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Dietary sodium and risk of stroke in the Northern Manhattan Study

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Abstract

Background and purpose—The American Heart Association (AHA) recommends limiting sodium intake to ≤1500 mg/day for ideal cardiovascular health. Although sodium intake has been linked to vascular disease by direct relationship with hypertension, few studies have supported an association with stroke risk.

Methods—Participants were from the Northern Manhattan Study (mean age 69±10 years, 64% women, 21% white, 53% Hispanic, 24% black), a population-based cohort study of stroke incidence. Sodium intake was assessed with a food frequency questionnaire at baseline and evaluated continuously and categorically: ≤1500 mg/day (12%), 1501–2300 mg/day (24%), 2301–3999 mg/day (43%), ≥4000 mg/day (21%). Over a mean follow-up of 10 years we examined the association between sodium consumption and 235 strokes using Cox models, adjusting for sociodemographics, diet, behavioral/lifestyle and vascular risk factors.

Results—Of 2657 participants with dietary data, the mean sodium intake was 3031±1470 mg/day, median 2787 (IQR 1966–3815) mg/day. Participants who consumed ≥4000 mg/day sodium had an increased risk of stroke (HR=2.59; 95% CI=1.27–5.28) vs. those who consumed ≤1500 mg/day, with a 17% increased risk of stroke for each 500 mg/day increase (95% CI=1.07–1.27).

Conclusions—High sodium intake was prevalent and associated with an increased risk of stroke independent of vascular risk factors. The new AHA dietary sodium goals will help reduce stroke risk.

Keywords

sodium; stroke; diet; epidemiology

Introduction

The American Heart Association (AHA) lowered its recommended level of sodium consumption to ≤1500 mg/day for all Americans. This recommendation is based largely on the well-established relationship between excess sodium intake and hypertension[1]. Although hypertension is a risk factor for vascular disease, and is associated with an approximately threefold increased risk of stroke[2], studies on the relationship between sodium consumption and risk of stroke and cardiovascular disease have shown inconsistent results. A recent

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meta-analysis suggested that elevated salt intake was associated with an increased risk of stroke, and, to a lesser extent, with an increased risk of cardiovascular disease (CVD)[3]. However, there was significant heterogeneity in effect estimates across studies, and the majority of included studies did not show an association between high sodium intake and risk of stroke or cardiovascular disease, or did so only for certain population subsets. In fact, a recent cohort study of white Europeans even showed a higher rate of CVD mortality among those with lower sodium excretion[4]. Although the latter study had several limitations, including potential exposure misclassification and a relatively young population resulting in a small number of outcome events, it raised some doubts about the association of sodium and cardiovascular events. Limited research has been conducted within ethnically heterogeneous populations, including blacks and Hispanics, who are at an increased risk for hypertension and stroke[5, 6]. The controversial findings of this recent report and gaps in the literature regarding the association between sodium consumption and risk of CVD and stroke among Blacks and Hispanics, underscore the need for further research.

We examined the association between sodium consumption and risk of stroke and combined vascular events, stroke, myocardial infarction (MI), and vascular death, in a multiethnic population-based prospective cohort study.

Methods

Study population

The Northern Manhattan Study (NOMAS) is a cohort study designed to determine stroke incidence, risk factors, and prognosis in a multi-ethnic urban population. Study details have been published previously[7].

Eligible participants were: a) stroke-free; b) >40 years old; and c) resided in Northern Manhattan for ≥ 3 months with a household telephone. 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. For our analysis, we excluded participants without a completed diet questionnaire (N=132), with improbable total daily kilocalories or sodium 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). The study was approved by the Columbia University and University of Miami IRBs and all participants provided informed consent.

