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Association Between Hypertension Prevalence and Dietary Sodium-Potassium Ratio: A Case Study of CDC Database

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Abstract. Hypertension remains one of the most prevalent chronic diseases in modern society and a leading cause of cardiovascular mortality. According to data from the Centers for Disease Control and Prevention (CDC), nearly half of American adults have hypertension, and in 2023, hypertension was a primary contributing factor in 664,470 deaths in the United States. While previous research has established the individual impacts of sodium and potassium intake on blood pressure, there is a lack of independent association testing for the sodium-potassium (Na/K) ratio as a combined dietary metric. Furthermore, following the COVID-19 pandemic,residents' dietary patterns have undergone significant changes, altering the metabolic baseline risk for hypertension. This study aims to verify the impact of daily dietary Na/K ratio on hypertension risk and to construct a multi-factor prediction model. Based on the NHANES cross-sectional database, this study enrolled 2,586 participants and employed a comprehensive analytical approach integrating descriptive analysis, K-means clustering, multivariable-adjusted logistic models, restricted cubic spline (RCS) techniques, and LASSO regularization for variable selection. Model performance was assessed using ROC curves and Nomograms, with stratified analyses examining the Na/K ratio-hypertension association. The dietary Na/K ratio emerged as an independent risk marker for hypertension, with significant pathogenic effects observed exclusively in older adults. Moreover, the Na/K ratio exhibited synergistic effects with central adiposity in elevating hypertension risk. These findings support the development of age-stratified, Na/K ratio-focused personalized dietary interventions for hypertension prevention.

Keywords: Hypertension, Sodium-Potassium Ratio, Logistic Regression, NHANES, logistic regression

1. Introduction

Hypertension is the most common chronic disease in the general population and one of the leading causes of death in modern society. The condition poses a substantial public health burden globally, with its prevalence continuing to rise. Despite extensive research into the separate influences of dietary sodium and potassium consumption upon blood pressure control, a critical knowledge gap persists concerning the independent relation of the dietary Na/K ratio to hypertensive likelihood.

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Previous evidence suggests a complex interplay between sodium and potassium in cardiovascular health. Elevated sodium intake is a well-established driver of increased blood pressure [1], while adequate potassium intake exerts protective effects on the cardiovascular system [2, 3]. However, the Na/K ratio, as a combined metric, may better reflect true dietary electrolyte balance than either nutrient alone. Previous studies have suggested that the ratio provides a more accurate representation of the sodium-potassium interplay in relation to cardiovascular outcomes [4].

Adding to the complexity, the COVID-19 pandemic has significantly altered residents' dietary structures. Previous studies have documented post-pandemic shifts in eating patterns, including increased consumption of processed foods and changes in nutrient intake [5]. These post-pandemic dietary transitions, coupled with evolving metabolic characteristics, have modified the baseline risk for hypertension in the population [6]. Therefore, there is an urgent need to re-evaluate the association between dietary electrolyte balance and hypertension in the current post-pandemic context.

This study addresses three key research questions: (1) Does the dietary Na/K ratio serve as an independent predictor of hypertension following adjustment for confounding variables? (2) Does the relationship of the Na/K ratio to hypertensive status exhibit a non-linear, dose-dependent pattern? (3) Can distinct clinical subgroups explain heterogeneity in observed hypertension risk?

By integrating unsupervised clustering, multivariable logistic regression, restricted cubic splines (RCS), and LASSO regularization, this study aims to validate the utility of the Na/K ratio as a precise dietary metric and provide a robust theoretical foundation for evidence-based dietary guidance in personalized hypertension management.

2. Materials and methods

2.1. Data source

The final analytic sample included 2,586 participants after excluding those with missing values for key variables such as dietary intake, blood pressure measurements, and relevant covariates.

2.2. Study variables

Outcome Variable: Hypertension was defined as systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥90 mmHg, or self-reported use of antihypertensive medication.

Primary Exposure: Dietary sodium-to-potassium (Na/K) ratio was estimated from 24-hour dietary recall interviews conducted by trained interviewers.

