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Dietary Sodium-Potassium Ratio in Low-Sodium Diet Population and Its Association with Stroke: Insights from NHANES Database

Liguo Li[1*], Hongguang Fu[2*], Ya Su[1], Junsong Jing[1]

1 Institute of Rehabilitation Medicine, Henan Academy of Innovations in Medical Science, Zhengzhou 451162, China.
2 Zhengzhou Health Vocational College No. 69, Jingxiang Road, Xingyang City, Zhengzhou City, Henan Province 451162, China.
* The two authors contributed equally.

Correspondence to: Liguo Li
Institute of Rehabilitation Medicine, Henan Academy of Innovations in Medical Science, Huanghai Road and Biotechnology 2nd Street, Aviation Port Area, Zhengzhou 451162, China;
Huanghe University of Science and Technology, 666 Zijingshan South Road, Guancheng Hui District, Zhengzhou 450061, China
TEL.: 15649673120; E-MAIL: Doctorleee@hnsyxkxy.cn

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Abstract

OBJECTIVE: To delve into the potential association between the dietary sodium-potassium ratio and stroke in the low-sodium diet population.
METHODS: Cross-sectional analysis was done utilizing data from the National Health and Nutrition Examination Survey (NHANES) spanning 7 cycles from 2003 to 2016. 7141 samples were selected. Participants were categorized into stroke and non-stroke groups following population characteristics, and the association between dietary sodium-potassium ratio and stroke was analyzed across various categorical variables. Stratification of the dietary sodium-potassium ratio was done using quartiles. Weighted logistic regression models were constructed by adjusting various confounders to examine the sodium-potassium ratio's relationship with stroke. Subgroup analyses stratified by BMI were performed; interaction terms were evaluated with significance threshold $p < 0.1$. The relation between the sodium-potassium ratio and stroke risk was explored by Restricted Cubic Splines (RCS).
RESULTS: Among the 7,141 participants with a dietary sodium intake ≤ 2,300 mg, the dietary sodium-to-potassium ratio was significantly higher in the stroke group (mean = 1.19, SD=0.51) versus non-stroke group (mean = 1.11, SD = 0.52); $p = 0.037$. An increased sodium-to-potassium ratio was associated with a higher risk of stroke in the following subgroups: women (Odds Ratio (OR) = 1.31, 95% CI: 1.05–1.62, $p = 0.013$), individuals with a BMI of 25–30 kg/m² (OR = 2.02, 95% CI: 1.40–2.91, $p < 0.001$), those with a history of smoking (OR = 1.79, 95% CI: 1.20–2.67, $p = 0.004$), alcohol consumers (OR = 1.29, 95% CI: 1.00–1.67, $p = 0.047$), those without coronary heart disease (OR = 1.33, 95% CI: 1.10–1.62, $p = 0.003$) or diabetes (OR = 1.33, 95% CI: 1.08–1.63, $p = 0.006$), but with hypertension (OR = 1.28, 95% CI: 1.03–1.59, $p = 0.022$). Stratified analysis by BMI indicated that the

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association between the sodium-to-potassium ratio and stroke was strongest and most consistent among overweight individuals (BMI 25–30 kg/m²), with all ORs exceeding 2 and $p < 0.001$. Restricted cubic spline analysis revealed a U-shaped relationship; stroke risk was minimized at a ratio of 0.76 (95% CI: 0.65–0.88), with protective effects sustained across the range 0.46–1.00. **CONCLUSION:** In low-sodium diet population, association between dietary sodium-potassium ratio and stroke risk follows a "U" shaped correlation. However, prospective investigations are warranted to provide additional evidence to support findings.

INTRODUCTION

Stroke, also known as cerebrovascular accident, is a serious cardiovascular disease and ranks as the second leading cause of death globally, accounting for approximately 11.6% of total mortality (Collaborators 2021). During 1990-2019, global stroke incidence and mortality increased by 70% and 43%, respectively (Collaborators 2021). According to statistics, the economic loss attributable to stroke exceeds US$721 billion, representing about 0.66% of the global Gross Domestic Product (GDP), with a particularly heavy economic burden on low- and middle-income countries (Feigin *et al.* 2022). Numerous studies have linked high sodium intake to an increased risk of stroke and higher mortality rates (Aburto *et al.* 2013; Wang *et al.* 2023a). It is estimated that reducing daily salt intake by 3000 mg (equivalent to 1179 mg of sodium) could prevent 32,000-66,000 new stroke cases (Bibbins-Domingo *et al.* 2010). Judge *et al.* reported a J-shaped relationship between sodium intake and stroke risk, indicating that stroke risk rises significantly once sodium intake exceeds a certain threshold (Judge *et al.* 2021). However, some prospective studies have found no correlation between sodium intake and stroke, while demonstrating an inverse association between potassium intake and stroke events (Yang *et al.* 2011). Although the association between sodium intake and stroke remains controversial, evidence suggests that the sodium-to-potassium ratio is a more accurate predictor of cardiovascular disease and mortality risk than evaluating dietary sodium or potassium alone (Bailey *et al.* 2015; Mirmiran *et al.* 2018; Mosallanezhad *et al.* 2023).