Baseline evaluation

Data were collected through interviews with trained research assistants in English or Spanish. Study physicians conducted physical examinations. Race-ethnicity was based upon self-identification using questions modeled after the US census and conforming to standard definitions outlined by Directive 15[8]. Standardized questions were adapted from the Behavioral Risk Factor Surveillance System by the Centers for Disease Control regarding hypertension, diabetes, smoking, and cardiac conditions[9]. Measurement of blood pressure (BP) and fasting blood specimens for glucose and lipids, and the definitions of hypercholesterolemia, diabetes, moderate-heavy physical activity, and moderate alcohol use were described previously[10, 11]. Hypertension was defined as BP ≥140/90 mmHg, antihypertensive medication use, or the participant's self-report of hypertension.

Diet

At baseline, participants were administered a modified Block National Cancer Institute food frequency questionnaire by trained research assistants, in English or Spanish[12]. This

questionnaire assesses dietary patterns over the previous year, and was modified to include specific dietary items commonly consumed among Hispanics. Sodium intake was calculated based on self-reported food consumption using DIETSYS software (Block Dietary Data System: Dietsys+ analysis software, version 59, 1999).

Average sodium consumption was examined continuously, with 500 mg/day as the unit of measurement, and in prespecified categories: ≤1500 mg/day (reference, AHA recommendation), 1501–2300 mg/day (consistent with USDA recommendation of ≤2300 mg for those at standard risk), 2301–3999 mg/day, 4000–10000 mg/day (approximately the top quintile).

Outcomes

The primary outcome was confirmed incident stroke of all subtypes (infarcts, intracerebral hemorrhage, and subarachnoid hemorrhage). Secondary outcomes were confirmed (1) incident combined vascular event (stroke, MI, or vascular death), (2) incident MI, and (3) vascular death. Follow-up procedures and outcome classifications were detailed previously[10, 13]. Subjects were screened annually by telephone to determine changes in vital status, detect neurologic events, document interval hospitalizations, and review risk factor status, medication changes, and changes in functional status. Persons who screened positive were scheduled for in-person assessment, including chart review and examination by study neurologists. Ongoing hospital surveillance of admission and discharge data, including screening of ICD-9 codes, was reviewed to detect outcome events. The outcome surveillance network includes screening of all daily admissions, daily contacts with the neurology consult residents, reviewing bi-monthly hospital discharge lists, emergency room visits, and visits to the ambulatory care network. All hospitalizations for suspected stroke or MI were reviewed thoroughly and trigger more extensive data collection for outcome adjudication. Medical records of all hospitalizations were reviewed to verify the details of suspected events. Outcome events were reviewed by a specially trained research assistant and when available, medical records were reviewed for all outcome events, including death, by the study neurologists and cardiologists.

Stroke was defined by the first symptomatic occurrence of any type of stroke including infarct, intracerebral hemorrhage (ICH), and subarachnoid hemorrhage (SAH). Stroke was defined based on WHO criteria as “rapidly developing clinical signs of focal (at times global) disturbance of cerebral function, lasting more than 24 hours or leading to death with no apparent cause other than that of vascular origin.” Strokes were classified as ICH, SAH and cerebral infarction (atherosclerotic extracranial vessel, atherosclerotic intracranial vessel, lacunar small vessel, cardioembolic, cryptogenic, and other determined cause). MI was defined by criteria adapted from the Cardiac Arrhythmia Suppression Trial and the Lipid Research Clinics Coronary Primary Prevention Trial and requires at least 2 of the 3 following criteria: (a) ischemic cardiac pain determined to be typical angina; (b) cardiac enzyme abnormalities defined as abnormal CPK-MB fraction or Troponin values; and (c) EKG abnormalities. Stroke events were adjudicated by the study neurologists and cardiac events by the study cardiologists. The cause of death was classified as vascular or non-vascular and based on information obtained from the family, medical records and death certificate. Vascular death included death due to stroke, MI, heart failure, pulmonary embolus, cardiac arrhythmia, or other vascular cause. These are ICD-9 codes 390–459.

Statistical analysis

We examined the unadjusted associations of categories of sodium consumption with sociodemographics and vascular risk factors using ANOVA and chi-square tests.