Covariates: Potential confounding variables included demographic factors (age, sex, race/ethnicity), anthropometric measurements (BMI, waist circumference), and biochemical markers (uric acid, high-sensitivity C-reactive protein [hsCRP], HbA1c, HDL cholesterol, LDL cholesterol, triglycerides, total cholesterol) [7].

2.3. Analytical workflow

The analysis proceeded through six sequential steps:

Step 1: Data Cleaning. Exclusion of outliers, imputation of missing values, and validation of variable distributions were performed to ensure data integrity.

Step 2: Exploratory Data Analysis. Descriptive statistics, correlation matrices, and visualization of relationships among exposure, covariates, and outcome were conducted to understand data

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characteristics.

Step 3: K-means Clustering. Unsupervised machine learning was applied to identify distinct phenotypic clusters based on metabolic and anthropometric profiles, enabling exploration of clinical heterogeneity.

Step 4: Multivariable Regression. Logistic regression models adjusted for confounders were used to assess the independent association between Na/K ratio and hypertension.

Step 5: Restricted Cubic Splines (RCS). Flexible modeling was employed to explore potential non-linear dose-response relationships between the Na/K ratio and hypertension risk.

Step 6: Interpretation. Synthesis of findings, assessment of clinical relevance, and discussion of public health implications for dietary guidelines were conducted.

2.4. Statistical analysis

Logistic regression models were constructed with hypertension status (0 = normotensive, 1 = hypertensive) as the dependent variable. The Na/K ratio was entered as the primary independent variable, with progressive adjustment for covariates across three models: Model 1 (unadjusted), Model 2 (adjusted for demographic factors), and Model 3 (fully adjusted for all covariates). Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated.

Restricted cubic spline (RCS) curves specifying four knots were applied to examine the possible curvilinear dose–response pattern of the dietary Na/K ratio in relation to hypertension risk. The reference point was set at the median Na/K ratio value.

Variable selection was performed via LASSO (Least Absolute Shrinkage and Selection Operator) regression in constructing a parsimonious prediction model. The optimal penalty parameter (λ) was determined through 10-fold cross-validation to minimize the mean squared error.

Model performance was evaluated using the area under the receiver operating characteristic curve (AUC) and calibration plots. A Nomogram was constructed for visual representation of the prediction model.

All statistical analyses were performed using R software (version 4.6.0). Two-sided p-values <0.05 were considered statistically significant.

3. Results

3.1. Exploratory data analysis and population characteristics

A total of 2,586 participants were included in the final analysis. The dietary Na/K ratio exhibited a pronounced positively skewed distribution, indicating that while most participants clustered between 0.5 and 2.0, a subset exhibited dietary patterns characterized by high sodium and low potassium intake. Approximately 35% of the total study population was classified as hypertensive. Broad inter-individual variability was observed across key metabolic and inflammatory biomarkers. Triglycerides (TG) and HbA1c demonstrated heavily right-skewed distributions with substantial extreme outliers, whereas LDL-C showed a symmetric, approximately normal distribution. High-sensitivity C-reactive protein (hsCRP) was successfully normalized via a natural logarithmic transformation [ln(hsCRP)] to ensure statistical robustness in regression modeling. The cohort covered a broad age range, with participants predominantly concentrated in middle-aged and elderly adult groups, and a slight male predominance and a high concentration of BMI values falling within the overweight and obese categories.

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3.2. Identification of clinical phenotypes via K-means clustering

Unsupervised K-means clustering based on the dietary Na/K ratio and metabolic biomarkers segmented the population (n = 2,586) into five distinct phenotypic clusters. Principal Component Analysis (PCA) score plotting confirmed that these clusters occupied well-separated spaces, though partial overlap existed among intermediate sub-groups, reflecting the underlying continuum of metabolic risk.

The baseline burden of hypertension demonstrated profound heterogeneity across these five categories. Specifically, Cluster 1 and Cluster 5 exhibited the highest prevalence of hypertension, with 48.8% of individuals affected in each group. Cluster 3 represented an intermediate high-risk phenotype with a prevalence of 44.3%. Conversely, Cluster 2 and Cluster 4 represented lower-risk sub-populations, with significantly lower hypertension prevalence of 27.9% and 28.7%, respectively(Figure 1).

