THE RELATIONSHIP BETWEEN SUBJECTIVE SLEEP QUALITY AND BLOOD PRESSURE AMONG UNIVERSITY STUDENTS

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ABSTRACT

Poor sleep quality among university students is often associated with academic stress, lifestyle habits, and irregular routines, which may influence blood pressure (BP). This study aimed to examine the relationship between sleep quality and BP in Indonesian university students. A descriptive correlational study with a cross-sectional design was conducted among 207 students at a private university in North Sulawesi Province of Indonesia. Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI) and BP was measured with an aneroid sphygmomanometer. Data analysis included descriptive statistics and Spearman correlation. The mean sleep quality score was 8.50 (SD=3.51) and 84.1% of participants were classified as having poor sleep quality. The mean systolic and diastolic BP were 107.76 mmHg (SD=13.61) and 75.91 mmHg (SD=11.91), respectively. A significant but weak negative correlation was observed between sleep quality and systolic BP (r=-0.191, p=0.006). While these findings differ from much of the global evidence linking poor sleep to higher BP, they highlight the complexity of sleep-BP interactions in young and healthy populations. These findings emphasize the importance of promoting better sleep hygiene, healthier lifestyle behaviors, and cardiovascular health strategies within university settings.

Keywords: Blood Pressure, Sleep Quality, University Students

INTRODUCTION

Sleep quality has emerged as a significant global health concern in recent years. Insufficient or poor-quality sleep is highly prevalent across diverse populations and has even been characterized as a public health epidemic with substantial social and economic costs. Research in the past five years continues to link inadequate sleep to myriad adverse outcomes, including an elevated risk of cardiovascular disease, higher blood pressure (BP), obesity, and diminished quality of life (Onyegbule, Muoghalu, Ofoegbu, & Ezeorah, 2025). Good sleep is essential for physiological restoration and homeostasis, yet modern lifestyle factors including technology use, stress, irregular schedules contribute to widespread sleep disturbances.

BP is another critical health indicator with major public health implications globally. Hypertension affects an estimated 1.4 billion people worldwide and remains the leading cause of cardiovascular morbidity and mortality (Dai et al., 2021; World Health Organization, 2021). The prevalence of hypertension has been rising in many regions including in East and Southeast Asia. For instance, national surveys report hypertension rates ranging from about 20% in some countries to over 40% in others and Indonesia faces a considerable hypertension burden, with

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studies indicating that more than two-fifths of Indonesian adults in mid-life have high BP (Zhou et al., 2021). Uncontrolled hypertension is a well-established risk factor for heart disease, stroke, and kidney failure, underscoring its public health impact. By contrast, hypotension is less frequently discussed at the population level, but it can also pose health risks. While mild hypotension is often asymptomatic and underrecognized, severe drops in BP can compromise perfusion to vital organs, leading to dizziness, fainting, or shock in acute cases (Chen, Sharma, & Bhattacharya, 2025). Thus, maintaining an optimal BP range is crucial, and both extremes (hypertension and hypotension) are relevant to public health, including in the Indonesian context.

A growing body of research has examined the relationship between sleep patterns and BP. Numerous epidemiological studies suggest that poor sleep may contribute to higher BP and increased risk of hypertension. For example, a recent longitudinal study of adolescents reported that those who met recommended sleep duration guidelines had a 37% lower risk of developing hypertension compared to peers with inadequate sleep (De Moraes, Ma, Nascimento-Ferreira, Hunt, & Hoelscher, 2024). Such findings align with biological expectations, given that insufficient sleep can activate stress responses that drive up BP. However, not all studies have found consistent associations, especially in young adult populations. Several investigations focusing on students and young adults have reported mixed results. A cross-sectional study of university students in Indonesia found no significant relationship between self-reported sleep quality and BP levels (Irwan et al., 2024). Similarly, Suling and Batubara (2023) observed no correlation between sleep quality and either systolic or diastolic BP in a sample of young university security staff, despite a high prevalence of poor sleep in that group. These inconsistent findings highlight that the sleep-BP link may be more complex in younger, healthier populations, and they underscore the need for further research.

