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Dietary sodium to potassium ratio and risk of stroke in a multi-ethnic urban population: The Northern Manhattan Study

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Abstract

Background and Purpose—There is growing evidence that increased dietary sodium (Na) intake increases the risk of vascular diseases, including stroke, at least in part via an increase in blood pressure (BP). Higher dietary potassium (K), seen with increased intake of fruits and vegetables, is associated with lower BP. The goal of this study was to determine the association of a dietary sodium to potassium ratio (Na:K) with risk of stroke in a multi-ethnic urban population.

Methods—Stroke-free participants from the Northern Manhattan Study, a population-based cohort study of stroke incidence, were followed for incident stroke. Baseline food-frequency questionnaires were analyzed for Na and K intake. We estimated the hazard ratios and 95% confidence intervals (HR, 95% CI) for the association of Na:K with incident total stroke using multivariable Cox-proportional hazards models.

Results—Among 2570 participants with dietary data (mean age 69 ± 10 years, 64% women, 21% white, 55% Hispanic, 24% black), the mean Na:K ratio was 1.22 ±0.43. Over a mean follow-up of 12 years, there were 274 strokes. In adjusted models a higher Na:K ratio was associated with increased risk for stroke (HR 1.6, 95% CI 1.2–2.1), and specifically ischemic stroke (HR 1.6, 95% CI 1.2–2.1).

Conclusions—Na:K intake is an independent predictor of stroke risk. Further studies are required to understand the joint effect of Na and K intake on risk of CVD.

Keywords

sodium; potassium; diet; stroke; epidemiology

Subject terms

Epidemiology; Diet and Nutrition; Risk Factors; Ischemic Stroke

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Introduction

Dietary sodium (Na) and potassium (K) are increasingly recognized as important contributors to cardiovascular disease (CVD). There is substantial and growing evidence that an increase in dietary Na^+ increases morbidity and mortality from cardiovascular disease (CVD), at least in part via an increase in blood pressure (BP). [1] Much of the increased Na intake stems from processed and restaurant foods.[2] Dietary K, derived primarily from fruit and vegetable intake, has an opposite effect, with higher intake having a protective effect against elevated BP.[3] The 2010 US Dietary Guidelines, which few are able to meet, emphasized that dietary Na should be limited to 2300 milligrams per day (mg/d), with a lower limit of 1500 mg/d for adults aged >50 years, non-Hispanic blacks, and those with diabetes, hypertension, or chronic kidney disease; the K intake goals were ≥4700 mg/d.[4] Guidelines on NA and K intake are based on clinical trials showing that reducing Na intake from moderate to low levels results in modest reductions in BP.[5,6] Although the physiological mechanisms are not fully understood, a diet high in K+ may decrease the adverse effect of dietary Na+, resulting in lower BP and decreased risk of stroke.[7] There are fewer studies, however, examining the role of the Na:K ratio on risk of CVD and stroke[8]. Prior observational studies on the role of the Na:K ratio on the risk of CVD have been carried out in European and Asian populations, which have different dietary patterns than populations living in urban centers in the United States[9,10]. Furthermore, few of these studies have examined the role of the Na:K ratio in populations with large proportions of Caribbean Hispanics, who have an understudied dietary pattern. We have previously reported, for example, that few residents in Northern Manhattan meet dietary guidelines for Na intake or adhere to the Mediterranean diet[11,12]. In a previous publication we showed that Northern Manhattan Study (NOMAS) participants who consumed ≥4000 mg/day sodium had an increased risk of stroke in comparison to those who consumed ≤1500 mg/day, and there was a 17% increased risk of stroke for each 500 mg/day increase, independent of vascular risk factors[11]. The purpose of the current study therefore was to examine whether dietary K and the Na:K ratio are associated with the risk of stroke in the Northern Manhattan Study. We hypothesized that low K intake and high Na:K ratio would be associated with a higher risk of stroke.