The dietary sodium-to-potassium ratio refers to the relative proportion of sodium to potassium in the human diet. Maintaining an appropriate sodium-potassium balance is generally necessary for physiological health (Bailey *et al.* 2015). However, modern diets are often characterized by high salt intake (high sodium and low potassium), leading to an elevated sodium-to-potassium ratio. This imbalance has been closely associated with various health issues, including stroke (Neal *et al.* 2021; Li *et al.* 2012). A longitudinal follow-up study identified the dietary sodium-potassium ratio as an independent predictor of cardiovascular events, including stroke (Mosallanezhad *et al.* 2023). Similarly, the NIPPON DATA80 cohort study in Japan also demonstrated that the dietary sodium-to-potassium ratio is a risk factor for stroke (Okayama *et al.* 2016). Research has shown a positive correlation between the dietary sodium-potassium ratio and stroke risk (Jayedi *et al.* 2019), while increasing potassium intake to lower the ratio may help reduce the risk of stroke (Goncalves & Abreu 2020). It is worth noting that most existing studies have focused on general populations with typically high daily sodium intake. According to the Dietary Guidelines for Americans (2020–2025), it is recommended that individuals aged 14 and older consume no more than 2,300 mg of sodium per day (Agriculture *et al.* 2020). However, the relationship between the sodium-to-potassium ratio and stroke risk remains unclear among populations who adhere to this recommended low-sodium diet.

Building on previous research, we hypothesized that there is an association between the dietary sodium-potassium ratio and stroke risk among populations with low sodium intake (≤2300 mg/day). To test this hypothesis, this study conducted a cross-sectional analysis using summarized data from 7 cycles of the National Health and Nutrition Examination Survey (NHANES) to examine the association between the dietary sodium-potassium ratio and stroke risk in the low-sodium diet populations. This study aims to contribute to the existing literature by further enriching the evidence regarding the association across different population groups.

METHODOLOGY

Data Source

NHANES is nationally representative cross-sectional research conducted continuously for testing health and nutritional status among U.S. residents, with data released in two-year cycles. We did a cross-sectional study of data from 7 cycles of NHANES spanning from 2003 to 2016, utilizing complex sampling weights. 71,058 participants were involved. Participants were sequentially excluded based on inclusion and exclusion criteria, removing participants with missing medical questionnaire data (n = 31,895), those with missing dietary sodium-potassium intake data or sodium intake >2300 mg (n = 30,289), and those with missing multiple categorical variables data (n = 1,733), resulting in a final sample of 7,141 participants for analysis (Fig. 1). NHANES survey protocol obtained approval from National Center for Health Statistics (NCHS) Research Ethics Review Board, and all participants given written informed consent.

Sodium and Potassium Intake and Outcomes

NHANES included data on dietary sodium and potassium intake measured through two 24-hour dietary recalls. Initial recall was conducted face-to-face, and