Several physiological mechanisms and behavioral factors have been proposed to explain how poor sleep could influence BP regulation. Sleep deprivation is known to trigger sympathetic nervous system activation and stimulate the renin-angiotensin-aldosterone system (RAAs), leading to elevated catecholamine release, vasoconstriction, and increases in BP (Onyegbule et al., 2025). Concurrently, inadequate or fragmented sleep can disrupt the normal diurnal rhythm of the hypothalamic-pituitary-adrenal (HPA) axis, resulting in elevated stress hormone levels such as cortisol that further raise BP (De Moraes et al., 2024).

Despite the plausibility of this link, there are notable gaps in the literature, particularly regarding younger populations in non-Western settings. Relatively few studies have examined these associations in young adults, especially in Southeast Asia, which is undergoing epidemiologic transition and rising non-communicable disease burdens. This gap is salient given emerging data showing that a substantial proportion of young people in the region experience both poor sleep and BP issues. A recent systematic review found that approximately 64% of medical students in Southeast Asia have poor sleep quality, a prevalence higher than the global average for students (Satriono, How, Tahereh S, Wishwadewa, & Habilm, 2024). However, the interaction between sleep quality and BP in this demographic remains under-studied.

In light of existing gaps, this research addresses the underrepresentation of Southeast Asian youth in the sleep-BP literature by focusing on a young adult student population. The study is grounded in the rationale that university students

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often experience irregular schedules, academic stress, and lifestyle changes that can impair sleep, potentially influencing cardiovascular indicators such as BP. Understanding this relationship in the Indonesian context is important for developing targeted health promotion strategies, particularly through sleep hygiene education and stress management programs within campus health services, to improve sleep and support cardiovascular health. Ultimately, this study seeks to contribute local evidence to the global discussion on sleep and heart health and to inform culturally appropriate interventions for young adults in Indonesia. Therefore, the present study aimed to investigate the relationship between sleep quality and BP among university students in Indonesia.

RESEARCH METHODS

A descriptive correlational study with a cross-sectional design was conducted. The study population comprised students at a university in North Sulawesi, Indonesia, from January to March 2024. The required sample size was calculated using G*Power software version 3.1.9.7, with the following parameters: Exact test family, statistical test correlation (bivariate normal model), effect size 0.3 (medium), alpha error probability 0.05, and power 0.80 (Faul, Erdfelder, Buchner, & Lang, 2009). The minimum sample size required was 84 participants. However, a total of 207 students were recruited. Inclusion criteria were active undergraduate students in the first to fourth year who agreed to participate. Exclusion criteria included graduate students, those who were ill or absent during data collection, and those taking antihypertensive medications.

The study questionnaire consisted of two sections. The first section included demographic information such as age, gender, ethnic group, religion, relationship status, year of study, grade point average (GPA), major, student status, and place of residence. The second section assessed body mass index (BMI), exercise, caffeine consumption, parental history of hypertension, chronic illness, sleeping environment, prayer before sleep, and behaviors to facilitate sleep.

Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI) (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI is widely regarded as the international gold standard for measuring subjective sleep quality during the past month. It comprises 19 self-reported items that are categorized into seven components: Subjective sleep quality, latency, duration, efficiency, disturbances, use of sleep medication, and daytime dysfunction. Each component is rated from 0 to 3, generating a total score between 0 and 21, where values greater than 5 indicate poor sleep quality. The PSQI has been validated across multiple populations, including students with a reported Cronbach's alpha of 0.83 (Albqoor & Shaheen, 2021). In the present study, the Cronbach's alpha of the PSQI was 0.784.

Blood pressure (BP) was measured using a calibrated aneroid sphygmomanometer (OneMed) and a stethoscope (GEA). Measurements of BP were taken in a seated position after participants had rested for at least 5 minutes in a quiet room (in a campus setting during the day), with the arm supported at heart level. Both systolic and diastolic BP were recorded in mmHg (millimeters of mercury) and classified according to the guidelines of American Heart Association (2023). The categories included hypotension, normal, prehypertension, and hypertension stages 1, 2, and 3. Participants with systolic BP ≤90 mmHg and diastolic BP ≤60 mmHg were categorized as hypotensive. A normal BP classification was defined as systolic <120 mmHg and diastolic < 80 mmHg. Prehypertension

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was defined as systolic BP ranging from 120 to 129 mmHg with diastolic <80 mmHg. Hypertension stage 1 was identified when systolic BP was 130-139 mmHg or diastolic BP was 80-89 mmHg. Hypertension stage 2 was defined as systolic BP >140 mmHg or diastolic BP >90 mmHg, while hypertension stage 3 was categorized as systolic BP >180 mmHg or diastolic BP >120 mmHg.

