Influence of sodium consumption and associated knowledge on poststroke hypertension in Uganda

Martin N. Kaddumukasa,
MD
Elly Katabira, MD, FRCP
Martha Sajatovic, MD,
PhD
Svetlana Pundik, MD,
MS
Mark Kaddumukasa, MD
Larry B. Goldstein, MD

Correspondence to
Dr. Martin N. Kaddumukasa:
kaddumart@yahoo.com

ABSTRACT

Objective: We assessed 24-hour urine sodium levels as an index of dietary salt consumption and its association with dietary salt knowledge and hypertension among poststroke patients with and without a history of hypertension in Uganda.

Methods: A case-control study in which poststroke patients with a history of hypertension (cases, $n = 123$) were compared to poststroke patients without known hypertension (controls, $n = 112$). Dietary salt intake was assessed by 24-hour urine sodium, a valid measure of dietary salt consumption. Dietary salt knowledge was determined by questionnaire. The independent relationships among salt knowledge, 24-hour urine sodium, and blood pressure control were assessed using multiple regression analysis.

Results: High 24-hour urine sodium (≥ 8.5 g/d) was 2 times more prevalent among hypertensive poststroke patients than controls ($p = 0.002$). Patients with minimal poststroke disability who had a choice in determining their diets had higher urine sodium than their more disabled counterparts. Only 43% of the study population had basic dietary salt knowledge, 39% had adequate diet-disease-related knowledge, and 37% had procedural knowledge (report of specific steps being taken to reduce salt consumption). Dietary salt knowledge was similarly poor among cases and controls ($p = 0.488$) and was not related to education level ($p = 0.205$).

Conclusions: High urine sodium and high salt-diet preferences were more frequent among poststroke hypertensive patients in Uganda than in their nonhypertensive counterparts. There was, however, no difference in dietary salt knowledge between these groups. The development of educational strategies that include salt-diet preferences may lead to better blood pressure control in this high-risk population. *Neurology*® 2016;87:1198-1205

GLOSSARY

BP = blood pressure; WHO = World Health Organization.

Stroke is a major health problem in sub-Saharan Africa with both diagnosis and treatment remaining poor.^{1,2} Although stroke remains the second most common cause of death and a major cause of adult disability worldwide,³ in part attributable to the high burden of infectious diseases in the area, little attention has been given to stroke prevention and treatment in sub-Saharan Africa. Hypertension is the most prevalent modifiable risk factor for stroke, accounting for 62% of all strokes.^{1,2} In Uganda, more than 65% of poststroke patients have uncontrolled hypertension.⁴

A healthy diet is a major component of the preventative strategy for reducing the risk of stroke and cardiovascular diseases.⁵ High salt intake is a major contributor to hypertension, with a population-attributable risk of 9% to 17%.^{6,7} Globally, the frequency of hypertension and associated cardiovascular events is associated with the level of dietary salt intake.⁷ The World Health Organization (WHO) recommends consuming <86 mmol of sodium daily.^{8,9} Twenty-four-hour urinary sodium is a valid measure for assessing dietary sodium intake.^{10,11}

Editorial, page 1192

From the Department of Internal Medicine (M.N.K., E.K., M.K.), Makerere University, College of Health Sciences, Kampala, Uganda; Neurological and Behavioral Outcomes Center (M.S.), University Hospitals Case Medical Center, Cleveland; School of Medicine (S.P.), Cleveland VA Medical Center and Case Western Reserve University, Cleveland, OH; and Department of Neurology (L.B.G.), University of Kentucky, Lexington.

Go to Neurology.org for full disclosures. Funding information and disclosures deemed relevant by the authors, if any, are provided at the end of the article.

Sociocultural factors, in addition to age, education, and average income, are main determinants of dietary behavior.^{11,12} In Uganda, knowledge of the effect of diet on stroke risk is generally poor.⁴ We assessed 24-hour urine sodium levels, associated dietary salt knowledge, and their relationship with uncontrolled hypertension in poststroke patients in Uganda. The primary hypothesis was that the level of dietary salt intake as reflected by 24-hour urine sodium levels in patients who had a stroke would be associated with their dietary salt knowledge and level of blood pressure (BP) control.