We used Cox proportional hazards models to examine the association between sodium consumption (continuously and categorically) and vascular events. Person-time of follow-up was accrued from baseline to the end of follow-up (March, 2011), the time of outcome event, death or loss to follow-up, whichever came first. We constructed the following models sequentially: (1) adjusted for demographics: age, sex, race/ethnicity, highschool completion; (2) adjusted for demographics and behavioral risk factors: smoking (never, former, current), moderate-heavy physical activity, moderate alcohol consumption, daily consumption of total kcal, protein, total fat, saturated fat, carbohydrates; (3) adjusted for demographics, behavioral risk factors, and vascular risk factors: diabetes, hypercholesterolemia, hypertension, previous cardiac disease, body mass index (BMI). We assessed potential effect modification by age, sex, race/ethnicity, hypertension status, and continuous BP measurements (in the overall sample and among those not taking anti-hypertensive medications), by including interaction terms between sodium consumption and these variables in model 3.

Results

This study included 2,657 NOMAS participants. The mean age at baseline was 69±10 years, 36% of participants were men, 21% white, 24% black, and 53% Hispanic. Over a mean follow-up of ten years, 615 vascular events accrued, including 235 strokes (202 ischemic strokes), 209 MIs, and 371 vascular deaths. The mean sodium consumption was 3031±1470 mg/day, median 2787 mg/day (inter-quartile range 1966–3815). Only 12% consumed the AHA recommended level of ≤1500 mg/day sodium, while 24% consumed 1501–2300, 43% 2301–3999, and 21% 4000–10000 mg/day.

Table 1 shows the risk factor profile of the study population overall and in relation to sodium consumption. In unadjusted analyses, lower sodium consumption was associated with older age, female sex, black race, never smoking, and anti-hypertensive use, while higher sodium consumption was associated with Hispanic ethnicity, moderate alcohol use, increased BMI and consumption of total kilocalories, protein, carbohydrates, total fat, saturated fat ($p<0.05$). There was no significant association between sodium consumption and continuous BP measurements or hypertension status at baseline.

We observed an increased risk of stroke with greater sodium consumption, and this relationship became stronger after adjusting for behavioral and vascular risk factors (Table 2). The analysis of sodium as a continuous variable showed a 17% increase in stroke risk for each 500 mg/day increase in sodium consumption (model 3, 95% CI=1.07–1.27). Those who consumed ≥4000 mg/day sodium had a 2.6-fold increase in stroke risk vs. those who consumed ≤1500 mg/day (model 3, 95% CI=1.27–5.28). Intake of sodium >1500 but < 4000 mg/day, had a HR of 1.3 for stroke which did not reach significance. We did not observe an interaction between sodium and age (interaction p=0.68), sex (interaction=0.84), race/ethnicity (interaction p=0.47 black vs. white, p=0.73 Hispanic vs. white), hypertension status (interaction p=0.99), or BP (interaction p=0.98 for systolic BP and p=0.73 for diastolic BP) at baseline in relation to stroke risk. When the outcome was restricted to ischemic stroke, the results remained consistent. A 16% increased risk of ischemic stroke was seen for each 500 mg/day sodium increase, and there was a 2.4-fold greater risk among those who consumed ≥4000 vs. ≤1500 mg/day sodium.

Table 3 shows the relationship between sodium consumption and combined vascular events. Consumption of ≥4000 mg/day sodium was associated with an elevated risk of combined vascular events vs. ≤1500 mg/day. Intake of 1501–2300 mg/day was associated with an increased risk of stroke, MI or vascular death compared to ≤1500 mg/day. There was no interaction between sodium consumption and age (interaction p=0.10), sex

(interaction=0.30), race/ethnicity (interaction p=0.19 black vs. white, p=0.18 Hispanic vs. white), hypertension status (interaction p=0.28), or BP (interaction p=0.18 for systolic BP and p=0.49 for diastolic BP) in relation to vascular events. No association was observed between sodium consumption and risk of MI or risk of vascular death (Table 3).