Data were collected by trained research assistants (RA) under the supervision of the investigators to ensure consistency and minimize observer bias. Prior to data collection, assistants were briefed on standardized procedures for administering questionnaires and measuring BP. All data were recorded anonymously, and participants were assured that their responses would be kept confidential.

Ethical clearance was granted by the Research and Ethics Committee of a private university in West Java, Indonesia (No. 358/KEPK-FIK.UNAI/EC/I/24). All eligible students were informed about the study's aims and procedures, and those who agreed to take part signed written informed consent forms. Anonymity and confidentiality were assured, and participants retained the right to withdraw from the study at any stage without any consequences.

Data analysis was performed using the Statistical Package for the Social Sciences (SPSS), version 27 (IBM Corp., Armonk, NY). Data distribution was assessed through skewness, kurtosis, and histogram plots. Descriptive statistics were used to summarize the characteristics of participants. Spearman correlation tests were employed to assess the relationship between sleep quality and BP, as the data were not normally distributed. Statistical significance was set at $p<0.05$.

RESULTS AND DISCUSSION

RESULTS

The demographic characteristics, health status, and sleep habits of the participants are portrayed in Table 1. The mean age of the respondents was 21.01 years (SD=2.14). Most participants were female (62.3%) and from Minahasa (62.3%). In terms of religion, the majority identified as Adventist (58.9%). More than half of the respondents were single (61.4%) and in the fourth year of study (33.8%). Over half of the students reported a GPA between 3.51-4.00 (55.1%). Regarding labor status, most participants were labor students (66.2%), with the highest proportion living in their house (42.5%).

With respect to health and lifestyle, the mean BMI was 22.43 (SD=4.12), with most participants categorized as normal weight (62.8%). The majority did not engage in regular exercise (75.8%) and reported no chronic illness (93.7%). Most students denied caffeine consumption (77.8%) and parental history of hypertension (63.8%). Sleep habit findings indicated that most participants slept in a dark environment (85.5%), prayed before sleep (52.7%), and frequently facilitated sleep by watching (44.9%) or listening to music (35.3%).

Table 1 Distribution of Demographic Characteristics, Health Status, and Sleep Habits of Participants (n=207)

Variables	Categories	n (%)	Mean ± SD	Min-Max
Age			21.01 ± 2.14	17-27
Gender	Male	78 (37.7)		
	Female	129 (62.3)		
Ethnic group	Minahasan	129 (62.3)		
	Sangirnese	15 (7.4)		
	Mollucan	8 (3.9)		

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Variables	Categories	n (%)	Mean ± SD	Min-Max
	Torajanese	9 (4.3)		
	Papuanes	14 (7.2)		
	Javanese	6 (2.9)		
	Mongondownese	6 (2.9)		
	Bataknese	4 (1.9)		
	Others	15 (7.2)		
Religion	Adventist	122 (58.9)		
	Protestant	80 (38.6)		
	Others	5 (2.5)		
Relationship status	Single	127 (61.4)		
	Dating	77 (37.2)		
	Married	3 (1.4)		
Year of study	1st Grade	47 (22.7)		
	2nd Grade	47 (22.7)		
	3rd Grade	43 (20.8)		
	4th Grade	70 (33.8)		
Grade Point Average	≤2,50	1 (0.5)		
	2,51-2,99	25 (12.0)		
	3,00-3,50	67 (32.4)		
	3,51-4,00	114 (55.1)		
Major	Economics	47 (22.7)		
	Computer Science	43 (20.7)		
	Nursing	47 (22.7)		
	Philosophy	38 (18.4)		
	Agriculture	6 (2.9)		
	Education	14 (6.8)		
	Secretarial Studies	12 (5.8)		
Labor status	Non-Labor	70 (33.8)		
	Labor	137 (66.2)		
Place of residence	Dorm	61 (29.5)		
	House	88 (42.5)		
	Rent	55 (26.6)		
	Others	3 (1.4)		
BMI (kg/m^2)			22.43 ± 4.12	16-37
BMI category	Underweight	35 (16.9)		
	Normal	130 (62.8)		
	Overweight	31 (15.0)		
	Obesity	11 (5.3)		
Regular exercise	No	157 (75.8)		
	Yes	50 (24.2)		
Regular caffeine consumption	No	161 (77.8)		
	Yes	46 (22.2)		
Parental history of hypertension	No	132 (63.8)		
	Yes	75 (36.2)		
Chronic illness	No	194 (93.7)		
	Yes	13 (6.3)		
Sleeping in darkness environment	No	30 (14.5)		
	Yes	177 (85.5)		
Praying before sleep	Always	109 (52.6)		
	Often	66 (31.9)		
	Rarely	32 (15.5)		
Behaviors to facilitate sleep	Watching	93 (44.9)		
	Listening to music	73 (35.3)		