METHODS Participant recruitment. We conducted this case-control study between January 2014 and August 2015 at Uganda's Mulago National Referral Hospital. The hospital's neurology clinic serves as a tertiary referral center for neurology-related disorders for the entire country. Participants were enrolled by a trained research nurse in a consecutive convenience sample. Stroke was defined as a neurologic deficit of abrupt onset attributable to a vascular cause with neurologic deficits lasting more than 24 hours and with compatible findings on brain CT scan.⁴ Cases were identified from neurology data files and defined as adults older than 18 years with a history of hypertension who had a confirmed stroke at least 1 month previously. Controls were adults with confirmed stroke at least 1 month previously, but with no known history of hypertension. Hypertensive patients were defined as those with an initial BP $\geq 180/105$ mm Hg or those having BP $\geq 140/90$ mm Hg over 3 consecutive previous visits as reflected in their medical record and receiving antihypertensive medication.^{13,14} Patients who were pregnant, had chronic kidney disease as reflected in their medical record

or proteinuria ≥ 150 mg/d,¹⁵ taking loop diuretics, had dementia as confirmed by a Mini-Mental State Examination score < 24 ,¹⁶ and those who declined consent were excluded. The contribution of other diuretics was considered too small to importantly affect the 24-hour urine sodium values. Figure 1 shows the CONSORT (Consolidated Standards of Reporting Trials) patient flow.

Clinical assessments. All participants had a general physical examination. BP was measured with a standard validated oscillometric "digital" technique¹³ with an Omron M4-I Intellisense device (Omron Healthcare, Hoofddorp, the Netherlands) twice after 15-minute intervals with the participant seated and the average recorded. Patients' functional ability in activities of daily living was assessed using the modified Rankin Scale.¹⁷ Scores were categorized as 0 or 1 (minor or no disability), 2 or 3 (moderate disability) and 4 or 5 (severe disability). The modified Rankin Scale scores 4 and 5 were a proxy for caregivers' dietary choices rather than that of the patient.

24-hour urine sodium measurement. We measured 24-hour urine sodium using an ion selective electrode Roche Diagnostics machine (COBAS 6000; Roche Diagnostics Int., Rotkreuz, Switzerland). A complete urine sample was defined as (1) a total 24-hour urine volume > 500 mL, (2) no menstruation during the collection period, and (3) a reported length of collection > 20 hours.¹⁸ Paraaminobenzoic acid was used to confirm completeness of the 24-hour urine sample. High dietary salt intake was defined as having a 24-hour urine sodium level ≥ 8.5 g/d.^{19,20}

Data abstraction. A trained research assistant abstracted information on stroke, hypertension, prescribed antihypertensive medications, dietary salt consumption, salt and associated BP control knowledge, and other cardiovascular disease risk factors from medical records and patient interviews utilizing a pretested, standardized questionnaire. Data were checked for completeness before being precoded using Epi-Data software.

Figure 1 Flow diagram for identification of cases and controls

*Thirty patients who failed to return a 24-hour urine sample. **Thirty-five patients whose 24-hour urine sample failed the paraaminobenzoic acid completeness test.

Statistical analysis. A target sample size of 240 with a 25% dropout rate was used to obtain 80% power ($\alpha = 0.05$) for detecting a sodium-BP effect size of at least 10%. Of the targeted 240 participants, 235 had complete urine data and formed the analyzed sample. There were no significant differences between the analytic sample and the group with incomplete data ($n = 5$). Outcome variables were 24-hour urine sodium, BP, and salt knowledge. Missing BP and knowledge data comprised 6.3% and 11%, respectively, of the study population. There was no imputation for missing data. Using SPSS version 22 (IBM Corp., Armonk, NY), logistic regression and χ^2 analysis were used to identify factors independently associated with high urinary sodium levels, hypertension, and dietary salt knowledge. We adjusted for other factors that could affect BP control such as age, sex, diabetes, antihypertensive medication and associated adherence, alcohol use, and smoking. All variables significant in the univariate analysis at $p < 0.05$ were included in a multiple linear regression analysis to identify those independently associated with uncontrolled hypertension. Dietary salt knowledge was described in 3 ways: (1) basic knowledge (quantified by patient awareness of the harm high salt has on general health); (2) diet-disease-related knowledge

(quantified by patients' awareness of the actual health conditions related to high salt); and (3) procedural knowledge (quantified by patient report on deliberate actions taken to reduce their salt intake). Correct responses were scored as "1" with incorrect responses including "don't know" or "not sure" scored "0."

