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COCHRANE COMMENTARIES

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Altered dietary salt for people with CKD

What is this review about?

This review evaluated the benefits and harms of altering dietary salt intake in people with chronic kidney disease (CKD). We included randomized controlled trials (RCTs) and quasi-RCTs that measured the effect of a low compared with a high salt intake in people with any stage of CKD.

What are the key findings?

There were no studies examining the effect of reducing salt in CKD on outcomes such as mortality, cardiovascular events or progression to end-stage kidney disease, therefore we reviewed the evidence examining the effect of altering salt intake on surrogate outcomes for cardiovascular disease or kidney disease progression.

Reducing salt intake significantly reduced systolic and diastolic blood pressure (Fig. 1), with a mean reduction in systolic/diastolic blood pressure of 8.8/3.7 mm Hg (95% confidence interval (CI) 6.2–11.3/2.3–5.1) when urinary sodium was reduced by 106 mmol per day (95% CI 93–119) from the high salt to the low salt groups (eight studies, 258 participants). Salt restriction significantly reduced urinary protein or albumin excretion in all four studies that reported these outcomes, with median reduction in 24 h urinary protein excretion of 34% (these data were not meta-analysed). Antihypertensive medication dosage was reduced with a low-salt diet (two studies, 52 participants; risk ratio 5.5, 95% CI 1.3–24). Salt restriction increased plasma renin activity (two studies, 71 participants; mean difference (MD) 1.1 ng/mL/h, 95% CI 0.5–1.7) and serum aldosterone (two studies, 71 participants; MD 6.2 ng/dL, 95% CI 3.8–8.6). Reducing salt did not change eGFR (two studies, 68 participants; MD −1.1 mL/min/1.73 m^2, 95% CI −4.4 to 2.1), creatinine clearance (three studies, 85 participants; MD −4.6 mL/min, 95% CI −11.8 to 2.6), serum creatinine (five studies, 151 participants: MD 5.1 µmol/L, 95% CI −9.0 to 19.3) or bodyweight (five studies, 139 participants: MD −1.5 kg; 95% CI −4.6 to 1.6).

What are the findings based on?

Eight studies (three parallel and five cross-over; 258 participants) reported the effect of altering salt intake in people