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Variables	Categories	n (%)	Mean ± SD	Min-Max
	Others	41 (19.8)		

Note: BMI=Body Mass Index; Kg=Kilogram; m=Meter.

The descriptive statistics of sleep quality components are shown in Table 2. The mean sleep duration of participants was 6.18 hours (SD=0.97), with the majority reporting 6-7 hours of sleep (38.2%). The mean sleep latency was 34.64 minutes (SD=47.82), and most students fell asleep within 16-30 minutes (41.1%). Regarding sleep efficiency, the mean habitual sleep efficiency was 84.1% (SD=17.21), with the majority achieving ≥85% (60.4%). Most students reported fairly good subjective sleep quality (51.7%) and almost half experienced 1-9 sleep disturbances (47.8%). More than half reported using sleep medication less than once a week (53.6%), while daytime dysfunction was mostly described as only a very slight problem (55.6%). Overall, the majority of participants had poor sleep quality (84.1%).

Table 2 Descriptive Statistics of Seven Sleep Quality Components (n=207)

Components	n (%)	Mean ± SD	Min-Max
Subjective Sleep Quality			
Very Good	31 (15.0)		
Fairly Good	107 (51.7)		
Fairly Bad	60 (29.0)		
Very Bad	9 (4.3)		
Sleep Latency (minutes)		34.64 ± 47.82	5-300
≤15 minutes	35 (16.9)		
16-30 minutes	85 (41.1)		
31-60 minutes	62 (30.0)		
>60 minutes	25 (12.0)		
Sleep Duration (hours)		6.18 ± 0.97	3-8
>7 hours	66 (31.9)		
6-7 hours	79 (38.1)		
5-6 hours	53 (25.6)		
<5 hours	9 (4.4)		
Habitual Sleep Efficiency (%)		84.1 ± 17.21	28-100
≥85%	125 (60.4)		
75-84%	31 (15.0)		
65-74%	27 (13.0)		
<65%	24 (11.6)		
Sleep Disturbances			
0	4 (1.9)		
1-9	99 (47.8)		
10-18	95 (45.9)		
19-27	9 (4.4)		
Use of Sleeping Medication			
Not during the past month	14 (6.8)		
Less than once a week	111 (53.6)		
Once or twice a week	69 (33.3)		
Three or more times a week	13 (6.3)		
Daytime Dysfunction			
No problem at all	29 (14.0)		
Only a very slight problem	115 (55.6)		

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Components	n (%)	Mean ± SD	Min-Max
Somewhat of a problem	57 (27.5)		
A very big problem	6 (2.9)		
Overall Sleep Quality			
Poor (6-21)	174 (84.1)		
Good (0-5)	33 (15.9)		

The scores of the seven components and overall sleep quality are presented in Table 3. The mean global score of sleep quality was 8.50 (SD=3.51), ranging from 1 to 18. The highest mean scores were observed in sleep disturbances component with a mean of 1.53 (SD=0.61) and use of sleeping medication at 1.39 (SD=0.71). In contrast, the lowest scores were found in sleep duration at 1.02 (SD=0.86) and habitual sleep efficiency at 0.76 (SD=1.07). All component scores ranged from 0 to 3.