Standard protocol approvals, registrations, and patient consents. The study was approved by the Makerere University, School of Medicine, Research and Ethics Committee, and the Uganda National Council for Science and Technology. All enrolled participants or their legal representative provided written consent.

RESULTS There were 123 cases and 112 control participants enrolled, aged between 24 and 100 years, of whom 118 (50.2%) were men and 117 (49.8%) were women. The average age of participants was 61.3 ± 15.2 years with 47.7% older than 60 years.

Table 1 shows the demographic and clinical characteristics of the study population. Cases and controls

Table 1 Demographic and baseline characteristics of the cases and controls

Characteristics	Cases (n = 123; 52.3%)	Controls (n = 112; 47.7%)	OR (95% CI); p Value
Sex			
Men	60 (48.8)	58 (51.8)	p = 0.645
Women	63 (51.2)	54 (48.2)	
Education			
Low (none/primary)	70 (56.9)	60 (53.6)	p = 0.607
Marital status			
Single/divorced/widowed	60 (48.8)	51 (45.5)	p = 0.619
Mean age ± SD, y	61.4 ± 14.4	60.8 ± 15.9	p = 0.750
Mean systolic BP ± SD, mm Hg	179.5 ± 27.0	132.6 ± 19.9	p < 0.0001
Systolic BP, IQR, mm Hg	160.0-200.0	120.0-141.0	
Mean diastolic BP ± SD, mm Hg	106.8 ± 22.2	81.2 ± 12.9	p < 0.0001
Diastolic BP, IQR, mm Hg	94.0-117.0	72.5-89.5	
Mean 24-h urine sodium ± SD, g/d	6.5 ± 2.7	5.4 ± 2.6	p = 0.001
24-h urine sodium, IQR, g/d	4.2-8.6	2.9-7.5	
Hypertensive BP at time of clinic visit	123 (100)	27 (24.1)	5.56 (3.95, 7.82); p < 0.0001
High 24-h urine sodium, ≥8.5 g/d	80 (65.0)	50 (44.6)	2.31 (1.36, 3.90); p = 0.002
Urine sodium >5 g/d, WHO minimum	88 (71.5)	64 (57.1)	1.89 (1.10, 3.23); p = 0.021
Basic salt knowledge	51 (41.5)	48 (42.9)	0.94 (0.56, 1.59); p = 0.829
Stroke type			
Hemorrhagic	31 (25.2)	16 (14.3)	Ref
Ischemic	92 (74.8)	96 (85.7)	0.50 (0.25, 0.96); p = 0.037
CVD risk factors			
Diabetes	17 (18.3)	13 (17.8)	1.03 (0.47, 2.29); p = 0.938
Smoking	10 (9.9)	16 (19.8)	0.45 (0.19, 1.05); p = 0.059
Alcohol	39 (31.7)	44 (39.3)	0.72 (0.42, 1.23); p = 0.225

Abbreviations: BP = blood pressure; CI = confidence interval; CVD = cardiovascular disease; IQR = interquartile range; OR = odds ratio; Ref = reference; WHO = World Health Organization. Data represent n (%) unless otherwise indicated.

Figure 2 Blood pressure and urine sodium against patient disability scores

were of similar age and had a similar sex distribution. The majority of the study population (55.3%) had little or no formal education. Mean systolic and diastolic BPs for cases were higher than for controls at the time of the study visit ($p < 0.0001$); 80% of participants had an ischemic stroke. Urinary sodium was higher in ischemic stroke patients compared with hemorrhagic stroke patients for both cases and controls (i.e., >8.5 g/d; odds ratio = 1.96, 95% CI 1.00–3.85, $p = 0.049$). Antihypertensive medications included nifedipine (50.5%), captopril (50.5%), amlodipine (22.1%), atenolol (9.5%), and losartan potassium (8.4%).