Methods

Study Population

NOMAS is a population based cohort study designed to determine risk factors for stroke and CVD in a multi-ethnic urban population. Eligible participants were: 1) stroke free; 2) resident of at least 3-months duration of Northern Manhattan as defined by zip-codes 10031, 10032, 10033, 10034, & 10040; 3) randomly derived from a household with a telephone; 4) age 40 or older (changed to age 55 or older in 1998) at the time of first in-person assessment. Participants were identified by random-digit dialing (91% telephone response rate) and recruited to have an in-person baseline interview and assessment between 1993–2001. The enrollment response rate was 75%, and 3,298 participants were enrolled. The study was approved by the institutional review boards at Columbia University Medical

Center and the University of Miami. All participants gave informed consent to participate in the study.

Baseline Risk Factors

Data regarding baseline status and risk factors were collected through interviews of participants. Race-ethnicity was determined by self-identification based on a questionnaire adapted from the 2000 U.S census. Standardized questions were asked regarding hypertension, diabetes, cigarette smoking, alcohol intake and cardiac comorbidities. Smoking was categorized as current (within the past year), former, or never smoker of cigarettes, cigars or pipes. Moderate alcohol use was defined as current drinking of >1 drink per month and ≤2 drinks per day. Moderate to heavy physical activity level was defined as engaging in one or more of selected rigorous physical activities in a typical 14-day period, as described previously [13]. Blood pressure was measured twice, before and after each examination, and averaged. Hypertension was defined as a blood pressure ≥140/90 mmHg, the patient's self-report of hypertension, or use of anti-hypertensive medications. Diabetes mellitus was defined by the patient's self-report of a history of diabetes, use of insulin or oral anti-diabetic medication, or fasting glucose ≥126 mg/dl. Hypercholesterolemia was defined as having a total cholesterol level of greater than 200 mg/dl, use of cholesterol lowering medications, or self-reported history of hypercholesterolemia. Calculation of the estimated glomerular filtration rate (eGFR) was performed using the CKD-epi formula.

Diet

At baseline, participants were administered a modified Block National Cancer Institute food frequency questionnaire by trained research assistants, in English or Spanish. This questionnaire assesses dietary patterns over the previous year, and was modified to include specific dietary items commonly consumed among Hispanics. Dietary Na and K intake were calculated based on self-reported food consumption using DIETSYS software (Block Dietary Data System: Dietsys+ analysis software, version 59, 1999) and reported as mean milligrams per day. Potassium consumption was examined continuously per 100 mg and in quartiles. The ratio of Na to K was examined continuously. For descriptive analyses we identified the median values for Na+ (2803 mg) and K+ (2446 mg), and we divided the cohort into 4 categories: low sodium (<median) and high potassium (>median) as the reference, low sodium (<median) and low potassium (<=median), high sodium (>=median) and high potassium (>median), and high sodium (>=median) and low potassium (<=median). Details on the NOMAS dietary assessments have been previously published.[11] A score to represent adherence to a Mediterranean-style diet has been described previously for this cohort.

Cardiovascular Disease Outcomes and Mortality

The primary outcomes of interest were all stroke and ischemic stroke. Participants were followed annually via phone screening to detect any new neurological symptoms, hospitalizations, or death. Potential strokes were adjudicated by two neurologists independently after review of all data. Cause of death was ascertained through phone discussion with the participant's family, review of medical records, and when available, a copy of the death certificate. Complete loss to follow up occurred in less than 1%.[14]

Statistical analysis

For our analysis, we excluded participants without a completed diet questionnaire (N=132), with improbable total daily kilocalories or Na+ consumption based on food frequency responses (<500 or >4000 kcal/day or >10,000 mg/day sodium, N=272), and those with an MI before baseline (n=237) due to concerns that they may have altered their data after the initial event, and because their baseline higher risk of recurrent events may have made this proportion of our sample biased. We examined the unadjusted associations of categories of sodium and potassium consumption with sociodemographics and vascular risk factors using ANOVA and chi-square tests. The association of K consumption and Na:K with incident stroke was examined using Cox proportional hazards models in a series of models, unadjusted and adjusted for potential confounders, after confirming the appropriateness of the proportional hazards assumption. Follow-up accrued from baseline to the date of stroke or death, loss to follow-up, or until March 2017, whichever came first, and this time from baseline to event or end of follow-up was used as the time scale in the Cox models. Model 1 for the analysis of potassium consumption included the following covariates: age, sex, high school completion, race-ethnicity, total calories, Mediterranean diet score, moderate alcohol use, moderate-heavy physical activity, smoking, and sodium consumption. Model 2 additionally adjusted for eGFR, body mass index, hypertension, hypercholesterolemia, and diabetes. The analysis of Na:K ratio only included Na+ consumption as a covariate in a sensitivity analysis in which it was added to model 2. We hypothesized a potential interaction between Na and K in relation to stroke, so we first looked at interactions between K+ (assessed continuously) and Na (<2300 mg/d vs >=2300 mg/day).