Discussion

Excessive sodium intake was prevalent in this population-based multi-ethnic cohort, with only 12% meeting the AHA recommended level of ≤1500 mg/day, only 36% meeting the USDA recommended level of ≤2300 mg/day, and 21% consuming ≥4000 mg/day, based on self-reported food consumption using a food frequency questionnaire. Excessive sodium intake was associated with an increased risk of vascular events, but in our event-specific analysis sodium consumption ≥4000 mg/day was associated mainly with stroke and less with MI or vascular death. There was a slight increased risk of stroke among those in the two daily sodium consumption categories between 1501–3999 mg in comparison to ≤1500 mg, but this did not reach statistical significance. For combined events, there was a significantly increased risk among those consuming 1501–2300 mg/day compared to ≤1500 mg/day. Although we found 17% relative increase in the hazard of stroke for every 500mg/day increase in dietary sodium intake, our data did not suggest a linear dose-response relationship between sodium consumption and stroke risk.

A meta-analysis supported a strong relationship between sodium consumption and stroke risk[3] although many previous prospective cohort studies did not show an association, or did so only for a subset of the study population. Specifically, a 23% increased risk of stroke was reported among those with higher salt intake (approximately 5 g/day more salt than those classified as consuming less salt). A 14% increased risk of cardiovascular disease was also associated with higher salt intake (p=0.07). Effect estimates across studies were heterogeneous, as were the methods used. Some studies also used food frequency questionnaires to assess sodium consumption, while others used 24-hour dietary recall or urinary sodium excretion analysis. The strength of the association with stroke risk was often different for men vs. women, but the direction of this difference was inconsistent. In our study, we did not observe effect modification by sex. Possible reasons for the lack of association between sodium and stroke risk in other studies include small sample size, misclassification of sodium intake, and short follow-up.

Our results are consistent with the meta-analysis[3] indicating a stronger association for sodium consumption with stroke than with cardiovascular disease. The majority of previous prospective studies also did not observe a significant relationship with global cardiovascular disease risk. The stronger relationship between sodium consumption and risk of stroke as compared to MI is likely due to the fact that BP is etiologically more important for stroke than MI[14]. Our findings do not support the conclusions of a recent study suggesting an increased risk of cardiovascular events among those with low sodium excretion levels in a predominantly white, younger European cohort.[4]

The current study includes a large proportion of blacks and Hispanics, who have been under-represented in the literature. As dietary behavior may vary across race/ethnic groups, even those living in the same community, and evidence suggests that blacks and Hispanics are at an increased risk of stroke and hypertension[5, 6], examination of the relationship between sodium consumption and stroke and cardiovascular disease risk in an ethnically heterogeneous population was needed. Sodium consumption differed by race/ethnicity in our study with Hispanics consuming the most, and therefore the attributable risk of sodium consumption for stroke among Hispanics is likely to be higher. The power to detect effect modification by race/ethnicity was modest, however, and we did not observe a significant

difference in the relationship between sodium consumption and stroke risk across race/ethnic groups.

Our study uses the new AHA recommended sodium consumption guideline as its reference value. The results show that a large proportion of our study population (88%) consumed more than the AHA recommendation of ≤1500 mg/day and that lowering their sodium consumption may have a substantial effect on lowering their stroke risk. Although the USDA level for the general population is set at 2300 mg/day, a lower level of ≤1500 mg/day has been recommended for certain population groups including those age >51 years, all blacks, and all patients who have hypertension, chronic kidney disease, or diabetes. Our study supports the importance of reducing sodium consumption to this level for most Americans. The association between sodium consumption and stroke risk was independent of behavioral and vascular risk factors, including hypertension, at baseline, and was observed among those with and without hypertension and across age groups, suggesting that lowering sodium consumption can have beneficial effects on stroke risk for all.

Although we controlled for hypertension at baseline, BP may still be on a causal pathway underlying the association between sodium consumption and stroke risk, as sodium consumption may influence changes in BP during follow-up. Excess sodium intake is directly related to elevated BP, and dose-response trials have shown that the BP response to sodium reduction is progressive and nonlinear[1]. Likewise, elevated BP is an established risk factor for cardiovascular disease and stroke, and primary and secondary prevention strategies support BP reduction to decrease vascular events[15–19].

