Response to Letter Regarding Article, “Reducing Sodium Intake to Prevent Stroke: Time for Action, Not Hesitation”

I appreciate your interest in my commentary on population-wide sodium intake published in Stroke. The letter by DiNicolantonio et al advised inaction rather than aggressive efforts to reduce sodium intake. Their piece ignores the public health reality that approximately two third of adults worldwide have either prehypertension or hypertension and that blood pressure (BP) is the leading cause of stroke and preventable deaths worldwide. It is widely recognized in the prevention community that drug therapy for hypertension, while effective, is a reactive approach and an incomplete solution to the pandemic of elevated BP and its consequences. A proactive approach that focuses on excess sodium intake, a key determinant of elevated BP, is warranted.

As for specific comments, there are several issues that warrant a response. First, they dismiss results from ecological studies, yet such evidence from populations with low BP provides important insights; especially when combined with other types of evidence, such results from trials document the BP-lowering effects of reducing sodium intake. Achieving lifelong levels of optimal BP is almost exclusively found in isolated populations, which with rare exception consume extremely low sodium intake. Their comment that refined carbohydrates and sedentary lifestyle might be responsible for BP changes observed in Kenyan migrants is perplexing. There is no convincing evidence that refined carbohydrates have a major effect on BP. Furthermore, the evidence on the BP-lowering effect of physical activity is not nearly as compelling as the evidence on sodium reduction.

Second, they claim that it is impossible to disentangle the effects of sodium reduction from other aspects of diet in the Dietary Approaches to Stop Hypertension (DASH)-sodium trial. In fact, the DASH-sodium trial explicitly tested 2 factors: dietary pattern and sodium. Individuals were randomized to 1 of 2 diets (DASH or control). Within each diet, each participant was fed low, medium, and high sodium intake in random order. The results document that sodium reduction significantly lowers BP in the setting of either the DASH or control diet.

A third matter deals with the use of BP for policy making. I too am skeptical of most surrogate outcomes. Yet BP stands apart as a clinically relevant variable to guide clinical decision making and policy. They comment that sodium reduction has adverse effects on renin, aldosterone, noradrenaline, adrenaline, triglycerides, and high-density lipoprotein cholesterol. However, none of these variables are relevant for clinical decision making. In fact, thiazide diuretics, a class of medications that adversely affect most of these variables, actually prevents cardiovascular disease.

A fourth issue is their reliance on a set of trials of sodium reduction in patients with heart failure reported from a single center. These studies are irrelevant for the general population and likely irrelevant to the management of heart failure, given substantial design flaws, unconventional heart failure management, and data irregularities. A corresponding meta-analysis that relied almost exclusively on these articles was retracted.

Kasprowicz et al comment that physicians and other healthcare providers are poorly educated about the beneficial effects of improved diet and ill equipped to provide nutrition counseling. I concur. It is vitally important for physicians to understand that the vast majority of preventable deaths are related to dietary factors. However, education of health professionals, while necessary, is clearly not sufficient. Our healthcare delivery system has focused on the treatment of existing disease and medical management of risk factors. We need much stronger public health approaches to create an environment in which healthy living is the default rather than the exception.

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