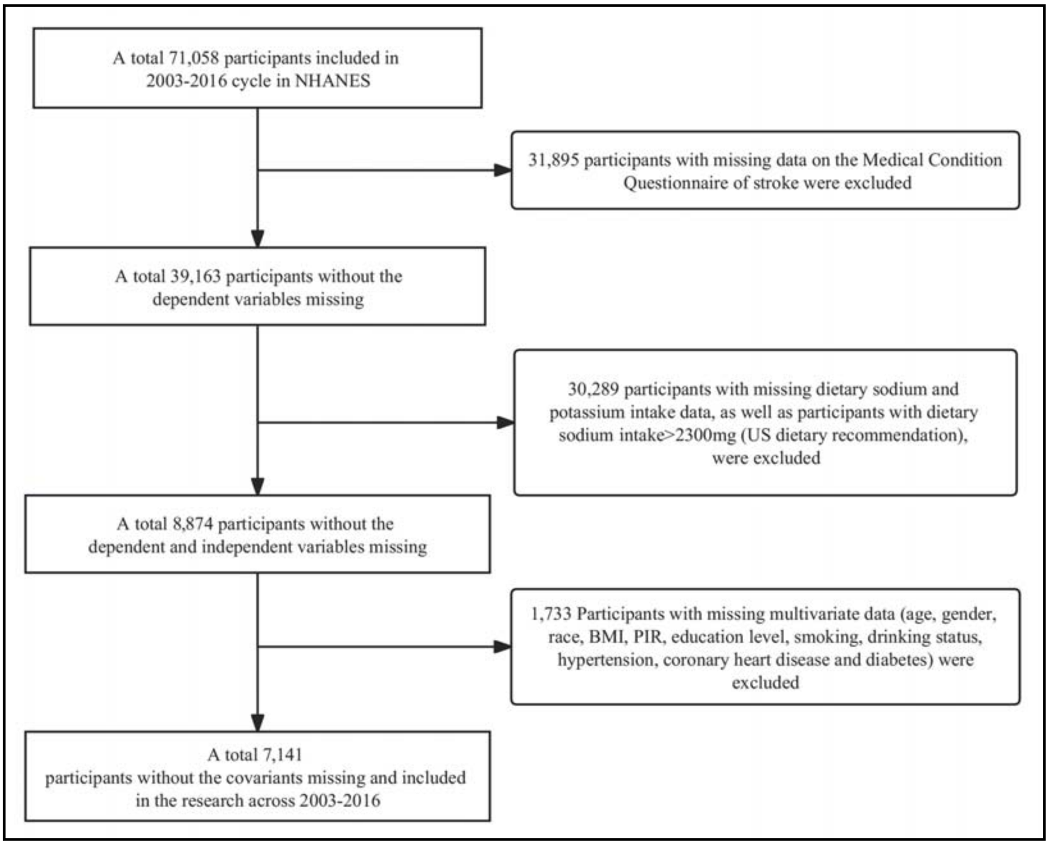

Fig. 1. Analysis Sample Flowchart

the second recall was done over the phone follow-up 3-10 days later. A 24-hour recall captured all food and beverages consumed from midnight to midnight the day before the interview. The average sodium and potassium intake (mg/day) for each participant was estimated as the mean of two dietary recalls. These intake values were subsequently converted to g/day for all analyses (Hu *et al.* 2020; Chmielewski & Carmody 2017).

Stroke was defined based on the response to "Has a doctor or other health professional ever told you that you had a stroke?" (Yang *et al.* 2022)

Covariates

Covariates included race, age, gender, poverty-income ratio (PIR) (≤1.3 as low-to-middle income, 1.3–3.5 as middle income, >3.5 as high income) (Stebbins *et al.* 2019), education level (college education or above, did not graduate from high school, graduated from high school (Huang *et al.* 2021)), Body Mass Index (BMI) (≤25 kg/m², 25–30 kg/m², and >30 kg/m²) (Tian *et al.* 2022), alcohol drinking (non-drinker and drinker) (Liu *et al.* 2022), and smoking (now, former, never) (Zhang *et al.* 2022). Hypertension (Lee & Kim 2022) was defined as meeting any of criteria: (a) being informed by a physician that they have hypertension; (b) being advised to take or already taking antihypertensive medication; or (c) achieving an average systolic blood pressure of 130 mmHg or diastolic blood pressure of 80 mmHg during NHANES assessment (measured three times consecutively after participant sat quietly for 5 minutes with maximum inflation level determined)(Miao *et al.* 2020). Diabetes (Zhao & Li 2022) was defined as meeting any of the criteria: (a) being diagnosed with diabetes by a physician; (b) taking glucose-lowering drugs at the moment; (c) Hemoglobin A1c (HbA1c) > 6.5%; or (d) fasting plasma glucose levels > 126 mg/dL. Coronary heart disease (CHD) (Wu *et al.* 2022) was defined as a "yes" response to "Whether a doctor or other health professional has ever told you that you had CHD." Detailed information on variables can be obtained by referencing modules through variable name definitions (https://wwwn.cdc.gov/Nchs/Nhanes/).

