Less Salt and Less Risk of Stroke
Further Support to Action

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See related article, p 1200.

High blood pressure is 1 of the major causes of morbidity, death, and disability worldwide, accounting for >60% of deaths from stroke. The risk of stroke increases with the levels of blood pressure. Blood pressure-lowering causes a reduction in risk within a few years, the gains being larger and faster in older people. Salt intake is 1 of the major determinants of blood pressure levels and a reduction in salt intake causes a dose-dependent reduction in blood pressure. This effect is seen in both sexes, all age and ethnic groups, and with all starting blood pressures. In populations, when salt intake is reduced, blood pressure in the community falls. It is therefore conceivable that if blood pressure falls, also stroke rates will fall. Given the complexities of controlling dietary salt supply and the need for long-term follow-up of vital outcomes, a randomized clinical trial of salt reduction on stroke outcomes in populations is not available and is not feasible. However, prospective longitudinal studies have been used to estimate the risk of the exposure to high levels of salt intake on the development of fatal and nonfatal strokes. This is similar to the evidence supporting public health policies on smoking, obesity, physical activity, and many forms of water sanitation.

A meta-analysis of prospective population studies (>150 000 participants and >5000 events) estimated a 23% difference in fatal and nonfatal stroke for a 5-g per day difference in salt intake (approximately 2000 mg of sodium) and a dose-response of a 6% increase in the risk of stroke for every 50 mmol (approximately 1150 mg) sodium per day, irrespective of the methods used to assess daily salt intake. Since then, a debate has started with the publication of new cohort studies and their controversial interpretations concerning the effect on coronary events.6,7

The prospective study by Gardener et al published in Stroke8 provides further support to the notion that high salt intake is a risk factor for stroke and that lower population dietary sodium goals will help reduce stroke risk. They examined >2600 men and women (mean age, 69 years) from multiethnic backgrounds, who took part in the Northern Manhattan Study, a prospective study with baseline assessment of diet through a food frequency questionnaire and 10-year follow-up of cause-specific fatal and nonfatal strokes and other vascular events. Both categorical and continuous analyses of dietary salt intake and stroke showed a statistically significant increased risk (hazard ratio, 2.59) in those reporting eating ≥4000 mg sodium per day (≥10 g salt) compared with those eating within the American Heart Association-recommended goals of ≤1500 mg per day (≤3.75 g salt; only 12% of the total population). Moreover, a 16% increased risk was detected for every 500-mg/d sodium increment (1.5 g salt). The use of a food frequency questionnaire will have not detected discretionary sources of salt, which in the US diet accounts for <20% of the total salt intake.

The present study is in agreement with the international recommendations. The following question now remains: how to reduce population salt intake to save lives?9

There is global consensus among policymakers for action to implement a population reduction in salt intake as an important strategy for the prevention of noncommunicable disease in the world.10 It is estimated that a 15% reduction in salt intake would avert 8.5 million deaths over 10 years worldwide, making it a preventive imperative. At the same time this policy satisfies stringent cost-effectiveness criteria. It is estimated that a mean population reduction of 3 g of salt per day (approximately 1200 mg of sodium) would result in an annual gain of 194 000 to 392 000 quality-adjusted life-years and savings of $10 billion to $24 billion in healthcare costs, representing a $6 to $12 return for each dollar spent. This is an economic imperative.

Finally, what would a program of policy changes look like? The details will vary according to localities, circumstances, resources, and more: communication, by establishing and evaluating public awareness campaigns, reformulation, by setting salt targets for reformulating existing food and producing new ones, monitoring, by measuring salt consumption in populations and content in food, and regulation. Any effective public health policy using a population approach is often dependent on the government actively using legislation or regulation.12 Previous examples include safe drinking water, effective sanitation, immunization, seatbelts, and smoke-free public places. We are now healthier and live much longer than a century ago. A well-grounded self-regulatory voluntary system has apparent benefits: it spares government resources, is less confrontational, more flexible, and quicker than government regulation.13 Indeed, softer voluntary codes of practice between governments and the food industry have been introduced in many countries.14
However, although less confrontational than regulation, self-regulation tends to be less effective than legislation. When promises are not fulfilled, harm occurs. For instance, the continuing and intense industry lobbying for the rejection of the “traffic light” proposal for food labeling at the European level or the attempts to derail nutritional public health initiatives indicate the unsurprising behavior of an industry focused on profit, not public health. It clearly demonstrates the predictable limitations of self-regulation. There is also a clear ethical justification for public health interventions to reduce salt consumption, because governments have a clear duty of care and because such an intervention would reduce inequalities.

In conclusion, the present study is another small tile of an impressive mosaic of evidence supporting a reduction of population salt intake as an urgent strategy for the global prevention of cardiovascular disease around the world.

Disclosures
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References
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