Dietary Sodium and Risk of Stroke in the Northern Manhattan Study

Hannah Gardener, ScD; Tatjana Rundek, MD; Clinton B. Wright, MD; Mitchell S.V. Elkind, MD; Ralph L. Sacco, MD

Background and Purpose—The American Heart Association recommends limiting sodium intake to \(\leq 1500\) 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: \(\leq 1500\) mg/day (12%), 1501 to 2300 mg/day (24%), 2301 to 3999 mg/day (43%), and \(\geq 4000\) 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; interquartile range, 1966–3815 mg/day). Participants who consumed \(\geq 4000\) mg/day sodium had an increased risk of stroke (hazard ratio, 2.59; 95% CI, 1.27–5.28) versus those who consumed \(\leq 1500\) 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 American Heart Association dietary sodium goals will help reduce stroke risk. (Stroke. 2012;43:1200-1205.)

Key Words: diet ■ epidemiology ■ sodium ■ stroke

See related article, pages 1195–1196

The American Heart Association (AHA) lowered its recommended level of sodium consumption to \(\leq 1500\) 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 3-fold 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 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.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 cardiovascular disease 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 cardiovascular disease 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
multietnic urban population. Study details have been published previously. Eligible participants were: (1) stroke-free; (2) >40 years old; and (3) 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 and 2001. The enrollment response rate was 75%, and 3298 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 Institutional Review Boards 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 on self-identification using questions modeled after the US census and conforming to standard definitions outlined by Directive 15. Standardized questions were adapted from the Behavioral Risk Factor Surveillance System by the Centers for Disease Control and Prevention regarding hypertension, diabetes, smoking, and cardiovascular conditions. Measurement of blood pressure (BP) and fasting blood specimens for glucose and lipids and the definitions of hypercholesterolemia, diabetes, moderate to heavy physical activity, and moderate alcohol use were described previously. Hypertension was defined as BP ≥140/90 mm Hg, anti-hypertensive 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. 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 to 2300 mg/day (consistent with US Department of Agriculture recommendation of ≤2300 mg for those at standard risk), 2301 to 3999 mg/day, and 4000 to 10 000 mg/day (approximately the top quintile).

Outcomes
The primary outcome was confirmed incident stroke of all subtypes (infarct, 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. Subjects were screened annually by telephone to determine changes in vital status, detect neurological 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 International Classification of Diseases, 9th Revision 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 bimonthly 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, and subarachnoid hemorrhage. Stroke was defined based on World Health Organization 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 intracerebral hemorrhage, subarachnoid hemorrhage, 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: (1) ischemic cardiac pain defined to be typical angina; (2) cardiac enzyme abnormalities defined as abnormal creatine–phosphokinase MB isoenzyme fraction or troponin values; and (3) electrocardiographic 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 nonvascular and based on information obtained from the family, medical records, and death certificates. Vascular death included death due to stroke, MI, heart failure, pulmonary embolus, cardiac arrhythmia, or other vascular cause. These are International Classification of Diseases, 9th Revision codes 390 to 459.

Statistical Analysis
We examined the unadjusted associations of categories of sodium consumption with sociodemographics and vascular risk factors using analysis of variance and χ² 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, and high school completion; (2) adjusted for demographics and behavioral risk factors: smoking (never, former, current); moderate to heavy physical activity, moderate alcohol consumption, daily consumption of total kcal, protein, total fat, saturated fat, and carbohydrates; and (3) adjusted for demographics, behavioral risk factors, and vascular risk factors: diabetes, hypercholesterolemia, hypertension, previous cardiovascular disease, and body mass index. 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 antihypertensive medications) by including interaction terms between sodium consumption and these variables in Model 3.

Results
This study included 2657 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 10 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; interquartile range, 1966–3815). Only 12% consumed the AHA-recommended level of ≤1500 mg/day sodium, whereas 24% consumed 1501 to 2300, 43% 2301 to 3999, and 21% 4000 to 10 000 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 associ-
ated with older age, female sex, black race, never smoking, and antihypertensive use, whereas higher sodium consumption was associated with Hispanic ethnicity, moderate alcohol use, increased body mass index, and consumption of total kilocalories, protein, carbohydrates, total fat, and 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 \(\geq 4000\) mg/day had a 2.6-fold increase in stroke risk versus those who consumed \(\leq 1500\) mg/day (Model 3; 95% CI, 1.27–5.28). Intake of sodium \(> 1500\) but \(< 4000\) mg/day had a hazard ratio 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 versus white, \(P = 0.73\) Hispanic versus 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 \(\geq 4000\) versus \(\leq 1500\) mg/day of sodium.

