Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

HHS Public Access

Author manuscript

Circulation. Author manuscript; available in PMC 2018 May 09.

Published in final edited form as:

Circulation. 2017 May 09; 135(19): 1784–1787. doi:10.1161/CIRCULATIONAHA.117.027933.

Sources of Dietary Sodium: Implications for Patients, Physicians, and Policy

Lawrence J. Appel, MD, MPH[1,2,3] and **Kathryn Foti, MPH**[1,2]

[1]Welch Center for Prevention, Epidemiology, and Clinical Research, Baltimore, MD 21205, USA

[2]Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD 21205, USA

[3]Division of General Internal Medicine, Johns Hopkins University, Baltimore, MD 21205, USA

The best available evidence strongly supports population-wide sodium reduction as a means to prevent cardiovascular disease and stroke. Excess sodium intake raises blood pressure, the leading preventable cause of mortality worldwide. Well-controlled trials have documented a direct progressive relationship of sodium intake with blood pressure. On average, as sodium intake is lowered, so is blood pressure, both in hypertensive and non-hypertensive individuals. By lowering blood pressure, sodium reduction should prevent cardiovascular disease. Given that elevated blood pressure is a global pandemic affecting 1.4 billion people worldwide[1] and that cardiovascular risk is elevated at blood pressure levels below drug treatment thresholds, the critical issue is not whether, but how to lower sodium intake in the general population. Any such strategy must start with knowledge of the dietary sources of sodium.

The study by Harnack and colleagues,[2] published in this issue of the journal, provides such information, updating a highly cited but relatively small study published in 1991 by Mattes and Donnelly,[3] who documented that 77% of sodium is added outside the home during food processing.[4] In contrast to western societies, the majority of sodium is added at home in some regions of Asian countries. For example, in Guangxi, China, over 80% of sodium is added during home cooking.[4] Differences in sodium sources have important policy implications because strategies to reduce sodium in the food supply are distinct from those aimed at reducing sodium added at home.

In the study by Harnack and colleagues, 450 adults were recruited in three geographic locations in the U.S.: Birmingham, Alabama; Palo Alto, California; and Minneapolis-St. Paul, Minnesota. Equal numbers of women and men from each of four race/ethnic groups (African American, Asian, Hispanic, and non-Hispanic whites) were enrolled. Four 24-hour dietary recalls over 11 days were collected from each participant using special procedures, which included the collection of duplicate samples of salt added to food at the table and in

Corresponding Author: Lawrence J. Appel, MD, MPH, Welch Center for Prevention, Epidemiology, and Clinical Research, 2024 E. Monument St, Baltimore, MD 21205, USA (lappel@jhmi.edu, 410-955-4156 (phone), 410-955-0476 (fax)).

Conflict of Interest Disclosures

None

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

food preparation. Harnack and colleagues implemented the same methodology for categorizing sodium sources developed by Mattes and Donnelly. Overall, sodium added during food manufacturing and processing was the leading source of sodium, accounting for 71% of total sodium intake. Sodium inherent to food was the next highest contributor (14.2%), followed by salt added in home food preparation (5.6%), and salt added to food at the table (4.9%). Home tap water consumed as a beverage and dietary supplement and non-prescription antacids each contributed <0.5%. These findings are virtually identical to those of Mattes and Donnelly (Table 1).

Several other findings were notable. First, sodium added during food manufacturing and processing was the leading contributor for all subgroups examined, including Asians. This is an important observation that supports reducing the content of commercially processed and prepared foods as a broadly effective strategy for reducing sodium intake in the U.S. If sodium added at home had been the leading source, or even a larger contributor, such a finding would necessitate a different approach to population-wide sodium reduction. Second, the percent (and amount) of sodium added during home preparation increased with age from 3.7% in those aged 18–29 to 8.7% in those aged 60–75. Whether these findings result from secular trends or from age-associated reductions in sensory perception is unclear. Third, total sodium intake was higher in a few subgroups, specifically, in those with less education and in those from the Alabama site. It is reasonable to speculate that differences in sodium intake might account, at least in part, for the higher risk of stroke in these subgroups. Fourth, in a subsample (n=150), mean urinary sodium excretion was similar to mean estimated intake from the dietary recalls. This finding documents the accuracy of multiple record-assisted 24-hour dietary recalls and supports their utility in the National Health and Nutrition Examination Survey.

