

## Salt and cardiovascular disease

Legislation to cut levels of salt in processed food is necessary and justified



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### RESEARCH, p 885

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**Competing interests:** None declared.

**Provenance and peer review:** Commissioned; not externally peer reviewed.

**BMJ 2007;334:859-60**

doi:10.1136/bmj.39175.364954.BE

Blood pressure is the most powerful predictor of stroke and other cardiovascular events. The importance of salt (sodium chloride) intake in determining blood pressure and the incidence of hypertension is well established. Furthermore, randomised controlled clinical trials of moderate reductions in salt intake show a dose dependent cause-effect relation and lack of a threshold effect within usual levels of salt intake in populations worldwide.<sup>1</sup> The effect is independent of age, sex, ethnic origin, baseline blood pressure, and body mass.

Prospective studies,<sup>2-5</sup> with one exception,<sup>6</sup> also indicate that higher salt intake predicts the incidence of cardiovascular events. While widespread support exists for reducing salt intake to prevent cardiovascular disease, the lack of large and long randomised trials on the effects of salt reduction on clinical outcomes has encouraged some people to argue against a policy of salt reduction in populations.<sup>6</sup>

In this week's *BMJ*, Cook and colleagues<sup>7</sup> report the long term effects of reduced dietary sodium on cardiovascular disease in people participating in the controlled randomised trials of hypertension prevention follow-up studies (TOHP I and II). More than 3000 participants without hypertension were randomised to a reduced sodium intake for 18 months (TOHP I) or 36-48 months (TOHP II), or to a control arm. The reductions in sodium intake were 44 mmol/day and 33 mmol/day (equivalent to ~2.6 g and ~2.0 g of salt), respectively. The results show that people originally allocated to either sodium reduction group had a 30% lower incidence of cardiovascular events in the next 10-15 years, irrespective of sex, ethnic origin, age, body mass, and blood pressure. The benefits exceed those estimated by a recent meta-analysis.<sup>8</sup> Cook and colleagues' study is the first to report a beneficial effect of dietary salt reduction on cardiovascular outcomes based on randomised trial data.

The study strengthens the support for dietary recommendations for lower salt intake to prevent cardiovascular disease in the general population. In 1985, the World Health Organization recommended that the average salt intake should be reduced to 5 g per day or less. However, few countries have policies for targeted reduction in salt intake.

Differences exist between developed and developing countries. In Westernised countries, people derive salt mostly from bread and processed food and only a small proportion comes from discretionary use (up to 20%). A

population wide policy of salt reduction in developed countries can only be implemented with the collaboration of the food industry. Over the years, however, the need to sustain a profitable market has led to opposition from the food industry or slow progress.<sup>9</sup>

In England and Wales some progress has been made, but levels of salt intake are still far from the government's recommended 6 g of salt per day. Future options are to do nothing, to establish voluntary target levels of salt for a wide range of foods, or to legislate so that the food industry has to comply. Given the inertia of the past 20 years, the first option would not contribute to progress. The "voluntary" option would support existing work, but it is unlikely to achieve the set targets. The recent position of the industry in rejecting the "traffic light" proposal for labelling, whereby highly salted foods would carry a red alert warning, is one measure of the gap still remaining. The legislation option would require the food industry to reduce the salt content of processed food to within set levels. The experience in Finland suggests that legislation has added value to the previous option and at this stage is necessary and justified.

Conversely, in many developing countries, like those of sub-Saharan Africa, where the main source of salt is still discretionary, community based and context specific initiatives can be effective and should be pursued,<sup>10</sup> given the increasing burden of cardiovascular disease related to hypertension.

Without considerably modifying the environment by allowing greater availability of low salt foods, people in developed countries will find it difficult to exercise their "choice" when trying to reduce dietary salt. Doctors and health professionals have long used dietary counselling to deliver non-pharmacological management of hypertension. Advising patients to reduce salt intake with a lifestyle package quickly delivered in a busy primary care setting is ineffective, however.<sup>11</sup> A baseline assessment of salt intake (through a 24 h urinary collection for the measurement of sodium) is not part of the UK's National Service Framework requirements and is not included in the Quality and Outcome Framework. The current system is therefore unlikely to make health professionals and consumers more aware of how much salt people eat or to increase motivation and knowledge on how to reduce it. In a climate of scarce healthcare resources, one of the most cost effective ways to reduce the burden of cardiovascular disease is being overlooked.<sup>12</sup> And yet the evidence gets stronger.

