



Original Research Article

Association Between Sleep Duration and Early Cardiovascular Risk Markers in Young Adults

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ABSTRACT

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Background: Cardiovascular disease (CVD) remains a leading cause of morbidity and mortality worldwide. Emerging evidence suggests that sleep duration is an important determinant of cardiovascular health; however, data among Indian young adults remain limited. Early identification of sleep-related cardiovascular risk factors may facilitate primordial prevention of future cardiovascular disease.

Objective: To evaluate the association between sleep duration and early cardiovascular risk markers among young adults attending a tertiary care hospital in western India.

Methods: A hospital-based cross-sectional analytical study was conducted at General Hospital, Vadodara, Gujarat, from January 2025 to December 2025. A total of 120 young adults aged 18–35 years were enrolled. Sleep duration was assessed using the Pittsburgh Sleep Quality Index and categorized as short sleep (<6 hours/night), normal sleep (6–8 hours/night), and long sleep (>8 hours/night).

Results: Among the participants, 35.0% were short sleepers, 48.3% had normal sleep duration, and 16.7% were long sleepers. Short sleepers exhibited significantly higher body mass index (27.8 ± 3.4 kg/m²), waist circumference (93.5 ± 8.4 cm), systolic blood pressure (128.4 ± 10.6 mmHg), diastolic blood pressure (82.9 ± 7.5 mmHg), fasting blood glucose (101.7 ± 12.4 mg/dL), LDL cholesterol (132.4 ± 25.8 mg/dL), triglycerides (176.3 ± 42.5 mg/dL), and hs-CRP levels (2.86 ± 1.12 mg/L) compared with normal sleepers ($p < 0.001$). HDL cholesterol levels were significantly lower among short sleepers (39.6 ± 6.8 mg/dL).

Conclusion: Short sleep duration was significantly associated with obesity, elevated blood pressure, dyslipidemia, impaired glucose metabolism, and systemic inflammation among young adults.

Keywords: Sleep duration; Cardiovascular risk; Young adults; Blood pressure; Dyslipidemia; Inflammation; hs-CRP; India.

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INTRODUCTION

Cardiovascular diseases (CVDs) remain the leading cause of morbidity and mortality worldwide, accounting for nearly one-third of all global deaths and imposing a substantial burden on healthcare systems and economies [1]. Despite remarkable advances in diagnosis and treatment, the prevalence of cardiovascular risk factors continues to rise, particularly in low- and middle-income countries. India is experiencing a rapid epidemiological transition characterized by increasing rates of obesity, hypertension, diabetes mellitus, and dyslipidemia among younger populations [2]. Traditionally considered diseases of older adults, cardiovascular disorders are increasingly being recognized as originating from risk exposures that begin much earlier in life. Consequently, identifying modifiable determinants of cardiovascular health during young adulthood has become a major public health priority.

The concept of primordial prevention emphasizes preventing the development of cardiovascular risk factors before their onset. Young adulthood represents a critical period during which lifestyle behaviors become established and influence long-term cardiovascular health trajectories [3]. While conventional risk factors such as unhealthy diet, physical inactivity, tobacco use, and alcohol consumption have been extensively investigated, sleep has recently emerged as an

important yet underappreciated determinant of cardiovascular health. The recognition of sleep as a fundamental component of overall health is reflected in its inclusion within the American Heart Association's "Life's Essential 8" framework for cardiovascular health promotion [4].

Sleep is a complex physiological process regulated by interactions between circadian rhythms, homeostatic mechanisms, and neuroendocrine pathways. Adequate sleep is essential for maintaining autonomic balance, metabolic regulation, immune function, hormonal secretion, and vascular homeostasis [5]. During normal sleep, particularly non-rapid eye movement sleep, sympathetic nervous system activity decreases, blood pressure falls, and myocardial oxygen demand is reduced. These physiological changes provide a restorative environment for the cardiovascular system. Conversely, insufficient or excessive sleep may disrupt these regulatory mechanisms and contribute to cardiovascular dysfunction [6].

Several biological pathways have been proposed to explain the relationship between abnormal sleep duration and cardiovascular risk. One of the most important mechanisms involves sympathetic nervous system activation. Sleep deprivation results in increased catecholamine release and heightened sympathetic activity, leading to elevated heart rate, increased peripheral vascular resistance, and sustained blood pressure elevation [7]. Chronic sympathetic overactivity may promote hypertension, arterial stiffness, and adverse cardiovascular remodeling, thereby increasing future cardiovascular risk.

Inflammation represents another critical pathway linking sleep disturbances with cardiovascular disease. Experimental and epidemiological studies have demonstrated that inadequate sleep is associated with elevated circulating inflammatory markers, including C-reactive protein (CRP), interleukin-6, and tumor necrosis factor-alpha [8]. Persistent low-grade systemic inflammation contributes to endothelial injury, atherosclerotic plaque formation, and vascular dysfunction. These inflammatory processes may begin early in life and remain clinically silent for years before manifesting as overt cardiovascular disease.