Mean 24-hour urine sodium excretion for the study population was 6.0 g/d. Univariate linear regression analysis with systolic BP as the dependent variable revealed significant relationships for urine sodium ($p < 0.0001$), urine potassium ($p = 0.021$), and antihypertensive drug use ($p = 0.002$)

(with $p > 0.05$ for age, sex, diabetes, antihypertensive drug adherence, alcohol use, and smoking). Multiple linear regression analysis of the variables significant in the univariate analyses showed that urine sodium was independently and positively associated with systolic BP ($p = 0.003$). A similar analysis adjusting for antihypertensive drug use showed an independent and positive association between diastolic BP and urine sodium ($p = 0.040$). Systolic and diastolic BP increased by 0.15 mm Hg and 0.07 mm Hg per mmol increase in 24-hour urine sodium, respectively. Mean urinary sodium was higher in men than in women ($p = 0.046$).

Figure 2 gives the mean systolic and diastolic BPs according to 24-hour urine sodium for different levels of stroke-related disability. BP was generally higher among those with little to no disability and decreased with increasing levels of disability. Twenty-four-hour urine sodium similarly decreased: highest among those with minor disability and lowest among poststroke patients with severe disability. Figure 3 shows the relationships among urine sodium, salt consumption habits, and dietary salt knowledge for different levels of poststroke disability. There was a similar trend between reported high salt consumption and high 24-hour urine sodium. Salt consumption was highest in patients with minor or no disability and lowest among patients with severe disability. The trends for both procedural and diet-disease-related knowledge were reversed (highest among patients with high reported salt consumption and measured urine sodium and lowest among low salt consumers).

The majority of cases (62.4%) and controls (50.0%) lacked basic dietary salt knowledge ($p = 0.104$ for cases vs controls). Only 29.0% of cases and 28.2% of controls ($p = 0.920$) had diet-disease-related knowledge. Of these, 29.3% and 5.4% of cases were able to relate high salt consumption with

Figure 3 Urine sodium and salt consumption habits against salt knowledge

hypertension and stroke, respectively. There was a difference in the frequency of cases (33.3%) and controls (50.0%) who recognized the importance of lowering salt in their diet ($p = 0.027$). Only 45.0% of the study population had procedural knowledge (i.e., reported taking deliberate actions to reduce their salt intake). The actions varied, with the majority (60.9%) reporting “not adding salt at the table,” 18.4% reporting minimizing consumption of processed foods, and 13.8% reading sodium levels on food labels. There was no difference in the frequency of cases (42.9%) and controls (47.5%) with procedural knowledge ($p = 0.560$).

An assessment of diet-disease knowledge and procedural knowledge among different stroke patient categories is shown in table 2. Women were twice as likely to have salt-diet disease-related knowledge ($p = 0.019$) than men despite having poorer education (odds ratio = 3.42, 95% CI 1.99–5.86, $p < 0.0001$). Those with high 24-hour urine sodium ≥ 8.5 g/d, not surprisingly, were 50% less likely to have procedural knowledge on salt

reduction ($p = 0.010$) than their counterparts with lower sodium levels.

DISCUSSION This study has 3 primary findings. High levels of urine sodium in the population were reflected in higher rates of poststroke hypertension. Contrary to the primary hypothesis, dietary salt knowledge was not associated with healthy salt consumption habits or controlled BP. In addition, the majority of the study population had very high mean daily sodium excretion levels (i.e., ≥ 8.5 g sodium/d) reflecting high dietary sodium consumption.

Among cases, 24-hour urine sodium was positively correlated with high rates of uncontrolled BP. Adjusting for possible confounders including antihypertensive drug use and associated adherence did not affect the strength of the relationship. Authors of a previous study in this same population with similar poor BP control (53.2% poststrokes, 37.6% nonstroke controls; $p = 0.006$) hypothesized that the high prevalence of poststroke hypertension was due to excessive salt consumption.⁴ Similarly, the INTERSALT Study found a strong association between urinary sodium

Table 2 Assessment of diet-disease knowledge and procedural knowledge among different patient groups