Results

Baseline demographics

There were a total of 2496 participants in NOMAS without a history of MI at baseline who had dietary data from the food frequency questionnaire available. The mean age of the cohort was 69 ± 10 years, with approximately 64% women and 55% Hispanic. Baseline demographics of the cohort are outlined in table 1. The mean dietary Na intake in the cohort was high (3057 ± 1510 mg/d, median=2803, with only a small proportion of the population being below dietary Na guidelines (<2300 mg/d 36%; < 1500 mg/d 12%). The dietary K intake was low (mean 2591 ± 1099 mg/d; median=2446, interquartile range=1805–3211), with only a small proportion of the population meeting K intake guidelines (>4700 mg/d = 5%). The mean Na:K ratio was 1.22 ± 0.43 with 38% of the sample having both high Na and K intake (above the median for both).

Participants were followed for a mean of 12 ± 5 years and there were 268 strokes (227 ischemic).

Risk of stroke with potassium intake

Tests of interaction suggested effect modification by Na (<2300 mg vs >=2300 mg) for the relationship between K with stroke ($p<0.10$), so K was examined within strata of Na+ consumption, as shown in Table 2. Dietary K intake was evaluated as a continuous variable and by quartiles (with the reference the highest quartile). Among participants with Na intake

<2300 mg/day there was a trend toward a positive association between K intake and total stroke risk after adjusting for confounders. K was associated with ischemic stroke risk. Among participants with higher Na intake (2300 mg/day or more), there was a marginally significant (p<0.10) inverse association between K intake and all stroke that was stronger in relation to ischemic stroke only.

Risk of stroke with the sodium to potassium ratio

Table 3 outlines the association of the Na:K ratio with the risk of stroke. We found that an increased Na:K ratio was associated with a greater risk of ischemic and all strokes independently of other confounders. When we further adjusted for Na in sensitivity analysis model 3, the effect estimates were attenuated for all stroke. There were 346 study participant who reported taking a diuretic, which could modify Na and K levels. When we exclude these participants in sensitivity analyses the conclusions remained the same. For example, the hazard ratio (95% CI) for Na:K ratio in relation to all stroke in model 2 was 1.50 (1.11–2.03).

The increased risk of stroke among participants with both high sodium consumption (>median) and low potassium (<median) vs all others was of borderline significance in model 2 (HR=1.38, 95% CI=0.98–1.93, p=0.06).

Effect modification by race/ethnicity was not observed for either K nor Na:K in relation to the outcomes.

Discussion

In our elderly multi-ethnic urban dwelling population with an overall high dietary intake of Na and low intake of K we found that an increase in the ratio of Na:K was associated with an increased risk of stroke. The relationship between K+ intake and stroke appeared to be dependent on Na+ intake, such that an unexpected positive association was observed for K intake among those with <2300 mg Na/day, and an expected inverse association was observed for K intake among those with ≥2300 mg Na/day. Our findings support the notion that Na and K dietary intake's influence on risk of stroke may be best understood in the context of the amount of both in combination. This approach is similar to other research on dietary intake with risk of CVD, where patterns of diet such as the Mediterranean diet provide more meaningful information on risk in comparison to analyses on single micronutrients. Our results also suggest that among those with high sodium consumption, who are therefore at an increased risk for hypertension and stroke, increased consumption of potassium-rich foods may help lower stroke risk, but further research to confirm this hypothesis is needed.

