DEVELOPMENTAL MEDICO-LIFE-SCIENCES

ISSN (P): 3007-2786, (E): 3007-2794 DOI: https://doi.org/10.69750/dmls.03.01.0178

ORIGINAL RESEARCH ARTICLE Open Access

Blood Pressure Variability and Stress Hormone Alterations in Chronically Sleep-Deprived Adults: A Comparative Study

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ABSTRACT

Background: Chronic sleep deprivation has emerged as a common lifestyle-related condition with important implications for cardiovascular health. Beyond absolute blood pressure values, blood pressure variability and neuroendocrine stress activation are increasingly recognized as early markers of cardiovascular risk. However, integrated clinical evidence linking chronic sleep deprivation with blood pressure variability and stress hormone alterations remains limited.

Objective: To assess and compare blood pressure variability and stress hormone levels in chronically sleep-deprived adults and individuals with normal sleep duration.

Methods: This comparative cross-sectional study was conducted from January 2025 to July 2025 at a tertiary care hospital. Eighty adults aged 25–55 years were enrolled and divided into two groups: chronically sleep-deprived participants (sleep duration <6 hours/night for ≥6 months; n=40) and normal sleepers (7–8 hours/night; n=40). Twenty-four-hour ambulatory blood pressure monitoring was performed to evaluate mean blood pressure and blood pressure variability indices. Morning fasting blood samples were collected for estimation of serum cortisol, plasma epinephrine, and norepinephrine. Statistical analysis included independent sample t-tests and Pearson correlation analysis.

Results: Mean ambulatory blood pressure values were comparable between groups. However, systolic and diastolic blood pressure variability were significantly higher in chronically sleep-deprived adults ($p<0.001$). Stress hormone levels were markedly elevated in the sleep-deprived group ($p<0.001$). Blood pressure variability showed strong positive correlations with cortisol and norepinephrine levels.

Conclusion: Chronic sleep deprivation is associated with increased blood pressure variability and heightened neuroendocrine stress, indicating early cardiovascular dysregulation.

Keywords: Sleep deprivation; Blood pressure variability; Cortisol; Catecholamines; Cardiovascular risk

Received: 10/08/2025
Revised: 11/12/2025
Accepted: 22/01/2026
Published: 31/01/2026

INTRODUCTION

Sleep is a primal biological activity that plays a critical role in cardiovascular, metabolic, and neuroendocrine homeostasis [1]. The effective regulation of the activity of the autonomic nervous system, the balance of hormones, the vascular tone, and the circadian rhythms necessitate proper sleep duration and the level of sleep quality. But one of the most recent and disturbing phenomena in the global population has been the chronic sleep inadequacy that has been caused by modern patterns of living, long working hours, psychosocial factors, screen time, and urbanization. The increasing epidemic of inadequate sleep has brought serious health questions in the minds of the general population, especially in the long-term cardiovascular outcomes [2,3].

This has been established by epidemiological and experimental studies that show that short duration of sleep is associated with a higher likelihood of developing hypertension, ischemic heart disease, stroke, and general cardiovascular mortality [4]. Although the mean blood

pressure values were a conventional measure of cardiovascular risk, recent studies indicate that blood pressure variability is a highly predictive measure of cardiovascular morbidity and mortality, just as the mean blood pressure values. The blood pressure variability is the manifestation of dynamic interrelation between the neural, hormonal, and vascular processes and indicates both short-term autonomic instability and long-term vascular dysfunction [5].

Physiologically, the normal sleep is very important in blood pressure dipping at night and sympathetic withdrawal. Persistent sleep deprivation impairs this balance causing continuous sympathetic nervous system and lowered parasympathetic tone [6]. This autonomic imbalance is a contributor to the exaggerated changes in blood pressure, dysfunction of the baroreceptors, and disturbed circadian rhythms in blood pressure. The linkage between heightened variability of blood pressure and the endothelial dysfunction, arterial hypertonicity, left ventricular hypertrophy, and rapid atherosclerosis has been observed even in people without ongoing hypertension [7].

Simultaneously, sleep deprivation has severe neuroendocrine stress impacts, in particular, the hypothalamic-pituitary-adrenal axis and the sympathetic-adrenomedullary system. The long-term sleep deprivation causes high concentrations of stress hormones: cortisol, epinephrine, and norepinephrine [8]. These hormones stimulate vasoconstriction, sodium retention, accelerated heart rate, and inflammation of the vasculature and can lead to an unstable blood pressure regulation. Continuously high levels of cortisol also disrupt normal circadian rhythms and lead to metabolic and inflammatory dysregulation [9].

