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Response to Invited Commentary

Prentice et al. Respond to "Improving Estimation of Sodium Intake"

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We thank Drs. Appel and Jacobs for their thoughtful comments (1) on our paper (2) on sodium and potassium intakes in relation to the risk of cardiovascular disease. In response, we would like to emphasize that our objective in exercising a biomarker calibration approach is to strengthen analyses of cardiovascular disease associations rather than analyses of group mean intake estimation.

Intake estimation using self-reported diet is problematic whether based on food frequency questionnaires, food records, or 24-hour recalls (3). The regression calibration approach adjusted estimates from food frequency questionnaires for both random and systematic error using a 24-hour urinary measure of sodium and potassium intakes in a biomarker substudy in Women's Health Initiative cohorts. Our major assumption was that (log-transformed) urinary measures, as estimates of (log-transformed) intake over a relatively short time period (e.g., 1 year), adhere to a classical measurement model; that is, these values are equal to their targeted values plus measurement error that is unrelated to such targets or other study subject characteristics. Even though these biomarkers incorporate a noteworthy "noise" component, they serve to "anchor" intake estimates for individual study subjects. Additional days of urine collection could reduce the noise component.

The calibrated intake estimates are derived from linear regression of biomarker values on corresponding self-report values and other study subject characteristics. The principal purpose of the other variables (e.g., age and race) is to allow correction for systematic bias related to these variables, rather than borrowing intake estimation information. The variables needed in the disease risk model for confounding control also need to be considered for inclusion in the corresponding calibration model. Conceptually, a calibrated intake estimates the expectation of the targeted intake conditional on the self-reported intake and other relevant measured variables. The one exception in our analyses concerns body mass index (BMI). BMI is a complex construct that could confound and/or mediate a disease association. Because the self-report signal for sodium is quite weak, we chose to leave BMI out of the disease risk model but use it in our calibration equations. As explained (2), doing so avoided overcorrection relative to this mediating variable but could allow some residual confounding.

On the basis of cited feeding studies (2), we think that a single 24-hour urinary excretion provides biomarkers that plausibly adhere to this classical measurement model for sodium, potassium, and their ratio. We also cited reports in which it was suggested that there are racial differences in potassium excretion. If such dependence is confirmed in a context of objectively measured potassium intake, then our analyses could result in incomplete measurement error correction for potassium in disease association analyses. More generally, our analyses were based on calibration equations that met objective criteria and could be expected to yield association analyses of much improved reliability compared with those based only on self-reported intakes. Hence, we think that our odds ratio and hazard ratio estimates, which mostly differ more strongly from the null (typically by a factor of 2–6) than do those based on self-report alone, merit serious consideration. Yet, quite a lot of uncertainty attends the biomarker calibrated association parameter estimates, and research is needed to further examine analyses that suggest the absence of clear positive association between stroke risk and higher sodium intake and our surprising associations of hemorrhagic stroke with all 3 dietary variables. Note that these estimated associations, including that shown for hemorrhagic stroke and potassium in our earlier paper (4), were suggested in analyses using self-reported intake data alone (Web Table 7 in Prentice et al. (2)), and therefore do not result from use of a biomarker calibration approach. In summary, combining self-reported data with substudy biomarker data provides a useful research strategy on this important topic, as we await large cohort studies that include both objective intake measures and cardiovascular disease outcomes.

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