Statistical Analysis

All statistical analyses were completed on R (V4.2.2). Participants were grouped based on stroke status according to population characteristics, and a baseline table was constructed using the tableone package. Categorical variables were shown as sample size and percentage (n (%)), and continuous variables were presented as mean(sd). Categorical variables were stratified in an unadjusted model using the survey package to build a weighted logistic regression model for the association between the sodium-potassium ratio and stroke. In the unadjusted model, a stratified analysis was completed on categorical variables. *P* of the interaction term in the stratified logistic regression model adjusted for all confounders was assessed by the chi-square

Tab. 1. Characteristics of NHANES participants between 2003-2016

Characters	Total	Non-stroke	Stroke	*p* Value
Overall	7141	6764 (95.8)	377 (4.2)	
Gender				0.585
Female	4922.0 (73.3)	4685.0 (73.4)	237.0 (71.4)	
Male	2219.0 (26.7)	2079.0 (26.6)	140.0 (28.6)	
Age	50.67 (18.10)	49.99 (17.95)	66.24 (13.94)	<0.001
Race				0.003
Mexican American	1306.0 (8.9)	1270.0 (9.2)	36.0 (3.8)	
Other Hispanic	719.0 (5.8)	695.0 (5.9)	24.0 (3.6)	
Non-Hispanic White	2986.0 (65.9)	2797.0 (65.7)	189.0 (70.0)	
Non-Hispanic Black	1704.0 (14.1)	1592.0 (13.9)	112.0 (18.2)	
Other race	426.0 (5.4)	410.0 (5.4)	16.0 (4.4)	
PIR				0.004
≤1.3	2692.0 (28.5)	2536.0 (28.2)	156.0 (35.4)	
1.3-3.5	2796.0 (38.0)	2639.0 (37.9)	157.0 (42.0)	
>3.5	1653.0 (33.5)	1589.0 (34.0)	64.0 (22.6)	
Education				0.003
Did not graduate from high school	2316.0 (22.5)	2172.0 (22.2)	144.0 (29.5)	
Graduated from high school	1674.0 (24.3)	1579.0 (24.1)	95.0 (28.3)	
College education or above	3151.0 (53.2)	3013.0 (53.7)	138.0 (42.2)	
BMI (kg/m²)				0.571
≤25	2050.0 (32.1)	1948.0 (32.3)	102.0 (28.7)	
25-30	2411.0 (33.3)	2278.0 (33.3)	133.0 (34.2)	
>30	2680.0 (34.6)	2538.0 (34.5)	142.0 (37.1)	
Smoking				0.012
Never smoking	4030.0 (54.4)	3865.0 (54.8)	165.0 (46.2)	
Former smoking	1672.0 (22.7)	1539.0 (22.3)	133.0 (31.0)	
Now Smoking	1439.0 (23.0)	1360.0 (23.0)	79.0 (22.8)	
Alcohol drinking				0.016
No	2766.0 (34.2)	2609.0 (33.9)	157.0 (42.2)	
Yes	4375.0 (65.8)	4155.0 (66.1)	220.0 (57.8)	
Coronary heart disease				<0.001
No	6791.0 (95.9)	6482.0 (96.5)	309.0 (80.8)	
Yes	350.0 (4.1)	282.0 (3.5)	68.0 (19.2)	
Diabetes				<0.001
No	5796.0 (86.6)	5553.0 (87.4)	243.0 (69.6)	
Yes	1345.0 (13.4)	1211.0 (12.6)	134.0 (30.4)	
Hypertension				<0.001
No	2951.0 (46.8)	2903.0 (48.1)	48.0 (18.3)	
Yes	4190.0 (53.2)	3861.0 (51.9)	329.0 (81.7)	
Sodium (g)	1.78 (0.41)	1.78 (0.40)	1.73 (0.42)	0.097
Potassium (g)	1.85 (0.78)	1.86 (0.78)	1.68 (0.71)	<0.001
Ratio (Na : K)	1.11 (0.52)	1.11 (0.52)	1.19 (0.51)	0.009

Note: Categorical variables are presented as n (%), and continuous variables as mean (SD). The counts (n) are unweighted, while percentages (%), means, and standard deviations (SD) are adjusted for survey weights.

test. $p < 0.05$ indicated significant differences. Dietary sodium-potassium ratio was stratified using quartiles, and weighted logistic regression models for the association were established using survey package. Subgroup analysis for confounder BMI, with interaction term $p < 0.1$, was conducted. The association was explored using RCS in a weighted logistic regression model adjusted for all confounders.

RESULTS

Baseline Characteristics

7,141 participants with dietary sodium intake ≤2300 mg were included. Overall characteristics distribution is presented in Table 1. In the low-sodium diet population, individuals with stroke tended to be older (66.24 (13.94)) and predominantly female (71.4%). Significant variances were observed between the 2 groups in terms of age, race, PIR, education level, smoking, alcohol drinking, CHD, diabetes, and hypertension ($p < 0.05$). Dietary sodium-potassium ratio in the stroke group (1.19 (0.51)) was significantly higher than in the non-stroke group (1.11 (0.52)) ($p < 0.05$).