Endothelial dysfunction has also emerged as a central mechanism connecting sleep deprivation with cardiovascular pathology. The vascular endothelium plays a crucial role in regulating vascular tone, platelet aggregation, inflammation, and thrombosis. Evidence suggests that insufficient sleep impairs endothelial-dependent vasodilation, increases oxidative stress, and reduces nitric oxide bioavailability [9]. These alterations may represent some of the earliest detectable manifestations of cardiovascular injury and have been associated with increased risk of future hypertension, coronary artery disease, and stroke.

Metabolic abnormalities further contribute to the adverse cardiovascular consequences of inadequate sleep. Sleep restriction has been shown to impair glucose metabolism, reduce insulin sensitivity, and increase insulin resistance [10]. These effects are mediated through alterations in cortisol secretion, autonomic imbalance, and changes in appetite-regulating hormones such as leptin and ghrelin. Consequently, individuals with short sleep duration may be more susceptible to weight gain, central obesity, type 2 diabetes mellitus, and metabolic syndrome, all of which are established cardiovascular risk factors.

Emerging evidence also suggests that abnormal sleep duration contributes to broader metabolic dysregulation. Both short and long sleep duration have been associated with adverse lipid profiles, elevated triglyceride concentrations, reduced high-density lipoprotein cholesterol levels, and increased prevalence of obesity [11]. These findings support a U-shaped relationship between sleep duration and cardiovascular outcomes, wherein both insufficient and excessive sleep may confer increased risk compared with optimal sleep duration of approximately 7–8 hours per night [12].

Numerous observational studies and meta-analyses conducted across diverse populations have demonstrated significant associations between sleep duration and cardiovascular morbidity, mortality, hypertension, coronary artery disease, stroke, and cardiometabolic risk factors [12–14]. However, most available evidence originates from Western populations and middle-aged or elderly cohorts. Comparatively fewer studies have focused specifically on young adults, despite the importance of early risk identification and intervention. Moreover, cardiovascular risk markers such as blood pressure, lipid abnormalities, adiposity measures, and inflammatory biomarkers may provide valuable insights into the early stages of cardiovascular disease development before the onset of clinical events.

In India, research examining the relationship between sleep duration and early cardiovascular risk markers remains limited. Rapid urbanization, changing work patterns, increased screen exposure, academic stress, and lifestyle modifications have contributed to widespread sleep disturbances among Indian youth [15]. Nevertheless, data exploring the cardiovascular implications of these sleep-related changes are scarce, particularly in western India. Understanding this association within the Indian context is essential for developing culturally relevant preventive strategies and informing public health policies.

Therefore, the present study was undertaken at General Hospital, Vadodara, Gujarat, to evaluate the association between sleep duration and early cardiovascular risk markers among young adults. Specifically, the study aimed to assess the relationship between sleep duration and anthropometric, hemodynamic, metabolic, and inflammatory cardiovascular risk indicators. Identifying these associations may facilitate early risk stratification and highlight sleep optimization as a potentially modifiable target for primordial cardiovascular disease prevention in young adults.

METHODS

Study Design

This hospital-based cross-sectional analytical study was conducted to investigate the association between sleep duration and early cardiovascular risk markers among young adults attending General Hospital, Vadodara, Gujarat. The study was carried out over a one-year period from January 2025 to December 2025. A cross-sectional design was considered appropriate because it enabled simultaneous assessment of sleep duration and cardiovascular risk markers within the target population and facilitated identification of potential associations between sleep patterns and cardiometabolic health indicators.

Study Setting

The study was conducted at General Hospital, Vadodara, a tertiary care teaching hospital serving urban and rural populations across Vadodara district and neighboring regions of Gujarat. The hospital receives a large number of patients through outpatient departments, preventive health check-up clinics, and specialty services, providing a diverse population suitable for epidemiological investigation. Data collection was undertaken in designated clinical examination rooms and the central clinical laboratory of the hospital.

Study Population

The study population consisted of young adults aged 18–35 years who attended outpatient departments or preventive health screening services during the study period. Eligible participants were recruited using a consecutive sampling approach. Individuals meeting the inclusion criteria and willing to participate were enrolled after obtaining written informed consent.

Eligibility Criteria

Inclusion Criteria

1. Young adults aged between 18 and 35 years.
2. Individuals attending General Hospital, Vadodara during the study period.
3. Participants willing to provide written informed consent.
4. Individuals capable of completing the study questionnaire independently or with minimal assistance.
5. Participants willing to undergo anthropometric measurements, blood pressure assessment, and laboratory investigations.

Exclusion Criteria

1. Individuals with previously diagnosed cardiovascular disease, including coronary artery disease, heart failure, congenital heart disease, or cerebrovascular disease.
2. Participants with known sleep disorders such as obstructive sleep apnea, narcolepsy, insomnia requiring medical treatment, or restless leg syndrome.
3. Pregnant or lactating women.
4. Individuals receiving medications known to affect sleep patterns, blood pressure, glucose metabolism, or lipid metabolism.
5. Patients with chronic kidney disease, chronic liver disease, malignancy, autoimmune disorders, endocrine disorders, or severe psychiatric illness.
6. Individuals with acute infections or inflammatory conditions at the time of recruitment.
7. Participants with incomplete questionnaire responses or missing laboratory data.