Table 3 shows the relationship between sodium consumption and combined vascular events. Consumption of \(\geq 4000\) mg/day was associated with higher risk of vascular events, whereas lower sodium intake was associated with lower risk. A 17% increase in risk was observed for each 500-mg/day increase in sodium consumption (Model 3; 95% CI, 1.07–1.27). The hazard ratio for \(\geq 4000\) mg/day sodium was 2.6-fold higher than for \(\leq 1500\) mg/day (Model 3; 95% CI, 1.27–5.28). There was no significant interaction between sodium and age, sex, race/ethnicity, hypertension status, or BP at baseline in relation to vascular events.
mg/day sodium was associated with an elevated risk of combined vascular events versus ≤1500 mg/day. Intake of 1501 to 2300 mg/day was associated with an increased risk of stroke, MI, or vascular death compared with ≤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 versus white, \( P = 0.18 \) Hispanic versus 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 multiethnic cohort with only 12% meeting the AHA-recommended level of ≤1500 mg/day, only 36% meeting the US Department of Agriculture-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 2 daily sodium consumption categories between 1501 to 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 to 2300 mg/day compared with ≤1500 mg/day. Although we found a 17% relative increase in the hazard of stroke for every 500-mg/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, 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, whereas others used 24-hour dietary recall or urinary sodium excretion analysis. The strength of the association with stroke risk was often different for men versus 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 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 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.

Strengths of our study include its population-based prospective design, multiethnic 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 questionnaire12,20,21 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 >10 000 mg. However, possible underreporting 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. Because our study population was all aged >40 years at baseline food frequency assessment, we were not able to examine the effect of sodium consumption before enrollment at earlier stages in life.

Our study provides evidence for a strong relationship between excess sodium intake and increased stroke risk in a multiethnic population. Our findings contribute to a body of literature indicating the high sodium intake in the United States 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.

Sources of Funding

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Disclosures

None.
References


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Northern Manhattan Studyにおける食事中のナトリウムと脳卒中のリスク

Dietary Sodium and Risk of Stroke in the Northern Manhattan Study

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Abstract

背景および目的: 米国心臓協会は、心血管を理想的な健康状態に保つためにはナトリウム摂取量を≦ 1,500 mg/日に制限することを推奨している。ナトリウム摂取は、高血圧との直接的な関係によって血管疾患と関連づけて考えられてきたが、脳卒中のリスクとの関連を裏付ける試験はほとんど存在しない。

方法: 被験者は、脳卒中の発生率に関する地域住民を対象としたコホート研究、Northern Manhattan Study（平均年齢69±10歳、女性64%、白人21%、ヒスパニック53%、黒人24%）から組み入れた。ナトリウム摂取量はベースライン時に食物摂取頻度調査票を用いて評価し、連続的およびカテゴリー別に評価した：≦ 1,500 mg/kg/日（12%）、1,501~2,300 mg/日（24%）、2,301~3,999 mg/日（43%）および≧ 4,000 mg/日（21%）。平均10年間の追跡期間にわたり、ナトリウム摂取量と235件の脳卒中の関係を、社会人口統計学的要素、食事、行動/ライフスタイルおよび血管危険因子について補正し、Coxモデルを用いて検討した。

結果: 食事のデータが得られた2,657例の被験者では、平均のナトリウム摂取量が3,031±1,470 mg/日（中央値：2,787 mg/日、四分位範囲：1,966~3,815 mg/日）であった。≧ 4,000 mg/日のナトリウム摂取をしていた被験者は、摂取量が≦ 1,500 mg/日の患者に比べて脳卒中のリスクが高く（ハザード比=2.59, 95% CI:1.27 ~ 5.28）、脳卒中の発現リスクは摂取量が500 mg/日増加するごとに17%上昇した（95% CI:1.07 ~ 1.27）。

結論: ナトリウムの多量摂取は血管危険因子とは無関係に脳卒中のリスク上昇に関連していた。米国心臓協会の食事中ナトリウムの新しい目標は、脳卒中のリスク軽減に役立つであろう。