Other issues were not specifically addressed in this paper, but hopefully will be addressed in forthcoming publications from this study. For example, the study did not report the anion that accompanied sodium. While it is assumed that 95% or more of sodium is consumed as sodium chloride and <5% from non-chloride forms, particularly from preservatives, there are no contemporary data from national surveys. Second, the authors did not report the percent of sodium consumed at home and outside of the home at restaurants and other venues. Third, the study did not report intra- and inter-individual variability in sodium intake or excretion. Such data would be useful in informing the number of collections needed to reliably estimate individual-level intake, as opposed to average group intake which can be estimated from just a single collection. Because of the high intra-individual variability of sodium intake and excretion, it has been estimated that as many as 10–15 separate measurements are needed to estimate an individual's usual sodium intake.[5]

Finally, the authors did not report the sodium density of food consumed. Total sodium intake is the product of sodium density (mg of sodium per kilocalorie) times kilocalorie intake. The authors did, however, provide total energy intake from which we calculated mean sodium density (Table 2). Mean sodium density (~1.8 mg of Na per kcal) was similar to the highest level tested in the DASH-Sodium trial, while the range in subgroups varied from 1.6 to 2.1 mg of Na per kcal.[6] In those with less education and those residing in Alabama, high sodium intake reflected higher sodium density rather than higher calorie intake.

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

This study has several important strengths, including rigorous assessment of sodium intake with four 24-hour dietary recalls, as well as special efforts to understand sodium intake from the water supply and pills. The population was large and diverse, with men and women from a broad age range (18 – 74), in 4 race-ethnic groups in 3 geographic regions. Still, some subgroups were small, resulting in limited statistical power to detect significant differences, e.g., differences by age and hypertensive status. Further, because race-ethnicity differed by site, e.g., 75% of African Americans came from the Alabama site, it is unclear whether some differences were related to site or race-ethnicity. Another limitation was the generalizability of the study population, sampled in large part from university employees in the 3 urban locations. Total intake and sources of dietary sodium of individuals residing in rural and lower socioeconomic status areas might differ from the estimates in this paper, especially given higher blood pressure and higher stroke mortality in these high-risk groups.[7]

This study has implications for patients, physicians, and policy. About half of adults report trying to reduce their sodium intake.[8] Given the prevailing food supply, patients attempting to lower their sodium intake must focus on product selection. In some instances, it is possible to identify (and swap) higher sodium products for alternative products lower in sodium. However, in many other settings, particularly restaurants, such information is not routinely available. Resources to assist consumers are available from organizations such as the American Heart Association.[9] Patients also have an escalating role in advocating for changes to the food supply.[9] Physician advice to reduce sodium is associated with patients taking action to reduce sodium, yet only about a quarter of patients report having received such advice from their doctor.[8]

Physicians should emphasize product selection with their patients as a primary means to reduce dietary sodium. Concurrently physicians can encourage their patients to limit use of the salt shaker, but such advice will have minimal impact on its own. Importantly, special efforts are needed to target younger persons, given their high calorie intake and hence their high sodium intake.

For policy makers, this study reinforces recommendations of the Institute of Medicine, which highlighted, as a principal strategy, targets for industry to reduce the sodium content of commercially prepared foods.[10] The Food and Drug Administration has released draft voluntary targets for reducing sodium in commercially processed and prepared food.[11] If short-term reduction goals were met, sodium intake would be reduced by about 400 mg per day, which would have a large impact on cardiovascular health at the population-level.[12] Even if it were possible to eliminate salt use at the table and in cooking, the maximum reduction that could be reached is about 400 mg per day, and such a reduction would be very difficult to achieve. Meanwhile, other countries with a similar pattern of dietary sodium sources have successfully implemented voluntary sodium reduction targets and have reduced blood pressure and cardiovascular disease mortality.[13]

In conclusion, sodium added during food manufacturing and processing remains the leading source of sodium, accounting for 71% of total intake. This finding was consistent across population subgroups. Efforts to reduce the sodium content in our food supply have tremendous potential to lower blood pressure and prevent cardiovascular disease. As

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

highlighted in two presidential statements from the American Heart Association,[14,15] now is the time for action.