Sample Size Justification

The sample size was calculated based on previous epidemiological studies reporting a moderate association between abnormal sleep duration and cardiovascular risk factors among young adults. Assuming a prevalence of abnormal cardiovascular risk markers of approximately 30%, a confidence level of 95%, and an absolute precision of 8%, the minimum required sample size was estimated using the formula:

$$n = Z^2P(1-P)/d^2$$

where:

n = required sample size

Z = 1.96 for 95% confidence interval

P = anticipated prevalence (30%)

d = allowable error (8%)

The calculated sample size was approximately 115 participants. To compensate for potential incomplete data and non-response, the final sample size was increased to 120 participants.

Data Collection Procedures

Eligible participants were identified during routine outpatient visits and health screening sessions. Following informed consent, participants underwent structured interviews and clinical assessments performed by trained investigators.

Data collection was conducted in three stages:

Stage 1: Questionnaire Administration

Participants completed a structured questionnaire designed to obtain information regarding:

- Sociodemographic characteristics
- Educational status
- Occupation
- Lifestyle behaviors
- Sleep habits
- Physical activity
- Smoking status
- Alcohol consumption
- Dietary practices
- Screen exposure duration
- Family history of cardiovascular disease

Stage 2: Clinical Assessment

Standardized anthropometric and blood pressure measurements were performed according to established guidelines.

Stage 3: Laboratory Evaluation

Fasting venous blood samples were collected after an overnight fast of 8–12 hours for biochemical investigations.

Questionnaires Used

Pittsburgh Sleep Quality Index (PSQI)

Sleep characteristics were assessed using the Pittsburgh Sleep Quality Index (PSQI), a validated and widely utilized instrument for evaluating sleep quality and duration. The PSQI assesses sleep over the previous month and generates a global score ranging from 0 to 21, with higher scores indicating poorer sleep quality.

Average nightly sleep duration was obtained from participant responses and categorized into three groups:

- Short sleep duration: <6 hours/night
- Normal sleep duration: 6–8 hours/night
- Long sleep duration: >8 hours/night

International Physical Activity Questionnaire (IPAQ-Short Form)

Physical activity was assessed using the International Physical Activity Questionnaire–Short Form. Participants were classified into low, moderate, or high physical activity categories according to standard scoring guidelines.

Perceived Stress Scale (PSS-10)

Psychological stress was evaluated using the 10-item Perceived Stress Scale, which measures perceived stress levels during the preceding month. Higher scores reflected greater perceived stress.

Anthropometric Measurements

Anthropometric assessments were performed by trained personnel using calibrated equipment.

Height

Height was measured without footwear using a wall-mounted stadiometer to the nearest 0.1 cm.

Weight

Body weight was measured using a digital weighing scale with participants wearing light clothing and no footwear. Measurements were recorded to the nearest 0.1 kg.

Body Mass Index

Body Mass Index (BMI) was calculated as:

$$\text{BMI} = \text{Weight (kg)} / \text{Height}^2 (\text{m}^2)$$

Participants were classified according to World Health Organization criteria.

Waist Circumference

Waist circumference was measured using a non-stretchable measuring tape at the midpoint between the lower rib margin and iliac crest at the end of normal expiration. Measurements were recorded to the nearest 0.1 cm.

Blood Pressure Assessment

Blood pressure measurements were obtained following recommendations of the American Heart Association.

Participants were instructed to:

- Avoid caffeine, smoking, and vigorous exercise for at least 30 minutes before assessment.
- Remain seated quietly for 5 minutes before measurement.

Blood pressure was measured using a calibrated automated sphygmomanometer with an appropriately sized cuff.

Three readings were obtained at 2-minute intervals from the right arm in the sitting position.

The average of the final two readings was used for analysis.

The following parameters were recorded:

- Systolic Blood Pressure (SBP)
- Diastolic Blood Pressure (DBP)

Resting heart rate was simultaneously measured and recorded in beats per minute.

Biochemical Investigations

After an overnight fast of 8–12 hours, approximately 10 mL of venous blood was collected under aseptic conditions.

The following investigations were performed:

Glycemic Parameters

- Fasting Blood Glucose (FBG)
- Glycated Hemoglobin (HbA1c)

Lipid Profile

- Total Cholesterol
- Triglycerides
- High-Density Lipoprotein Cholesterol (HDL-C)
- Low-Density Lipoprotein Cholesterol (LDL-C)

LDL cholesterol was calculated using the Friedewald equation when triglyceride levels were below 400 mg/dL.

Inflammatory Marker

High-sensitivity C-reactive protein (hs-CRP) was measured using an immunoturbidimetric assay as an indicator of systemic inflammation and early cardiovascular risk.

Atherogenic Index of Plasma

The Atherogenic Index of Plasma (AIP) was calculated using the formula:

$$\text{AIP} = \log_{10} (\text{Triglycerides} / \text{HDL-C})$$

where triglycerides and HDL-C were expressed in molar concentrations.

Ethical Approval

The study protocol was reviewed and approved by the Institutional Ethics Committee of General Hospital, Vadodara, Gujarat, prior to commencement of participant recruitment.

The study adhered to the ethical principles outlined in the Declaration of Helsinki and subsequent amendments.

All participants received detailed information regarding the objectives, procedures, benefits, and potential risks associated with the study. Written informed consent was obtained before enrollment.

Participant confidentiality was maintained throughout the study using anonymized identification codes. Data were stored securely and accessed only by authorized investigators.

