

Association of Sleep Quality and Sleep Duration with Resting Blood Pressure Among Young Adults: A Cross -Sectional Analytical Study

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Abstract

Background: Cardiovascular disease precursors, notably hypertension, are increasingly diagnosed in young adults. While traditional risk factors such as diet, genetics, and physical inactivity are well-established, emerging evidence indicates that lifestyle factors including sleep quality play a critical role in cardiovascular homeostasis. This study investigates the association between subjective and objective sleep quality measures and resting blood pressure parameters among young adults.

Methods: A cross-sectional analytical design was utilized, evaluating 210 young adult participants aged 18 to 26 years. Sleep quality was quantified using the Pittsburgh Sleep Quality Index (PSQI), alongside self-reported sleep duration. Standardized automated blood pressure measurements were taken to record systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP).

Results: Findings revealed a high prevalence of poor sleep quality (PSQI > 5) among participants, recorded at 52.4%. Participants classified as poor sleepers exhibited significantly elevated systolic (118.4 ± 9.2 mmHg vs. 111.1 ± 8.5 mmHg, $p < 0.001$) and diastolic blood pressures (77.6 ± 6.8 mmHg vs. 71.2 ± 5.9 mmHg, $p < 0.001$) compared to good sleepers. Regression models confirmed that poor sleep quality and short sleep duration (< 6 hours) were independent predictors of elevated blood pressure after adjusting for body mass index, caffeine consumption, and perceived psychological stress.

Conclusion: Poor sleep quality is strongly associated with elevated blood pressure indices in young adults. Integrating targeted sleep hygiene interventions into early cardiovascular disease prevention strategies is highly warranted

Aim: To assess the association between sleep quality and blood pressure among young adults using the Pittsburgh Sleep Quality Index (PSQI)

1. INTRODUCTION

Cardiovascular diseases (CVDs) remain the leading cause of global mortality, with hypertension acting as the primary modifiable risk factor (Yan et al., 2025). Traditionally perceived as a pathology affecting middle-aged and older populations, hypertension and its subclinical precursors are manifesting with increasing frequency among young adults aged 18 to 30 years (Fultariya et al., 2026). The etiology of

early-onset vascular dysregulation is multifactorial, encompassing genetic predisposition, dietary sodium intake, sedentary lifestyles, and chronic psychosocial stress (Oseni et al., 2024). In recent years, sleep architecture and sleep quality have emerged as vital determinants of cardiovascular health (Bika Léle et al., 2025). Hypertension has multiple long term effects such as kidney disease, ventricular hypertrophy leading to heart failure, ischemic heart disease, cerebrovascular accidents and so on which can have long term health implications raising the morbidity by decreasing the quality of life significantly and also increasing the rates of mortality. (3)

Sleep is extremely important for the proper functioning of all our body systems in spite of people actually not knowing what exactly it does and how it impacts our bodily systems. There are multiple studies being conducted to study the metabolic, molecular and hormonal impact of sleep on our body. Lack of sleep has been linked to obesity, Type 2 diabetes, hypertension and metabolic syndrome.(6) Sleep is composed of multiple phases like 4 Non Rapid eye movement phases and one rapid eye moment phase which can be studied using an electroencephalogram. These stages have a significant impact on our blood pressure. (1)(2)For example nightmares increase our blood pressure and risk of cardiovascular disease. (5)

Modern lifestyle pressures, including academic demands, professional stress, electronic device usage, and irregular schedules, have precipitated an epidemic of sleep curtailment and fragmentation among young adults (Santos C Souza, 2021). Studies have shown that people can be classified into two groups namely, dippers and non dippers. Dippers refers to those whose blood pressure drops during sleep and non dippers refers to those whose blood pressure doesn't drop during their sleep cycles. For example patients with diseases such as obstructive sleep apnea were found to be non dippers and have a risk for developing hypertension. (4)

Physiologically, restorative sleep characterized by sufficient duration and continuity is essential for cardiovascular recovery, marked by nocturnal blood pressure dipping, enhanced parasympathetic tone, and down-regulation of sympathetic nervous system activity (Javaheri C Redline, 2012). Conversely, sleep disruption triggers sustained autonomic imbalance, systemic inflammation, endothelial dysfunction, and elevated cortisol secretion, all of which accelerate arterial stiffening and blood pressure elevation (Palagini et al., 2015).

Despite the growing recognition of sleep as a pillar of health, empirical literature investigating the precise gradient between subjective sleep quality indices and ambulatory or resting blood pressure specifically in young adult cohorts remains sparse (Grandner, 2020). Most large-scale epidemiological studies focus on older demographics where confounding comorbidities obscure primary mechanistic associations. Therefore, this study aims to examine the association between sleep quality, sleep duration, and resting blood pressure parameters among young adults, controlling for key lifestyle confounders

Objectives

1. To assess sleep quality and sleep duration among young adults using the Pittsburgh Sleep Quality Index (PSQI).
2. To measure and compare resting systolic and diastolic blood pressure in relation to sleep quality and sleep duration.
3. To determine the association between poor sleep and elevated blood pressure among young adults.