Sodium consumption was not associated with systolic or diastolic BP or defined hypertension in our study. In fact, anti-hypertensive use was associated with lower sodium consumption. The lack of association between sodium consumption and BP was likely due to the cross-sectional nature of the analysis, as BP and diet were both assessed at baseline. Participants with hypertension, particularly those taking anti-hypertensive medication, may have been advised by their physicians to limit sodium consumption. In addition, the majority of our cohort (73%) had hypertension at baseline, which could have inhibited our ability to detect an association with sodium intake.

The biological mechanisms by which sodium might influence stroke risk independent of BP are speculative. Adverse health effects of heavy sodium consumption, independent of BP, include increased oxidative stress, impaired renal function, left ventricular hypertrophy, arterial fibrosis, increased large elastic artery stiffness, vascular endothelial dysfunction, and vascular remodeling, all of which are associated with vascular disease risk[1].

Strengths of our study include its population-based prospective design, multi-ethnic population, high follow-up, validated outcomes, and comprehensive collection of vascular and other behavioral/lifestyle risk factors. However, despite the use of a well-established valid and reliable food frequency questionnaire[12, 20, 21] to calculate sodium consumption, a potential for both random misclassification and recall bias persists. We lacked independent verification of dietary sodium intake using an objective measurement such as urinary sodium excretion. The prospective design suggests that most misclassification would likely be random. We tried to limit the effect of inaccurate recall of diet by excluding participants with improbably low or high total daily kilocalories (<500 or >4000) or sodium consumption >10000 mg. However, possible under-reporting of total diet is suggested by the low mean caloric consumption particularly in the ≤1500 mg sodium category. In addition, the calculation of sodium consumption using the food frequency questionnaire was not able to fully capture the contribution of salt added to foods at the table. Sodium consumption was based on self-reported food consumption at a single time point, but participants were asked

to indicate their average food consumption over the last year. Dietary patterns may change over time, and possibly during follow-up. As our study population was all over the age of 40 at baseline food frequency assessment, we were not able to examine the effect of sodium consumption prior to enrollment at earlier stages in life. We lacked verification of sodium intake using an objective measurement such as urinary sodium excretion.

Our study provides evidence for a strong relationship between excess sodium intake and increased stroke risk in a multi-ethnic population. Our findings contribute to a body of literature indicating the high sodium intake in the US has negative health consequences. The new AHA strategic dietary goals for 2020, which include sodium reduction to ≤1500 mg/day, will help promote ideal cardiovascular and brain health. Our findings underscore the need for public health initiatives to reduce the sodium level in the food supply.

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Table 1

Demographics and vascular risk factors and sodium consumption

		Sodium Consumption (mg/day)		
	Full cohort (N=2657)	**≤1500 (N=320)**	**1501–3999 (N=1779)**	**≥4000 (N=558)**
Categorical factors, N (%)				
Male sex *	965 (36)	68 (21)	622 (35)	275 (49)
Race/ethnicity *				
White	552 (21)	49 (15)	397 (22)	106 (19)
Black	637 (24)	104 (33)	418 (24)	115 (21)
Hispanic	1407 (53)	160 (50)	925 (52)	322 (58)
Other	61 (2)	7 (2)	39 (2)	15 (3)
High school completion	1124 (46)	140 (44)	836 (47)	248 (44)
Smoking *				
Current	452 (17)	54 (17)	298 (17)	100 (18)
Former	946 (36)	92 (29)	625 (35)	229 (41)
Never	1259 (47)	174 (54)	856 (48)	229 (41)
Moderate-heavy physical activity	232 (9)	18 (6)	163 (9)	51 (9)
Moderate alcohol use *	895 (34)	84 (26)	610 (34)	201 (36)
Previous cardiac disease	482 (18)	58 (18)	310 (17)	114 (20)
Hypertension	1928 (73)	244 (76)	1276 (72)	408 (73)
Anti-hypertensive use *	1139 (43)	161 (50)	751 (42)	227 (41)
Diabetes	552 (21)	64 (20)	364 (20)	124 (22)
Hypercholesterolemia	1501 (56)	193 (60)	1008 (57)	300 (54)
Continuous factors, mean (SD)				
Age *	69 (10)	70 (10)	69 (10)	68 (9)
Total kilocalories/day *	1561 (648)	814 (238)	1429 (401)	2413 (594)
Total fat g/day *	61 (31)	31 (12)	54 (20)	99 (32)
Saturated fat g/day *	20 (12)	9 (4)	18 (8)	34 (13)
Protein g/day *	62 (28)	30 (11)	57 (18)	96 (28)
Carbohydrates g/day *	187 (80)	100 (37)	175 (57)	277 (79)
Systolic blood pressure	143 (21)	144 (20)	143 (21)	144 (21)
Diastolic blood pressure	83 (11)	83 (11)	83 (11)	84 (11)
Low-density lipoprotein	128 (36)	131 (35)	129 (36)	126 (36)
High-density lipoprotein *	47 (15)	49 (16)	47 (15)	45 (14)
Body mass index *	28 (6)	28 (5)	28 (5)	29 (6)