Table 3 Scores of the Seven Components and Overall Sleep Quality (n=207)

Sleep Quality Components	Mean ± SD	Min-Max
Sleep Disturbances	1.53 ± 0.61	0-3
Use of Sleeping Medication	1.39 ± 0.71	0-3
Sleep Latency	1.37 ± 0.90	0-3
Subjective Sleep Quality	1.23 ± 0.75	0-3
Daytime Dysfunction	1.19 ± 0.71	0-3
Sleep Duration	1.02 ± 0.86	0-3
Habitual Sleep Efficiency	0.76 ± 1.07	0-3
Overall Sleep Quality Score	8.50 ± 3.51	1-18

The descriptive data of participants' blood pressure (BP) are displayed in Table 4. The mean systolic BP was 107.76 (SD=13.61), ranging from 80 to 180 mmHg, while the mean diastolic BP was 75.91 (SD=11.91), ranging from 50 to 190 mmHg. Based on classification, the majority of participants had normal BP 110 (53.1%), followed by prehypertension 38 (18.4%) and hypotension 36 (17.4%), while the remaining classifications were observed only in a few cases.

Table 4 Descriptive Data of Participants' Blood Pressure (n=207)

Variables	n (%)	Mean ± SD	Min-Max
Systolic BP (mmHg)		107.76 ± 13.61	80-180
Diastolic BP (mmHg)		75.91 ± 11.91	50-190
BP Classification			
Hypotension	36 (17.4)		
Normal	110 (53.1)		
Prehypertension	38 (18.4)		
Hypertension Stage 1	22 (10.6)		
Hypertension Stage 2	0 (0.0)		
Hypertension Stage 3	1 (0.5)		

Note: BP=Blood Pressure; mmHg=Millimeters of Mercury.

The correlation matrix of sleep quality components and participants' BP is depicted in Table 5. Overall sleep quality showed a significant negative correlation with systolic BP (r=-0.191, p=0.006). Similarly, sleep disturbances component were significantly and negatively correlated with systolic BP (r=-0.188, p=0.007).

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Other sleep quality components, including sleep latency, sleep duration, habitual sleep efficiency, use of sleeping medication, and daytime dysfunction did not show significant associations with either systolic or diastolic BP p>0.05).

Table 5 Correlation Matrix of Sleep Quality Components and Participants' Blood Pressure (n=207)

Variable	r/p	Systolic BP	Diastolic BP
Subjective Sleep Quality	r	-0.024	0.040
	p	0.726	0.570
Sleep Latency	r	-0.121	-0.078
	p	0.082	0.261
Sleep Duration	r	-0.133	-0.117
	p	0.056	0.093
Habitual Sleep Efficiency	r	-0.092	-0.045
	p	0.188	0.519
Sleep Disturbances	r	**-0.188****	-0.136
	p	**0.007**	0.051
Use of Sleeping Medication	r	-0.091	-0.089
	p	0.190	0.200
Daytime Dysfunction	r	-0.100	0.003
	p	0.150	0.971
Overall Sleep Quality Score	r	**-0.191****	-0.133
	p	**0.006**	0.057

Note: BP=Blood Pressure.

DISCUSSION

The present study found a modest but significant inverse relationship between sleep quality and systolic blood pressure (SBP) in a sample of Indonesian university students. Notably, poorer sleep quality was associated with lower SBP, meaning students who reported worse sleep tended to have slightly lower resting systolic pressures. This finding is somewhat counterintuitive in light of the broader literature. Numerous studies worldwide have documented that inadequate or disturbed sleep is usually linked to higher BP and increased hypertension risk. For example, a recent cohort study in Mexico reported that individuals with poorer self-reported sleep quality had about a 50% higher risk of developing hypertension over 30 months (Amezcua-Guerra et al., 2024). Similarly, Makarem et al. (2021) summarized that short sleep duration and sleep disturbances (like insomnia or sleep apnea) are associated with elevated BP and greater hypertension incidence. In adolescents, those with objectively poor sleep efficiency (low sleep quality) showed systolic pressures about 4 mmHg higher than their better-sleeping peers (Kohyama, 2021). A study in Japan by Taira, Fukumine, and Nakamura (2024) likewise found that adults under 50 who reported poor sleep quality had significantly higher mean SBP than those with good sleep (127.8 vs 121.0 mmHg). Taken together, the prevailing evidence from global cohorts to regional studies indicates that poor sleep is typically a risk factor for elevated BP, not a protective factor.