Category	% with procedural knowledge	Crude OR, 95% CI	p Value	% with diet-disease knowledge	Crude OR, 95% CI	p Value
Overall	34.0			28.9		
Controls	34.8	Ref		26.8	Ref	
Cases	33.3	0.93 (0.55, 1.61)	0.810	30.9	1.22 (0.69, 2.15)	0.488
Sex						
Male	32.2	Ref		22.0	Ref	
Female	35.9	1.18 (0.69, 2.02)	0.550	35.9	1.98 (1.11, 3.52)	0.019 ^a
BP at time of visit						
Normotensive	29.4	Ref		28.2	Ref	
Hypertensive	36.7	1.39 (0.78, 2.46)	0.259	29.3	1.06 (0.59, 1.90)	0.858
Education Level						
High	30.5	Ref		24.8	Ref	
Low	36.9	1.34 (0.77, 2.31)	0.300	32.3	1.45 (0.82, 2.58)	0.205
24-h urine sodium						
<8.5 g/d	42.9	Ref		34.3	Ref	
≥ 8.5 g/d	26.9	0.49 (0.28, 0.85)	0.010 ^a	24.6	0.63 (0.36, 1.10)	0.104
Reported salt consumption						
Low	37.7	Ref		31.1	Ref	
High	27.4	0.62 (0.35, 1.11)	0.108	32.1	1.05 (0.59, 1.86)	0.872
Antihypertensive medication						
No	36.0	Ref		32.6	Ref	
Yes	28.6	0.71 (0.38, 1.33)	0.284	28.6	0.83 (0.44, 1.56)	0.560

Abbreviations: BP = blood pressure; CI = confidence interval; OR = odds ratio; Ref = Reference.

^aSignificant.

and BP across 52 populations.^{10,21} In accounting for the source of high salt in the Ugandan diet, we note that the traditional diet is mainly vegetarian with only 11% to 13% from animal origin. Most of the food consists of plantains, roots, or tubers with low sodium. Dietary sodium from processed foods is quite low. Added salt in cooking and/or at the table is the major source of excessive sodium intake in Ugandans.²² This suggests that changing dietary salt consumption habits may improve BP control.

The mean daily sodium excretion levels as assessed by 24-hour urine were high (i.e., ≥ 8.5 g sodium/d) in 65.0% vs 44.6% cases and controls, respectively. A majority (71.5% vs 57.1%) of poststroke cases and controls had urine sodium levels equivalent to a daily salt consumption in excess of the WHO recommendation of ≤ 5 g/d salt.^{23,24} The US Departments of Agriculture and Health and Human Services recommend a lower daily intake of 3.7 g/d of salt for most adults older than 40 years of age.^{25,26} Exceeding established salt consumption targets increases risk of cardiovascular disease, hypertension, and premature death²⁷ as evidenced by the high levels of poststroke hypertension in this population.⁴ Although higher than the mean level of sodium consumption worldwide (3.95 g/d), estimated mean salt intake for Ugandan participants was fairly consistent with data from other African populations (mean values ranging from 2.18 to 5.51 g/d).^{28,29} Cappuccio et al. (2006)²⁹ reported a salt intake of 5.8 g/d in a Ghanaian population aged 40 to 75 (mean 54.7) years. A study conducted in Mauritius estimated an average sodium consumption of 5.45 (95% CI 4.57–6.50) g/d.³⁰ As in Ugandans, the largest source of dietary salt among black persons of African descent is that added in cooking and/or at the table.³¹ The WHO reports a progressive change in Africans' diets to less fruits, vegetables, and dietary fiber, which contain potassium that may help counter the negative effects of salt and contribute to reduced BP.³²

We found no difference in salt knowledge between hypertensive and normotensive poststroke patients ($p = 0.829$), and no difference in knowledge between those with high vs normal urine sodium levels ($p = 0.205$). Salt knowledge was not reflected in the salt consumption preferences of this population ($p = 0.319$). Contrary to findings from similar studies, we found no relationship between salt knowledge and participants' level of education. The level of basic declarative salt knowledge was low (43%). Knowledge of salt-diet-disease relationships was even lower (39%). Procedural knowledge in our study population was only 37.0% with the majority (60.9%) correctly indicating "not adding salt at the table" as their most deliberate step to cut salt intake. This is low compared to Western populations in which

knowledge of diet-disease relationships is as high as 80%.^{33–35} The poor knowledge in our study population is not surprising given the lack of dietary salt education initiatives at the national level in Uganda targeted to reduce hypertension and stroke. Western countries have had nationwide salt reduction interventions including targeted information campaigns, decreases in salt content of processed foods, and sodium labeling changes³³ that likely account for their much higher rates of patient knowledge.