表2 ナトリウム摂取量と脳卒中リスクの関係

<table>
<thead>
<tr>
<th>1日あたりの食事中のナトリウム量, mg</th>
<th>人・年</th>
<th>イベント</th>
<th>モデル1*</th>
<th>モデル2†</th>
<th>モデル3‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>500 mg/日（増加）</td>
<td>27,048</td>
<td>235</td>
<td>1.08 (1.04 ~ 1.13)</td>
<td>1.17 (1.07 ~ 1.27)</td>
<td>1.17 (1.07 ~ 1.27)</td>
</tr>
<tr>
<td>≦ 1,500</td>
<td>3,408</td>
<td>24</td>
<td>1.0 (参照基準)</td>
<td>1.0 (参照基準)</td>
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</tr>
<tr>
<td>1,501~2,300</td>
<td>6,620</td>
<td>56</td>
<td>1.24 (0.77 ~ 2.01)</td>
<td>1.33 (0.81 ~ 2.18)</td>
<td>1.38 (0.84 ~ 2.27)</td>
</tr>
<tr>
<td>2,301~3,999</td>
<td>11,752</td>
<td>89</td>
<td>1.15 (0.73 ~ 1.81)</td>
<td>1.31 (0.78 ~ 2.22)</td>
<td>1.32 (0.78 ~ 2.23)</td>
</tr>
<tr>
<td>4,000~10,000</td>
<td>5,262</td>
<td>66</td>
<td>1.99 (1.24 ~ 3.20)</td>
<td>2.50 (1.23 ~ 5.07)</td>
<td>2.59 (1.27 ~ 5.28)</td>
</tr>
</tbody>
</table>

*人口統計学的要素（年齢、性別、人種/民族、教育）について補正。
†人口統計学的要素+行動に関する危険因子（飲酒、喫煙、運動量、総摂取カロリー、総脂質、飽和脂肪、炭水化物、蛋白）について補正。
‡人口統計学的要素+行動に関する危険因子+血管危険因子（糖尿病、高コレステロール血症、高血圧、心臓疾患の既往、肥満指数）について補正。

表3 ナトリウムと複合血管イベントリスクとの関係ならびに心筋梗塞および血管死それぞれとの関係

<table>
<thead>
<tr>
<th>1日あたりの食事中のナトリウム量, mg</th>
<th>人・年</th>
<th>イベント</th>
<th>モデル1*</th>
<th>モデル2†</th>
<th>モデル3‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>500 mg/日（増加）</td>
<td>26,278</td>
<td>615</td>
<td>0.95 (0.86 ~ 1.04)</td>
<td>0.94 (0.85 ~ 1.04)</td>
<td>0.94 (0.85 ~ 1.04)</td>
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<tr>
<td>≦ 1,500</td>
<td>3,306</td>
<td>67</td>
<td>1.0 (参照基準)</td>
<td>1.0 (参照基準)</td>
<td>1.0 (参照基準)</td>
</tr>
<tr>
<td>1,501~2,300</td>
<td>6,432</td>
<td>157</td>
<td>1.43 (1.08 ~ 1.87)</td>
<td>1.35 (1.00 ~ 1.82)</td>
<td>1.35 (1.00 ~ 1.82)</td>
</tr>
<tr>
<td>2,301~3,999</td>
<td>11,447</td>
<td>253</td>
<td>1.09 (0.87 ~ 1.37)</td>
<td>1.07 (0.86 ~ 1.32)</td>
<td>1.07 (0.86 ~ 1.32)</td>
</tr>
<tr>
<td>4,000~10,000</td>
<td>5,095</td>
<td>138</td>
<td>1.82 (1.08 ~ 2.98)</td>
<td>1.68 (1.05 ~ 2.67)</td>
<td>1.68 (1.05 ~ 2.67)</td>
</tr>
</tbody>
</table>

*人口統計学的要素+行動に関する危険因子（飲酒、喫煙、運動量、総摂取カロリー、総脂質、飽和脂肪、炭水化物、蛋白）について補正。
†人口統計学的要素+行動に関する危険因子+血管危険因子（糖尿病、高コレステロール血症、高血圧、心臓疾患の既往、肥満指数）について補正。
소금 섭취량이 뇌졸중 위험에 미치는 영향: 북부 멘하탄 연구

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Key Words: diet ■ epidemiology ■ sodium ■ stroke

대표과 목적
미국심장협회에서 이례적인 심혈관계 질환 예방을 위해 하루 소금 섭취량만 1,500 mg 이하로 제한하는 것을 권고하였다. 소금 섭취량은 고혈압과의 직접적 관련을 통해 혈관질환의 발생과 관련되어 있으나, 소금 섭취량과 뇌졸중 위험도에 대해서는 거의 알려진 바가 없다.