Acknowledgments

Funding Support: Kathryn Foti is supported by Grant Number T32 HL007024 from the National Heart, Lung, and Blood Institute, National Institutes of Health.

References

1. Mills KT, Bundy JD, Kelly TN, Reed JE, Kearney PM, Reynolds K, Chen J, He J. Global Disparities of Hypertension Prevalence and Control: A Systematic Analysis of Population-Based Studies From 90 Countries. Circulation. 2016; 134:441–450. [PubMed: 27502908]
2. Harnack L, Cogswell M, Shikany J, Gardner C, Gillespie C, Loria C, Zhou X, Yuan K, Steffen L. Sources of Sodium in US Adults from Three Geographic Regions. Circulation. 2017; XX:XXX–XXX.
3. Mattes RD, Donnelly D. Relative Contributions of Dietary Sodium Sources. J Am Coll Nutr. 1991; 10:383–393. [PubMed: 1910064]
4. Anderson CAM, Appel LJ, Okuda N, Brown IJ, Chan Q, Zhao L, Ueshima H, Kesteloot H, Miura K, Curb JD, Yoshita K, Elliott P, Yamamoto ME, Stamler J. Dietary Sources of Sodium in China, Japan, the United Kingdom, and the United States, Women and Men Aged 40 to 59 Years: The INTERMAP Study. J Am Diet Assoc. 2010; 110:736–745. [PubMed: 20430135]
5. Weaver CM, Martin BR, McCabe GP, McCabe LD, Woodward M, Anderson CA, Appel LJ. Individual variation in urinary sodium excretion among adolescent girls on a fixed intake. J Hypertens. 2016; 34:1290–1297. [PubMed: 27176144]
6. Svetkey LP, Sacks FM, Obarzanek E, Vollmer WM, Appel LJ, Lin PH, Karanja NM, Harsha DW, Bray GA, Aickin M, Proschan MA, Windhauser MM, Swain JF, McCarron PB, Rhodes DG, Laws RL. The DASH Diet, Sodium Intake and Blood Pressure Trial (DASH-sodium): rationale and design. DASH-Sodium Collaborative Research Group. J Am Diet Assoc. 1999; 99:S96–104. [PubMed: 10450301]
7. Moy E, Garcia MC, Bastian B, Rossen LM, Ingram DD, Faul M, Massetti GM, Thomas CC, Hong Y, Yoon PW, Iademarco MF. Leading Causes of Death in Nonmetropolitan and Metropolitan Areas — United States, 1999–2014. MMWR Surveill Summ. 2017; 66:1–8.
8. Jackson SL, Coleman King SM, Park S, Fang J, Odom EC, Cogswell ME. Health Professional Advice and Adult Action to Reduce Sodium Intake. Am J Prev Med. 2016; 50:30–39. [PubMed: 26163171]
9. American Heart Association. Sodium Breakup. https://sodiumbreakup.heart.org/. Accessed March 20, 2017
10. Institute of Medicine. Strategies to Reduce Sodium Intake in the United States. Washington, DC: The National Academies Press; 2010.
11. US Food and Drug Administration. Draft Guidance for Industry: Voluntary Sodium Reduction Goals: Target Mean and Upper Bound Concentrations for Sodium in Commercially Processed, Packaged, and Prepared Foods. https://www.fda.gov/Food/GuidanceRegulation/GuidanceDocumentsRegulatoryInformation/ucm494732.htm. Published June 1, 2016. Accessed March 20, 2017
12. Bibbins-Domingo K, Chertow GM, Coxson PG, Moran A, Lightwood JM, Pletcher MJ, Goldman L. Projected Effect of Dietary Salt Reductions on Future Cardiovascular Disease. New Engl J Med. 2010; 362:590–599. [PubMed: 20089957]
13. He FJ, Pombo-Rodrigues S, MacGregor GA. Salt reduction in England from 2003 to 2011: its relationship to blood pressure, stroke and ischaemic heart disease mortality. BMJ Open. 2014; 4:e004549.
14. Appel LJ, Frohlich ED, Hall JE, Pearson TA, Sacco RL, Seals DR, Sacks FM, Smith SC, Vafiadis DK, Van Horn LV. The Importance of Population-Wide Sodium Reduction as a Means to Prevent Cardiovascular Disease and Stroke. Circulation. 2011; 123:1138–1143. [PubMed: 21233236]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