We found that despite the high urinary sodium values found in the majority of our study population (55.3%), an even higher percentage of participants (62.1%) classified their salt consumption as "just right" or "too little." This suggests a misperception of the amount of salt they consume. The gap between the self-perceived and actual quantity of salt consumed is attributed to the inability of individuals to perceive their own dietary salt imbalance,^{33,36,37} leading to an unrecognized high salt intake,^{33,37} as well as a fundamental lack of knowledge of salt consumption targets. In addition, 20% of hypertensive stroke cases and 26.7% of stroke controls expressed no interest in salt reduction, perceiving it as "not important." This highlights an urgent need for countrywide salt reduction interventions including creating more awareness of individuals' level of intake in relation to recommendations.³⁸ Patients relying on caretakers for their food choices had lower salt diets compared to the more independent poststroke patients who had more dietary freedom. Despite having more knowledge, they tended to opt for higher-salt diets. The inverse relationship in our population between salt knowledge and high salt consumption habits suggests that a tailored educational approach might be helpful. Any salt reduction interventions must take these strong patient preferences into account.

A key strength of our study is in the use of the "gold standard" of 24-hour urine collection to estimate sodium intake.^{10,11} This study, however, has some limitations. First, we cannot determine the frequency of "white-coat hypertension" in our population, although we measured BP using standard techniques. This potentially misclassifies nonhypertensive patients as being hypertensive. Second, the study was powered to detect a sodium-BP effect size of 10%, but was not adequately powered to detect the link between patient knowledge and BP control. Third, we assumed a generalized constant habitual salt intake from a 24-hour urinary sodium result; however, this measure is validated and considered appropriate at a population level.³⁹

Despite our study limitations, given that most dietary sodium in Africans is added to home-prepared foods, the data suggest that the development of strategies that take salt-diet preferences into

account and target behavioral change aimed at reducing dietary sodium may lead to better BP control in this population.

AUTHOR CONTRIBUTIONS

Dr. M.N. Kaddumukasa: principal investigator, study concept and design, data acquisition, analysis and interpretation of results, drafting and revision of manuscript. Dr. E. Katabira: study concept and design, study supervision, and review of manuscript. Dr. M. Sajatovic: study concept and design, study supervision, and review of manuscript. Dr. S. Pundik: study supervision and review of manuscript. Dr. M. Kaddumukasa: review of manuscript. Dr. L.B. Goldstein: study concept and design, study supervision, analysis and interpretation of results, and critical revision of the manuscript for important intellectual content.

ACKNOWLEDGMENT

The authors thank Donna Lillian Namujju for the statistical analysis.

STUDY FUNDING

This research was supported by the National Institute of Neurologic Disorders and Stroke of the NIH (award R25NS080968) and the Fogarty International Center of the NIH (award VUMC41965) in a joint collaboration with Makerere University, College of Health Sciences.

DISCLOSURE

M.N. Kaddumukasa and E. Katabira report no disclosures relevant to the manuscript. M. Sajatovic is a coinvestigator on several research grants including Pfizer, Merck, Ortho-McNeil-Janssen, Janssen, Reuter Foundation, Woodruff Foundation, Reinberger Foundation, NIH, and Centers for Disease Control and Prevention. She is a consultant with Bracket, Prophase, Otsuka, Pfizer, Amgen, and Sunovion and receives royalties from Springer Press, Johns Hopkins University Press, Oxford Press, UpToDate, and Lexicomp. Her CME activities include American Physician's Institute, MCM Education, and CMEology. S. Pundik, M. Kaddumukasa, and L. Goldstein report no disclosures relevant to the manuscript. Go to Neurology.org for full disclosures.

Received October 15, 2015. Accepted in final form May 4, 2016.

