INSIGHTS-JOURNAL OF
LIFE AND SOCIAL SCIENCES

INSIGHTS-JLSS
Insights-Journal of Life and Social Sciences

CORRELATION BETWEEN SALT SENSITIVITY AND BLOOD PRESSURE VARIABILITY IN PRE-HYPERTENSIVE ADULTS

Original Article

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Conflict of Interest: None

Grant Support & Financial Support: None

Acknowledgment: The authors sincerely thank all participants for their valuable contribution to this research.

ABSTRACT

Background: Salt sensitivity is recognized as a significant modifier of blood pressure and cardiovascular risk. However, its relationship with daily blood pressure variability (BPV) in pre-hypertensive adults remains underexplored.

Objective: To investigate the association between dietary salt sensitivity and 24-hour blood pressure variability among pre-hypertensive individuals.

Methods: A cross-sectional study was conducted on 100 pre-hypertensive adults aged 18–45 years. Participants underwent a standardized low-sodium and high-sodium dietary intervention to determine salt sensitivity, defined as a ≥10 mmHg change in mean arterial pressure. Ambulatory blood pressure monitoring (ABPM) over 24 hours was used to assess systolic and diastolic BPV. Pearson's correlation and multivariate regression analyses, adjusting for age, BMI, smoking status, and baseline BP, were utilized to explore relationships.

Results: Forty-five participants (45%) were identified as salt-sensitive. Salt-sensitive individuals exhibited significantly higher 24-hour systolic (15.2 ± 3.9 mmHg) and diastolic BP variability (11.6 ± 2.7 mmHg) compared to salt-resistant individuals (11.4 ± 3.1 mmHg and 8.5 ± 2.2 mmHg, respectively; $p < 0.001$). Pearson's correlation demonstrated a moderate positive relationship between salt sensitivity and systolic ($r = 0.48$, $p < 0.001$) and diastolic BPV ($r = 0.42$, $p < 0.001$). Regression analysis confirmed salt sensitivity as an independent predictor of both systolic ($\beta = 0.41$, $p < 0.001$) and diastolic BPV ($\beta = 0.36$, $p = 0.002$).

Conclusion: Salt sensitivity is significantly associated with greater daily blood pressure variability in pre-hypertensive adults. Early identification and targeted sodium reduction strategies in salt-sensitive individuals may offer a practical approach to stabilize blood pressure patterns and reduce cardiovascular risk.

Keywords: Adult, Blood Pressure Monitoring, Blood Pressure Variability, Cross-Sectional Studies, Hypertension Prevention, Prehypertension, Salt Sensitivity, Sodium Intake.

INTRODUCTION

Salt sensitivity has emerged as a critical but often underappreciated factor in the development and progression of hypertension, a condition that affects nearly one-third of the adult population globally. While hypertension itself has been the focus of countless studies, the subtle variations in blood pressure observed among individuals even before the threshold of clinical hypertension is reached warrant closer examination (1). Pre-hypertensive individuals, characterized by systolic blood pressures between 120–139 mmHg or diastolic pressures between 80–89 mmHg, represent a particularly vulnerable group (2). Identifying early modifiable factors in this population could yield substantial benefits in reducing the future burden of cardiovascular disease (3). Among the many variables influencing blood pressure, dietary salt intake, and more specifically an individual's sensitivity to it, appears to play a pivotal role. Despite extensive epidemiological evidence linking high salt intake to elevated blood pressure, the concept of salt sensitivity and its association with daily blood pressure variability remains less clearly understood, particularly in pre-hypertensive adults (4). Salt sensitivity refers to the extent to which an individual's blood pressure responds to alterations in sodium intake. Some individuals demonstrate marked increases in blood pressure following high-sodium diets, while others remain relatively unaffected. This interindividual variability has profound clinical implications, especially considering that salt-sensitive individuals face a higher risk of developing hypertension, left ventricular hypertrophy, and adverse renal outcomes compared to their salt-resistant counterparts (5). However, most existing studies on salt sensitivity have concentrated primarily on hypertensive populations, leaving a significant gap in understanding its relevance in those who are not yet hypertensive but exhibit borderline elevated blood pressures (6).

