Salt—too much or too little?

When apparent dogma is challenged, we should speak not of controversy but rather accede to the all-encompassing expression of so-called scientific uncertainty, so as to avoid unbecoming rhetoric. The issue of population strategies for salt consumption is a good case in point. There is no argument other than "excessive salt in the diet raises blood pressure", and that strategies to reduce salt in individuals with hypertension prevent the cardiovascular consequences of the disease. However, the corollary that reducing sodium intake across populations will be beneficial to all, has been challenged with the assertion that doing so might indeed be harmful.

Until relatively recently there has been tacit acceptance that governments across the world should be lobbied to lower salt in the diet for the betterment of the health of all.

The first serious challenge to this approach came from a European population study in which low sodium excretion predicted an increased cardiovascular mortality.[1] The study was dismissed by *The Lancet* as contributing "little to our understanding of salt and disease" and that "the results of this work should neither change thinking nor practice".[2] Then, as often happens in science, a refutation to this dismissal came from another large population study, confirming that a low sodium intake was associated with increased cardiovascular risk.[3] These studies prompted the US Centers for Disease Control and Prevention to evaluate the evidence for possible harm in reducing population salt intake; it was concluded that most evidence supported a positive relation between high sodium intake and the risk of cardiovascular disease but that there was some, albeit inconclusive evidence, suggesting that a low sodium intake was associated with adverse health effects in some subgroups of the population.[4]

In 2014, the PURE study, which was done in 157 543 adults in 18 countries, showed that both higher (>5 g/day) and lower levels (<3 g/day) of sodium excretion were associated with increased risk of cardiovascular disease, resulting in a J-shaped association curve.[5] A well-reasoned editorial on this study acknowledged that because hypertension is the most common modifiable risk factor for cardiovascular disease, accounting for more than 9 million deaths annually, it behoves the scientific community to evaluate any population-based strategy, such as salt reduction, that might halt this epidemic. The editorial argued that the issue could only be decided by doing a randomised, controlled outcome trial, and that "in the absence of such a trial, the results argue against reduction of dietary sodium as an isolated public health recommendation".[6]

Support for this viewpoint has been added to by a large meta-analysis[7] and a cohort study,[8] but the most persuasive evidence is reported in this issue of *The Lancet* by Andrew Mente and colleagues.[9] Sodium excretion in 133 118 individuals from more than 49 countries, of whom half were hypertensive and half normotensive, showed that cardiovascular disease and death were increased with low sodium intake irrespective of hypertension status, whereas there was an increased risk of cardiovascular disease and death only in individuals with hypertension consuming more than 7 g of sodium per day. Based on these findings, it is concluded that any population strategy for lowering sodium would be best targeted at a minority (about 11%) of the population with hypertension who also consume high sodium, and that such a policy might be harmful for those with a low sodium intake and lower blood pressures.

These provocative findings will be challenged. It will be argued that the method used to estimate urinary sodium excretion is not representative of 24-h urinary excretion. The counter viewpoint is that, whereas the technique is unsuitable for estimation of 24-h sodium excretion in individuals, it has been validated against 24-h urine collections in both healthy individuals and those with hypertension, and serves, therefore, as a valid measure of mean population sodium intake in epidemiological studies of association.[9] It will be contended that randomised controlled trials to determine the intake of sodium on outcome are simply not feasible, and that the benefits of a low-salt-for-all strategy outweigh any potentially harmful effects. However, without defining precisely the latter risk, this approach is inherently flawed in that the assumption on which the premise is based is scientifically incorrect. Although large prospective observational studies give supportive evidence, they do not prove causality and it is encouraging, therefore, to see that a pilot randomised controlled trial has been initiated (NCT02458248).

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In conclusion, we must acknowledge that given the dependency of so many physiological systems on the sodium cation, it should come as no surprise that a low-salt-for-all policy would benefit some and disadvantage others. So rather than allowing contrary evidence to dispel the positive efforts that have been made to reduce the salt content of foods, we must now direct our efforts to formulating a policy that will benefit the majority in society without comprising the minority.

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