- 1 He FJ, MacGregor GA. How far should salt intake be reduced? *Hypertension* 2003;42:1093-9.
- 2 Sasaki S, Zhang X-H, Kesteloot H. Dietary sodium, potassium, saturated fat, alcohol and stroke mortality. *Stroke* 1995;26:783-9.
- 3 He J, Ogden LG, Vupputuri S, Bazzano LA, Loria C, Whelton PK. Dietary sodium intake and subsequent risk of cardiovascular disease in overweight adults. *JAMA* 1999;282:2027-34.
- 4 Tuomilehto J, Jousilahti P, Rastenyte D, Moltchanov V, Tanskanen A, Pietinen P, et al. Urinary sodium excretion and cardiovascular mortality in Finland: a prospective study. *Lancet* 2001;357:848-51.
- 5 Nagata C, Takatsuka N, Shimizu N, Shimizu H. Sodium intake and risk of death from stroke in Japanese men and women. *Stroke* 2004;35:1543-7.
- 6 Cohen HW, Hailpern SM, Fang J, Alderman MH. Sodium intake and mortality in the NHANES II follow-up study. *Am J Med* 2006;119:275.e7-14.
- 7 Cook NR, Cutler JA, Obarzanek E, Buring JE, Rexrode KM, Kumanyika SK, et al. Long term effects of dietary sodium reduction on cardiovascular disease outcomes: observational follow-up of trials of hypertension prevention. *BMJ* 2007 doi: 10.1136/bmj.39147.604896.55.
- 8 He FJ, MacGregor GA. Effect of modest salt reduction on blood pressure: a meta-analysis of randomized trials. Implications for public health. *J Hum Hypertens* 2002;16:761-70.
- 9 Karppanen H, Mervaala E. Sodium intake and hypertension. *Prog Cardiovasc Dis* 2006;49:59-75.
- 10 Cappuccio FP, Kerry SM, Micah FB, Plange-Rhule J, Eastwood JB. A community programme to reduce salt intake and blood pressure in Ghana. *BMC Public Health* 2006;6:13.
- 11 Little P, Kelly J, Barnett J, Doward M, Margetts B, Warm D. Randomised controlled factorial trial of dietary advice for patients with a single high blood pressure reading in primary care. *BMJ* 2004;328:1054-7.
- 12 Selmer RM, Kristiansen IS, Haglerod A, Graff-Iversen S, Larsen HK, Meyer HE, et al. Cost and health consequences of reducing the population intake of salt. *J Epidemiol Community Health* 2000;54:697-702.

## Postmenopausal hormone therapy

Symptoms should be treated with lowest effective dose of hormone therapy for the shortest time possible

### SHORT CUTS, p 872

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**Competing interests:** DG has received research support from Berlex, Bionovo, Eli Lilly, and Pfizer. EB-C has received research support from NIH, Amgen, Lilly, Merck, Wyeth, Proctor and Gamble, Roche, and GlaxoSmithKline.

**Provenance and peer review:** Commissioned; not externally peer reviewed.

**BMJ 2007;334:860-1**

doi:10.1136/bmj.39185.380405.BE

In March 2007, the North American Menopause Society (NAMS) published an updated position statement on the use of hormone therapy in postmenopausal women.<sup>1</sup> NAMS recommends hormone therapy, which is a highly effective treatment for hot flushes and vaginal atrophy,<sup>2</sup> as first line treatment for women with moderate to severe symptoms. It is also effective for preventing osteoporotic fractures,<sup>3,4</sup> but NAMS recommends that hormone therapy for this purpose should be weighed against potential harm and that other approved preventive treatments such as bisphosphonates should be considered. These recommendations are clear, simple, and based on solid evidence from many randomised controlled trials.

However, NAMS recommendations are less clear in several other areas. For example, after clearly stating that hormone therapy increases risk of venous thromboembolic events and stroke, no advice is provided about how clinicians and patients should use this information. Similarly, NAMS notes that risk of breast cancer is increased in women who use oestrogen plus progestin for five years or more, but no recommendation is given about its use in women at high risk of breast cancer. The statement also notes that treatment with hormone therapy in women over 65 years increases risk for dementia,<sup>5,6</sup> and that no evidence is available regarding effects on dementia from clinical trials in younger women, but there is no clear statement that hormone therapy should not be used to prevent dementia.

NAMS published a position statement on use of postmenopausal hormone therapy in 2004, and since then no large randomised trials have been published that would require revision of guidelines. What then has changed since the earlier statement? The main changes in the new position statement reflect the belief of NAMS panelists that, if used during or shortly after the menopause, hormone therapy may not increase risk of coronary heart disease. Evidence to support

this “timing hypothesis” comes from studies of castrated animals and post hoc analyses of observational studies, but primarily rests on subgroup analyses of data from the two women’s health initiative randomised trials.<sup>7</sup> Analyses that pooled data from the women’s health initiative trial of oestrogen alone and of oestrogen plus progestin show no clear difference in risk for coronary heart disease associated with use of hormone therapy in women in their 50s compared with older women. However, women treated with hormone therapy within 10 years of the menopause seemed to have a reduced risk of heart disease (hazard ratio 0.76, 95% confidence interval 0.50 to 1.16), whereas those who had undergone the menopause more than 20 years ago had an increased risk (1.28, 1.03 to 1.58; P value for interaction 0.02). These data are not entirely convincing, as about 137 comparisons were performed, and several statistically significant findings would be expected to occur by chance. The “timing hypothesis” will probably



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