Association between Dietary Sodium-Potassium Ratio and Stroke

Stratified analysis of categorical variables was conducted using a weighted logistic model (Table 2). An elevated dietary sodium-potassium ratio significantly raised stroke risk in populations comprising females, BMI of 25-30 kg/m², with a history of alcohol drinking, smoking, without CHD and diabetes, but with hypertension ($p < 0.05$). However, constructing interaction terms between confounders and dietary sodium-potassium ratio, adjusting for all confounders, and conducting interaction tests showed non-significant interaction terms (*p for interaction* > 0.05).

Association between Different Dietary Sodium-Potassium Ratios and Stroke

In all four models, regardless of adjusting for confounders, an elevated dietary sodium-potassium ratio significantly raised stroke risk (Odds Ratio (OR)>1, $p < 0.05$). Stratification of dietary sodium-potassium ratio into quartiles revealed that in all four models, an elevated dietary sodium-potassium ratio in Q4 (>1.31) significantly elevated stroke risk (OR>1, $p < 0.05$) compared to Q1, showing a trend of increasing stroke risk with each quartile change (*p for trend* < 0.05) (Table 3).

Relationship between Sodium-Potassium Ratio and Stroke across Different BMIs

Weighted logistic regression models were established for various dietary sodium-potassium ratios and stroke across BMI groups (Table 4). For individuals with a BMI of 25-30 kg/m², in all four models, an increased dietary sodium-potassium ratio significantly raised stroke risk

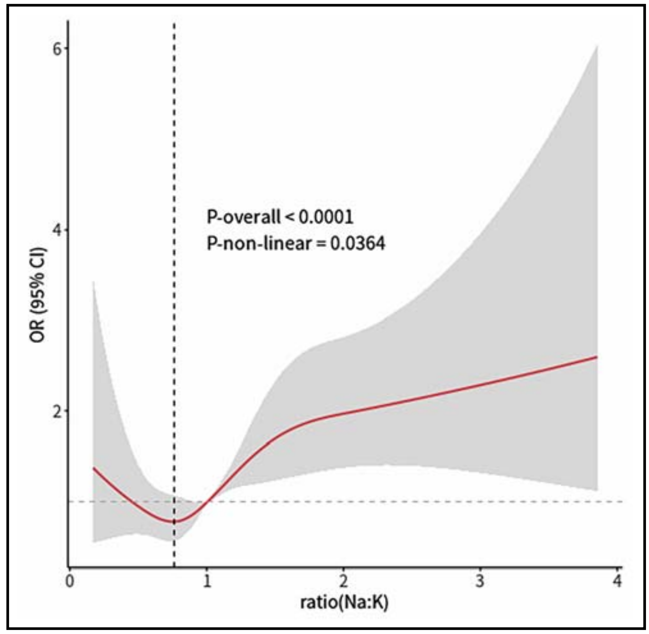

Fig. 2. The Odds Ratio of sodium-potassium Ratio with Stroke by Covariates, NHANES 2003-2016
Note: RCS line is adjusted for gender, age, race, PIR, education level, BMI, smoking, alcohol drinking, CHD, diabetes, and hypertension. The OR is represented by the red line, and the shaded part represents the 95% CI. OR: Odds ratio; CI: Confidence interval. NHANES: National Health and Nutrition Examination Survey.

(OR>1, $p < 0.05$). Stratification of dietary sodium-potassium ratio revealed that in all four models, in Q4 (>1.26) relative to Q1, the ratio was significantly associated with stroke risk (OR>1, $p < 0.05$). Consistent associations were observed when BMI was >30 kg/m², after adjusting for different confounders (OR>1, $p < 0.05$).

Nonlinear Relationship between Sodium-Potassium Ratio and Stroke

In the weighted logistic regression model adjusted for all confounders, we used RCS to delve into the relation between dietary sodium-potassium ratio and stroke (Fig. 2). Overall trend was significant (*p*-overall < 0.05), showing a non-linear association (*p-non-linear* = 0.0364 < 0.05). As the dietary sodium-potassium ratio increased, stroke risk followed a "U"-shaped curve. The ratio within the range of 0.46-1.00 significantly reduced stroke risk, with the lowest odds ratio observed at a ratio of 0.76.