Although the detrimental effect of sleep deprivation on the cardiovascular system has become widely recognized, the lack of clinical studies that constantly compare blood pressure changes with the indicators of biochemical stress in the chronically sleep-deprived adults remains rather minimal [10]. The vast majority of the current studies have paid attention to either the average values of blood pressure or hormonal variations independently without understanding fully its underlying pathophysiological correlations. Moreover, there is little information on data of the hospital-based adult populations in the developing world, although sleep deprivation is a prevalent issue in this context [11].

Hence, the current research was aimed at comparatively assessing blood pressure change and stress hormone dynamics among sleep-deprived adults in the chronic state compared to healthy normal sleepers. Harnessing the synergy of ambulatory blood pressure measurement with biochemical measurement of stress hormones, this study will shed more light on the early cardiovascular dysregulation related to chronic sleep deprivation and will draw attention to its possible status as a modifiable risk factor of cardiovascular disease [12].

MATERIALS AND METHODS

The study was a comparative cross-sectional study conducted within the Department of Physiology in partnership with the Department of Medicine at a tertiary care hospital within a duration of seven months, between January 2025 and July 2025. The aim was to assess the fluctuations of blood pressure and changes in stress hormones in the chronically sleep-deprived adults compared to those with the normal time of sleep.

Eighty adult individuals were recruited in the research applying a non-probability consecutive sampling method. The participants were separated into two equal groups. Group A was comprised of 40 sleep-deprived adults, characterized as people who had an average of less than 6 hours of nocturnal sleep per night on six or more months of continuous sleep deprivation. Group B was comprised of 40 age- and sex-matched normal sleepers, those who indicated that they have a normal sleep duration of 7-8 hours a night and that they have had 6 months of that duration.

Participants were adults aged 2555 years of both sexes, who appeared healthy and were ready to sign informed consent in writing. The participants had to have a consistent sleeping pattern in the past six months. Individuals were not included when they were shift workers, had a known diagnosis of sleep disorders, including obstructive sleep apnea as well as a previous diagnosis of hypertension, diabetes mellitus, chronic kidney disease, chronic liver disease, ischemic heart disease, or any other chronic systemic illness. Individuals who have been taking any drug that is known to alter blood pressure or the level of stress hormone such as antihypertensives, corticosteroids, antidepressants, or sympathomimetic medication were also excluded. There were also exclusion criteria of pregnant women, smokers, substance abusers, and acute-illness and people within four weeks of hospitalization.

A validated sleep questionnaire was used to assess sleep duration and, the participants were requested to keep a 14 days sleep diary before enrolling in order to establish habitual sleep duration. The operational definition of chronic sleep deprivation was an average of less than six hours per night of sleep in a period of six months and more and normal sleep was defined as an average of seven to eight hours per night during the same time.

The baseline demographic and clinical data were taken after the enrollment (age, gender, occupation, body mass index, caffeine consumption, and physical activity level). Office blood pressure was recorded by use of a calibrated sphygmomanometer and the average of two was recorded following a period of five minutes of rest in a seated position.

The subjects were all subjected to 24 hours of ambulatory blood pressure on an approved automated machine on the non dominating arm. Accuracy of blood pressure measurements was recorded after every 30 minutes during the day (07:0022:00) and after every 60 minutes during the night (22:0007:00). There was a recommendation that the participants should carry on with their daily

activities and should not engage in strenuous exercises during the period of monitoring and that the arm should be kept still when taking measurements. Ambulatory blood pressure monitoring supplied mean blood pressure (systolic and diastolic), and the index of the variability of blood pressure. The change in blood pressure was determined using the standard deviation of the systolic and diastolic blood pressure during the 24 hour, day and night.

To conduct a biochemical analysis, morning venous blood samples were taken (at 08:00-09:00 AM) following 8 -10 hours of overnight fast to reduce circadian changes. Respondents were advised not to consume caffeine and engage in intensive physical exercise within a period of 12 hours prior to the collection of samples. Cortisol levels were determined in serum and catecholamine (epinephrine and norepinephrine) were measured in plasma by the standardized use of enzyme-linked immunosorbent assay (ELISA) and immunoassay methods according to the manufacturer specifications. The procedure was applied and samples were centrifuged and stored under the relevant temperatures until further analysis and the internal quality control procedure was adhered to throughout the process.

Variations in blood pressure variability measures and stress hormone levels before and after the experiment were the major measures of outcome. The secondary outcomes were the correlations of the parameters of blood pressure variability with the levels of circulating stress hormones.

