

LETTERS

ATTEMPTING TO REDUCE SODIUM INTAKE MIGHT DO HARM AND DISTRACT FROM A GREATER ENEMY

Take it with a grain of salt: despite assertions to the contrary,¹ we do not know that reducing mean population sodium intake would decrease the risk of cardiovascular disease or save lives. Yes, we know that “excess sodium consumption raises blood pressure”¹ (at least transiently, for most people,² to a clinically minor degree^{3,4}). But based on the intermediate outcome of blood pressure, we cannot extrapolate that lowering sodium consumption would reduce cardiovascular risk or premature death. In fact, randomized controlled trial evidence suggests just the opposite: that lower sodium intake may lead to worsened cardiovascular disease and earlier death.⁵

It turns out that biological response to sodium intake is complex. Reducing sodium intake may lower blood pressure; but it may also decrease insulin sensitivity, alter lipids, and stimulate a variety of neurohormonal pathways detrimental to the cardiovascular system.^{4,6,7}

For these reasons, our bodies may work to keep our sodium intakes higher than the levels the Institute of Medicine and other authorities now advise. In fact, mean sodium

consumption—well above recommended levels^{1,8}—has been fairly constant across diverse populations for multiple generations.⁸ Thus, although it may be true that “individuals have little control over their sodium intake,”¹ it is probably not for the reasons Angell and Farley contend in their editorial blaming the food industry and consumers’ inability to avoid high-sodium processed and prepared foods.¹ Sodium consumption may have much more to do with human physiology than food formulations.⁸

If food reformulations were to make processed foods less palatable and discourage their consumption, this might be the only way the National Salt Reduction Initiative could be good for public health. Indeed, it is probably not the sodium in processed foods but the foods themselves that are the problem. Among other issues, these “foods” are generally engineered from dizzying arrays of highly refined carbohydrates; the consumption of which is associated with obesity, unhealthy lipids, high blood pressure, and insulin resistance, all as part of a broader metabolic syndrome^{9,10} (likely much more a risk for cardiovascular disease and early death than any modest effect of “excess” sodium intake).

When it comes to preventing cardiovascular disease and early death, refined carbohydrates are a greater enemy. Although there has been some action targeting refined carbohydrates, current action falls short. If sugar-sweetened beverages are a public-health problem, then why aren't sugar-laden cookies? If an energy drink is unhealthy, why not an energy bar? If reformulating products to have less sodium results in more sugar, will that be good for public health?

New York City could build on the momentum of its sugar-sweetened beverage work to refocus national action. Targeting high-sugar prepared and processed foods, as opposed to sodium, would be a decided start in the right direction. ■

Sean C. Lucan, MD, MPH, MS

About the Author

Sean C. Lucan is with the Department of Family and Social Medicine, Albert Einstein College of Medicine/Montefiore Medical Center, Bronx, NY.

Correspondence should be sent to Sean C. Lucan, Department of Family and Social Medicine, Albert Einstein College of Medicine/Montefiore Medical Center, 1300 Morris Park Ave, Mazer Building, Room 410, Bronx, NY 10461 (e-mail: slucan@yahoo.com). Reprints can be ordered at <http://www.ajph.org> by clicking the “Reprints” link. This letter was accepted October 6, 2012. doi:10.2105/AJPH.2012.301105

References

1. Angell SY, Farley TA. Can we finally make progress on sodium intake? *Am J Public Health*. 2012;102(9):1625–1627.
2. Alderman MH. Presidential Address: 21st Scientific Meeting of the International Society of Hypertension: dietary sodium and cardiovascular disease: the ‘J’-shaped relation. *J Hypertens*. 2007;25(5):903–907.
3. He FJ, MacGregor GA. Effect of longer-term modest salt reduction on blood pressure. *Cochrane Database Syst Rev*. 2004;(3):CD004937.
4. Graudal NA, Galloe AM, Garred P. Effects of sodium restriction on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride: a meta-analysis. *JAMA*. 1998;279(17):1383–1391.
5. DiNicolantonio JJ, Di Pasquale P, Taylor RS, Hackam DG. Low sodium versus normal sodium diets in systolic heart failure: systematic review and meta-analysis. *Heart*. 2012; Epub ahead of print.
6. Grassi G, Dell’Oro R, Seravalle G, Foglia G, Trevano FQ, Mancia G. Short- and long-term neuroadrenergic effects of moderate dietary sodium restriction in essential hypertension. *Circulation*. 2002;106(15):1957–1961.
7. Petrie JR, Morris AD, Minamisawa K, et al. Dietary sodium restriction impairs insulin sensitivity in noninsulin-dependent diabetes mellitus. *J Clin Endocrinol Metab*. 1998; 83(5):1552–1557.
8. Taubes G. Salt, we misjudged you. *New York Times*. June 2, 2012;The Opinion Pages.
9. Grundy SM, Hansen B, Smith SC Jr, Cleeman JJ, Kahn RA. Clinical management of metabolic syndrome: report of the American Heart Association/National Heart, Lung, and Blood Institute/American Diabetes Association conference on scientific issues related to management. *Circulation*. 2004;109(4):551–556.
10. Reaven GM. The insulin resistance syndrome: definition and dietary approaches to treatment. *Annu Rev Nutr*. 2005;25:391–406.

ANGELL AND FARLEY RESPOND

In his letter, Lucan argues that the relationship between excess sodium consumption and adverse health effects is not supported by the evidence. He concludes that public health actions to improve population health should instead focus on sugar.

Letters to the editor referring to a recent Journal article are encouraged up to 3 months after the article's appearance. By submitting a letter to the editor, the author gives permission for its publication in the Journal. Letters should not duplicate material being published or submitted elsewhere. The editors reserve the right to edit and abridge letters and to publish responses.

Text is limited to 400 words and 10 references. Submit online at www.editorialmanager.com/ajph for immediate Web posting, or at ajph.edmgr.com for later print publication. Online responses are automatically considered for print publication. Queries should be addressed to the Editor-in-Chief, Mary E. Northridge, PhD, MPH, at men6@nyu.edu.