Reduced dietary salt for the prevention of cardiovascular disease

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ABSTRACT

BACKGROUND: This is an update of a Cochrane review that was first published in 2011 of the effects of reducing dietary salt intake, through advice to reduce salt intake or low-sodium salt substitution, on mortality and cardiovascular events.

OBJECTIVES: 1. To assess the long-term effects of advice and salt substitution, aimed at reducing dietary salt, on mortality and cardiovascular morbidity. 2. To investigate whether a reduction in blood pressure is an explanatory factor in the effect of such dietary interventions on mortality and cardiovascular outcomes.

METHODS:

Search methods: We updated the searches of CENTRAL (2013, Issue 4), MEDLINE (OVID, 1946 to April week 3 2013), EMBASE (OVID, 1947 to 30 April 2013) and CINAHL (EBSCO, inception to 1 April 2013) and last ran these on 1 May 2013. We also checked the references of included studies and reviews. We applied no language restrictions.

Selection criteria: Trials fulfilled the following criteria: (1) randomised, with follow-up of at least six months, (2) the intervention was reduced dietary salt (through advice to reduce salt intake or low-sodium salt substitution), (3) participants were adults and (4) mortality or cardiovascular morbidity data were available. Two review authors independently assessed whether studies met these criteria.

Data collection and analysis: A single author extracted data and assessed study validity, and a second author checked this. We contacted trial authors where possible to obtain missing information. We extracted events and calculated risk ratios (RRs) and 95% confidence intervals (CIs).

MAIN RESULTS: Eight studies met the inclusion criteria: three in normotensives (n = 3518) and five in hypertensives or mixed populations of normo- and hypertensives (n = 3766). End of trial follow-up ranged from six to 36 months and the longest observational follow-up (after trial end) was 12.7 years.

The risk ratios (RR) for all-cause mortality in normotensives were imprecise and showed no evidence of reduction (end of trial RR 0.67, 95% confidence interval (Cl) 0.40 to 1.12, 60 deaths; longest follow-up RR 0.90, 95% Cl 0.58 to 1.40, 79 deaths n = 3518) or in hypertensives (end of trial RR 1.00, 95% Cl 0.86 to 1.15, 565 deaths; longest follow-up RR 0.99, 95% Cl 0.87 to 1.14, 674 deaths n = 3085).

There was weak evidence of benefit for cardiovascular mortality (hypertensives: end of trial RR 0.67, 95% CI 0.45 to 1.01, 106 events n = 2656) and for cardiovascular events (hypertensives: end of trial RR 0.76, 95% CI 0.57 to 1.01, 194 events, four studies, n = 3397; normotensives: at longest follow-up RR 0.71, 95% CI 0.42 to 1.20, 200 events; hypertensives: RR 0.77, 95% CI 0.57 to 1.02, 192 events; pooled analysis of six trials RR 0.77, 95% Cl 0.63 to 0.95, n = 5912). These findings were driven by one trial among retirement home residents that reduced salt intake in the kitchens of the homes, thereby not requiring individual behaviour change.

Advice to reduce salt showed small reductions in systolic blood pressure (mean difference (MD) -1.15 mmHg, 95% CI -2.32 to 0.02 n = 2079) and diastolic blood pressure (MD -0.80 mmHg, 95% CI -1.37 to -0.23 n = 2079) in normotensives and greater reductions in systolic blood pressure in hypertensives (MD -4.14 mmHg, 95% CI -5.84 to -2.43 n = 675), but no difference in diastolic blood pressure (MD -3.74 mmHg, 95% CI -8.41 to 0.93 n = 675).

Overall many of the trials failed to report sufficient detail to assess their potential risk of bias. Health-related quality of life was assessed in one trial in normotensives, which reported significant improvements in well-being but no data were presented.

AUTHORS' CONCLUSIONS: Despite collating more event data than previous systematic reviews of randomised controlled trials, there is insufficient power to confirm clinically important effects of dietary advice and salt substitution on cardiovascular mortality in normotensive or hypertensive populations. Our estimates of the clinical benefits from advice to reduce dietary salt are imprecise, but are larger than would be predicted from the small blood pressure reductions achieved. Further well-powered studies would be needed to obtain more precise estimates. Our findings do not support individual dietary advice as a means of restricting salt intake. It is possible that alternative strategies that do not require individual behaviour change may be effective and merit further trials.

The abstract of this review is available from: http://onlinelibrary.wiley. com/doi/10.1002/14651858.CD009217.pub3/abstract

REFERENCE

 Adler AJ, Taylor F, Martin N, Gottlieb S, Taylor RS, Ebrahim S. Reduced dietary salt for the prevention of cardiovascular disease. Cochrane Database Syst Rev. 2014;12:CD009217.

COMMENTS

Dietary sodium restriction and cardiovascular disease prevention: should we quit?

High salt consumption (> 2.0 g/day) is possibly responsible for one in every ten deaths attributed to cardiovascular disease worldwide.¹ There is a log-linear association between sodium intake and blood pressure values, and hypertension is the most important risk factor for cardiovascular diseases (CVDs), because of its elevated prevalence, underdiagnosis and undertreatment.² The question that ensues is how efficacious dietary sodium restriction is in relation to blood pressure control and consequent prevention of cardiovascular events. Sodium restriction is a lifestyle change that is universally proposed for hypertensive individuals, in addition to weight loss, physical exercise and cessation of smoking, with the ultimate goal of preventing CVDs and prolonging life.

However, recent evidence from a Cochrane systematic review has cast doubts on the efficacy of dietary sodium restriction.³ The authors evaluated eight studies: three on normotensive populations (n = 3,518) and five on hypertensive or mixed populations of normotensive and hypertensive individuals (n = 3,766). The patients were followed up for periods ranging from six months to 12.7 years. There were modest changes in systolic blood pressure: mean differences of -1.15 mmHg and -4.14 mmHg, respectively among normotensive and hypertensive individuals. However, although there was a change in diastolic blood pressure of -0.8 mmHg among normotensive individuals, there were non-significant reductions

among hypertensive individuals. There were non-significant reductions in cardiovascular mortality among both normotensive and hypertensive individuals, and CVD events were found to be reduced only when the studies were pooled together (-23%).

This review is limited by the lack of an objective measurement for sodium intake, such as urinary sodium excretion, for testing treatment compliance. However, it suggests that behavioral changes are hard to attain and that their effects may be limited. The results shown here can be compared with those from the randomized Look AHEAD study, which failed to show any benefits from lifestyle changes among diabetic patients, simply because they did not follow the recommendations over the long term.⁴ It can be concluded that better ways of implementing lifestyle changes need to be developed, including in relation to dietary sodium restriction. The results from this review do not mean that dietary sodium restriction should simply no longer be recommended and that reliance should be placed only on medications for attaining CVD reduction.

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