DISCUSSION

This study aimed to investigate the association between the dietary sodium-potassium ratio and the risk of stroke in a population adhering to a low-sodium diet. Our findings revealed a significant correlation between a high dietary sodium-potassium ratio and an increased risk of stroke within this population. This association was particularly pronounced among females, individuals with a BMI between 25 and 30 kg/m², those with

Tab. 2. Relationship between sodium-potassium ratio and stroke in categorical variables

Participants	OR	95% CI	*p*-value	*p* for interaction
Gender				0.704
Female	1.31	1.05-1.62	0.013	
Male	1.24	0.96-1.61	0.101	
Race				0.239
Mexican American	1.46	0.92-2.33	0.107	
Other Hispanic	1.41	0.59-3.34	0.431	
Non-Hispanic White	1.29	0.97-1.71	0.077	
Non-Hispanic Black	1.19	0.96-1.47	0.116	
Other race	0.74	0.23-2.40	0.606	
PIR				0.918
≤1.3	1.23	0.86-1.76	0.258	
1.3-3.5	1.19	0.98-1.45	0.079	
>3.5	1.38	0.72-2.67	0.329	
Education				0.709
Did not graduate from high school	1.43	0.97-2.09	0.066	
Graduated from high school	0.96	0.69-1.34	0.809	
College education or above	1.34	0.92-1.97	0.124	
BMI (kg/m^2)				0.066
≤25	1.04	0.66-1.62	0.869	
25-30	2.02	1.40-2.91	<0.001	
>30	1.17	0.90-1.52	0.230	
Smoking				0.497
Never smoking	1.27	0.91-1.78	0.157	
Former smoking	1.79	1.20-2.67	0.004	
Now Smoking	1.15	0.84-1.56	0.375	
Alcohol drinking				0.653
No	1.26	0.98-1.62	0.064	
Yes	1.29	1.00-1.67	0.047	
Coronary heart disease				0.983
No	1.33	1.10-1.62	0.003	
Yes	1.62	0.72-3.67	0.237	
Diabetes				0.240
No	1.33	1.08-1.63	0.006	
Yes	1.12	0.78-1.61	0.529	
Hypertension				0.534
No	1.48	0.99-2.21	0.051	
Yes	1.28	1.03-1.59	0.022	

Note: The P-values for interaction terms were adjusted for gender, age, race, PIR, education level, BMI, smoking, alcohol drinking, coronary heart disease, diabetes, and hypertension.

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Tab. 3. Association between sodium-potassium ratio and odds ratios (95% CIs) of stroke, NHANES 2003-2016

	OR (95% CI)							
Participants	**Crude**	***p*-value**	**Model I**	***p*-value**	**Model II**	***p*-value**	**Model III**	***p*-value**
All participants	1.28 (1.09-1.52)	0.003	1.66 (1.32-2.08)	<0.001	1.62 (1.29-2.03)	<0.001	1.61 (1.27-2.03)	<0.001
Ratio (Na: K)								
Q1 (≤0.77)	Ref.		Ref.		Ref.		Ref.	
Q2 (0.77-1.01)	1.05 (0.26-4.30)	0.942	0.73 (0.17-3.04)	0.657	0.74 (0.17-3.20)	0.688	0.68 (0.15-3.06)	0.609
Q3 (1.01-1.31)	1.85 (0.85-4.05)	0.118	1.94 (0.82-4.61)	0.129	1.88 (0.80-4.41)	0.144	1.76 (0.74-4.18)	0.197
Q4 (>1.31)	1.26 (1.05-1.51)	0.010	1.55 (1.24-1.95)	<0.001	1.52 (1.22-1.90)	<0.001	1.52 (1.21-1.92)	<0.001
P for trend	0.021		<0.001		<0.001		<0.001	

Note: Crude model: No adjustments. Model I: Adjusted for gender, age, race, PIR, and education level. Model II: Adjusted for gender, age, race, PIR, education level, BMI, smoking, and alcohol drinking. Model III: Adjusted for gender, age, race, PIR, education level, BMI, smoking, alcohol drinking, coronary heart disease, diabetes, and hypertension.

a history of alcohol consumption or smoking, those without CHD or diabetes, but with hypertension. Using restricted cubic spline (RCS) analysis, we identified a U-shaped relationship between the sodium-potassium ratio and stroke risk. For individuals on a low-sodium diet, maintaining a dietary sodium-potassium ratio between 0.46 and 1.00 may effectively reduce the risk of stroke, with the lowest risk observed at a ratio of 0.76. These findings underscore the importance of monitoring the sodium-potassium ratio in low-sodium diets and provide actionable dietary guidance for stroke prevention.