The significant negative correlation between sleep disturbances and SBP in this study indicates that students reporting more fragmented or disturbed sleep tended to have slightly lower resting SBP. This finding is somewhat counterintuitive in light of broader research, which generally links poorer sleep with higher BP. Numerous recent global studies have documented that insufficient or disrupted sleep correlates with elevated SBP and greater hypertension risk. For example,

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meta-analytic data and large cohort studies show that short sleep duration or poor sleep quality is associated with increased odds of developing hypertension (C. Li & Shang, 2021) and hypertensive individuals often report worse sleep than normotensives (L. Li et al., 2020). Asian findings echo this pattern, research in China and Southeast Asia has found a high prevalence of poor sleep among patients with hypertension and linked sleep complaints to higher BP and cardiovascular risk (L. Li et al., 2020; Saat et al., 2021). Not all studies, however, are uniform in their conclusions. A cross-sectional study of young adults including a recent Indonesian sample found no significant sleep-BP relationship (Suling & Batubara, 2023), hinting that age, lifestyle, or methodological factors can modulate the sleep-BP nexus.

One possible reason for this discrepancy may lie in the unique characteristics of our young, normotensive sample. The majority of participants were healthy students in their late teens or twenties, an age group that may respond differently to sleep challenges than older adults. Indeed, some research suggests that acute sleep deprivation raises BP in older individuals but has a blunted effect in younger adults. In the Japanese study mentioned above, the association between poor sleep quality and higher SBP was present in those aged ≤49 but not in those ≥50, with a significant interaction by age (Taira et al., 2024). Young adults might experience more short-term resilience or adaptive physiological responses to sleep loss. For instance, fatigue and reduced alertness from insufficient sleep could lead to transient decreases in daytime sympathetic tone or activity levels, thereby lowering BP in the short run. It is notable that chronically hypotension can itself cause symptoms like dizziness, lethargy, and fatigue, which might negatively affect one's perceived sleep quality (American Heart Association, 2024). In our sample, students with relatively lower SBP could have experienced more daytime fatigue or sluggishness, potentially contributing to complaints of poor sleep. This raises the intriguing possibility of reverse causation or a bi-directional link i.e. individuals with lower BP (within a normal range) might sleep worse due to subtle symptoms of tiredness, even as, conventionally, poor sleep would be expected to raise BP. Such complexities highlight that the sleep-BP relationship is not purely one-directional and may differ by context and population.

Behavioral and lifestyle factors in this university student cohort may also help explain the unexpected inverse correlation. It is plausible that students reporting poor sleep were those juggling greater academic stress, irregular schedules, or multiple commitments. Paradoxically, these same individuals might engage in more daytime physical activity such as commuting, part-time work, sports or skip meals due to time constraints, factors that could lower their resting BP. In contrast, students with good sleep quality might have more regular routines that include greater caloric intake or less daytime activity, possibly resulting in slightly higher SBP. Some unmeasured confounders such as detailed data on diet, exercise, or body mass index (BMI) could create a scenario where the usual positive link between poor sleep and high BP is dampened or even reversed. Supporting this notion, an Indonesian study on nursing students found that high BMI was associated with both poorer sleep and higher BP, suggesting that obesity can mask or modify the sleep-BP relationship (Irwan et al., 2024). In our generally lean student sample, the absence of many hypertension risk factors might have led the minor influences of sleep to manifest differently. Another consideration is the distinction between acute and chronic effects of poor sleep. Our measurement captured current sleep quality and current BP. It is possible that acute sleep loss,

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for example cramming for exams, produces a temporary drop in BP due to exhaustion, whereas long-term habitual poor sleep would, if sustained, contribute to hypertension via cumulative stress on the cardiovascular system (Makarem et al., 2021).