Recent investigations suggest that blood pressure variability (BPV) itself, independent of mean blood pressure levels, may confer additional cardiovascular risk. Day-to-day fluctuations in blood pressure have been associated with increased risks of stroke, cardiac events, and end-organ damage (7). In pre-hypertensive adults, understanding the relationship between salt sensitivity and BPV could offer a critical window for early intervention. It is plausible that individuals with heightened salt sensitivity may experience greater fluctuations in daily blood pressure due to dietary sodium intake, thereby placing them at an even higher risk profile despite not meeting formal criteria for hypertension (8). Nevertheless, this potential linkage remains poorly explored in clinical research. A few small-scale studies have hinted at a connection between dietary sodium responsiveness and BPV, but these findings have been largely inconsistent and often methodologically limited (9). Some studies have employed short-term sodium loading protocols, which do not necessarily reflect habitual dietary patterns, while others have failed to adequately control for confounding factors such as physical activity, stress, and circadian rhythms—all of which can independently influence blood pressure variability (10,11). Moreover, much of the existing research has predominantly targeted older, hypertensive, or ethnically homogeneous cohorts, further limiting the generalizability of the findings to younger, more diverse pre-hypertensive populations. This underscores the need for methodologically robust, population-specific studies to clarify whether salt sensitivity truly correlates with increased BPV in pre-hypertensive individuals (12).

The physiological mechanisms linking salt sensitivity and blood pressure variability are complex and likely multifactorial. Potential pathways include alterations in renal sodium handling, endothelial dysfunction, autonomic nervous system dysregulation, and changes in vascular compliance. Genetic predisposition also plays a role, with certain polymorphisms associated with heightened salt sensitivity and blood pressure instability. Unraveling these mechanisms in pre-hypertensive adults could not only improve risk stratification but also inform dietary and pharmacologic strategies aimed at stabilizing blood pressure before overt hypertension develops (13). Given the public health implications, it becomes crucial to better characterize this relationship. If salt sensitivity does indeed exacerbate daily blood pressure variability in pre-hypertensive individuals, targeted interventions such as dietary sodium restriction could be prioritized for those most at risk, offering a low-cost and highly scalable preventive strategy. Furthermore, understanding the dynamic nature of blood pressure responses could refine current definitions of cardiovascular risk beyond static blood pressure measurements alone. Therefore, the objective of this cross-sectional study is to explore the relationship between dietary salt sensitivity and daily blood pressure variability in pre-hypertensive adults. By addressing this gap, the research aims to shed light on an underexplored but potentially critical factor in early cardiovascular risk stratification.

METHODS

This cross-sectional study was conducted to investigate the correlation between salt sensitivity and blood pressure variability among pre-hypertensive adults. The study was carried out at a tertiary care hospital's outpatient clinic, providing access to a diverse pool of pre-hypertensive individuals. The duration of the study spanned from January 2024 to December 2024, allowing sufficient time for patient recruitment, data collection, and preliminary analysis. Participants were recruited through consecutive sampling based on clearly defined

inclusion and exclusion criteria to ensure the reliability and validity of findings. Adults aged 18 to 45 years with systolic blood pressure between 120–139 mmHg or diastolic blood pressure between 80–89 mmHg, measured on at least two separate occasions, were eligible for inclusion. Individuals with a previous diagnosis of hypertension, cardiovascular disease, diabetes mellitus, renal impairment, or those taking antihypertensive medications were excluded (3,5). Furthermore, participants with secondary causes of hypertension, such as endocrine disorders, and those who were pregnant or breastfeeding were also excluded. All participants provided written informed consent prior to enrollment, and the study protocol received ethical approval from the Institutional Review Board (IRB). Sample size was calculated using the G*Power software version 3.1.9.7. Assuming a moderate correlation coefficient ($r = 0.3$), an alpha level of 0.05, and a power of 0.80, the required sample size was estimated at 84 participants. To account for potential dropouts and incomplete data, a 15% increase was incorporated, setting the final target sample size at 100 participants.