15. Whelton PK, Appel LJ, Sacco RL, Anderson CAM, Antman EM, Campbell N, Dunbar SB, Frohlich ED, Hall JE, Jessup M, Labarthe DR, MacGregor GA, Sacks FM, Stamler J, Vafiadis DK, Van Horn LV. Sodium, Blood Pressure, and Cardiovascular Disease. Circulation. 2012; 126:2880–2889. [PubMed: 23124030]

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Table 1

Comparison of Two Studies Assessing Sources of Dietary Sodium Intake in United States Adults.

	Mattes and Donnelly (1991)[3]		**Harnack, et al (2017)[2]**	
Sample characteristics				
Sample size (n)	62		450	
Female (%)	74		50	
Race/ethnicity	Category (%)		Category (%)	
	Black	23	African American	22
	White	71	Non-Hispanic White	34
	Native American	5	Asian	22
	Unknown	2	Hispanic	22
Age	Mean years (SD)		Category (%)	
	30.1 (9.0)		18–29	27
			30–44	23
			45–59	30
			60–74	21
Hypertension (%)	0		24	
Study design				
Inclusion criteria	Normotensive, not adhering to any therapeutic diet		Free from chronic kidney disease or diabetes insipidus	
Sites	Philadelphia, PA		Birmingham, AL; Palo Alto, CA; Minneapolis-St. Paul, MN	
Diet measurement	Food records over 7 consecutive days		Four record-assisted telephone 24-hour dietary recall interviews over 11 days	
Measurement of salt added in cooking and at the table	Pre-weighed salt shakers		Collection of duplicate samples	
Validation of sodium intake	24-hour urines on days 5, 6, 7		24-hour urines on 4 days in a subset of participants (n=150)	
Reliability of sodium sources	Procedures repeated 8 and 25 weeks later among a subset of participants (n=20)		Not assessed	
Mean sodium intake (mg Na/ day)	3938		3501	
Sodium sources (%)				
Processing-added	77		71	
Inherent	12		14	
Cooking	6		6	

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

	Mattes and Donnelly (1991)[3]	**Harnack, et al (2017)[2]**
Table	5	5
Water	<1	<1
Dietary supplements or non-prescription antacids	Not assessed	<1

Author Manuscript Author Manuscript Author Manuscript Author Manuscript

Table 2

Mean Total Sodium Intake, Energy Intake, and Sodium Density by Demographic and Health Characteristics.

	Total sodium intake (mg Na/day)	Energy intake (kcal/day)	Na density (mg Na/kcal)
Sex			
Female	3123	1762	1.77
Male	3886	2191	1.77
Age			
18–29	3551	2080	1.71
30–44	3545	1990	1.78
45–59	3510	1998	1.76
60–74	3383	1840	1.84
Race/ethnicity			
Asian	3551	1936	1.83
African American	3798	2023	1.88
Hispanic	3138	1843	1.70
Non-Hispanic White	3503	2105	1.66
Highest education			
High school graduate or less	3854	1874	2.06
Some college or technical school	3809	2088	1.82
College and above	3302	1964	1.68
Site			
Alabama	3899	1907	2.04
California	3247	1955	1.66
Minnesota	3346	2068	1.62
History of hypertension			
Yes	3715	1994	1.86
No	3402	1964	1.73
Body weight status			
Normal or underweight	3254	1905	1.71
Overweight	3439	2002	1.72
Obese	3805	2022	1.88