The SPSS version 26 was used in data analysis. Continuous variables were stated as mean, standard deviation, whereas the categorical variables were stated as frequencies and percentages. They were compared on the basis of independent samples t-test, and Pearson correlation analysis was conducted to determine the relationship between the variability of blood pressure and the level of stress hormones. The p-value below 0.05 was taken to be statistically significant.

Institutional Ethical Review Committee granted ethical approval of the study (ERC/PRCMDC/2024/129). All the participants were provided with informed consent in written form before collecting data. Participant information was strictly held in confidence and informed consent was given to any clinically important information, and participants were referred to seek the necessary medical attention when necessary.

RESULTS

The final analysis involved 80 participants (40 sleep-deprived chronically and 40 normal sleepers). Every registered participant passed through ambulatory blood pressure monitoring and biochemical evaluation and did not miss any data because of technical and procedural reasons.

Demographic and clinical baseline data: Table 1 summarizes the baseline demographic and clinical characteristics of the study population. The average age of participants in the chronically sleep-deprived group was 41.3 +/- 7.2 years, vs. 40.6 +/- 6.9 years in the control and there was no statistically significant difference ($p = 0.64$). The gender was similar in terms of representation in the groups where males had majority of 55 percent of the sleep-deprived group and 52.5 percent of the control group ($p = 0.82$). The mean body mass index was insignificantly different among sleep-deprived group (26.1 ± 3.4 kg/m 2) and the control group (25.7 ± 3.2 kg/m 2, $p = 0.56$). Both groups had resting office systolic and diastolic blood pressure values within the normotensive range, and no statistically significant differences were observed. The mean sleep time was found to differ significantly among the groups so that the group classification was correct.

Ambulatory blood pressure parameters and blood pressure variability: Table 2 presents the data on 24-hours ambulatory blood pressure monitoring. The 24-hour systolic and diastolic mean values of blood pressure were marginally more in the chronically sleep-deprived condition compared to the controls; nevertheless, the results were not statistically significant. On the contrary, the indices of blood pressure variability showed significant and statistically significant differences across groups.

The sleep-deprived group had a significant standard deviation of systolic blood pressure variability (14.8 ± 3.1 mmHg) than the control group (10.9 ± 2.4 mmHg, $p < 0.001$). On the same note, the 24-hour diastolic blood pressure changes were found to be significantly higher in sleep-deprived (11.6 ± 2.6 mmHg) than normal sleepers (8.7 ± 1.9 mmHg, $p < 0.001$). Daytime systolic blood pressure variability in sleep-deprived group (13.9 ± 2.9 mmHg) was also significantly high than in controls (10.2 ± 2.2 mmHg, $p < 0.001$). The same was the case with night-time systolic blood pressure change with sleep-deprived people (12.1 ± 2.7 mmHg) demonstrating higher values compared to normal sleepers (8.8 ± 1.8 mmHg, $p < 0.001$).

Circadian blood pressure patterns were analyzed, and they found that 45% of sleep-deprived individuals also demonstrated the non-dipping blood pressure pattern, whereas 20% of control group participants did not ($p = 0.02$).

Stress hormone levels: Table 3 presents the comparison of circulating stress hormone concentrations in study groups. The sleep-deprived participants showed a significantly greater level of serum cortisol (19.612 ± 4.3 3g/dl) than normal sleepers (13.813 ± 3.1 3g/dl, $p = 0.001$). There was also a significant rise of plasma epinephrine levels in the sleep-deprived (72.4 ± 15.8 pg/mL) as compared to the control group (49.7 ± 12.3 pg/mL, $p = 0.001$). In the same fashion, plasma norepinephrine levels were very high in sleep-deprived (412.6 ± 86.4 pg/mL) than normal sleepers (298.9 ± 74.2 pg/mL, $p < 0.001$).

Correlation between blood pressure variability and stress hormones: Correlation analysis of the whole study population reported significant positive relationships between the indexes of variations of blood pressure and the level of the stress hormone, and they can be summarized in

Table 4. The 24h systolic blood pressure variation had a significant positive relationship with serum cortisol ($r = 0.62$, $p < 0.001$) and plasma norepinephrine ($r = 0.58$, $p < 0.001$). The systolic blood pressure variability and plasma epinephrine levels were found to have a moderate yet statistically significant relationship ($r = 0.44$, $p = 0.001$).