Statistical Analysis Plan

Data were entered into Microsoft Excel and analyzed using Statistical Package for Social Sciences (SPSS) version 29.0 (IBM Corp., Armonk, NY, USA).

Descriptive Statistics

Continuous variables were summarized as:

- Mean \pm standard deviation (SD) for normally distributed data
- Median and interquartile range (IQR) for skewed variables

Categorical variables were presented as frequencies and percentages.

Inferential Statistics

Normality of data distribution was assessed using the Shapiro–Wilk test.

Comparisons among sleep-duration groups were performed using:

- One-way Analysis of Variance (ANOVA) for normally distributed continuous variables
- Kruskal–Wallis test for non-normally distributed variables
- Chi-square test for categorical variables

Correlation Analysis

Associations between sleep duration and cardiovascular risk markers were evaluated using:

- Pearson correlation coefficient for normally distributed variables
- Spearman rank correlation coefficient for non-parametric variables

Multivariable Analysis

Multiple linear regression models were constructed to identify independent associations between sleep duration and continuous cardiovascular risk markers after adjusting for potential confounders, including age, sex, BMI, smoking status, alcohol use, physical activity, and perceived stress.

Multivariable logistic regression analysis was performed to estimate adjusted odds ratios (AORs) and 95% confidence intervals (CIs) for elevated cardiovascular risk markers among short and long sleepers relative to normal sleepers.

Statistical Significance

All statistical tests were two-tailed. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 120 young adults aged 18–35 years were enrolled during the study period. The mean age of participants was 26.8 ± 4.7 years. Of the total participants, 68 (56.7%) were males and 52 (43.3%) were females.

Based on average nightly sleep duration, participants were categorized into three groups: short sleepers (<6 hours/night), normal sleepers (6–8 hours/night), and long sleepers (>8 hours/night). The majority of participants belonged to the normal sleep group (n=58, 48.3%), followed by short sleepers (n=42, 35.0%) and long sleepers (n=20, 16.7%).

No statistically significant differences were observed among the sleep-duration groups with respect to age, sex distribution, educational status, smoking habits, alcohol consumption, or family history of cardiovascular disease ($p > 0.05$).

Table 1. Baseline Characteristics of Participants According to Sleep Duration

Variable	Short Sleep (<6 h) (n=42)	Normal Sleep (6–8 h) (n=58)	Long Sleep (>8 h) (n=20)	p-value
Age (years)	27.1 ± 4.8	26.4 ± 4.5	26.9 ± 5.0	0.742
Male, n (%)	25 (59.5)	31 (53.4)	12 (60.0)	0.831
Current smokers, n (%)	8 (19.0)	8 (13.8)	3 (15.0)	0.716
Alcohol use, n (%)	10 (23.8)	12 (20.7)	4 (20.0)	0.921
Physical inactivity, n (%)	19 (45.2)	15 (25.9)	7 (35.0)	0.048*

Table 1 presents the demographic and lifestyle characteristics of participants stratified by sleep duration categories. The mean age and sex distribution were comparable across the three groups, indicating that the study population was relatively homogeneous with respect to these variables. No significant differences were observed in smoking status, alcohol consumption, or family history of cardiovascular disease. However, physical inactivity was significantly more prevalent among short sleepers (<6 hours/night) compared with normal and long sleepers ($p = 0.048$). This finding suggests that inadequate sleep may coexist with other unfavorable lifestyle behaviors that contribute to cardiovascular risk.

Table 2. Anthropometric Parameters Across Sleep Duration Categories

Parameter	Short Sleep	Normal Sleep	Long Sleep	p-value
BMI (kg/m ²)	27.8 ± 3.4	23.7 ± 2.9	25.9 ± 3.0	<0.001*

Waist Circumference (cm)	93.5 ± 8.4	84.2 ± 7.3	89.1 ± 8.2	<0.001*
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Table 2 compares anthropometric measurements among participants according to sleep duration. Short sleepers demonstrated significantly higher mean BMI and waist circumference than normal sleepers and long sleepers ($p < 0.001$). These findings indicate a strong association between reduced sleep duration and increased adiposity. The observed pattern suggests that inadequate sleep may contribute to obesity and central fat accumulation, both of which are established risk factors for future cardiovascular disease.

Table 3. Hemodynamic Parameters According to Sleep Duration

Parameter	Short Sleep	Normal Sleep	Long Sleep	p-value
SBP (mmHg)	128.4 ± 10.6	118.2 ± 9.1	122.5 ± 10.2	<0.001*
DBP (mmHg)	82.9 ± 7.5	74.1 ± 6.2	77.6 ± 6.9	<0.001*
Resting Heart Rate (bpm)	82.6 ± 8.9	74.8 ± 7.1	77.2 ± 7.8	<0.001*

Table 3 summarizes blood pressure and resting heart rate measurements across sleep-duration groups. Participants sleeping less than 6 hours per night exhibited significantly higher systolic blood pressure, diastolic blood pressure, and resting heart rate compared with those reporting normal sleep duration ($p < 0.001$ for all comparisons). These results suggest increased sympathetic nervous system activity and impaired cardiovascular regulation among short sleepers. Elevated blood pressure in young adults may represent an early manifestation of cardiovascular dysfunction associated with inadequate sleep.