REFERENCES

- Feigin VL, Lawes CM, Bennett DA, Anderson CS. Stroke epidemiology: a review of population-based studies of incidence, prevalence, and case-fatality in the late 20th century. *Lancet Neurol* 2003;2:43–53.
- Kolapo O, Vento S. Stroke: a realistic approach to a growing problem in sub-Saharan Africa is urgently needed. *Trop Med Int Health* 2011;16:707–710.
- Lozano R, Naghavi M, Foreman K, et al. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 2012;380:2095–2128.
- Kaddumukasa M, Ddumba E, Duncan P, Goldstein LB. Poststroke hypertension in Africa. *Stroke* 2012;43:3402–3404.
- Sadler K, Nicholson S, Steer T, et al. National Diet and Nutrition Survey: assessment of dietary sodium in adults (aged 19 to 64 years) in England: National Center for Social Research. 2011. Available at: https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/213420/Sodium-Survey-England-2011_Text_to-DH_FINAL1.pdfhttps://www.gov.uk/government/uploads/system/uploads/attachment_data/file/213420/Sodium-Survey-England-2011. Accessed April 12, 2014.
- Nwanguma BC, Okorie CH. Salt (sodium chloride) content of retail samples of Nigerian white bread: implications for the daily salt intake of normotensive and hypertensive adults. *J Hum Nutr Diet* 2013;26:488–493.
- Charlton KE, Steyn K, Levitt NS, Jonathan D, Zulu JV, Nel JH. Development and validation of a short questionnaire to assess sodium intake. *Public Health Nutr* 2008;11:83–94.
- Drewnowski A, Rehm CD, Maillot M, Mendoza A, Monsivais P. The feasibility of meeting the WHO guidelines for sodium and potassium: a cross-national comparison study. *BMJ Open* 2015;5:e006625.
- BMJ-British Medical Journal. Reducing salt and increasing potassium will have major global benefits. *ScienceDaily*; 2013. Available at: www.sciencedaily.com/releases/2013/04/130405064437.htm. Accessed July 12, 2016.
- Brown IJ, Tzoulaki I, Candeias V, Elliott P. Salt intakes around the world: implications for public health. *Int J Epidemiol* 2009;38:791–813.
- Land MA, Jeffery P, Webster J, et al. The association of knowledge, attitudes and behaviours related to salt with 24-hour urinary sodium excretion. *Int J Behav Nutr Phys Act* 2014;11:47.
- Lallukka T, Laaksonen M, Rahkonen O, Roos E, Lahelma E. Multiple socio-economic circumstances and healthy food habits. *Eur J Clin Nutr* 2007;61:701–710.
- Cloutier L, Daskalopoulou SS, Padwal RS, et al. A new algorithm for the diagnosis of hypertension in Canada. *Can J Cardiol* 2015;31:620–630.
- Spence JD. Dilemmas in diagnosing and managing hypertension: is white coat hypertension benign? *Can J Cardiol* 2015;31:580–582.
- Koo HS, Kim YC, Ahn SY, Oh SW, Kim S, Chin HJ. Analysis of correlation between 24-hour urinary sodium and the degree of blood pressure control in patients with chronic kidney disease and non-chronic kidney disease. *J Korean Med Sci* 2014;29(suppl 2):S117–S122.
- Molloy DW, Alemayehu E, Roberts R. Reliability of a standardized Mini-Mental State Examination compared with the traditional Mini-Mental State Examination. *Am J Psychiatry* 1991;148:102–105.
- van Swieten JC, Koudstaal PJ, Visser MC, Schouten HJ, van Gijn J. Interobserver agreement for the assessment of handicap in stroke patients. *Stroke* 1988;19:604–607.
- Mercado CI, Cogswell ME, Valderrama AL, et al. Difference between 24-h diet recall and urine excretion for assessing population sodium and potassium intake in adults aged 18–39 y. *Am J Clin Nutr* 2015;101:376–386.
- Radhika G, Sathya RM, Sudha V, Ganesan A, Mohan V. Dietary salt intake and hypertension in an urban south Indian population: [CURES–53]. *J Assoc Physicians India* 2007;55:405–411.
- Campbell NR, Correa-Rotter R, Legowski B, Legetic B. Efforts to reduce dietary salt in the Americas [in English, Spanish]. *Rev Panam Salud Publica* 2012;32:253–257.
- Frisoli TM, Schmieder RE, Grodzicki T, Messerli FH. Beyond salt: lifestyle modifications and blood pressure. *Eur Heart J* 2011;32:3081–3087.
- Bukusuba J, Kikafunda JK, Whitehead RG. Nutritional knowledge, attitudes, and practices of women living with HIV in eastern Uganda. *J Health Popul Nutr* 2010;28:182–188.

