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EDITORIAL COMMENT

Time for a Renewed Focus on the DASH-Low Sodium Diet*

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Although the Dietary Approaches to Stop Hypertension (DASH) diet and its sodium-restricted variation were initially conceived as strategies to combat hypertension, it has become clear over the past several decades that the benefits of these dietary patterns extend beyond hypertension alone. The original DASH trial in 1997 demonstrated that a diet that emphasizes fruits, vegetables, low-fat dairy, and whole grains and limits saturated fat, red and processed meats, and sugar results in a decrease in systolic and diastolic blood pressure of 11.4 mm Hg (95% confidence interval: 6.9 to 15.9 mm Hg) and 5.5 mm Hg (95% confidence interval: 2.7 to 8.2 mm Hg), respectively, among individuals with hypertension (1). Further, the DASH-Sodium trial in 2001 showed that a combination of the DASH diet and a low sodium diet (0.5 mg/kcal) resulted in greater blood pressure lowering than either diet alone (2). Beyond this, numerous analyses of randomized trials have shown that the DASH diet also positively impacts other cardiometabolic risk factors, including lipids, glycemic control, and body weight (3). Although there are no randomized trials to assess the impact of the DASH and/or low sodium diets on hard cardiovascular (CV) outcomes, there is ample observational evidence to suggest that both dietary patterns are associated with significantly improved cardiovascular disease and stroke risk (3,4).

Beyond improved blood pressure control, the mechanism(s) by which the DASH diet and sodium restriction may improve CV outcomes is not clear. The DASH diet may exert blood pressure benefit through interactions with the renin-angiotensin-aldosterone system (5) or through upregulation of nitric oxide bioavailability (a vasodilatory stimulus) and decreased pulse wave velocity (a measure of arterial stiffness) (6). However, benefits on CV outcomes may involve additional mechanisms yet undetermined. The mechanisms of CV benefit from reduced sodium intake are even more obscure, but may be related to alterations in the autonomic nervous system, vascular function, renal function, or cardiac function (4). The impact of the DASH diet, alone or in combination with a low sodium diet, on markers of cardiac strain, injury, and inflammation has not been assessed, until now.

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In this issue of the *Journal*, Juraschek et al. (7) report a secondary analysis of the DASH-Sodium trial to determine the impact of DASH diet and sodium restriction, individually and combined, on high-sensitivity cardiac troponin I (hs-cTnI, measure of cardiac injury), N-terminal b-type pro natriuretic peptide (NT-proBNP, measure of cardiac strain), and high-sensitivity C-reactive protein (hs-CRP, measure of inflammation) (7). The DASH-Sodium trial was a controlled feeding study that enrolled adults with untreated systolic blood pressure 120 to 159 mm Hg and diastolic blood pressure of 80 to 95 mm Hg, and randomized them to either a control diet (reflective of a typical American diet) or the DASH diet. Further, participants in both arms were assigned to each of 3 sodium intake levels (high, medium, low) for 30 days in random order using a crossover design with washout periods in-between. Energy intake was adjusted to keep the weight of each participant constant throughout the trial. Serum specimens were collected

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at baseline and after each of the 30-day sodium feeding periods.

The design of the DASH-Sodium trial allowed these authors to tease apart the effects of the DASH diet and of sodium reduction, both independently and in combination, on the previously mentioned cardiac biomarkers. With respect to the effect of the DASH diet versus the control diet, the authors found that the DASH diet reduced hs-cTnI by 14.0% and hs-CRP by 13.2%, but interestingly there was no difference between diets in change in NT-proBNP. Thus, the DASH diet alone appeared to have a beneficial effect on myocardial injury and on inflammation, but not on cardiac strain. With respect to the effect of sodium reduction (independent of the underlying dietary pattern), the low sodium diet decreased NT-proBNP by 19.2% compared with the high sodium diet, but did not affect hs-cTnI. Of note, the low sodium diet led to an increase in hs-CRP by 8.8%, suggesting that a low sodium diet decreases cardiac strain but increases inflammation. With respect to the combined effect of the DASH diet with sodium reduction compared with the control-high sodium diet, the DASH-low sodium diet led to a reduction in hs-cTnI by 20.1% and NT-proBNP by 22.9%, but did not significantly impact hs-CRP. Thus, the combination of DASH diet and low sodium appears to improve cardiac injury and strain, and does not affect overall inflammation.

These data indicate, for the first time, that the combination of the DASH diet and sodium reduction directly affects the heart in 2 distinct ways: through decreased myocardial injury and decreased cardiac strain. Whether these effects are mediated through blood pressure lowering alone or through other mechanisms was not evaluated and would be important for future study. However, evidence of impact on cardiac injury and strain are important because of their known strong correlation with CV events (8,9), further bolstering the argument that the DASH-low sodium diet is likely to reduce the risk of CV events. The possible deleterious effect of low sodium alone on markers of inflammation further strengthens the argument for combining these dietary interventions. Importantly, effects on cardiac injury and strain were demonstrated in individuals without any clinical evidence of coronary artery disease or heart failure at baseline, suggesting that this dietary combination can improve subclinical metrics of cardiac health. Further, the impact on these markers was seen within weeks, indicating a relatively rapid impact on cardiac damage.

What are the clinical and public health implications of these findings? These data should spur a renewed focus on the critical need for widespread adoption of the DASH-low sodium diet in the United States. We have known for several decades now that the DASH diet and sodium restriction can prevent and treat hypertension (the most prevalent CV disease risk factor), reflected in multiple guideline recommendations (10,11). Unfortunately, the overall uptake of the DASH-low sodium diet in the United States remains woefully low (12). With these new data suggesting a direct cardiac benefit of the DASH-low sodium diet, the authors should be congratulated for continuing to shine a light on a dietary pattern that deserves greater attention from both a policy and an implementation science perspective. We need to approach the adoption of the DASH-low sodium diet in diverse communities with the same scientific rigor and intensity that we have used to understand its biological benefits. The challenge remains moving the DASH-low sodium diet from the research world into the real world, where its significant health benefits can be fully realized.

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