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The Article by Andrew Mente and colleagues¹ raises serious concerns of the credibility and rigour of the review process. The paper methodology suffers from flaws that have been repeatedly addressed in the medical literature in recent years and that are ignored.²

The use of sodium concentrations from morning urine fasting samples extrapolated to 24-h urinary sodium excretion is an inappropriate method for estimating salt intake.³ Mente and colleagues' reference to their validation,⁴ critiqued at the time of its publication,⁵ ignores the presence of a significant bias when estimating individuals' sodium excretion. They also avoid to mention that a similar validation in a Chinese cohort⁶ presents the results with less confidence. They use data on individuals when assessing risk prediction in a cohort study design, which is highly misleading as several 24-h urine collections are needed to approximate an individual's salt intake with a high degree of confidence and without bias. Not surprisingly, cohort studies using repeated 24-h urine collections to assess salt intake show a linear graded association between sodium excretion and cardiovascular outcomes with no increase at lower sodium intakes.^{7,8}

The authors split a continuously distributed biological variable in the population (blood pressure) into a dichotomy of hypertension

and normotension, which reduces the statistical power of detecting associations, particularly when studying trends.

Sick populations and patient groups are consistently being used to study the implications of a moderate reduction in salt consumption in the general population. None of these studies' results can be generalised to inform current public health strategies for a moderate reduction in sodium consumption in populations or to be considered of good quality to support a causal association between low sodium intake and increased cardiovascular mortality.⁹ The evidence supporting global actions for a moderate reduction in salt consumption to prevent cardiovascular disease is strong and such studies should not overturn the concerted public health action to reduce salt intake globally.

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Andrew Mente and colleagues¹ ignore a possible source of bias in their analysis of the association of sodium excretion with cardiovascular events. At any measure of blood pressure, the population will comprise individuals with varying extents of sodium sensitivity, and those with higher sensitivity will achieve that blood pressure if they ingest less sodium.² As a result, within any blood pressure range—for example, normotensive—there will be an anticorrelation between sodium intake and sodium sensitivity.

If sodium sensitivity is causal of cardiovascular events or death, this anticorrelation could explain the observational anticorrelation between sodium intake and these events. Such a causal association between sodium sensitivity and adverse outcomes is plausible: sensitive individuals are likely to have been hypertensive in the past and are likely to be so in the future. As a result, the observational association of low sodium excretion with cardiovascular events might not be causal, but an artefact of the anticorrelation between sodium intake and sensitivity within the normotensive group.

I declare no competing interests.

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