In the same way, the variability of the diastolic blood pressure showed significant positive relationships with serum cortisol ($r = 0.55$, $p < 0.001$), plasma norepinephrine ($r = 0.51$, $p < 0.001$), and plasma epinephrine ($r = 0.41$, $p = 0.002$).

Table 1: Baseline demographic and clinical characteristics of study participants

Variable	Sleep-Deprived Group (n=40)	Control Group (n=40)	p-value
Age (years)	41.3 ± 7.2	40.6 ± 6.9	0.64
Male/Female (n)	22 / 18	21 / 19	0.82
Body mass index (kg/m²)	26.1 ± 3.4	25.7 ± 3.2	0.56
Office SBP (mmHg)	122.4 ± 8.1	120.9 ± 7.6	0.38
Office DBP (mmHg)	78.6 ± 5.9	77.8 ± 5.6	0.51
Average sleep duration (hours/night)	5.2 ± 0.6	7.4 ± 0.5	<0.001

Table 2: Ambulatory blood pressure parameters and blood pressure variability

Parameter	Sleep-Deprived Group (n=40)	Control Group (n=40)	p-value
24-hour mean SBP (mmHg)	124.8 ± 9.3	121.6 ± 8.7	0.09
24-hour mean DBP (mmHg)	80.2 ± 6.4	78.9 ± 6.1	0.28
24-hour SBP variability (SD)	14.8 ± 3.1	10.9 ± 2.4	<0.001
24-hour DBP variability (SD)	11.6 ± 2.6	8.7 ± 1.9	<0.001
Daytime SBP variability (SD)	13.9 ± 2.9	10.2 ± 2.2	<0.001
Night-time SBP variability (SD)	12.1 ± 2.7	8.8 ± 1.8	<0.001
Non-dipper pattern, n (%)	18 (45%)	8 (20%)	0.02

Table 3: Comparison of stress hormone levels between study groups

Hormone	Sleep-Deprived Group (n=40)	Control Group (n=40)	p-value
Serum cortisol (µg/dL)	19.6 ± 4.3	13.8 ± 3.1	<0.001
Plasma epinephrine (pg/mL)	72.4 ± 15.8	49.7 ± 12.3	<0.001
Plasma norepinephrine (pg/mL)	412.6 ± 86.4	298.9 ± 74.2	<0.001

Table 4: Correlation between blood pressure variability and stress hormone levels (n=80)

Variable	Serum Cortisol	Epinephrine	Norepinephrine
24-hour SBP variability	r = 0.62, p<0.001	r = 0.44, p=0.001	r = 0.58, p<0.001
24-hour DBP variability	r = 0.55, p<0.001	r = 0.41, p=0.002	r = 0.51, p<0.001

Comprehensively, sleep-deprived adults showed a tremendous high blood pressure variability at 24 hours, during the day, and during the night, as well as the prevalence of the non-dipping blood pressure patterns. These changes were also accompanied by an extreme high level of circulating stress hormone. There existed strong positive relationships between indices of variability of blood pressure and levels of stress hormones.

DISCUSSION

A current comparative analysis shows that chronic sleep deprivation is linked with pronounced irregularities in blood pressure regulation and neuroendocrine reactions to stress, even in adults who are normotensive by office criteria [13]. The most notable discoveries of this research are the significantly higher blood pressure variability in short term, the more pronounced non-dipping blood pressure profile, and the considerably higher levels of the circulating stress hormones- cortisol, epinephrine, and norepinephrine in sleep deprived people as compared to normal sleepers. Noteworthy, there was a positive correlation that was strong, between the indices of variability in blood pressure and the stress hormones, indicating that there exists a tight physiological relationship between the autonomic endocrine activation and the variability in blood pressure [14].

Blood pressure variability has developed as a strong indicator of cardiovascular risk, even without considering the average blood pressure values [15]. Although mean 24-hour systolic and diastolic blood pressure were marginally increased and statistically insignificant between the groups in this study, blood pressure change was greatly high across 24-hour, daytime, and nighttime in sleep-deprived subjects. The implication of this discovery is that continuous sleep deprivation has a more profound impact on dynamic blood pressure regulation than on the stable blood pressure level. This instability is evidence of a dysfunctional autonomic buffering, decreased parasympathetic, and an increase in sympathetic drive, all of which predispose to a vascular injury and target-organ damages in the long term [16].