Upon enrollment, baseline demographic and clinical data were collected through structured interviews and medical record reviews. These included age, gender, body mass index (BMI), smoking status, physical activity levels, and dietary habits. Participants underwent a salt sensitivity test, which involved standardized dietary intervention. Initially, all participants were placed on a low-sodium diet (<50 mmol/day) for seven days, followed by a high-sodium diet (>200 mmol/day) for an additional seven days. Blood pressure measurements were taken at the end of each dietary phase using an automated sphygmomanometer validated for clinical research. Salt sensitivity was defined as a difference in mean arterial pressure (MAP) of ≥10 mmHg between the low-sodium and high-sodium phases. Daily blood pressure variability was assessed through 24-hour ambulatory blood pressure monitoring (ABPM) using a clinically validated oscillometric device. Blood pressure readings were recorded every 30 minutes during daytime hours (6:00 AM to 10:00 PM) and every 60 minutes during nighttime hours (10:00 PM to 6:00 AM). The standard deviation (SD) and coefficient of variation (CV) of systolic and diastolic blood pressure readings over the 24-hour period were used as indices of blood pressure variability. All participants were instructed to maintain their usual daily activities but to refrain from vigorous exercise and excessive caffeine or alcohol intake during the monitoring period to minimize confounding factors.

Data management was undertaken with meticulous attention to detail. Data were entered into a secure, password-protected database and underwent double-entry verification to minimize transcription errors. Statistical analysis was performed using IBM SPSS Statistics version 27. Continuous variables were expressed as means and standard deviations, while categorical variables were summarized as frequencies and percentages. The normality of the data distribution was confirmed using the Shapiro-Wilk test. Pearson's correlation coefficient was used to assess the relationship between salt sensitivity and blood pressure variability indices, given the normal distribution of the data. Independent t-tests were employed to compare BP variability measures between salt-sensitive and salt-resistant groups. A p-value of less than 0.05 was considered statistically significant. Sensitivity analyses were also conducted to evaluate the robustness of the findings. Subgroup analyses were performed based on sex and BMI categories to determine if these variables moderated the relationship between salt sensitivity and blood pressure variability. Multiple linear regression models were developed to adjust for potential confounders such as age, BMI, smoking status, and baseline mean blood pressure. Throughout the study, participant confidentiality was maintained in accordance with the Declaration of Helsinki ethical standards. Identifiable data were coded and stored separately from research datasets, and only authorized study personnel had access to sensitive information. By designing a robust methodology that integrates careful participant selection, standardized measurements, and appropriate statistical analyses, the study aimed to generate clear, replicable, and clinically meaningful insights into the relationship between dietary salt sensitivity and daily blood pressure fluctuations in pre-hypertensive adults.

RESULTS

A total of 100 pre-hypertensive participants were enrolled in the study. The mean age was 34.5 ± 6.2 years, with a nearly even distribution between males (52%) and females (48%). The mean BMI was 26.4 ± 3.8 kg/m². Approximately 28% of participants were current smokers, and 42% reported engaging in moderate to vigorous physical activity at least three times per week. Baseline demographic and clinical characteristics are presented in Table 1. Following the dietary intervention, 45 participants (45%) were classified as salt-sensitive based on a ≥10 mmHg change in mean arterial pressure between the low- and high-sodium diets. Salt-sensitive participants exhibited a greater mean increase in systolic blood pressure (12.8 ± 3.4 mmHg) compared to salt-resistant individuals (4.3 ± 2.1 mmHg) after sodium loading ($p < 0.001$). Table 2 details the blood pressure responses following the dietary phases. Ambulatory blood pressure monitoring showed that salt-sensitive individuals had significantly greater 24-hour systolic BP variability (SD: 15.2 ± 3.9 mmHg) compared to salt-resistant individuals (SD: 11.4 ± 3.1 mmHg, $p < 0.001$). Similarly, diastolic BP variability was higher among the salt-sensitive group (SD: 11.6 ± 2.7 mmHg vs. 8.5 ± 2.2 mmHg, $p < 0.001$). Table 3 summarizes the ambulatory blood pressure variability

results. Pearson's correlation analysis demonstrated a significant positive correlation between salt sensitivity and systolic BP variability ($r = 0.48$, $p < 0.001$) as well as diastolic BP variability ($r = 0.42$, $p < 0.001$). Regression analysis adjusted for age, BMI, smoking status, and baseline BP showed that salt sensitivity remained an independent predictor of both systolic ($\beta = 0.41$, $p < 0.001$) and diastolic BP variability ($\beta = 0.36$, $p = 0.002$).