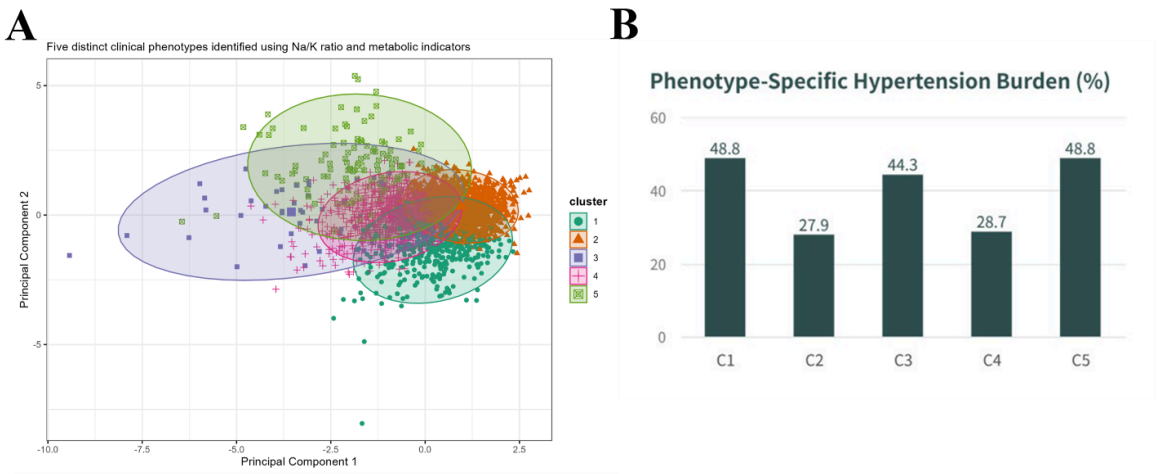

Figure 1. Age-stratified non-linear dose-response relationships between dietary sodium-to-potassium (Na/K) ratio and hypertension risk

3.3. Multivariable logistic regression analysis of hypertension

A multivariable logistic regression model was fitted to evaluate the independent effects on hypertension. Age was positively and strongly associated with the outcome (OR = 1.03, 95% CI: 1.03–1.04, $P < 0.001$). Central adiposity, captured by waist circumference, also exhibited an independent hazardous effect (OR = 1.02, 95% CI: 1.01–1.04, $P < 0.01$). Furthermore, substantial racial disparities were uncovered: individuals classified under Race 4 exhibited an 88% increase in the odds of hypertension compared to the reference racial category (OR = 1.88, 95% CI: 1.23–2.89, $P < 0.01$). The comprehensive odds ratios and corresponding confidence intervals for all evaluated biochemical and clinical parameters are graphically summarized in Figure 2. Multivariable logistic regression analysis of independent risk factors associated with hypertension prevalence.

Notably, the dietary Na/K ratio demonstrated a positive but statistically non-significant linear correlation within the global fully adjusted model (OR value = 1.12, 95% CI: 0.96–1.30, $P > 0.05$). No independent linear significance was detected for BMI, HbA1c, LDL-C, TG, hsCRP, or total cholesterol, suggesting potential multi-collinearity or non-linear masking effects within the aggregated population.

