.48 ± 8.24 mg/dL, p < 0.001). Total cholesterol was significantly higher among irregular sleepers (182.36 ± 27.12 mg/dL) than regular sleepers (166.82 ± 24.46 mg/dL, p = 0.002). Triglyceride levels were also significantly higher in the irregular sleep group (132.62 ± 34.28 mg/dL) compared with the regular sleep group (108.54 ± 28.36 mg/dL, p < 0.001). HDL cholesterol, which is considered protective, was significantly higher among regular sleepers (48.22 ± 6.54 mg/dL) compared with irregular sleepers (42.84 ± 5.92 mg/dL, p < 0.001). LDL cholesterol was significantly lower in the regular sleep group (96.46 ± 18.14 mg/dL) compared with the irregular sleep group (112.38 ± 21.26 mg/dL, p < 0.001).

Table 5 presents the correlation between sleep regularity score and selected HRV and cardiometabolic parameters among all 105 participants. Sleep regularity showed a significant positive correlation with SDNN (r = 0.462, p < 0.001), RMSSD (r = 0.514, p < 0.001), and HF power (r = 0.438, p < 0.001). Sleep regularity was negatively correlated with LF/HF ratio (r = -0.421, p < 0.001) and resting heart rate (r = -0.396, p < 0.001). This suggests that better sleep regularity was associated with lower sympathetic predominance and lower resting heart rate. A significant negative correlation was also observed between sleep regularity and BMI (r = -0.308, p = 0.001), indicating that participants with better sleep regularity tended to have lower body mass index. Among cardiometabolic parameters, sleep regularity showed a significant negative correlation with systolic blood pressure (r = -0.372, p < 0.001), fasting blood glucose (r = -0.281, p = 0.004), and triglycerides (r = -0.336, p < 0.001). These findings suggest that better sleep regularity was associated with lower blood pressure, better glycemic status, and lower triglyceride levels. Sleep regularity showed a significant positive correlation with HDL cholesterol (r = 0.294, p = 0.002), suggesting a favorable association between regular sleep and protective lipid profile.

Table 1: Baseline demographic and anthropometric characteristics of study participants

Variable	Total (n=105)	Regular Sleep (n=56)	Irregular Sleep (n=49)	p-value
Age (years)	22.84 ± 2.31	22.61 ± 2.18	23.10 ± 2.45	0.284
Male, n (%)	58 (55.24%)	30 (53.57%)	28 (57.14%)	0.712
Female, n (%)	47 (44.76%)	26 (46.43%)	21 (42.86%)	0.712
Height (cm)	167.42 ± 8.54	167.96 ± 8.23	166.81 ± 8.92	0.503
Weight (kg)	64.28 ± 10.16	62.11 ± 9.42	66.76 ± 10.54	0.019*
BMI (kg/m²)	22.91 ± 3.08	22.04 ± 2.74	23.91 ± 3.19	0.002*
Waist circumference (cm)	81.47 ± 8.62	78.94 ± 7.86	84.36 ± 8.52	0.001*
Hip circumference (cm)	94.11 ± 7.31	92.84 ± 6.95	95.56 ± 7.48	0.054
Waist-Hip Ratio	0.87 ± 0.06	0.85 ± 0.05	0.88 ± 0.06	0.006*

Statistically significant (p < 0.05)

Table 2: Sleep profile characteristics among study groups

Variable	Regular Sleep (n=56)	Irregular Sleep (n=49)	p-value
Average sleep duration (hours)	7.28 ± 0.64	6.54 ± 0.78	<0.001*
Bedtime variability (minutes)	18.62 ± 6.15	74.38 ± 18.46	<0.001*
Wake-up time variability (minutes)	21.44 ± 7.21	69.55 ± 20.12	<0.001*
Sleep latency (minutes)	14.86 ± 5.32	24.41 ± 8.56	<0.001*
Nocturnal awakenings/night	0.84 ± 0.56	1.68 ± 0.74	<0.001*
Sleep efficiency (%)	91.72 ± 3.64	84.26 ± 5.11	<0.001*
Social jetlag (hours)	0.48 ± 0.26	1.61 ± 0.58	<0.001*