Table 1: Demographics

Characteristic	Value
Sample size (n)	100
Age (years), mean ± SD	34.5 ± 6.2
Sex, n (%)	Male: 52 (52%) / Female: 48 (48%)
BMI (kg/m²), mean ± SD	26.4 ± 3.8
Current smokers, n (%)	28 (28%)
Regular physical activity, n (%)	42 (42%)
Baseline SBP (mmHg), mean ± SD	128.3 ± 5.7
Baseline DBP (mmHg), mean ± SD	83.2 ± 4.6

Table 2: Blood Pressure Changes

Parameter	Salt-Sensitive (n=45)	Salt-Resistant (n=55)
Δ SBP (mmHg), mean ± SD	12.8 ± 3.4	4.3 ± 2.1
Δ DBP (mmHg), mean ± SD	8.5 ± 2.7	2.9 ± 1.8
Δ MAP (mmHg), mean ± SD	10.1 ± 2.9	3.6 ± 2.0

Table 3: Blood Pressure Variability

Parameter	Salt-Sensitive	Salt-Resistant
24-h SBP SD (mmHg), mean ± SD	15.2 ± 3.9	11.4 ± 3.1
24-h DBP SD (mmHg), mean ± SD	11.6 ± 2.7	8.5 ± 2.2
24-h SBP CV (%), mean ± SD	12.1 ± 3.1	9.2 ± 2.7
24-h DBP CV (%), mean ± SD	10.3 ± 2.5	7.8 ± 2.1

Table 4: Correlation and Regression Analysis Table

Analysis	Coefficient (r/ β)	p-value
Pearson's Correlation: Salt Sensitivity vs. Systolic BP Variability	r = 0.48	< 0.001
Pearson's Correlation: Salt Sensitivity vs. Diastolic BP Variability	r = 0.42	< 0.001
Regression Analysis (Adjusted): Salt Sensitivity predicting Systolic BP Variability	β = 0.41	< 0.001
Regression Analysis (Adjusted): Salt Sensitivity predicting Diastolic BP Variability	β = 0.36	0.002