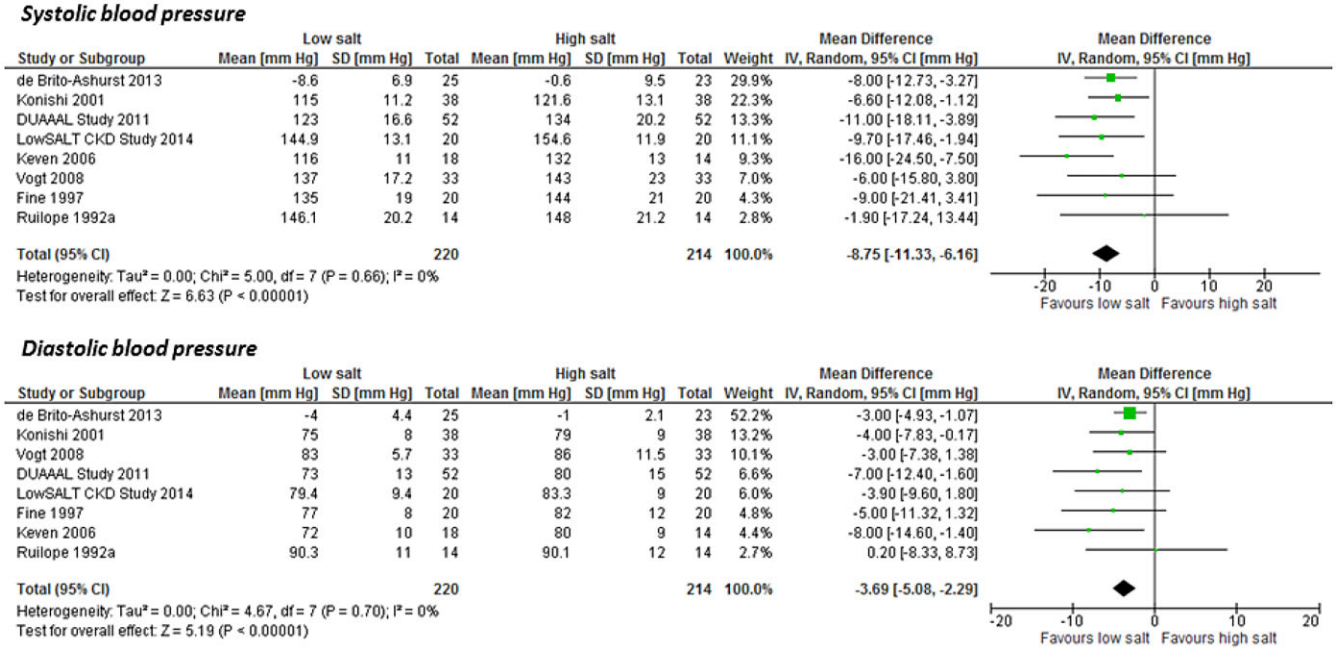

Fig. 1 Change in blood pressure with altering salt intake in people with CKD

with CKD. Median urinary sodium excretion was 203.5 (range 166–246) mmol/day in the high salt period and 98 (range 48–155) mmol/day in the low-salt period. Six studies (206 participants) were conducted in people with stage 3–4 CKD, one post-transplant and one in people on peritoneal dialysis. There were no studies in haemodialysis patients. The limited number of studies in end-stage kidney disease made it difficult to compare the effects of reducing salt intake across stages of CKD. Study duration was relatively short, ranging from 1 to 26 weeks (median 6 weeks). Measurement of potential dietary confounders such as potassium intake, protein intake or change in bodyweight because of muscle or fat loss was a limitation of most studies. Risk of bias was low or unclear for most studies. One cross-over study had the potential for high carryover effect; three had high risk of bias from potential confounders (change of medication or diet) and two studies were industry funded.

Implications for practice

- In people with CKD, reducing salt intake considerably reduces blood pressure
- There is consistent evidence that dietary salt restriction reduces proteinuria in people with CKD
- In the short term, salt restriction does not change eGFR
- Impact of salt restriction on mortality, incidence of CVD or progression to ESKD is unknown.

Clinical perspective

The relationship between salt intake and BP, as well as other cardiovascular risk factors, has been studied extensively since the 1950s. In this time, several meta-analyses have been conducted, however these mostly excluded people with kidney disease. As salt-sensitivity is thought to be at least partially dependent on kidney function, it is likely that people with kidney disease are more vulnerable to the detrimental effects of excess salt intake. This was evident when comparing our results to previous meta-analyses in those without CKD. We found a reduction in blood pressure of 9/4 mm Hg with salt reduction of approximately 100 mmol (2300 mg per day) in people with CKD whereas reduction of 5–6/2–3 mm Hg are commonly reported in meta-analyses of studies in people without kidney disease. In addition to BP reductions, salt restriction consistently reduced proteinuria with median reduction of 34%. If these reductions were maintained long term, this would contribute to clinically significant reductions in cardiovascular events and progression to ESKD. Research to determine whether these benefits are sustained with long-term adherence to a low salt diet is needed.

Reducing salt intake is likely to be a cost-effective and low-risk strategy for reducing cardiovascular risk and risk of worsening renal function. Current evidence-based clinical guidelines recommend target of less than 6 g of salt (100 mMol; 2300 mg sodium) per day for people with CKD, although most people consume far above this and achieving long-term adherence to salt restriction can be challenging. Referral to an accredited dietician who can provide individualized strategies to reduce salt intake should be considered.

McMahon EJ, Campbell KL, Bauer JD, Mudge DW. Altered dietary salt intake for people with chronic kidney disease. *Cochrane Database of Systematic Reviews* 2015, Issue 2. Art. No.: CD010070. DOI: 10.1002/14651858.CD010070.pub2.