This study identified a positive association between the dietary sodium-potassium ratio and the risk of stroke in a low-sodium diet population. This association remained robust after adjusting for multiple confounding factors and is consistent with previous findings in other populations. For instance, Jayedi *et al.* demonstrated that among adults aged 18 and older, stroke risk increases with higher dietary sodium-potassium ratios. Each unit increase (in mmol/mmol) in the dietary sodium-potassium ratio is associated with a pooled relative risk of 1.22 (95% Confidence Interval (CI): 1.04–1.41) for stroke (Jayedi *et al.* 2019). Similarly, Gonçalves *et al.* reported that a higher dietary sodium-potassium ratio and lower potassium intake are associated with an increased risk of stroke among older adults (Goncalves & Abreu 2020), a phenomenon also observed in the elderly Japanese population (Yamori & Horie 1994). Therefore, even in populations consuming a low-sodium diet, attention should be paid to balancing sodium and potassium intake to reduce stroke risk.

The American College of Cardiology recommends that adults maintain a daily sodium-potassium intake ratio well below 1 (2300 mg/4700 mg) (Eckel *et al.* 2014). Consistently, the Multi-Ethnic Study of Atherosclerosis demonstrated that a urinary sodium-potassium ratio ≤1 is associated with a 40–50% reduction in stroke risk (Averill *et al.* 2019). Our study further revealed that a dietary sodium-potassium ratio exceeding 1.31 significantly increased stroke risk in populations adhering to a low-sodium diet. This threshold was even lower (>1.26) among individuals with a BMI between 25–30 kg/m². Based on multivariate RCS analysis, maintaining a dietary sodium-potassium ratio between 0.46 and 1.00 may confer protective effects against stroke in low-sodium diet populations. The National Institutes of Health recommends a daily potassium intake of 2600 mg for adult women and 3400 mg for adult men (Potassium). Moderately increasing consumption of potassium-rich foods, such as fresh fruits, vegetables, legumes, nuts, fish, and dairy products (Weaver 2013), or using potassium-enriched salt substitutes may help maintain the sodium-potassium ratio within an optimal range (Neal *et al.* 2021).

The Global Burden of Disease study identified high BMI as one of the most rapidly growing risk factors for stroke (Collaborators 2021). Our findings are consistent with those of Liu *et al.* who reported a J-shaped relationship between BMI and stroke risk, with risk significantly increasing once BMI exceeds 25 kg/m² (Liu *et al.* 2018). A potential mechanism for this association may involve urinary albumin excretion, which is elevated in individuals with higher BMI at comparable sodium intake levels (Verhave *et al.* 2004; Tagawa *et al.* 2023). This can indicate endothelial damage and increased susceptibility to cardiovascular diseases (Deckert *et al.* 1989). Adequate dietary potassium intake may offer protection against albuminuria (Meneely & Ball 1958). Animal and clinical studies suggest that high-potassium diets protect blood vessels from sodium-induced damage, possibly by inhibiting sympathetic activity in salt-sensitive hypertension (Fujita & Ando 1984). Therefore, targeted dietary management is essential

Tab. 4. Relationship between sodium-potassium ratio and stroke by BMI (95% CI), NHANES, 2003-2016

	OR (95% CI)							
Participants	**Crude**	***p*-value**	**Model I**	***p*-value**	**Model II**	***p*-value**	**Model III**	***p*-value**
≤25 (kg/m²)	1.04 (0.66-1.62)	0.869	1.46 (0.98-2.16)	0.057	1.39 (0.95-2.03)	0.089	1.37 (0.93-2.01)	0.104
Q1 (≤0.76)	Ref.		Ref.		Ref.		Ref.	
Q2(0.76-0.99)	3.93 (0.19-81.5)	0.371	3.8 (0.14-105)	0.425	3.53 (0.14-89.3)	0.438	2.95 (0.13-68.1)	0.494
Q3 (0.99-1.30)	1.13 (0.27-4.70)	0.868	1.09 (0.27-4.35)	0.902	1.03 (0.26-4.08)	0.971	1.34 (0.29-6.21)	0.704
Q4 (>1.30)	1.13 (0.72-1.75)	0.593	1.46 (0.96-2.21)	0.074	1.42 (0.96-2.10)	0.073	1.37 (0.88-2.12)	0.157
25-30 (kg/m²)	2.02 (1.40-2.91)	<0.001	2.44 (1.59-3.74)	<0.001	2.4 (1.56-3.70)	<0.001	2.34 (1.52-3.60)	<0.001
Q1 (≤0.76)	Ref.		Ref.		Ref.		Ref.	
Q2 (0.76-0.98)	1.11 (0.09-14.2)	0.933	0.42 (0.05-3.66)	0.427	0.36 (0.05-2.81)	0.325	0.34 (0.04-3.00)	0.327
Q3 (0.98-1.26)	2.76 (0.88-8.65)	0.078	2.77 (0.81-9.54)	0.101	2.72 (0.80-9.24)	0.105	2.65 (0.76-9.27)	0.123
Q4 (>1.26)	1.93 (1.33-2.80)	<0.001	2.29 (1.57-3.35)	<0.001	2.22 (1.51-3.26)	<0.001	2.18 (1.45-3.28)	<0.001
>30 (kg/m²)	1.17 (0.90-1.52)	0.230	1.45 (1.05-2.00)	0.021	1.43 (1.05-1.96)	0.023	1.54 (1.09-2.16)	0.012
Q1 (≤0.79)	Ref.		Ref.		Ref.		Ref.	
Q2 (0.79-1.05)	0.22 (0.04-1.13)	0.066	0.22 (0.04-1.26)	0.085	0.23 (0.04-1.32)	0.094	0.2 (0.03-1.29)	0.087
Q3 (1.05-1.38)	1.44 (0.45-4.57)	0.535	1.65 (0.47-5.81)	0.429	1.7 (0.48-6.07)	0.407	1.67 (0.44-6.40)	0.448
Q4 (>1.38)	1.09 (0.83-1.44)	0.534	1.35 (0.98-1.88)	0.066	1.34 (0.98-1.83)	0.063	1.39 (1.02-1.92)	0.038