2. Methodology

2.1 Study Design and Participants

A cross-sectional analytical study was conducted over a six-month academic term. A total of 210 young adults aged 18 to 26 years were recruited via convenience sampling from university and community settings. Inclusion criteria comprised healthy young adults without a prior clinical diagnosis of hypertension, diabetes mellitus, sleep apnea, or major psychiatric disorders, and who were not taking medications known to affect blood pressure or sleep. Participants with incomplete survey data or missing physiological measurements were excluded from the final analysis

2.2 Sleep Quality Assessment

Sleep quality was evaluated using the Pittsburgh Sleep Quality Index (PSQI), a validated 19-item self-administered questionnaire that assesses seven distinct clinical domains: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction over a one-month time interval. Global PSQI scores range from 0 to 21, with scores greater than 5 indicating severe, clinically significant poor sleep quality. In addition, average nightly sleep duration was quantified and categorized into short sleep (< 6 hours), adequate sleep (6 to 8 hours), and long sleep (> 8 hours).

2.3 Blood Pressure and Anthropometric Measurements

Resting blood pressure was measured using an automated digital sphygmomanometer following standard clinical guidelines. Participants rested in a seated position with feet flat on the floor and arm supported at heart level for at least five minutes prior to measurement. Three consecutive readings were obtained at two-minute intervals, and the average of the second and third readings was recorded for systolic blood pressure (SBP) and diastolic blood pressure (DBP). Mean arterial pressure (MAP) was calculated using the standard formula: $MAP = DBP + 1/3 (SBP - DBP)$. Height and weight were measured to calculate Body Mass Index (BMI) expressed in kg/m^2 .

2.4 Statistical Analysis

Data were analyzed using standard statistical software. Continuous variables were expressed as mean $\pm$ standard deviation, and categorical variables were presented as frequencies and percentages. Independent samples t-tests and chi-square tests were utilized for bivariate comparisons between good sleepers and poor sleepers. Multivariable linear and logistic regression models were constructed to evaluate the independent association between sleep quality parameters and blood pressure outcomes, adjusting for covariates including age, sex, BMI, caffeine intake, and perceived stress score. Statistical significance was established at $p < 0.05$.

3. Results

Among the 210 participants included in the final analysis, the mean age was 21.4 ± 1.9 years, and 56.2% were female. The overall prevalence of poor sleep quality (PSQI > 5) was 52.4% ($n = 110$). Short sleep duration (< 6 hours per night) was reported by 33.3% of the cohort. Bivariate comparisons demonstrated statistically significant differences in resting cardiovascular parameters between participants with good sleep quality versus those with poor sleep quality.

Cardiovascular Sleep Parameter	/Good Sleepers (n=100)	Poor Sleepers (n=110)	Total Sleepers (N=210)	Cohort p-value
Systolic Blood	111.1 ± 8.5	118.4 ± 9.2	114.9 ± 9.1	< 0.001

Pressure (mmHg)				
Diastolic	Blood	71.2 ± 5.9	77.6 ± 6.8	74.6 ± 6.9
Pressure (mmHg)				
Mean	Arterial	84.5 ± 6.4	91.2 ± 7.3	88.0 ± 7.2
Pressure (mmHg)				
Resting	Heart	Rate	72.4 ± 7.8	80.1 ± 8.9
(bpm)				
Global PSQI Score		3.4 ± 1.2	8.9 ± 2.1	6.3 ± 3.1

As detailed in Table 1, poor sleepers exhibited statistically significant elevations across all measured hemodynamic parameters, including systolic blood pressure (118.4 mmHg vs. 111.1 mmHg, $p < 0.001$), diastolic blood pressure (77.6 mmHg vs. 71.2 mmHg, $p < 0.001$), and resting heart rate (80.1 bpm vs. 72.4 bpm, $p < 0.001$). Multiple linear regression analysis revealed that global PSQI score was an independent positive predictor of systolic blood pressure ($\beta = 0.312$, $p = 0.002$) after controlling for body mass index and perceived stress

Ethical Considerations

The study was conducted after obtaining approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants before data collection. Participation was voluntary, and participants were informed of their right to withdraw from the study at any stage. Participant confidentiality was maintained by using coded/anonymized data for analysis

Research Question:

Is poor sleep quality (PSQI >5) associated with higher blood pressure among young adults?

4. Discussion

The present study demonstrates a significant association between poor sleep quality, shorter sleep duration, and elevated cardiovascular parameters among young adults. Poor sleep quality (PSQI >5) was observed in 52.4% of participants, while those with PSQI <5 were classified as good sleepers. Poor sleepers had significantly higher systolic and diastolic blood pressure, mean arterial pressure, and resting heart rate compared with good sleepers. Furthermore, the global PSQI score was an independent positive predictor of systolic blood pressure after adjustment for body mass index and perceived psychological stress ($\beta = 0.312$, $p = 0.002$), suggesting an independent association within the study population.