* $p<0.05$ across categories of sodium consumption (chi-square test for categorical variables, ANOVA for continuous variables)

Table 2

Sodium intake in relation to stroke risk

			HR (95% CI) for stroke		
Daily dietary sodium (mg)	**Person-years**	**Events**	**Model 1***	**Model 2†**	**Model 3‡**
500 mg/day increase	27048	235	1.08 (1.04–1.13)	1.17 (1.07–1.27)	1.17 (1.07–1.27)
≤1500	3408	24	1.0 (ref)	1.0 (ref)	1.0 (ref)
1501–2300	6620	56	1.24 (0.77–2.01)	1.33 (0.81–2.18)	1.38 (0.84–2.27)
2301–3999	11752	89	1.15 (0.73–1.81)	1.31 (0.78–2.22)	1.32 (0.78–2.23)
4000–10000	5262	66	1.99 (1.24–3.20)	2.50 (1.23–5.07)	2.59 (1.27–5.28)

*Adjusted for demographics (age, sex, race/ethnicity, education)

†Adjusted for demographics + behavioral risk factors (alcohol use, smoking, physical activity, total calories, total fat, saturated fat, carbohydrates, protein)

‡Adjusted for demographics + behavioral risk factors + vascular risk factors (diabetes, hypercholesterolemia, hypertension, previous cardiac disease, body mass index)

Table 3

Sodium in relation to risk of combined vascular events, and of MI and Vascular death separately

Daily dietary sodium (mg)		HR (95% CI)								
		Stroke, MI, or vascular death			MI			Vascular death		
	Person-years	Events	Model 2*	Model 3†	Events	Model 2*	Model 3†	Events	Model 2*	Model 3†
500 mg/day increase	26278	615	1.06 (1.00–1.12)	1.05 (0.99–1.11)	209	0.95 (0.86–1.04)	0.94 (0.85–1.04)	371	1.02 (0.95–1.10)	1.02 (0.95–1.10)
≤1500	3306	67	1.0 (ref)	1.0 (ref)	29	1.0 (ref)	1.0 (ref)	41	1.0 (ref)	1.0 (ref)
1501–2300	6432	157	1.32 (0.98–1.78)	1.35 (1.00–1.82)	53	0.88 (0.55–1.43)	0.93 (0.58–1.51)	95	1.39 (0.95–2.04)	1.43 (0.97–2.11)
2301–3999	11447	253	1.21 (0.87–1.67)	1.21 (0.87–1.67)	80	0.66 (0.39–1.11)	0.68 (0.40–1.15)	164	1.37 (0.91–2.07)	1.37 (0.90–2.07)
4000–10000	5095	138	1.70 (1.08–2.68)	1.68 (1.06–2.67)	47	0.79 (0.37–1.69)	0.78 (0.36–1.70)	71	1.49 (0.82–2.72)	1.49 (0.81–2.72)

*Adjusted for demographics + behavioral risk factors

†Adjusted for demographics + behavioral risk factors + vascular risk factors