Physiologically, several mechanisms have been established that link sleep quality with BP regulation. Sleep is a critical period for cardiovascular recovery: during normal restful sleep, BP typically dips 10-20% as parasympathetic activity increases and sympathetic output declines. Poor sleep quality, whether due to insomnia, fragmented sleep, or sleep disorders can disrupt this nightly BP dipping pattern (Su, Li, Long, He, & Ding, 2021). Insufficient or fragmented sleep triggers sustained sympathetic nervous system activation, elevating heart rate and peripheral vascular resistance, which in turn raises BP. Sleep deprivation also activates the hypothalamic-pituitary-adrenal (HPA) axis, leading to elevated cortisol and catecholamine levels that raise BP and heart rate beyond normal circadian levels. Additionally, chronic poor sleep is pro-inflammatory, increases inflammatory cytokines and oxidative stress, which promote endothelial dysfunction and arterial stiffness over time. These changes can blunt the vasodilatory capacity of blood vessels and contribute to sustained hypertension. Short or poor sleep has further been linked to metabolic dysregulation, including impaired glucose tolerance and increased appetite for calorie-dense foods, eventually contributing to weight gain and metabolic syndrome, which are major drivers of high BP (Makarem et al., 2021). In epidemiologic studies, people with chronically poor sleep often have greater odds of developing hypertension, as seen in the Chinese population where patients with hypertension are almost 3 times more likely to report poor sleep than normotensive individuals (L. Li et al., 2020). It is important to recognize, however, that these mechanisms typically manifest in the long term. In a young healthy population like ours, the acute physiological effects of one or a few nights of bad sleep might differ, and homeostatic systems (baroreflexes, renal fluid balance, etc.) may compensate to maintain BP or even overshoot in the opposite direction. Thus, while the global consensus is that poor sleep quality has deleterious effects on cardiovascular health (often raising BP) (Amezcua-Guerra et al., 2024; Makarem et al., 2021), our findings suggest that context such as age, health status, and acuteness of sleep disruption can modulate the direction and magnitude of the association.

It is also instructive to compare our results with those from similar cohorts in the region. Evidence from Asia and Southeast Asia largely aligns with global trends, with poor sleep linked to higher BP. A meta-analysis in China reported that over half of hypertensive patients had bad sleep quality, and case-control data confirmed significantly worse sleep among hypertensives compared to healthy controls (L. Li et al., 2020). In Japan, as noted, subjective sleep complaints even among individuals with normal sleep duration were associated with elevated SBP in younger adults (Taira et al., 2024).

Meanwhile, some Southeast Asian studies in young populations have found no significant relationship between sleep quality and BP, which is closer to a null finding rather than an inverse relationship. For instance, a recent survey of 45 college students in West Sulawesi, Indonesia found no association between PSQI-measured sleep quality and BP (Irwan et al., 2024). Likewise, Suling and Batubara (2023) reported no correlation between sleep quality and either systolic or diastolic BP in a group of Jakarta security officers working night shifts. Those null results were attributed to limited sample sizes and the young, predominantly

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normotensive status of participants. Our study, with a larger sample and a significant finding, adds a new dimension by revealing an inverse correlation in this context. One speculative explanation is that cultural or behavioral factors unique to our student sample such as communal living in dorms, academic stress patterns, and caffeine use produced an atypical physiological response. It is conceivable that mild sleep deprivation in these students led to lower morning BPs due to extended resting states or delayed diurnal activation. Alternatively, the finding could be a statistical anomaly or reflect subtler biases. For instance, students who perceived their sleep as poor might simply have been more health-conscious or relaxed during the BP measurement, inadvertently yielding lower readings. Importantly, the correlation was weak, indicating considerable individual variation. Thus, while our data show a significant trend, it should not be over-interpreted as evidence that poor sleep improves BP. Rather, it highlights that the relationship between sleep and cardiovascular physiology is complex and can be influenced by a web of factors in a given population.

Several limitations must be considered when interpreting these findings. The cross-sectional design precludes any causal inference. We measured sleep quality and BP at a single point in time, so it is unclear whether poor sleep led to changes in SBP, or if inherent BP levels (or other confounders) influenced sleep perceptions. Sleep quality was assessed via self-report questionnaire, which is subjective in nature. Participants' perceptions of sleep may be influenced by mood, stress, or other biases. We only examined resting BP during the day (in a campus setting) without accounting for potential 24-hour BP patterns. Our sample consisted of young adults in a specific region of North Sulawesi and was relatively homogeneous in age and lifestyle. This limits the generalizability of the results to other age groups or populations.

CONCLUSION AND RECOMMENDATION

The majority of participants in this study reported poor sleep quality, while most maintained normal BP. A significant negative correlation was identified between sleep quality and systolic BP, but no association was observed with diastolic BP, with students showing lower BP experiencing more sleep disturbances. These findings suggest the need for future longitudinal studies using objective sleep assessments to better clarify causal mechanisms. Broader samples across multiple institutions are also recommended to enhance generalizability. At the practical level, universities should promote programs targeting sleep hygiene, stress management, and healthy lifestyle behaviors to improve students' cardiovascular health.

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