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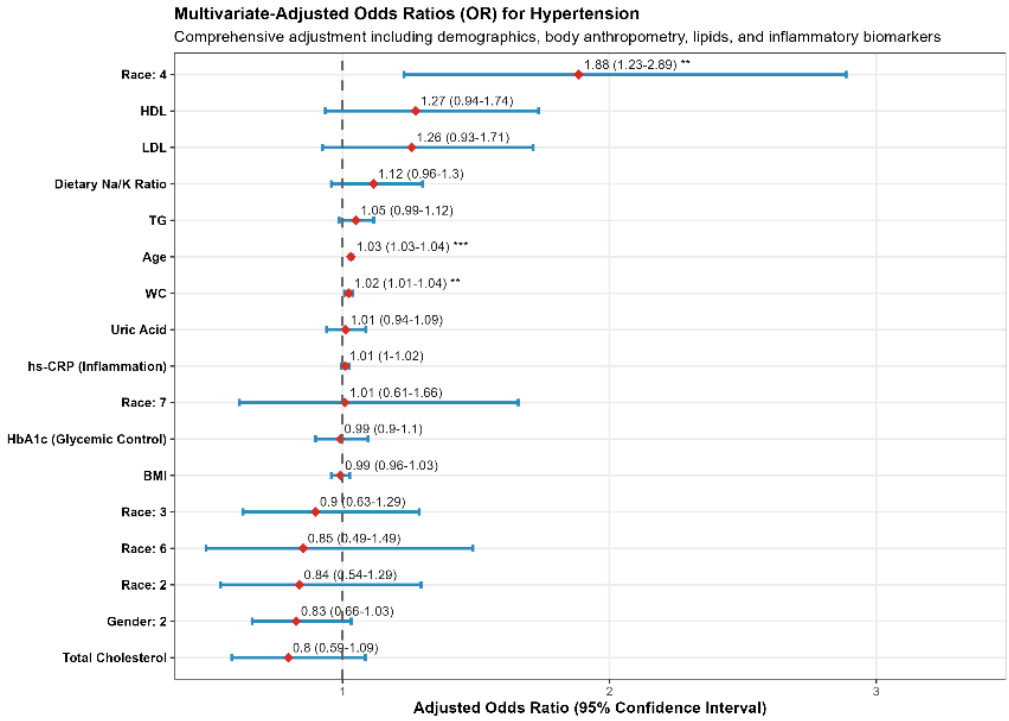

Figure 2. Multivariable logistic regression analysis of independent risk factors associated with hypertension prevalence

3.4. Age-stratified dose–response relationship between dietary Na/K ratio and hypertension

To decipher the non-significance observed in the global linear regression, restricted cubic spline (RCS) analysis adjusted for waist circumference was deployed across progressive age strata. The analysis unveiled a striking age-dependent interaction that modified the hazard profile of the dietary electrolyte ratio.

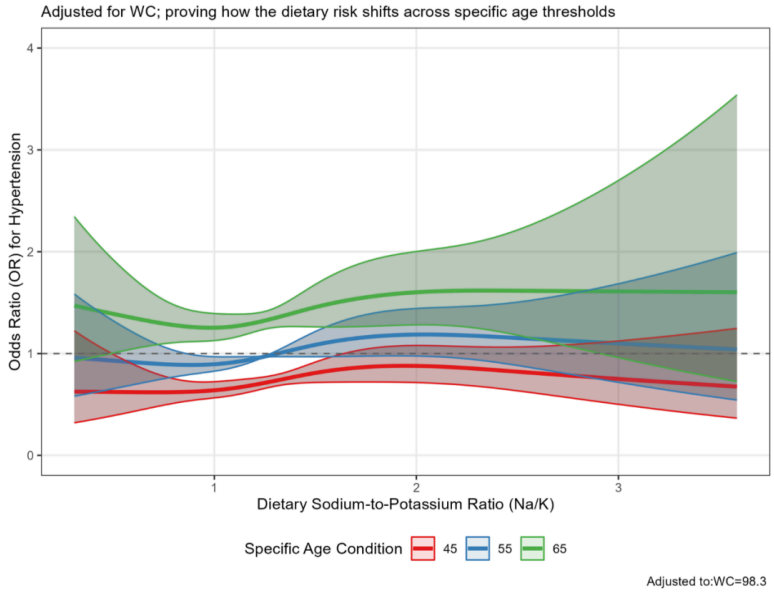

Figure 3. Age-stratified dose-response relationships between dietary Na/K ratio and hypertension risk modeled by restricted cubic splines (RCS)

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As illustrated in Figure 3, among older adults aged 65, the estimated odds of hypertension accelerated steeply and progressively in a linear-like trend with higher Na/K exposure, leading odds ratios to rise past 2.0 at the upper tier of the dietary exposure range. In stark contrast, among the younger cohorts aged 45 and 55 years, the dose-response trajectories remained entirely flat, maintaining odds ratios near unity (OR ≈ 1.0) across the span of the Na/K ratio. This clearly indicates that the cardiovascular toxicity of a high-sodium, low-potassium diet is selectively pronounced and and exerts its effects within the aging population.