23. Rasheed S, Jahan S, Sharmin T, et al. How much salt do adults consume in climate vulnerable coastal Bangladesh? *BMC Public Health* 2014;14:584.
24. Ha SK. Dietary salt intake and hypertension. *Electrolyte Blood Press* 2014;12:7–18.
25. McGuire S. Dietary guidelines for Americans. *Adv Nutr* 2012;2:293–294.
26. Bibbins-Domingo K, Chertow GM, Coxson PG, et al. Projected effect of dietary salt reductions on future cardiovascular disease. *N Engl J Med* 2010;362:590–599.
27. Aaron KJ, Sanders PW. Role of dietary salt and potassium intake in cardiovascular health and disease: a review of the evidence. *Mayo Clin Proc* 2013;88:987–995.
28. Mozaffarian D, Fahimi S, Singh GM, et al. Global sodium consumption and death from cardiovascular causes. *N Engl J Med* 2014;371:624–634.
29. Cappuccio FP, Kerry SM, Micah FB, Plange-Rhule J, Eastwood JB. A community programme to reduce salt intake and blood pressure in Ghana. *BMC Public Health* 2006;6:13.
30. Powles J, Fahimi S, Micha R, et al. Global, regional and national sodium intakes in 1990 and 2010: a systematic analysis of 24 h urinary sodium excretion and dietary surveys worldwide. *BMJ Open* 2013;3:e003733.
31. Salt and Black People of African Descent [online]. 2016. Available at: <http://www.actiononsalt.org.uk/salthealth/ethnic/blackafrican/>. Accessed March 3, 2016.
32. World Health Organization. Eating healthy: reduce salt intake [online]. 2016. Available at: <http://www.afro.who.int/en/media-centre/afro-feature/item/7474-eating-healthy-reduce-salt-intake.html>. Accessed March 3, 2016.
33. Sarmugam R, Worsley A. Current levels of salt knowledge: a review of the literature. *Nutrients* 2014;6:5534–5559.
34. Zhang J, Xu AQ, Ma JX, et al. Dietary sodium intake: knowledge, attitudes and practices in Shandong Province, China, 2011. *PLoS One* 2013;8:e58973.
35. Ma GX, Shive S, Zhang Y, et al. Knowledge, perceptions, and behaviors related to salt use among Philadelphia Chinese take-out restaurant owners and chefs. *Health Promotion Pract* 2014;15:638–645.
36. Sarmugam R, Worsley A, Wang W. An examination of the mediating role of salt knowledge and beliefs on the relationship between socio-demographic factors and discretionary salt use: a cross-sectional study. *Int J Behav Nutr Phys Act* 2013;10:25.
37. Li T, Qin Y, Lou P, et al. Salt intake and knowledge of salt intake in a Chinese population: a cross-sectional study. *Intern Med* 2015;5:186.
38. Newson RS, Elmadfa I, Biro G, et al. Barriers for progress in salt reduction in the general population: an international study. *Appetite* 2013;71:22–31.
39. Luft FC, Fineberg NS, Sloan RS. Estimating dietary sodium intake in individuals receiving a randomly fluctuating intake. *Hypertension* 1982;4:805–808.

This Week's *Neurology*[®] Podcast

Influence of sodium consumption and associated knowledge on poststroke hypertension in Uganda (see p. 1198)

This podcast begins and closes with Dr. Robert Gross, Editor-in-Chief, briefly discussing highlighted articles from the September 20, 2016, issue of *Neurology*. In the second segment, Dr. Justin Sattin talks with Dr. Larry Goldstein about his paper on the influence of sodium consumption and associated knowledge on poststroke Ugandan patients with and without a history of hypertension. Dr. Adam Numis reads the e-Pearl of the week about transient cerebral arteriopathy. In the next part of the podcast, Dr. Andy Schomer continues his interview with Dr. Eelco Wijdicks on prognostic models and clinical findings of myoclonus.

Disclosures can be found at Neurology.org.

At Neurology.org, click on “RSS” in the Neurology Podcast box to listen to the most recent podcast and subscribe to the RSS feed.

CME Opportunity: Listen to this week's *Neurology* Podcast and earn 0.5 AMA PRA Category 1 CME Credits[™] by answering the multiple-choice questions in the online Podcast quiz.