Statistically significant (p < 0.05)

Table 3: Comparison of heart rate variability parameters between study groups

Variable	Regular Sleep (n=56)	Irregular Sleep (n=49)	p-value
Resting heart rate (beats/min)	68.42 ± 6.14	74.58 ± 7.36	<0.001*
Mean RR interval (ms)	878.24 ± 74.61	806.58 ± 69.84	<0.001*
SDNN (ms)	52.64 ± 10.21	41.32 ± 9.48	<0.001*
RMSSD (ms)	44.86 ± 11.18	31.74 ± 8.95	<0.001*
pNN50 (%)	19.52 ± 6.74	11.26 ± 5.18	<0.001*
LF power (ms²)	612.48 ± 146.84	701.22 ± 165.31	0.004*
HF power (ms²)	548.16 ± 132.54	392.48 ± 118.67	<0.001*
LF/HF ratio	1.24 ± 0.42	1.98 ± 0.56	<0.001*
Total power (ms²)	1826.64 ± 384.56	1498.26 ± 352.81	<0.001*

Statistically significant (p < 0.05)

Table 4: Comparison of resting cardiometabolic parameters between study groups

Variable	Regular Sleep (n=56)	Irregular Sleep (n=49)	p-value
Systolic BP (mmHg)	114.28 ± 8.46	121.54 ± 9.12	<0.001*
Diastolic BP (mmHg)	72.18 ± 6.24	77.62 ± 7.18	<0.001*
Mean arterial pressure (mmHg)	86.21 ± 5.84	92.26 ± 6.36	<0.001*
Fasting blood glucose (mg/dL)	88.64 ± 7.18	94.48 ± 8.24	<0.001*
Total cholesterol (mg/dL)	166.82 ± 24.46	182.36 ± 27.12	0.002*
Triglycerides (mg/dL)	108.54 ± 28.36	132.62 ± 34.28	<0.001*
HDL cholesterol (mg/dL)	48.22 ± 6.54	42.84 ± 5.92	<0.001*
LDL cholesterol (mg/dL)	96.46 ± 18.14	112.38 ± 21.26	<0.001*

Statistically significant (p < 0.05)

Table 5: Correlation of sleep regularity score with HRV and cardiometabolic parameters (n=105)

Variable	Correlation coefficient (r)	p-value
SDNN	0.462	<0.001*
RMSSD	0.514	<0.001*
HF power	0.438	<0.001*
LF/HF ratio	-0.421	<0.001*
Resting heart rate	-0.396	<0.001*
BMI	-0.308	0.001*
Systolic BP	-0.372	<0.001*
Fasting blood glucose	-0.281	0.004*
Triglycerides	-0.336	<0.001*
HDL cholesterol	0.294	0.002*

Statistically significant (p < 0.05)

DISCUSSION

In the present study, the two sleep groups were comparable for age, sex distribution, and height, indicating that the observed physiological differences were less likely to be due to baseline demographic imbalance. The mean age was 22.61 ± 2.18 years in the regular sleep group and 23.10 ± 2.45 years in the irregular sleep group, with no significant difference (p = 0.284). Male distribution was also similar between groups, 53.57% in regular sleepers and 57.14% in irregular sleepers (p = 0.712). This comparability is important because sleep timing irregularity itself may influence biological rhythms. Phillips et al. (2017) reported that irregular sleep–wake patterns in university students were associated with delayed circadian timing and poorer sleep–wake organization, supporting the relevance of studying sleep regularity in young adults rather than focusing only on sleep duration.[7]