3.5. LASSO regularization for feature optimization

To provide an objective machine-learning validation for variable importance, LASSO logistic regression was performed on the 13 clinical and biochemical variables. The L1 penalty parameter selected via 10-fold cross-validation optimized the model by shrinking non-contributing parameters to zero. Remarkably, generalized obesity (BMI), long-term glycemic control (HbA1c), and atherogenic lipoproteins (LDL-C) were entirely eliminated from the predictive model (coefficients compressed to 0). Conversely, the dietary Na/K ratio was heavily selected and retained a robust positive coefficient (+0.0665). Along with age (+0.0300) and waist circumference (+0.0188), the retention of the Na/K ratio in the parsimonious model provides mathematical proof of its superior, independent predictive value over separate nutrient components and generalized anthropometric measurements(Table 1).

Table 1. Feature optimization and variable selection for hypertension risk via LASSO regularized logistic regression

Variable_Description	Lasso_Beta
Dietary Na/K Ratio	0.066473
Age	0.030021
Gender: Male vs Female	-0.14836
Race 2	-0.03443
Race 3	0
Race 4	0.642117
Race 6	0
Race 7	0.014251
BMI	0
Waist Circumference	0.0188
Uric Acid	0.007229
hsCRP	0.007165
HbA1c	0
HDL	0.008327
LDL	0
TG	0.002273
Total Cholesterol	0.003883

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4. Discussion

This study utilized an integrative approach—combining unsupervised K-means clustering, multivariable logistic regression, non-linear restricted cubic splines, and LASSO regularized selection—to systematically dissect the independent relationship linking the dietary Na/K ratio to hypertension risk within a contemporary post-pandemic population. Our multi-tiered analysis successfully resolved the apparent paradox of global linear non-significance by exposing a powerful age-dependent risk acceleration threshold and identifying highly susceptible clinical sub-phenotypes.

The global multivariable logistic model initially yielded a non-significant linear effect for the Na/K ratio (OR=1.12, P>0.05). In traditional epidemiological frameworks, such a finding might prematurely lead to the conclusion that the combined electrolyte metric lacks an association with blood pressure regulation. However, our subsequent stratified RCS analysis effectively unlocked this phenomenon as a classic case of demographic masking. The toxicological impact of a disrupted sodium-potassium balance is not uniform across the lifespan. For individuals aged 45 and 55, the risk curves remained notably flat, a pattern that largely reflects the robust cardiovascular resilience, intact renal compensatory natriuresis, and endothelium-dependent vasodilation that are characteristic of younger adults.

Conversely, for the 65-year-old cohort, the risk of hypertension demonstrated an aggressive, dose-dependent escalation, with odds ratios doubling (OR>2.0) at higher exposure ranges. Biologically, this age-selective vulnerability is highly plausible. Aging is inextricably linked with structural and functional vascular degradation, characterized by arterial stiffening, diminished renal blood flow, and a marked decline in the efficiency of the sodium-potassium adenosine triphosphatase (Na+/K+-ATPase) pump. Consequently, older adults exhibit pronounced "salt sensitivity." When exposed to an elevated dietary Na/K ratio, their compromised renal capacity fails to excrete excess sodium adequately, while the lack of concurrent potassium intake deprives the vasculature of its primary hyperpolarizing and vasodilatory defense, triggering volume expansion, sympathetic overactivity, and overt hypertension.

The application of unsupervised K-means clustering further illuminated the clinical heterogeneity of the study population. By separating the 2,586 individuals into five distinct clusters, we demonstrated that hypertension risk is mediated by holistic phenotypic profiles rather than isolated dietary exposures. The stark contrast between the high-prevalence clusters (Clusters 1 and 5 at 48.8%) and the low-prevalence clusters (Cluster 2 at 27.9%) underscores the synergistic impact of concurrent metabolic states.

