Sodium and Potassium Intake in US Adults

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Quantitative estimates of sodium intake in the general population provide a basis for clinical and public health decision making. For many years, National Health and Nutrition Examination Survey (NHANES) results have provided estimates for sodium and potassium intake in the US general noninstitutionalized population. However, these reports have been based on 24-hour dietary recalls, a method that is subject to a variety of systematic errors in the estimation of electrolyte intake and is known to underestimate dietary sodium intake and overestimate dietary potassium intake. In this issue of *Circulation*, Jackson et al[1] provide the first nationally representative estimates for sodium and potassium intake in US adults based on carefully collected 24-hour urine collections. All of the 765 adults surveyed had at least one 24-hour urine collection, and a second collection was obtained in more than half (436) of the participants, allowing for a between-visit random error correction in estimation. The availability of national estimates based on 24-hour urine collections represents an important step forward for NHANES and a new norm for reporting dietary electrolyte intake in US adults.

As one would expect, the intake of dietary sodium was higher, and of potassium was lower, in the current report in comparison with previous NHANES estimates. The average intake of sodium was well above the current federal *Dietary Guidelines for Americans* recommendations of <2300 mg/d for all adults and <1500 mg/d for those ≥51 years, and those of any age who are African American, or have hypertension, diabetes mellitus, or chronic kidney disease.[2] For example, the average intake of 3746 mg/d noted in those with hypertension is almost 2.5 times higher than the federal recommendation. The reported average intake of dietary potassium (1997 mg/d) was less than half the federal recommendation of 4700 mg/d for US adults. Based on the current and previous reports, almost all adults in the United States have a daily intake of sodium that exceeds federal recommendations and a potassium intake that fails to meet federal recommendations. In the current report, only 32 (4.2%) of the 765 participants had a dietary sodium intake <2300 mg/d, and the percent meeting the federal recommendation would be even lower if one accounted for the participant's age, race/ethnicity, and prevalence of hypertension, diabetes mellitus, or chronic kidney disease.

The report by Jackson et al identified a strong and statistically significant direct relationship between sodium intake and blood pressure (BP), and a corresponding inverse relationship between potassium intake and BP, as well. The magnitude of the associations for both dietary sodium and potassium with BP were considerably higher than in previous NHANES reports, with a 1000-mg-lower level of sodium intake being associated with a –4.4 mm Hg level of systolic BP and a 1000-mg-higher level of potassium intake being associated with a –3.4 mm Hg level of systolic BP. These relationships are also greater than one would expect from clinical

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trial experience,[3] albeit the effects of dietary reductions in sodium intake and potassium supplementation have varied depending on participant demographics, starting level of BP, and, in the case of potassium interventions, concurrent level of sodium intake. In contrast to the general population sample studied in NHANES, clinical trials tend to be conducted in selected samples of volunteers who may have a higher socioeconomic status. One should be cautious in assuming causality for cross-sectional associations, but, if this were to be the case for the findings by Jackson et al, a reduction in sodium intake to the federal recommendation might lower systolic BP by ≈10 mm Hg in adults with hypertension, and an increase in potassium intake to the federal recommendation might reduce systolic BP by ≈9 mm Hg. The effects of sodium reduction could be even greater in adults with obesity, based on the patterns for interaction noted between high body weight and sodium-BP effects in the current and some prior reports.[4] There was also a significant relationship between sodium-to-potassium ratio and BP, albeit the contrast in adjusted odds ratio with hypertension between the first and fourth quartile of the exposure variable was more striking for sodium than for the sodium-to-potassium ratio.

Previous cross-sectional and longitudinal studies have also identified a strong, progressive, and statistically significant direct relationship between dietary sodium and BP, independent of whether sodium intake was estimated by means of spot urine samples,[5] 24-hour recall methods,[6] or the gold-standard method of 24-hour urine collections.[7] Likewise, the inverse relationship between potassium intake and BP has been noted in many previous studies.[5,6,8] In addition, numerous randomized controlled trials and meta-analyses have demonstrated the capacity of sodium reduction and potassium supplementation to lower BP and provide an effective means to prevent and treat hypertension.[3] Level of BP is thought to serve as a good surrogate for cardiovascular disease (CVD) risk, especially for stroke,[3] and indirect evidence from long-term follow-up of BP-lowering trials suggests that sodium reduction provides a means to prevent CVD.[9,10] In contrast to the findings in the report by Jackson et al, one might expect the joint effects of sodium and potassium to provide a stronger relationship with BP than either electrolyte on its own. In randomized controlled trials, the BP-lowering effect of potassium has been greater in adults with a higher intake of sodium (higher sodium-to-potassium ratio)[11] and in a careful prospective analysis of long-term follow-up in Trials of Hypertension Prevention participants, sodium-to-potassium ratio was more strongly related to CVD than either sodium or potassium alone.[12]

A strong body of evidence documents the efficacy of sodium reduction and potassium supplementation in lowering BP,[3] especially in those who are older, of African or African American descent, and, in the case of potassium interventions, have a higher intake of dietary sodium. Cohort studies of the relationship between sodium and CVD have yielded mixed relationships, but most are difficult to interpret because of the poor quality of their methods.[3] Studies that have used multiple 24-hour urine collections to estimate dietary sodium have demonstrated a strong positive relationship between sodium intake and CVD,[3,10] even in cohorts where the potential for reverse causality has been high.[13] Meta-analyses of cohort studies have consistently noted a strong significant inverse relationship between dietary potassium intake and CVD.[14] Randomized controlled trials designed to test the effect of dietary sodium reduction and potassium supplementation on CVD are desirable but challenging to design and implement.[3] A cluster-designed randomized controlled trial would appear to be the only feasible option for testing the efficacy of dietary sodium reduction or potassium supplementation, alone or in combination. The SSaSS study (Salt Substitute and Stroke Study) is a large-scale cluster-designed randomized controlled trial being conducted in China to determine the effects of reducing the sodium-to-potassium ratio, achieved by means of a potassium-rich salt substitute, on stroke and all-cause mortality.[15] With 600 clusters and a total sample size of almost 21 000 adults (≈35 per cluster) at high risk for CVD, this trial is positioned to determine whether a reduction in the sodium-to-potassium ratio will be beneficial. Those enrolled had a mean age of 65 years at baseline and were selected on the basis of having had a prior stroke (73%) or being at high risk for stroke. The trial is being funded by the National Health and Medical Research Council of Australia. Results and follow-up are expected to be completed before the end of 2020. The results will be of considerable help in documenting the effect of a reduction in the sodium-to-potassium ratio on CVD.

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FOOTNOTES

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