

A low-cost salt substitute reduces blood pressure in high-risk individuals

Decreasing dietary salt intake is potentially a low-cost means of reducing the burden of blood-pressure-related disease. A recent paper reports on the blood pressure effects of using a salt substitute in rural Chinese individuals at high risk for vascular disease.

In a randomized double-blind trial, 608 individuals were provided with either normal salt (100% NaCl) or commercially available salt substitute (65% NaCl, 25% KCl, 10% MgSO₄) to cover all household uses. Over the 12-month follow-up period, systolic blood pressure (equivalent at baseline) was a mean of 3.7 mmHg lower in the salt-substitute group than in the normal-salt group ($P < 0.001$). The magnitude of this reduction seemed to increase with time ($P = 0.001$; maximal difference: 5.4 mmHg at 12 months). No differences in diastolic blood pressure were observed between the two groups at any time. First morning urine sodium concentrations were similar in the two groups at 6 and 12 months, but urine potassium concentrations were significantly higher in the salt-substitute group at both time points (by 6.8 mmol/l and 7.2 mmol/l, respectively). No incidences of severe hyperkalemia were recorded, and the rates of serious adverse events were similar in the two groups.

The study group concluded that salt substitution resulted in a sustained cost-effective clinically relevant reduction in systolic blood pressure. Salt substitution might be appropriate for high-risk individuals in developing countries, where the majority of dietary salt intake does not come from processed foods.

Original article The China Salt Substitute Study Collaborative Group (2007) Salt substitution: a low-cost strategy for blood pressure control among rural Chinese: a randomized, controlled trial. *J Hypertens* 25: 2011–2018

Restoring nightly blood pressure dip by altering timing of antihypertensive therapy in CKD

Individuals whose blood pressure does not drop by at least 10% at night ('nondippers') are at increased risk of cardiovascular morbidity and mortality. Nondipping status is common

among patients with chronic kidney disease (CKD) and is associated with an increased risk of end-stage renal disease. Minutolo *et al.* investigated whether altering the timing of administration of antihypertensive drugs can restore the normal circadian rhythm of blood pressure in CKD patients.

The study enrolled 32 outpatients with CKD who had an estimated glomerular filtration rate < 90 ml/min/1.73 m², nondipper status (night:day ratio of mean ambulatory blood pressure [ABP] > 0.9) and a mean ABP of $< 135/85$ mmHg. Patients' antihypertensive treatment regimens were modified by a shift in the dosing of one antihypertensive drug (not a diuretic) from the morning to the evening.

After 8 weeks of the modified treatment regimen, 28 of 32 patients (87.5%) had achieved dipping status. The average night:day ratio of ABP decreased significantly from baseline to 8 weeks after the drug shift (from 0.95 ± 0.04 to 0.87 ± 0.04 ; $P < 0.001$). Mean systolic and mean diastolic office blood pressures in the morning also dropped markedly over 8 weeks ($P = 0.02$ for both parameters). Mean urinary protein excretion decreased significantly from 271 ± 284 mg/day at baseline to 182 ± 225 mg/day 8 weeks after the drug shift ($P < 0.001$). The mean number of antihypertensive drugs taken per patient during the study was 2.4 ± 1.4 . No relationship was found between the number or type of drugs administered and the change in blood pressure following the drug shift.

Original article Minutolo R *et al.* (2007) Changing the timing of antihypertensive therapy to reduce nocturnal blood pressure in CKD: an 8-week uncontrolled trial. *Am J Kidney Dis* 50: 908–917

Coenzyme Q₁₀ deficiency with renal involvement: a newly characterized disorder

Primary coenzyme Q₁₀ (CoQ₁₀, or ubiquinone) is vital for mitochondrial energy production. Deficiency of this enzyme normally manifests as neurological and muscular symptoms. Diomedi-Camassei and colleagues describe the first four cases of primary CoQ₁₀ deficiency with renal involvement, linking the disorder to novel mutations of the COQ2 gene that encodes the para-hydroxybenzoate–polyprenyltransferase enzyme of the CoQ₁₀ synthesis pathway.