This systemic interplay was mathematically validated by the LASSO regression results. LASSO discarded generalized metrics such as BMI, while retaining waist circumference and the dietary Na/K ratio. This outcome supports the shift in modern clinical medicine from monitoring gross weight to evaluating visceral fat deposition (central obesity) and precision nutritional intake. A high dietary Na/K ratio does not act in a vacuum; its pathogenic potential is magnified when paired with the chronic low-grade inflammation and metabolic shifts characteristic of visceral adiposity, as reflected in our high-prevalence cluster profiles.

From a public health perspective, these findings carry profound implications for modern dietary guidelines. Traditional nutritional recommendations typically specify separate, absolute thresholds for daily sodium and potassium intake (e.g., restricting sodium to below 2,300 mg/day). However, such single-nutrient strategies are often difficult for the public to calculate and adhere to, particularly in a post-pandemic environment dominated by sodium-dense ultra-processed foods.

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Our study validates the Na/K ratio as a more comprehensive and reliable dietary metric. More importantly, the clear age stratification suggests that universal, population-wide dietary targets may be sub-optimal. Public health interventions should instead transition toward personalized nutritional guidance. For instance, while younger populations maintain a wider buffer zone, individuals over 65, or those with central obesity, must be provided with strict, targeted clinical recommendations to maintain their dietary Na/K ratio below a specific threshold to prevent catastrophic blood pressure acceleration.

This study possesses several notable strengths. First, it utilizes a large, high-quality, nationally representative database (NHANES), ensuring excellent statistical power and generalizability. Second, the analytical workflow is highly rigorous, utilizing advanced machine learning (K-means and LASSO) alongside traditional epidemiological models (RCS and multivariable regression) to cross-validate findings and prevent subjective variable selection bias.

However, certain limitations must be acknowledged. Due to the cross-sectional design of NHANES, absolute chronological causality between dietary shifts and hypertension onsetcannot be definitively established. Additionally, dietary intake data derived from 24-hour recalls are inherently vulnerable to recall bias and may not perfectly capture long-term, multi-year eating habits. Future prospective cohort studies tracking precise urinary sodium-to-potassium excretion over extended periods are required to fully map the causal trajectories within high-risk phenotypic subgroups.

5. Conclusion

In conclusion, this study establishes that the dietary Na/K ratio is a powerful, independent, and age-stratified indicator of hypertension risk. The non-linear relationship verified through RCS analysis highlights a severe cardiovascular risk acceleration selectively operational within the aging population. Furthermore, K-means clustering and LASSO regularized selection successfully mapped the clinical heterogeneity, proving that a high dietary Na/K ratio acts synergistically withcentral adiposity to drive hypertension susceptibility. These insights provide a compelling theoretical foundation for abandoning rigid, single-nutrient guidelines in favor of flexible, age-targeted, and personalized dietary ratio management for effective hypertension prevention.

However, several limitations should be acknowledged. First, a firm causal connection linking dietary Na/K ratio to hypertension incidence cannot be drawn, given the cross-sectional nature intrinsic to NHANES and its attendant constraints. Second, the reliance on 24-hour dietary recalls introduces potential recall bias, as participants may inaccurately report their dietary intake, and single-day assessments may not adequately reflect long-term eating patterns. Third, although we adjusted for multiple covariates, residual confounding from unmeasured factors cannot be entirely excluded. Future research should prioritize longitudinal cohort studies with repeated dietary assessments and urinary electrolyte measurements to validate the causal mechanisms underlying the Na/K ratio-hypertension association. Additionally, intervention studies targeting personalized dietary Na/K ratio management in high-risk populations, particularly older adults with central obesity, are warranted to develop evidence-based clinical guidelines for hypertension prevention. Further exploration of the interplay between genetic susceptibility, gut microbiota, and dietary electrolyte balance may also provide novel insights into precision nutrition strategies for cardiovascular health.

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