

Potassium citrate prevents increased urine calcium excretion and bone resorption induced by a high sodium chloride diet.

Sellmeyer DE, Schloetter M, Sebastian A.

The amount of sodium chloride in the diet of industrialized nations far exceeds physiological requirements. The impact of abundant dietary salt on skeletal health has yet to be established, but is potentially detrimental through increased urinary calcium losses. We examined the effect of increased dietary sodium chloride on urine calcium excretion and bone turnover markers in postmenopausal women and, further, whether potassium citrate attenuates the effects of increased dietary salt. Postmenopausal women (n = 60) were adapted to a low-salt (87 mmol/d sodium) diet for 3 wk, then randomized to a high-salt (225 mmol/d sodium) diet plus potassium citrate (90 mmol/d) or a high-salt diet plus placebo for 4 wk. Urine calcium, urine N-telopeptide, urine cAMP, serum osteocalcin, and fasting serum PTH were measured at the end of the low- and high-salt diets. On the high salt plus placebo diet, urine calcium increased 42 +/- 12 mg/d (mean +/- SEM), but decreased 8 +/- 14 mg/d in the high salt plus potassium citrate group (P = 0.008, potassium citrate vs. placebo, unpaired t test). N-telopeptide increased 6.4 +/- 1.4 nanomoles bone collagen equivalents per millimole creatinine in the high salt plus placebo group and 2.0 +/- 1.7 nanomoles bone collagen equivalents per millimole creatinine in the high salt plus potassium citrate group (P < 0.05, potassium citrate vs. placebo, unpaired t test). Osteocalcin, PTH, and cAMP were not significantly altered. The addition of oral potassium citrate to a high-salt diet prevented the increased excretion of urine calcium and the bone resorption marker caused by a high salt intake. Increased intake of dietary sources of potassium alkaline salts, namely fruit and vegetables, may be beneficial for postmenopausal women at risk for osteoporosis, particularly those consuming a diet generous in sodium chloride.