Letter by DiNicolantonio et al Regarding Article, “Reducing Sodium Intake to Prevent Stroke: Time for Action, Not Hesitation”

To the Editor:

In a recent review in Stroke, Appel1 states that the evidence supporting population-wide reduction in sodium intake is compelling; the time for action is now. Appel concludes that reducing sodium intake would be both safe and effective for preventing cardiovascular disease, stroke, and deaths. We take strong issue with several of Appel’s points and urge caution when considering his conclusions.

Most of arguments of Appel focus on evidence relating sodium intake to blood pressure (BP). Starting with the weakest evidence, Appel mentions that Kenyans who migrated to urban areas had higher mean sodium intakes and BPs than those who remained in rural areas. Such ecological association hardly implicates sodium. Urban living is also associated with other dietary factors (eg, refined-carbohydrate consumption) and lifestyle changes (eg, sedentary work) that are also likely to increase BP independent of sodium.

With respect to experimental evidence, Appel invokes the Dietary Approaches to Stop Hypertension (DASH)-Sodium trial. He notes that, in the trial, sodium reduction to a level of 1500 mg/d lowered BP. But DASH-Sodium was a multi-interventional trial; the experimental group received advice to increase their consumption of fruits, vegetables, whole grains, fish, nuts, potassium, calcium, magnesium, dietary fiber, and to reduce their intake of red meat, sweets, and sugar-containing beverages.2 It is impossible to conclude that results were because of a reduction in sodium. The same holds true for a meta-analysis Appel cites:3 included trials tested intervention conditions differing from control conditions by more than just sodium intake. Additionally, a Cochrane review of 167 diet trials showed only small reductions in BP (on the order of 1%–3.5%) with low-sodium intake.4

Ultimately, BP is not what we care about. Despite Appel’s reassurance that BP is considered one of the few surrogate outcomes with a sufficiently robust body of evidence to guide policy, this statement is simply unfounded. Surrogate outcomes, particularly for vascular diseases, have been repeatedly misguiding; BP in not an exception. In the randomized, double-blind, Hypertension-Stroke Cooperative Study Group trial, stroke survivors achieved a mean decrease in BP of 25.0/12.3 mm Hg with drug therapy but showed no significant reduction in stroke, myocardial infarction, or sudden death. Similarly, a 2012 Cochrane review of antihypertensive therapy trials showed a lack of cardiovascular benefit, despite a reduction in BP.5 Thus, even if reducing sodium intake could reduce BP, it might not improve vascular outcomes. Indeed, sodium reduction is also significantly associated with increases in renin, aldosterone, noradrenaline, adrenaline, cholesterol, and triglycerides.6 It is unclear why Appel feels BP is more important than these other surrogate outcomes.

In regards to patient-oriented outcomes, Appel dismisses randomized trials in patients with heart failure as irrelevant because of the unconventional treatment approach of the investigators.7 Yet these trials—showing increases in hospitalizations and mortality with low-sodium intake versus normal-sodium intake—tested identical diets in intervention and comparison arms with the only difference being the level of ingested sodium (making these trials more relevant than DASH-Sodium and other trials Appel cites). Also, Appel fails to cite 3 relevant heart failure trials, all consistently show harm with reduced sodium intake.7–9

Conclusions of Appel are based on—if not biased by—a single surrogate end point (BP) for which the evidence for sodium restriction is not compelling. Effects on other surrogate outcomes should give pause, and the only existing data on important patient-oriented outcomes should make a decided case for hesitation, not action, when it comes to reducing population sodium intake.

Perhaps the greatest concern with arguments of Appel is his concluding remark that “policymakers should redouble their efforts to lower sodium intake in processed foods.” Processed foods may be unhealthy for many reasons; sodium content may be—or may not be—just one of them. Given that sodium intake occurs in a remarkably narrow range across very diverse populations and eating habits,10 it is possible that if processed foods were reformulated to be lower in salt, people would just eat more of them to obtain the sodium human physiology demands. Would the extra doses of unhealthy fats, refined carbohydrates, artificial colors, flavors, preservatives, fillers, and any chemical substitutes for the reduced sodium accompanying larger portions of processed food be better for health?

When it comes to reducing population sodium intake, there is low likelihood of benefit and real potential for serious harm. The most reasonable conclusion is that it is not time for misdirected action. It is time to look before we leap.

Disclosures

None.

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*Stroke*. 2014;45:e106-e107; originally published online May 6, 2014;
doi: 10.1161/STROKEAHA.114.005067

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/45/6/e106