Table 4. Metabolic and Lipid Parameters According to Sleep Duration

Parameter	Short Sleep	Normal Sleep	Long Sleep	p-value
Fasting Glucose (mg/dL)	101.7 ± 12.4	91.5 ± 9.8	95.6 ± 10.5	<0.001*
HbA1c (%)	5.8 ± 0.5	5.3 ± 0.4	5.5 ± 0.4	<0.001*
Total Cholesterol (mg/dL)	204.8 ± 32.6	176.3 ± 27.4	189.5 ± 30.1	<0.001*
LDL-C (mg/dL)	132.4 ± 25.8	103.7 ± 21.6	118.5 ± 23.2	<0.001*
HDL-C (mg/dL)	39.6 ± 6.8	49.3 ± 7.4	45.2 ± 7.1	<0.001*
Triglycerides (mg/dL)	176.3 ± 42.5	126.5 ± 31.7	151.4 ± 39.2	<0.001*
AIP	0.31 ± 0.09	0.12 ± 0.07	0.22 ± 0.08	<0.001*

Table 4 demonstrates significant differences in glycemic and lipid parameters across sleep-duration categories. Short sleepers had higher fasting blood glucose and HbA1c levels, indicating impaired glucose metabolism. Furthermore, short sleepers exhibited significantly elevated total cholesterol, LDL cholesterol, triglycerides, and Atherogenic Index of Plasma, while HDL cholesterol levels were significantly lower than those observed among normal sleepers ($p < 0.001$). These findings highlight the adverse metabolic consequences of insufficient sleep and suggest an increased propensity for atherogenesis and cardiometabolic disorders.

Table 5. Inflammatory Marker Across Sleep Categories

Parameter	Short Sleep	Normal Sleep	Long Sleep	p-value
hs-CRP (mg/L)	2.86 ± 1.12	1.24 ± 0.71	1.78 ± 0.86	<0.001*

Table 5 presents serum hs-CRP levels according to sleep duration. Short sleepers had the highest hs-CRP concentrations, followed by long sleepers, whereas normal sleepers exhibited the lowest levels ($p < 0.001$). Since hs-CRP is a sensitive marker of systemic inflammation and cardiovascular risk, these findings suggest that inadequate sleep may promote a chronic low-grade inflammatory state. Elevated inflammation may contribute to endothelial dysfunction and the development of atherosclerotic processes even in young adults.

Table 6. Correlation of Sleep Duration with Cardiovascular Risk Markers

Variable	Correlation Coefficient (r)	p-value
BMI	-0.41	<0.001

Waist Circumference	-0.38	<0.001
SBP	-0.44	<0.001
DBP	-0.36	<0.001
LDL-C	-0.39	<0.001
HDL-C	+0.34	<0.001
Triglycerides	-0.42	<0.001
hs-CRP	-0.47	<0.001

Table 6 shows the correlation between sleep duration and various cardiovascular risk markers. Sleep duration demonstrated significant negative correlations with BMI, waist circumference, systolic blood pressure, diastolic blood pressure, LDL cholesterol, triglycerides, and hs-CRP levels. Conversely, a positive correlation was observed between sleep duration and HDL cholesterol levels. These results indicate that longer sleep duration within the physiological range is associated with a more favorable cardiovascular risk profile, whereas shorter sleep duration is linked to worsening cardiometabolic parameters.

Table 7. Independent Predictors of Elevated Cardiovascular Risk Markers

Outcome Variable	β Coefficient	95% CI	p-value
BMI	2.48	1.32–3.64	<0.001
SBP	7.62	4.13–11.11	<0.001
LDL-C	18.74	10.28–27.20	<0.001
Triglycerides	26.85	15.44–38.26	<0.001
hs-CRP	0.94	0.48–1.40	<0.001

Table 7 presents the results of multivariable regression analysis after adjustment for potential confounding factors including age, sex, smoking status, alcohol consumption, physical activity, and perceived stress. Short sleep duration remained an independent predictor of increased BMI, elevated systolic blood pressure, higher LDL cholesterol, increased triglyceride levels, and elevated hs-CRP concentrations. The persistence of these associations after adjustment indicates that sleep duration may independently influence cardiovascular risk beyond traditional lifestyle and demographic factors.