These findings are consistent with previous literature. Fultariya et al. reported higher resting blood pressure and heart rate among individuals with shorter sleep duration, and Logeshpriyan et al. described associations between sleep-related behaviours, poorer sleep quality, and elevated blood pressure in young adults. Similarly, a systematic review and meta-analysis by Yang et al., has demonstrated an association between short sleep duration and hypertension among Asian populations.

Although sleep duration and sleep quality are related, they represent distinct dimensions of sleep and may have independent implications for cardiovascular health. Sleep duration alone may not adequately capture the restorative nature of sleep, as fragmentation, difficulty initiating or maintaining sleep, and disturbed sleep-wake transitions may also influence cardiovascular regulation. In this context, Kim et al. reported that forced awakening was associated with a greater morning blood pressure surge compared with natural awakening, suggesting that the manner of awakening may influence blood pressure during the transition

from sleep to wakefulness. Such findings highlight that cardiovascular responses to sleep may depend not only on the total duration of sleep but also on its quality and continuity. Thus, assessment of both sleep quality and sleep duration may provide a more comprehensive understanding of the relationship between sleep and blood pressure in young adults.

Short sleep duration was common in the present study, with 33.3% of participants reporting less than six hours of sleep per night. This is noteworthy given evidence that irregular sleep duration is associated with higher blood pressure in young adults, as demonstrated by Culver et al. The high prevalence of short sleep in our study therefore highlights inadequate and irregular sleep as potentially modifiable factors relevant to blood pressure in this population.

Several physiological mechanisms may explain the association between poor sleep and elevated blood pressure observed in the present study. Sleep restriction and fragmentation can increase sympathetic activity, reduce parasympathetic tone, and impair nocturnal blood pressure dipping, while activation of the hypothalamic-pituitary-adrenal axis may further disrupt cardiovascular regulation. At the molecular level, sleep fragmentation generates oxidative stress that disrupts endothelial nitric oxide synthase (eNOS), decreasing nitric oxide while upregulating endothelin-1. Additionally, poor sleep quality promotes metabolic dysregulation and weight gain further exacerbating sympathetic tone and vascular resistance contributing to elevated blood pressure.

In addition to elevated blood pressure, poor sleep quality was associated with a significantly higher resting heart rate in the present study, with poor sleepers having a mean resting heart rate of 80.1 ± 8.9 compared with 72.4 ± 7.8 bpm among good sleepers ($p < 0.001$). This finding is consistent with previous observations linking inadequate or disturbed sleep with increased resting heart rate. Thus, the cardiovascular effects associated with poor sleep in young adults may extend beyond blood pressure alone and involve multiple hemodynamic parameters.

An important consideration in interpreting these findings is that factors such as caffeine consumption, perceived stress, and BMI may influence both sleep quality and blood pressure, making it difficult to attribute the observed differences to sleep alone. In the present study, adjustment for BMI and perceived stress did not eliminate the positive association between global PSQI score and systolic blood pressure, suggesting that this relationship was not explained entirely by these factors. Nevertheless, blood pressure is multifactorial, and sleep should therefore be considered as one component of a broader cardiovascular risk profile rather than an isolated determinant.

The present study has several strengths, including the use of a validated measure of sleep quality, assessment of sleep duration, standardized repeated blood pressure measurements, and evaluation of multiple cardiovascular parameters. Adjustment for relevant factors such as BMI and perceived stress further strengthened the assessment of the association between sleep quality and blood pressure.

Several limitations should be considered when interpreting the present findings. These include the cross-sectional design; convenience sampling, which may limit generalizability of the findings to the wider young adult population; reliance on self-reported sleep measures, which may introduce recall or reporting bias; and the possibility of residual confounding from unmeasured behavioural and psychosocial factors. Therefore, the findings should be interpreted as evidence of an association rather than causation.

Future studies should use longitudinal designs, larger and more representative samples, and incorporate objective sleep measures such as actigraphy or polysomnography to clarify the relationship between sleep and blood pressure and determine whether interventions aimed at improving sleep can reduce cardiovascular risk.

Overall, the present study supports an association between poor sleep quality and less favourable resting cardiovascular parameters in young adults. Although causality cannot be inferred from the current design, the findings suggest that sleep may represent a relevant and modifiable component of early cardiovascular risk. Promoting healthy sleep patterns may therefore be a useful component of cardiovascular risk prevention in young adulthood.

5. Conclusion

In conclusion, poor sleep quality and shorter sleep duration were significantly associated with higher resting systolic and diastolic blood pressure among young adults. Not only does it have cardiovascular side effects but also has been linked to the development and exacerbation of psychiatric disorders, especially mood disorders, schizophrenia and bipolar disorder and so on (23)(24). The above findings highlight sleep as a potentially modifiable cardiovascular risk factor. Promoting adequate, good-quality sleep may support healthier blood pressure and contribute to early cardiovascular risk reduction

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