The present study observed significantly higher anthropometric indices among irregular sleepers. Body weight was higher in the irregular sleep group than the regular sleep group (66.76 ± 10.54 kg vs. 62.11 ± 9.42 kg, p = 0.019), and BMI was also higher among irregular sleepers (23.91 ± 3.19 kg/m² vs. 22.04 ± 2.74 kg/m², p = 0.002). Waist circumference was greater in irregular sleepers (84.36 ± 8.52 cm vs. 78.94 ± 7.86 cm, p = 0.001), indicating greater central adiposity. These findings are comparable with Wong et al. (2022), who studied first-year college students and reported an average Sleep Regularity Index of 74.1 ± 8.7 and mean BMI of 22.0 ± 3.5 kg/m²; lower sleep regularity was significantly associated with greater baseline BMI (B = -0.06, 95% CI: -0.09 to -0.02, p = 0.001).[8]

In this study, irregular sleepers also had higher waist–hip ratio than regular sleepers (0.88 ± 0.06 vs. 0.85 ± 0.05, p = 0.006), while hip circumference showed a non-significant trend toward higher values in the irregular sleep group (95.56 ± 7.48 cm vs. 92.84 ± 6.95 cm, p = 0.054). These results suggest that irregular sleep may be associated more strongly with central fat distribution than with generalized body size alone. Huang et al. (2019), in the Multi-Ethnic Study of Atherosclerosis involving 2,003 participants followed for a median of 6.3 years, reported that each one-hour increase in variability in bedtime or sleep duration was associated with up to a 27% greater chance of metabolic abnormality, including higher waist circumference, higher triglycerides, higher fasting glucose, and lower HDL cholesterol.[9]

Sleep profile differences in the present study were marked and statistically significant. Regular sleepers had longer average sleep duration than irregular sleepers (7.28 ± 0.64 hours vs. 6.54 ± 0.78 hours, p <

0.001), much lower bedtime variability (18.62 ± 6.15 minutes vs. 74.38 ± 18.46 minutes, p < 0.001), and lower wake-up time variability (21.44 ± 7.21 minutes vs. 69.55 ± 20.12 minutes, p < 0.001). Sleep latency was also shorter, nocturnal awakenings were fewer, sleep efficiency was higher, and social jetlag was lower among regular sleepers. Sűdy et al. (2019), in 33 healthy young men aged 20–26 years, found that social jetlag was associated with altered sleep-related autonomic regulation; differences in pNN50, RMSSD, and HF power were significant in relation to workday–free day sleep timing shifts, supporting the present finding that social jetlag of 1.61 ± 0.58 hours in irregular sleepers may have physiological relevance.[10]

Heart rate variability findings in the present study showed better autonomic profile among regular sleepers. SDNN was higher in regular sleepers than irregular sleepers (52.64 ± 10.21 ms vs. 41.32 ± 9.48 ms, p < 0.001), RMSSD was also higher (44.86 ± 11.18 ms vs. 31.74 ± 8.95 ms, p < 0.001), and pNN50 was greater (19.52 ± 6.74% vs. 11.26 ± 5.18%, p < 0.001). These differences indicate higher overall HRV and better parasympathetic modulation among regular sleepers. Bolin et al. (2022), in young adults with mean age 22.7 ± 4.3 years, reported that improvement in HRV was clinically relevant, with significant increases in SDNN (p = 0.037) and total power (p = 0.026), and also found a significant correlation between diastolic blood pressure and high-frequency HRV (r = 0.41, p < 0.05).[11]

Frequency-domain HRV parameters in this study further supported autonomic imbalance among irregular sleepers. HF power was significantly higher in regular sleepers (548.16 ± 132.54 ms²) than irregular sleepers (392.48 ± 118.67 ms², p < 0.001), whereas LF power was higher in irregular sleepers (701.22 ± 165.31 ms² vs. 612.48 ± 146.84 ms², p = 0.004). The LF/HF ratio was also significantly higher among irregular sleepers (1.98 ± 0.56 vs. 1.24 ± 0.42, p < 0.001), suggesting relative sympathetic predominance. Scott et al. (2023), in a large global sample of 12,287 adults with more than two million nights of sleep data, found that irregular sleep timing was associated with hypertension; regular variation in bedtime by more than about 30 minutes was associated with a 32% higher hypertension risk, supporting the link between sleep timing instability and adverse cardiovascular regulation.[12]