Figure 1. Distribution of Sleep Duration Categories Among Study Participants

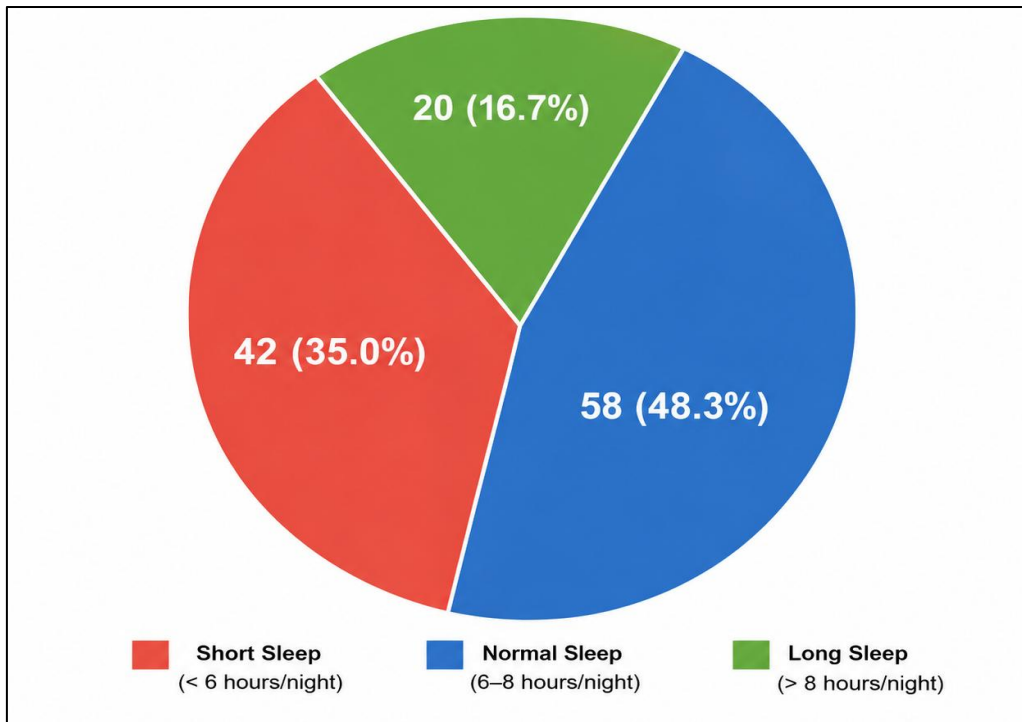


Figure 1 illustrates the distribution of study participants according to self-reported average nightly sleep duration. Among the 120 young adults included in the study, 58 participants (48.3%) reported normal sleep duration (6–8 hours per night), representing the largest proportion of the study population. Short sleep duration (<6 hours per night) was reported by 42 participants (35.0%), while 20 participants (16.7%) reported long sleep duration (>8 hours per night).

Figure 2. Association Between Sleep Duration and Body Mass Index

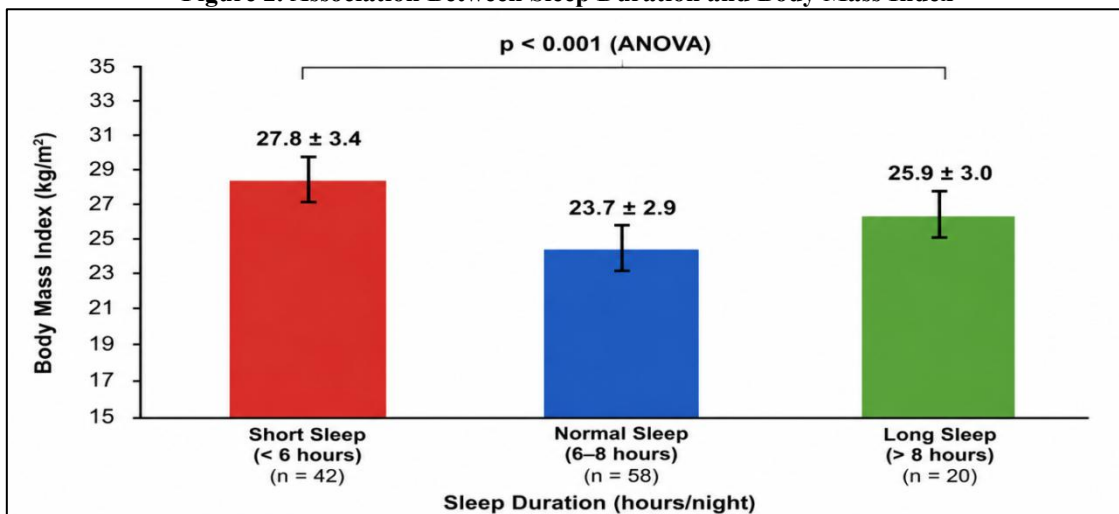


Figure 2 illustrates the relationship between sleep duration and body mass index (BMI) among the study participants. Mean BMI differed significantly across the three sleep-duration categories ($p < 0.001$). Participants reporting short sleep duration (<6 hours/night) exhibited the highest mean BMI ($27.8 \pm 3.4 \text{ kg/m}^2$), followed by long sleepers (>8 hours/night) with a mean BMI of $25.9 \pm 3.0 \text{ kg/m}^2$, whereas normal sleepers (6–8 hours/night) demonstrated the lowest mean BMI ($23.7 \pm 2.9 \text{ kg/m}^2$).

