Effects of oral potassium on blood pressure: meta-analysis of randomized controlled clinical trials


Authors' objectives
To assess the effects of supplementation with oral potassium on blood-pressure (BP) in humans.

Searching
MEDLINE was searched using the terms 'blood pressure', 'dietary potassium', 'potassium' and 'potassium chloride'. Bibliographies of original articles and reviews, and the authors' own reference files were also examined. Only English-language articles published before 1995 were considered.

Study selection
Study designs of evaluations included in the review
Randomised controlled trials (RCTs) of either parallel or crossover design were included. The duration of the studies varied from 4 days to 3 years.

Specific interventions included in the review
Potassium supplementation administered as a pill (in the form of potassium chloride, potassium citrate and bicarbonate) or as part of the diet. Dosage of potassium prescribed in the intervention arm was at least 60 mmol/day in all but 2 trials, and at least 100 mmol/day in 10 trials (median, 75 mmol/day). The intervention arm was compared to a control group of either placebo, lower dose of potassium, or no supplementation. Antihypertensive medications were administered concurrently in 4 of the trials.

Participants included in the review
Both hypertensive and normotensive participants (aged 19 years) were included in the review.

Outcomes assessed in the review
The net change from pre-treatment to end of follow-up in urinary excretion of sodium and potassium, body weight, and BP (systolic and diastolic) for treatment and control conditions. The mean 24-hour urinary sodium excretion during follow-up, recorded at the trial's end.

How were decisions on the relevance of primary studies made?
The primary studies were assessed independently for relevance by two of the review's authors. Areas of disagreement or uncertainty were adjudicated by other authors.

Assessment of study quality
No formal validity assessment was carried out, although study design details were recorded during the data extraction phase; these included whether the trial was of parallel, crossover or open design, single-blind or double-blind. Details of the study design were recorded independently by two of the authors who were blinded to each other's coding. This process was carried out during the data extraction phase.

Data extraction
The data were extracted independently by two of the authors who were blinded to each other's coding but not to treatment group.

Methods of synthesis
How were the studies combined?
For each trial, the net changes in BP, urinary electrolyte excretion and weight from baseline to follow-up were calculated. To calculate pooled effect size, each study was assigned weights consisting of the reciprocal of the total variance for BP change. Estimates of the mean effect of potassium supplementation on BP and corresponding 95% confidence intervals (CIs) were calculated by means of fixed-effect and random-effects models. To explore the influence of covariables on net changes in BP, a series of prestated subgroup analyses were performed. For each subgroup, pooled effects were calculated by the random-effects model and statistical significance was tested by analysis of variance. Univariate and multivariate linear regression models were used to explore the influence of a series of prestated covariables on net BP change.

How were differences between studies investigated?
The homogeneity of the effect size across studies was tested by Q-statistics.

Results of the review
Thirty-three RCTs (1,560 hypertensive and 1,005 normotensive participants) were included: 3 of crossover open design, 2 crossover single-blind, 16 crossover double-blind, 4 parallel open, 1 parallel single, and 7 parallel double-blind.

Due to significant variation in effect size across the 33 trials, only results from the random-effects model are presented.

Overall pooled estimates of effect of potassium supplementation on systolic and diastolic BP were -4.44 mmHg (95% CI: -2.53, -6.36, p<0.001) and -2.45 mmHg (95% CI: -0.74, -4.16, p<0.01), respectively. Exclusion of an outlier reduced overall pooled effect size estimates to -3.11 mmHg (95% CI: -1.91, -4.31, p<0.001) for systolic BP and -1.97 mmHg (95% CI: -0.52, -3.42, p<0.01) for diastolic BP. Effects of treatment appeared to be enhanced in studies in which participants were concurrently exposed to a high intake of sodium.

Linear regression analysis identified a significant, independent positive relationship between average 24-hour urinary sodium excretion during follow-up in each trial and corresponding net reduction in systolic (p=0.004) and diastolic (p=0.003) BP.

Authors' conclusions
The results support the premise that low potassium intake may play an important part in the genesis of high BP. Increased potassium intake should be considered as a recommendation for prevention and treatment of hypertension, especially in those who are unable to reduce their intake of sodium.

CRD commentary
The restriction of the review to English language article may have been a cause of publication bias, although the authors examine this within the review using a funnel plot analysis; no evidence was found to suggest that publication bias was a factor in the meta-analysis. The authors present a thorough and well-documented review.

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