Note: Crude model: No adjustments. Model I: Adjusted for gender, age, race, PIR, and education level. Model II: Adjusted for gender, age, race, PIR, education level, BMI, smoking, and alcohol drinking. Model III: Adjusted for gender, age, race, PIR, education level, BMI, smoking, alcohol drinking, coronary heart disease, diabetes, and hypertension.

for individuals with high BMI among low-sodium population.

A prospective cohort study found no significant differences in the association between dietary sodium-potassium ratio and cardiovascular disease based on sex or hypertension status (Yang *et al.* 2011). In contrast, our study revealed a strong association between this ratio and stroke risk specifically among women and hypertensive individuals. This discrepancy may be attributed to women's heightened susceptibility to stroke, which is influenced by factors such as longer life expectancy (Virani *et al.* 2020), reproductive factors (Wang *et al.* 2023b), higher rates of passive smoking (Pan *et al.* 2019), and increased prevalence of migraines (Tietjen & Maly 2020). Hypertension, a major risk factor for stroke, promotes stroke by increasing shear stress, arterial stiffness, and endothelial dysfunction, ultimately transmitting pulsatile flow to the cerebral microcirculation (Cipolla *et al.* 2018). Meng *et al.* proposed a mechanism through which high potassium intake may confer protection: by inhibiting sodium-chloride cotransporters in the distal tubules, potassium reduces sodium reabsorption in the kidneys, thereby lowering vascular tension and blood pressure (Meng *et al.* 2022). Thus, we conclude that women and hypertensive individuals represent high-risk subgroups for stroke in low-sodium populations. Maintaining an appropriate dietary sodium-potassium ratio is therefore particularly crucial for stroke prevention in these groups.

This study is the first to investigate the association between dietary sodium-potassium ratio and stroke risk specifically in a low-sodium diet population, providing new evidence on the impact of dietary patterns on stroke in this group. Furthermore, the analysis incorporated comprehensive data from seven cycles of the NHANES database, and the relatively large sample size helps mitigate potential biases associated with limited cohort size. However, several limitations should be acknowledged. First, the study population consisted exclusively of U.S. residents, which may limit the generalizability of the findings to other populations. Second, dietary data in NHANES were collected via 24-hour dietary recalls and telephone follow-ups, which are susceptible to recall bias. Additionally, certain subgroups (e.g., individuals with BMI ≤25 kg/m²) had a low number of stroke events, resulting in wide CIs and necessitating cautious interpretation. Finally, as a cross-sectional study, this research can only demonstrate associations rather than establish causality. Future prospective cohort studies or randomized controlled trials are warranted to validate the causal relationship between the sodium-potassium ratio and stroke risk in low-sodium diet populations, and to further elucidate its long-term effects and underlying mechanisms.

DECLARATION

Ethics approval and consent to participate

Ethical approval and consent were not required as this study was based on publicly available data

Data availability statement

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

Consent for publication

Not applicable

Competing Interests

Not applicable.

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Authors' contributions

Liguo Li and Ya Su contributed to data analysis, drafting and revising the article, gave the final approval of the version to be published, and agreed to be accountable for all aspects of the work.

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