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In Hypertension

Sodium, Potassium, and Cardiovascular Disease Several studies suggest that we should promote low-sodium, high-potassium diets — especially in patients with hypertension. Sodium restriction and generous potassium intake are associated with lower blood pressure (BP), but the ultimate effect on cardiovascular (CV)-related morbidity is less clear. In 2021, several studies addressed this issue and refocused attention on these dietary interventions. In a trial from China, 21,000 people with histories of stroke or inadequately controlled hypertension were randomized to replace regular salt with a salt substitute (75% sodium chloride, 25% potassium chloride) or to continue using regular salt; to further enhance sodium restriction, intervention participants were instructed to use the salt substitute more sparingly than they had used regular salt. At 5 years, rates of stroke, major adverse CV events, and mortality were each significantly lower in the salt-substitute group — by roughly 2 or 3 events per 100 participants. According to 24-hour urine electrolyte measurements, the intervention lowered sodium intake by only about 10% but increased potassium intake by about 50% (NEJM JW Gen Med Oct 1 and N Engl J Med Sep 16; 385:1067). In another study, researchers pooled data from 6 cohort studies in which people with no history of CV or renal disease submitted multiple 24-hour urine collections for sodium and potassium levels, reflecting dietary intake. At average follow-up of 9 years, those with sodium excretion of ≈2 g daily had 2 or 3 fewer CV events per 100 people, compared with those whose sodium excretion was ≈5 g daily. Conversely, people with high potassium excretion had 1 fewer CV event per 100 people, compared with those whose potassium excretion was low. These findings were adjusted for confounding factors, and they applied to people with or without diagnosed hypertension (NEJM JW Gen Med Dec 15 and N Engl J Med Nov 13; [e-pub]). Finally, a meta-analysis of 85 studies revealed a linear, dose-response relation between estimated sodium intake and BP during 1 to 36 months of observation. At the extremes of intake (6 g vs. 1 g of sodium intake), the systolic BP difference was ≈12 mm Hg; for diastolic BP, the difference was ≈5 mm Hg (NEJM JW Gen Med Jun 1 and Circulation Apr 20; 143:1562). Taken together, these studies suggest that we should redouble our efforts to promote low sodium, high-potassium diets for many patients, especially those with hypertension. A caveat is that excessive potassium intake should be avoided in patients at risk for hyperkalemia (e.g., those with advanced renal disease).

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