방법
뇌졸중 발생 코호트인 북부 멘하탄 연구에서 참여자(평균 연령 69±10, 64% 여성, 21% 백인, 53% 히스패닉, 24% 혼인)를 얻었다. 소금 섭취량은 연구 참여 시 식품영양조사 설문지를 이용하여 조사하였으며 연속형 및 범주형 변수(≤1,500 mg/일 [12%], 1,501~2,300 mg/일 [24%], 2,301~3,999 mg/일 [43%], 그리고 ≥4,000 mg/일 [21%])로 나누어 분석하였다. 평균 10년 동안 추적관찰 기간 동안 소금 섭취량과 235건의 뇌졸중 발생에 대해서, 사회경제적 요인, 식이, 생활패턴 및 심혈관계 위험 인자 등을 보정한 로지스틱 모델을 이용하여 분석하였다.

결과
2,657명의 식이자료를 분석하였고, 평균 소금 섭취량은 3,031 ±1,470 mg/일(중앙값, 2,787: 사분위수, 1,966~3,815 mg/일)이었으며, 하루 4,000 mg 이상을 섭취하는 사람들은 1,500 mg 이하를 섭취하는 사람들과 비교했을 때, 뇌졸중 위험도가 높았다(HR, 2.59: 95% CI, 1.27~5.28). 또한 하루 소금 섭취량이 500 mg씩 증가할 때마다 뇌졸중 위험도는 약 17% 증가하였다(95% CI, 1.07~1.27).

결론
소금 섭취량이 높은 경우가 일반적이었으며, 높은 소금 섭취량은 심혈관계 위험인자와 무관하게 뇌졸중 위험도 증가와 관련이 있었다. 미국심장협회에서 제시한 소금 목표 섭취량은 뇌졸중 위험도를 낮추는데 기여할 것이다.

Table 2. Sodium Intake in Relation to Stroke Risk

<table>
<thead>
<tr>
<th>Daily Dietary Sodium, mg</th>
<th>Person-Years</th>
<th>Events</th>
<th>Model 1*</th>
<th>Model 2†</th>
<th>Model 3‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>500 mg/d increase</td>
<td>27,048</td>
<td>235</td>
<td>1.08 (1.04—1.13)</td>
<td>1.17 (1.07—1.27)</td>
<td>1.17 (1.07—1.27)</td>
</tr>
<tr>
<td>≤1500</td>
<td>3408</td>
<td>24</td>
<td>1.0 (referent)</td>
<td>1.0 (referent)</td>
<td>1.0 (referent)</td>
</tr>
<tr>
<td>1501—2300</td>
<td>6620</td>
<td>56</td>
<td>1.24 (0.77—2.01)</td>
<td>1.33 (0.81—2.18)</td>
<td>1.38 (0.84—2.27)</td>
</tr>
<tr>
<td>2301—3999</td>
<td>11,752</td>
<td>89</td>
<td>1.15 (0.73—1.81)</td>
<td>1.31 (0.78—2.22)</td>
<td>1.32 (0.78—2.23)</td>
</tr>
<tr>
<td>4000—10,000</td>
<td>5,262</td>
<td>66</td>
<td>1.99 (1.24—3.20)</td>
<td>2.50 (1.23—5.07)</td>
<td>2.59 (1.27—5.28)</td>
</tr>
</tbody>
</table>

*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

<table>
<thead>
<tr>
<th>Daily Dietary Sodium, mg</th>
<th>Stroke, MI, or Vascular Death</th>
<th>MI</th>
<th>Vascular Death</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Person-Years</td>
<td>Events</td>
<td>Model 2*</td>
</tr>
<tr>
<td>500-mg/d increase</td>
<td>26,278</td>
<td>615</td>
<td>1.06 (1.00—1.13)</td>
</tr>
<tr>
<td>≤1500</td>
<td>3306</td>
<td>67</td>
<td>1.0 (referent)</td>
</tr>
<tr>
<td>1501—2300</td>
<td>6432</td>
<td>157</td>
<td>1.32 (0.98—1.78)</td>
</tr>
<tr>
<td>2301—3999</td>
<td>11,447</td>
<td>253</td>
<td>1.21 (0.87—1.67)</td>
</tr>
<tr>
<td>4000—10,000</td>
<td>5,095</td>
<td>138</td>
<td>1.70 (1.08—2.68)</td>
</tr>
</tbody>
</table>

MI indicates myocardial infarction.
*Adjusted for demographics + behavioral risk factors.
†Adjusted for demographics + behavioral risk factors + vascular risk factors.