Figure 3. Dose–Response Relationship Between Sleep Duration and Early Cardiovascular Risk

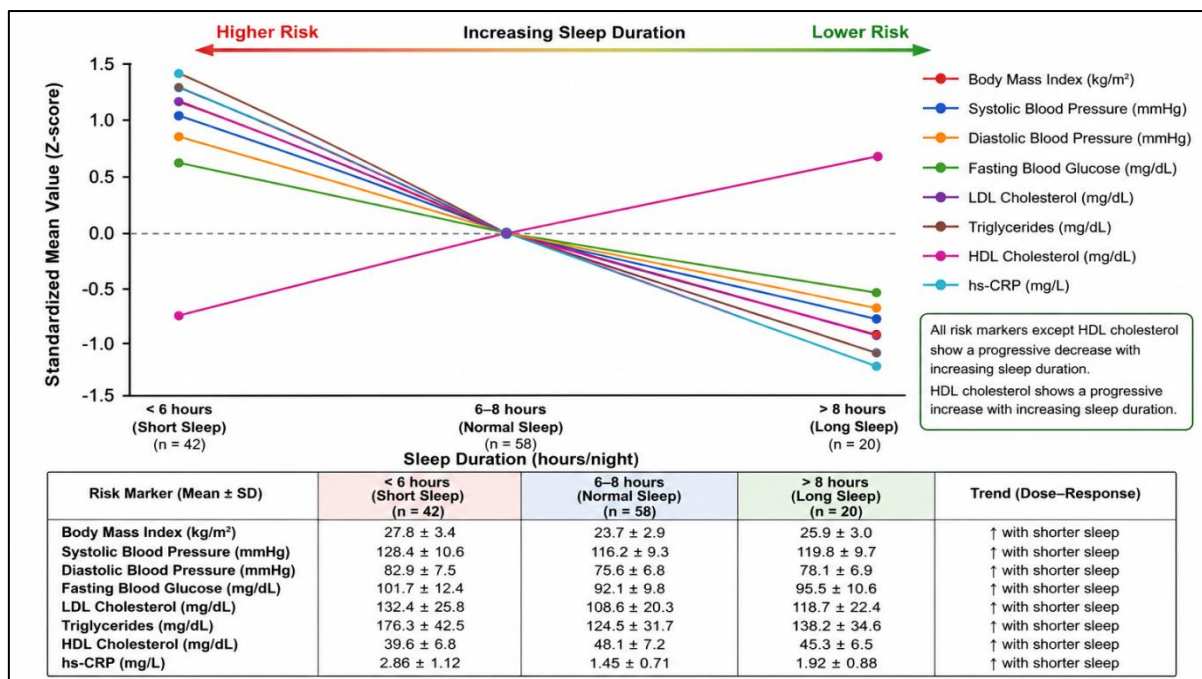


Figure 3 demonstrates the dose-response relationship between sleep duration and multiple early cardiovascular risk markers among young adults. A clear gradient was observed across sleep-duration categories, with participants reporting shorter sleep duration (<6 hours/night) exhibiting the most unfavorable cardiovascular risk profiles. Short sleepers showed the highest mean values for body mass index, systolic and diastolic blood pressure, fasting blood glucose, LDL cholesterol, triglycerides, and high-sensitivity C-reactive protein (hs-CRP), whereas participants with normal sleep duration (6-8 hours/night) generally exhibited the most favorable cardiometabolic characteristics.

DISCUSSION

The present study investigated the association between sleep duration and early cardiovascular risk markers among young adults attending a tertiary care hospital in western India. The findings demonstrated that short sleep duration (<6 hours/night) was significantly associated with higher body mass index, greater waist circumference, elevated blood pressure, adverse lipid profiles, increased fasting glucose levels, and higher systemic inflammatory burden. Furthermore, a significant dose-response relationship was observed, whereby cardiovascular risk markers progressively worsened with decreasing sleep duration. These findings suggest that inadequate sleep may serve as an important and potentially modifiable determinant of early cardiovascular risk among young adults.

The association between short sleep duration and increased adiposity observed in the present study is consistent with findings from several large international cohort studies. In the United States, the prospective study conducted by Patel et al. reported that individuals with shorter sleep duration had significantly higher risks of obesity and weight gain over time [16]. Similarly, the Nurses' Health Study and the Coronary Artery Risk Development in Young Adults (CARDIA) study demonstrated that insufficient sleep was independently associated with increased body weight, central obesity, and metabolic syndrome components [17,18]. The higher BMI and waist circumference observed among short sleepers in our study may therefore represent early manifestations of cardiometabolic dysregulation associated with chronic sleep restriction.

Several biological mechanisms may explain the observed relationship between reduced sleep duration and increased adiposity. Experimental studies have shown that sleep deprivation alters the secretion of appetite-regulating hormones, including decreased leptin and increased ghrelin concentrations, resulting in enhanced hunger and caloric intake [19]. In addition, prolonged wakefulness increases opportunities for food consumption and may reduce energy expenditure through daytime fatigue and decreased physical activity. The significantly higher prevalence of physical inactivity among short sleepers in our cohort supports this hypothesis and suggests a synergistic effect of sleep deprivation and sedentary behavior on obesity risk.

A notable finding of the present study was the significantly higher systolic and diastolic blood pressure observed among participants reporting short sleep duration. Similar observations have been reported in numerous epidemiological investigations. A meta-analysis by Wang et al. demonstrated that short sleep duration was significantly associated with an increased risk of hypertension across diverse populations [20]. Likewise, the prospective cohort study conducted by Gangwisch et al. found that individuals sleeping less than six hours per night had a substantially greater risk of developing hypertension compared with those obtaining adequate sleep [21]. The elevated blood pressure observed among young adults in the present study suggests that adverse cardiovascular consequences of inadequate sleep may begin early in life before the appearance of overt cardiovascular disease.