Resting cardiometabolic parameters in this study were less favorable among irregular sleepers. Systolic blood pressure was higher in the irregular sleep group than the regular sleep group (121.54 ± 9.12 mmHg vs. 114.28 ± 8.46 mmHg, p < 0.001), diastolic blood pressure was also higher (77.62 ± 7.18 mmHg vs. 72.18 ± 6.24 mmHg, p < 0.001), and mean arterial pressure followed the same pattern (92.26 ± 6.36 mmHg vs. 86.21 ± 5.84 mmHg, p < 0.001). Ogura et al. (2022), in a cross-sectional study of 3,880 participants, reported that subjective irregular sleep was significantly associated with metabolic syndrome after adjustment for age, sex, physical activity, sleep duration, bedtime, alcohol, smoking, and sleeping pill use (OR 1.231, 95% CI: 1.101–1.375).[13]

The lipid and glycemic findings of the present study also showed a clear adverse pattern among irregular sleepers. Fasting blood glucose was higher in irregular sleepers (94.48 ± 8.24 mg/dL vs. 88.64 ± 7.18 mg/dL, p < 0.001), total cholesterol was higher (182.36 ± 27.12 mg/dL vs. 166.82 ± 24.46 mg/dL, p = 0.002), triglycerides were higher (132.62 ± 34.28 mg/dL vs. 108.54 ± 28.36 mg/dL, p < 0.001), HDL cholesterol was lower (42.84 ± 5.92 mg/dL vs. 48.22 ± 6.54 mg/dL, p < 0.001), and LDL cholesterol was higher (112.38 ± 21.26 mg/dL vs. 96.46 ± 18.14 mg/dL, p < 0.001). Lunsford-Avery et al. (2018), using data from 1,978 older adults in the Multi-Ethnic Study of Atherosclerosis, showed that lower Sleep Regularity Index was associated with cardiometabolic risk indicators including obesity, diabetes, hypertension, and cardiovascular disease, which supports the present observation that sleep regularity is linked with multiple metabolic parameters rather than a single isolated marker.[14]

Correlation analysis in the present study demonstrated that better sleep regularity was positively associated with autonomic health and negatively associated with cardiometabolic risk. Sleep regularity was positively correlated with SDNN (r = 0.462, p < 0.001), RMSSD (r = 0.514, p < 0.001), HF power (r = 0.438, p < 0.001), and HDL cholesterol (r = 0.294, p = 0.002), while it was negatively correlated with LF/HF ratio (r = -0.421, p < 0.001), resting heart rate (r = -0.396, p < 0.001), BMI (r = -0.308, p = 0.001), systolic blood pressure (r = -0.372, p < 0.001), fasting blood glucose (r = -0.281, p = 0.004), and triglycerides (r = -0.336, p < 0.001). Windred et al. (2024), in 60,977 UK Biobank participants using more than 10 million hours of accelerometer data, reported that sleep regularity was a strong predictor of mortality risk, with a median Sleep Regularity Index of 81.0 and 1,859 deaths recorded during follow-up, emphasizing that sleep regularity has broader implications for long-term cardiovascular and metabolic health.[15]

CONCLUSION

This study demonstrated that irregular sleep patterns were associated with reduced heart rate variability, higher resting heart rate, and unfavorable cardiometabolic parameters among healthy young adults. Participants with regular sleep showed better parasympathetic activity, lower sympathetic predominance, lower blood pressure, and healthier glucose and lipid profiles. Sleep regularity was significantly correlated with HRV and cardiometabolic markers, suggesting its importance as an early indicator of physiological health. Promoting consistent sleep timing may help improve autonomic balance and reduce future cardiometabolic risk in young adults.

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