Review: reduced sodium intake results in small reductions in blood pressure among people with hypertension


Question
What is the effect of reduced sodium intake on systolic blood pressure (SBP) and diastolic blood pressure (DBP)?

Data sources
English language studies were identified using Medline (1966 to December 1997) with combinations of the search terms salt or sodium; restriction or dietary; blood pressure or hypertension; and randomised or randomized or random. 4 previous meta-analyses and the reference lists of retrieved citations were also reviewed.

Study selection
Studies were included if they randomly allocated patients to either a low or high sodium diet; sodium intake was assessed using 24 hour urinary sodium excretion; no confounding occurred; effects on SBP, DBP, and mean blood pressure were reported; and mean age of patients was >15 years.

Data extraction
Data were extracted on sample size, mean age and sex of participants, duration of intervention, sodium reduction, and outcomes. Main outcomes were SBP and DBP; other outcomes included hormone concentrations, blood lipid concentrations, and body weight.

Main results
83 references including 114 studies met the inclusion criteria. Separate meta-analyses were done for 58 studies involving people with hypertension (n = 2161, median mean age 49 y, median study duration 28 d) and 56 studies involving people with normal blood pressure (n = 2581, median mean age 27 y, median study duration 8 d). In trials of people with hypertension, a mean sodium reduction of 118 mmol/24 hours reduced both SBP (3.9 mm Hg, 95% CI 3.0 to 4.8) and DBP (1.9 mm Hg, CI 1.3 to 2.5). In trials of people with normal blood pressure, a mean sodium reduction of 100 mmol/24 hours reduced SBP (1.2 mm Hg, CI 0.6 to 1.8) but not DBP.

Because the authors did several analyses, they set a conservative level of significance (p < 0.005). Meta-analyses of the effects of sodium restriction on hormone concentrations and lipid concentrations showed that compared with a high sodium diet, a low sodium diet increased renin (53 studies, 1110 patients, p < 0.001), aldosterone (38 studies, 840 patients, p < 0.001), noradrenaline (29 studies, 700 patients, p < 0.001), cholesterol (19 studies, 653 patients, p < 0.001), and low density lipoprotein concentrations (13 studies, 517 patients, p = 0.003), but did not affect adrenaline (10 studies, 207 patients, p = 0.02), triglyceride (14 studies, 565 patients, p = 0.05) or high density lipoprotein (15 studies, 573 patients, p = 0.35) concentrations.

Conclusion
Reduced sodium intake results in small reductions in systolic and diastolic blood pressures in people with hypertension, and a small reduction in systolic blood pressure in people with normal blood pressure.


treatment.

Commentary

The role of salt in hypertension has been the subject of debate for decades. Graudal et al present a robust systematic review that attempts to control for confounding factors and potential biases in the analysis. The discussion provides well considered arguments illuminating both sides of the salt debate. The results about the effect of salt on blood pressure update the findings of a 1996 meta-analysis, and, additionally, identify the possibility that there may be adverse effects related to a low salt intake.

The limitations of the review include the short average length of the studies found, which was 28 days for the hypertensive studies and 8 days for the normotensive studies. It is unknown whether the effects observed would be present, absent, increased, or decreased with an extended period of time on a low salt diet (ie, several years or even decades). This is a major concern as these are the timespans over which people are being asked to lower their salt intake. Another issue not frequently addressed in such studies is that of the tolerability of such a diet in most cultures and the long term adherence to it. These issues could not have been addressed because of the short length of the studies found.

The message to practitioners is that lowering salt intake lowers systolic blood pressure, more so in people with hypertension. Trials that measured such variables indicate that lowering salt intake may adversely affect the renin-angiotensin-aldosterone system and lipid profiles through a compensatory mechanism. Professionals should be cautious therefore in advising a strict low sodium diet in either population until the potential adverse hormonal and lipid effects of such a dietary regimen are more fully characterised. Furthermore, even if sodium restriction does lower blood pressure, we do not know the extent to which such changes actually affect morbidity and mortality outcomes. The authors of this study do not support a general recommendation to reduce sodium intake, but suggest that sodium reduction be used only as a supplementary treatment for hypertension.

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