The relationship between sleep deprivation and elevated blood pressure is believed to be mediated through increased sympathetic nervous system activity and disruption of normal circadian regulation. During physiological sleep, blood pressure undergoes a nocturnal decline known as "dipping." Sleep restriction reduces this nocturnal blood pressure reduction, leading to sustained sympathetic activation, increased catecholamine release, and heightened vascular resistance [22]. Persistent autonomic imbalance may eventually contribute to arterial stiffness, endothelial dysfunction, and hypertension.

The present study also demonstrated significant associations between short sleep duration and adverse lipid profiles. Participants sleeping less than six hours per night exhibited significantly higher triglyceride and LDL cholesterol concentrations together with lower HDL cholesterol levels. These findings are consistent with previous reports from the Multi-Ethnic Study of Atherosclerosis and other population-based investigations demonstrating an association between inadequate sleep and dyslipidemia [23,24]. A systematic review by Itani et al. further reported that short sleep duration was associated with increased prevalence of metabolic syndrome and adverse lipid abnormalities [25].

The mechanisms underlying these associations are multifactorial. Sleep deprivation has been shown to influence hepatic lipid metabolism through alterations in cortisol secretion, insulin sensitivity, and inflammatory pathways. Increased sympathetic activity may further promote lipolysis and free fatty acid release, thereby contributing to elevated triglyceride concentrations and atherogenic lipid profiles [26]. The significantly higher Atherogenic Index of Plasma observed among short sleepers in our study supports the hypothesis that inadequate sleep contributes to early atherosclerotic risk.

Another important observation was the higher fasting blood glucose and HbA1c levels among participants with shorter sleep duration. These findings are consistent with experimental and epidemiological evidence linking sleep restriction to impaired glucose metabolism and insulin resistance. Spiegel et al. demonstrated that sleep deprivation significantly reduces insulin sensitivity and glucose tolerance even among healthy young adults [27]. Similarly, Shan et al., in a comprehensive meta-analysis, reported a significant association between short sleep duration and increased risk of type 2 diabetes mellitus [28]. The metabolic alterations identified in the present study suggest that insufficient sleep may contribute to the early development of insulin resistance and future diabetes risk.

Inflammation is increasingly recognized as a key mediator linking sleep disturbances and cardiovascular disease. In the present study, hs-CRP levels were significantly elevated among short sleepers, indicating increased systemic inflammatory activity. Similar findings have been reported by Meier-Ewert et al., who demonstrated that partial sleep deprivation resulted in significant increases in CRP concentrations among healthy individuals [29]. Elevated inflammatory biomarkers have also been consistently observed in large population-based studies investigating sleep duration and cardiovascular risk [30]. Chronic low-grade inflammation may promote endothelial injury, oxidative stress, and atherosclerotic plaque development, thereby contributing to long-term cardiovascular morbidity.

The inverse correlations observed between sleep duration and cardiovascular risk markers further strengthen the evidence supporting a dose-response relationship between sleep and cardiovascular health. Participants reporting shorter sleep duration consistently exhibited worse anthropometric, hemodynamic, metabolic, and inflammatory profiles. Similar dose-dependent associations have been reported in the UK Biobank study, which demonstrated progressive increases in cardiovascular risk with decreasing sleep duration [31]. Such findings reinforce the concept that sleep duration should be considered a continuous cardiovascular risk factor rather than a simple binary exposure.

The public health implications of these findings are substantial. Young adulthood represents a critical period for the establishment of lifelong health behaviors. Rapid urbanization, increased academic and occupational demands, prolonged screen exposure, and changing lifestyles have contributed to widespread sleep deprivation among young adults worldwide, including India [32]. Given that cardiovascular disease often develops silently over several decades, early identification and modification of sleep-related risk factors may provide an important opportunity for primordial prevention.

From a clinical perspective, routine assessment of sleep duration should be incorporated into cardiovascular risk evaluation among young adults. Sleep screening questionnaires can be easily implemented in primary care and preventive health settings. Individuals reporting chronic short sleep duration may benefit from targeted lifestyle interventions, sleep hygiene counseling, behavioral therapy, and cardiovascular risk monitoring. Incorporating sleep health into existing cardiovascular prevention programs may enhance the effectiveness of risk reduction strategies.

The present study possesses several strengths, including comprehensive assessment of anthropometric, hemodynamic, metabolic, and inflammatory markers within a relatively young population. However, certain limitations should be acknowledged. The cross-sectional design precludes causal inference, and sleep duration was assessed using self-reported measures, which may be subject to recall bias. The single-center nature of the study may limit generalizability to other populations. Nevertheless, the findings provide valuable evidence regarding the relationship between sleep duration and early cardiovascular risk among Indian young adults.

CONCLUSION

The present study demonstrated a significant association between sleep duration and early cardiovascular risk markers among young adults attending a tertiary care hospital in Gujarat, India. Participants reporting short sleep duration (<6 hours per night) exhibited significantly higher body mass index, waist circumference, systolic and diastolic blood pressure, fasting blood glucose, LDL cholesterol, triglycerides, Atherogenic Index of Plasma, and high-sensitivity C-reactive protein levels, along with lower HDL cholesterol concentrations, compared with individuals who reported normal sleep duration. Furthermore, a clear dose-response relationship was observed, indicating that cardiovascular risk progressively increased with decreasing sleep duration.

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