The more frequent incidence of non-dipping blood pressure patterns among sleep-deprived adults is also evidence pointing to the existence of circadian disregulation. Normal sleep is connected with nocturnal sympathetic withdrawing and physiological reduction of blood pressure [17]. The depletion of chronic sleep disturbs

this rhythm, causing long-term nocturnal sympathetic stimulation and inhibition of nocturnal blood pressure decrement. The clinical relevance of this finding has been underscored by consistent associations between non-dipping status and left ventricular hypertrophy, stroke, and renal damage, and even increased mortality of the heart [18].

Neuroendocrine assessment indicated that the level of serum cortisol and plasma catecholamine was severely high in sleep-deprived persons in the chronic state. The findings denote the continuity of the hypothalamic-pituitary-adrenal axis and sympathetic-adrenomedullary axis [19]. Cortisol is a key player in cardiovascular regulation by increasing the vascular sensitivity to catecholamines, increasing sodium and water retention, and endothelial dysfunction. In a similar manner, the presence of high levels of norepinephrine and epinephrine elevates heart rate, peripheral vascular resistance, and arterial stiffness which raise the blood pressure variability [20].

The close interrelations between the level of stress hormones and variability in blood pressure give a mechanistic understanding of how chronic sleep deprivation can be a contributing factor to premature cardiovascular malfunction [5]. An increase in cortisol and norepinephrine was significantly related to both systolic and diastolic blood pressure variation, which indicates that long-term neuroendocrine stress is a key contributor to blood pressure variability. These results are in line with experimental sleep restriction studies that reported augmented sympathetic nerve activity, diminished sensitivity of the baroreceptor and modified circadian hormonal rhythms after sleep deprivation [11,13].

Clinically, this study has implications of showing that even prior to the onset of overt hypertension, adverse cardiovascular changes may take place in sleep-deprived individuals [6]. Conventional clinic blood pressure measurements can be incapable of identifying this early risk, but ambulatory blood pressure measurements and variability enable more sensitive predictors of subclinical cardiovascular stress. These results have clear implications on the general health of the population in general given the high level of chronic sleep deprivation among working-age adults [14].

The duration of sleep is also identified as another risk factor that can be changed in the study. In contrast to most of the conventional cardiovascular risk factors, sleep behavior is subject to modification of lifestyle, behavior and change of workplace policies. The prompt detection of sleep-deprived people with coupled fluctuations in blood pressure has the potential to permit the implementation of preventive measures in time before it is too late to act upon the probable long-term cardiovascular risks [1-5].

It has some limitations that should be mentioned. The duration of sleep was measured through questionnaires, sleep diaries and not through objective measurement like actigraphy or polysomnography. The cross-sectional design does not allow causation and longitudinal research is required to establish whether normalization of the sleep duration results in a further increase in the blood pressure variances and stress hormone patterns. Other variables like psychological stress and dietary sodium were not directly measured either and could have played a role in the observed correlations [19,20].

CONCLUSION

This investigation indicates that chronic sleep deprivation is linked to strong amplification of blood pressure variability, a greater occurrence of non-dipping blood pressure types, and pronounced stress hormone elevations in adults with normotensive blood pressure. These close correlations between the variability of blood pressure and neuroendocrine stressors indicate that persistent autonomic and hypothalamic-pituitary-adrenal activation is to play a key role in blood pressure inconsistency regarding inadequate sleep. These results highlight the role of sleep in cardiovascular regulation and justify the use of sleep assessment in cardiovascular risk assessment. Treatment of chronic sleep deprivation could be one of the effective and non-pharmacological ways of preventing cardiovascular disease in the early stages.

Conflict of Interest: The authors report no conflicts of interest related to this study.

Funding: No external funding was received for the conduct of this study.

Acknowledgments:

The authors acknowledge the support of Pak Red Crescent Medical & Dental College, Lahore, Pakistan, for facilitating participant recruitment, data collection, and laboratory investigations. The contribution of all clinical, nursing, and technical staff involved in participant evaluation and data acquisition is gratefully appreciated.

Authors' Contributions:

A.S. conceived the study, designed the methodology, and supervised manuscript drafting.

H.I. contributed to data collection, biochemical analysis, and preliminary manuscript preparation.

M.H.G. performed data interpretation, literature review, statistical validation, and critical revision of the final manuscript.

All authors read and approved the final version of the manuscript.

Data Availability Statement: The data used in this study are available from the corresponding author upon reasonable request, in accordance with ethical and institutional guidelines.

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This article may be cited as: Shahid A, Ibrahim H, Gulzar MH. Blood pressure variability and stress hormone alterations in chronically sleep-deprived adults: a comparative study. *Dev Med Life Sci*. 2026;3(1):3-8. doi:10.69750/dmls.03.01.0178