There is a substantial body of research on the effects of Na and K intake when considered separately[15], but more limited research on both moieties in combination especially in populations with high Na intake such as African-Americans and Hispanics in urban centers.[16] The data on dietary Na is well known, and public health campaigns to reduce dietary Na intake in processed foods as well as in restaurants have made significant strides in reducing the burden of disease associated with hypertension and CVD.[17] Several researchers

have identified an association of higher dietary potassium, seen with increased intake of fruits and vegetables, with lower BP.[3] High K intake reduces BP in adults with hypertension and has no adverse effect on blood lipids and catecholamine concentration, or renal function.[18] The effect of increased K+ intake seems to act primarily through a reduction in blood pressure, making it not surprising that there is a particularly strong effect on the risk of stroke as we, and others, have noted[2]. For K, the data suggests that supplementation is best achieved through alterations in the diet, rather than by supplements, since interactions with other nutrients are associated with its health benefits.[8] The relative levels of both Na and K intake may provide additional information on risk of CVD such that increasing K intake in the face of high Na intake (thereby reducing the ratio) may be effective at reducing the risk of stroke. The findings of a higher K intake being associated with an increased risk of stroke was however an unexpected finding, though we note the overall high Na intake in our population this is unlikely to be the only explanation. One possible explanation is that Na and K intake can be associated with difficult to control hypertension or congestive heart failure (CHF), such that those participants considered at highest risk were counseled by a physician to decrease Na intake and increase K intake. In other words, participants with low sodium intake may have adopted this dietary pattern after having been counseled by a physician to lower sodium intake due to hypertension or other comorbidities. In NOMAS we did not collect measures of severity of HTN or CHF and may therefore have residual confounding.

The strengths of our study include examining the role of Na and K intake on the risk of stroke in a multi-ethnic population with a very high Na intake, noting that increasing K intake could offset the risk associated with a high Na intake. Our study however has several limitations. Our Na and K intake was based on answers to a food frequency questionnaire rather than objective measures such as 24 hour urinary excretion which may be more informative.[19] Food frequency questionnaires are particularly deficient in accurately capturing sodium consumption from salt added at the table, and are limited because the sodium content in prepared foods varies widely. However, we have previously shown Na consumption from this questionnaire to be associated with stroke risk in this study population. We only collected one measure of Na and K intake and do not have information on dietary patterns in mid-life or after enrollment which may provide additional information on the true effect on CVD. We were also unable to capture dietary changes that may result from preclinical diagnoses prior to baseline that may impact risk of the outcomes of interest. Lastly, our small sample size may limit power to detect more subtle effects on stroke subtypes in categorical analyses, resulting in wide confidence bounds.

In summary, we found that Na:K intake is an independent predictor of stroke risk in a multi-ethnic population with a low proportion meeting dietary guidelines. These findings emphasize the importance of considering dietary data as a whole versus examining micronutrients individually. Further studies are required to understand the joint effect of Na and K intake on risk of stroke.

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Table 1

Baseline Demographics

	Overall	High sodium and low potassium* (N=293)	High sodium and high potassium* (N=955)	Low sodium and low potassium* (N=955)	Low sodium and high potassium* (N=293)	P-value
Mean age (SD)	68.66 (10.29)	67.36 (9.75)	67.92 (10.08)	69.37 (10.51)	70.09 (10.46)	0.0002
Male (N, %)	902, 36	122, 42	412, 43	282, 30	86, 29	<0.0001
High school completion (N, %)	1123, 45	127, 43	446, 47	403, 42	147, 50	0.05
Race/ethnicity						<0.0001
Black (N, %)	592, 24	68, 23	199, 21	264, 28	61, 21	
White	529, 21	51, 17	216, 23	165, 17	97, 33	
Hispanic	1375, 55	174, 59	540, 57	526, 55	135, 46	
Mean Total calories (SD)	1570.00 (656.28)	1542.98 (340.13)	2155.05 (592.34)	1044.16 (302.60)	1403.98 (258.40)	<0.0001
Moderate alcohol use, (N, %)	857, 34	105, 36	350, 37	298, 31	104, 35	0.08
Moderate-heavy physical activity, (N, %)	213, 9	21, 7	97, 10	62, 7	33, 11	0.01
Smoking (N, %)						<0.0001
Never	1170, 47	123, 42	438, 46	454, 48	155, 53	
Former	906, 36	103, 35	361, 38	325, 34	117, 40	
Current	420, 17	67, 23	156, 16	176, 18	21, 7	
Mean BMI (SD)	27.93 (5.55)	28.14 (5.37)	28.07 (5.81)	27.78 (5.37)	27.75 (5.42)	0.57
Hypertension, (N, %)	1818, 73	208, 71	687, 72	702, 74	221, 75	0.55
Hypercholesterolemia, (N, %)	1451, 58	174, 59	550, 58	542, 57	185, 63	0.25
Diabetes, (N, %)	520, 21	66, 23	200, 21	185, 19	69, 24	0.38