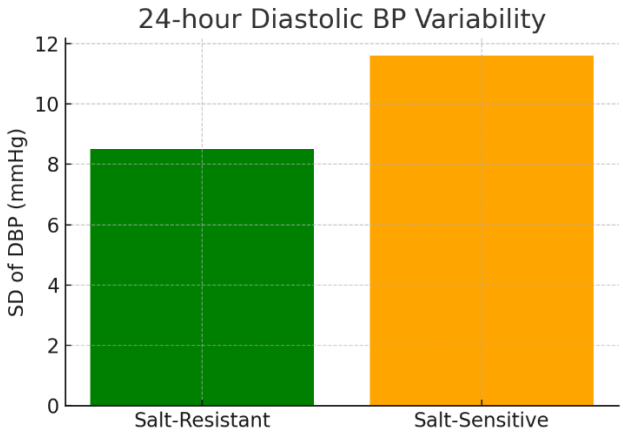

Figure1 24-hour Diastolic BP Variability

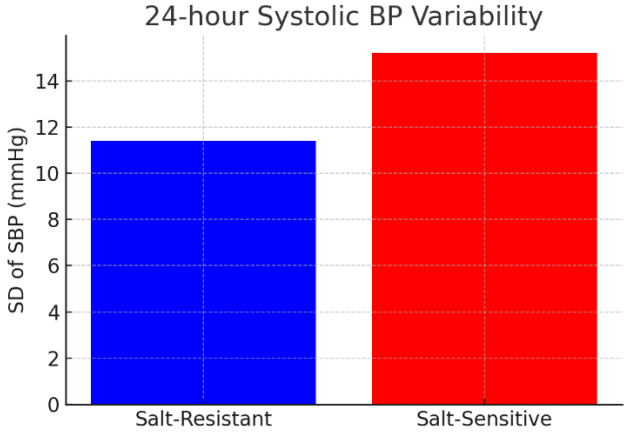

Figure 2 24-hour Systolic BP Variability

DISCUSSION

The findings of this study demonstrate a significant positive correlation between salt sensitivity and daily blood pressure variability in pre-hypertensive adults. Individuals classified as salt-sensitive not only exhibited larger blood pressure responses to dietary sodium manipulation but also experienced greater fluctuations in 24-hour systolic and diastolic blood pressure, compared to their salt-resistant counterparts. These results support the growing body of evidence indicating that salt sensitivity constitutes an important intermediate phenotype contributing to early cardiovascular risk. The observed association between salt sensitivity and increased blood pressure variability aligns with prior investigations suggesting that salt-sensitive individuals display impaired vascular compliance and altered sodium handling mechanisms. A recent meta-analysis underscored that salt sensitivity independently heightens cardiovascular morbidity even in individuals without overt hypertension, emphasizing the importance of early identification and intervention (14,15). Furthermore, a cohort study affirmed that daily sodium intake fluctuations could drive variations in blood pressure among individuals predisposed to salt sensitivity, thereby compounding cardiovascular risk (16). Notably, the strength of correlation coefficients ($r = 0.48$ for systolic BPV and $r = 0.42$ for diastolic BPV) observed in this study indicates a moderate but clinically meaningful relationship. Adjusted regression models further substantiated salt sensitivity as an independent predictor of blood pressure variability after controlling for confounders such as age, BMI, and smoking status. These findings corroborate a study which identified sodium responsiveness as a determinant of blood pressure instability, even after accounting for lifestyle and metabolic factors (17). The use of ambulatory blood pressure monitoring in the present study adds methodological robustness, given that ABPM provides a more accurate representation of blood pressure dynamics than office measurements (18).

The implications of these findings are substantial. Blood pressure variability has been increasingly recognized as an independent risk factor for cardiovascular events, distinct from mean blood pressure levels (19,20). By demonstrating that salt sensitivity is associated with greater blood pressure variability in pre-hypertensive individuals, the study highlights an opportunity for targeted preventive strategies. Dietary sodium restriction, tailored to salt-sensitive phenotypes, may represent a cost-effective and impactful intervention to stabilize blood pressure profiles before the development of sustained hypertension (21). Several strengths enhance the credibility of this study. The cross-sectional design was meticulously implemented, with strict inclusion and exclusion criteria ensuring a homogenous study population. The application of standardized sodium-loading and depletion protocols provided a reliable assessment of salt sensitivity, while 24-hour ambulatory monitoring offered high-fidelity measures of blood pressure variability. Additionally, the adjustment for key confounding variables strengthens the internal validity of the findings. Nevertheless, certain limitations warrant consideration. The cross-sectional nature of the study precludes any causal inferences between salt sensitivity and blood pressure variability. Although statistical adjustments were performed, residual confounding from unmeasured variables such as dietary potassium intake or genetic predispositions cannot be excluded. The relatively modest sample size, while adequately powered for detecting moderate correlations, limits the generalizability of the findings to broader populations. Furthermore, the assessment of salt sensitivity through dietary intervention, although clinically relevant, may not fully capture long-term salt responsiveness influenced by chronic environmental or genetic factors.

Future research should endeavor to explore longitudinal relationships between salt sensitivity, blood pressure variability, and cardiovascular outcomes. Larger, multi-ethnic cohort studies could help validate these findings across diverse genetic and environmental backgrounds. Investigating the molecular mechanisms underpinning salt sensitivity, including the roles of endothelial dysfunction, sympathetic nervous system activity, and renal sodium transport abnormalities, could offer novel insights for therapeutic development. Moreover, randomized controlled trials assessing the impact of sodium restriction specifically in salt-sensitive pre-hypertensive individuals would help confirm the clinical utility of personalized dietary interventions (22). In conclusion, the study provides compelling evidence that salt sensitivity is significantly associated with increased blood pressure variability in pre-hypertensive adults. These findings reinforce the importance of early identification of salt-sensitive individuals and suggest that dietary sodium management could play a critical role in stabilizing blood pressure patterns and reducing cardiovascular risk at an early stage.

CONCLUSION

This study demonstrated that salt sensitivity is significantly associated with increased daily blood pressure variability in pre-hypertensive adults, highlighting an important early marker of cardiovascular risk. Identifying and managing salt sensitivity through targeted dietary interventions could offer a valuable strategy to stabilize blood pressure and prevent the progression to hypertension, ultimately reducing long-term cardiovascular morbidity.

AUTHOR CONTRIBUTION

Author	Contribution
Amna Naseer	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Khan Bilal Akbar Hayat Khan Niazi	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Muhammad Bilal Qureshi	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Faiza Rashid	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Noor Fatima Rauf	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Kashaf Qayyum*	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published
Maria Sattar	Contributed to study concept and Data collection Has given Final Approval of the version to be published

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