*Sodium: high >=2803mg, low <2803mg. Potassium: high >2446mg, low <=2446mg

Table 2

Association of dietary potassium intake with risk of stroke stratified by sodium consumption in the Northern Manhattan Study.

		Sodium <2300mg (N=898)		Sodium ≥2300mg (N=1598)	
		Ischemic Stroke HR (95% CI)	**All stroke HR (95% CI)**	**Ischemic Stroke HR (95% CI)**	**All stroke HR (95% CI)**
Potassium intake per 100 mg/day	Model 1	1.05 (1.01–1.10)	1.04 (1.00–1.09)	0.97 (0.95–1.00)	0.98 (0.96–1.00)
	Model 2	1.05 (1.00–1.10)	1.04 (1.00–1.09)	0.97 (0.95–1.00)	0.98 (0.96–1.00)
Potassium intake (lowest versus highest quartile) *	Model 2	0.42 (0.08–2.08)	0.37 (0.10–1.43)	1.71 (0.89–3.28)	1.77 (0.98–3.20)
Potassium intake (2nd quartile versus highest quartile) †	Model 2	0.82 (0.18–3.73)	0.66 (0.19–2.32)	1.13 (0.65–1.96)	1.11 (0.67–1.84)
Potassium intake (3rd quartile versus highest quartile) ‡	Model 2	1.09 (0.24–4.88)	0.78 (0.22–2.76)	0.82 (0.51–1.33)	0.85 (0.55–1.32)

Model 1: adjusted for age, sex, high school completion, race-ethnicity, total calories, Mediterranean diet score, moderate alcohol use, moderate-heavy physical activity, smoking, sodium consumption

Model 2: variables in model 1+ estimated glomerular filtration rate, body mass index, hypertension, hypercholesterolemia, diabetes

* 207.9–1805.2 mg/day versus *3211.0–9543.5* mg/day

† *1805.8–2445.7* mg/day versus *3211.0–9543.5* mg/day

‡ *2446.8–3210.8* mg/day versus *3211.0–9543.5* mg/day

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Table 3

Association of the sodium and potassium ratio with risk of ischemic and all stroke in the Northern Manhattan Study

		Ischemic Stroke HR (95% CI)	**All stroke HR (95% CI)**
Sodium to potassium ratio (continuous)	Univariate	1.53 (1.17–2.02)	1.54 (1.20–1.98)
	Model 1	1.55 (1.17–2.06)	1.53 (1.18–1.99)
	Model 2	1.60 (1.19–2.14)	1.58 (1.20–2.06)
	Model 3	1.46 (1.00–2.13)	1.37 (0.97–1.95)

Model 1: adjusted for age, sex, high school completion, race-ethnicity, total calories, Mediterranean diet score, moderate alcohol use, moderate-heavy physical activity, smoking

Model 2: variables in model 1 + estimated glomerular filtration rate, body mass index, hypertension, hypercholesterolemia, diabetes

Model 3: variables in model 2 + sodium consumption. In this model, the covariates that were risk factors for all stroke at $p<0.10$ included low education level (<highschool completion), age, male sex, black race, smoking, hypertension, and diabetes.