Bone loss because of high sodium intake: Is there a connection to the acid-base balance?

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High dietary sodium intake is considered as a risk factor for osteoporosis. We examined the effect of increased dietary sodium on bone metabolism and acid-base balance in nine healthy male test subjects (mean age: 26 ± 1 years; body weight: 71.5 ± 1.3 kg). They received an individually tailored, exactly controlled nutrient intake. Only sodium intake was altered by variations in the Na^+- amount of the diet. During the first 6-day period they received a low Na^+ intake, 50 mmol Na^+/d (phase 1), during the following 6-day period 200 mmol Na^+/d (phase 2). The next 10-day period they received 550 mmol Na^+/d (phase 3) and in the following 6-day period we reduced the sodium intake to 50 mmol Na^+/d (phase 4) again. The bone resorption markers (C- and N- terminal telopeptide of type I collagen (CTX, NTX)) were measured in all 24-hour urine collections. The fasting morning blood was analyzed for the bone formation markers, bone specific alkaline phosphatase (bAP) and N-terminal propeptide of type I prokollagen (PINP). Parameters of the acid-base balance (pH, bicarbonate ($HCO3^-$) and base excess (BE)) were analyzed in the capillary blood from the fingertip two times in each study phase.

NTX increased significantly from phase 1 to 2 (NTX: $p = 0.04$), both of the bone resorption markers increased from phase 1 to 3 (CTX: $p < 0.001$, NTX: $p = 0.005$) and from phase 2 to 3 (CTX: $p = 0.01$; NTX: $p = 0.004$). The bone formation markers bAP and PINP remained unchanged in these study phases (bAP: $p = 0.47$, PINP: $p = 0.84$). Between low and very high sodium intake there was a significant fall in serum pH level ($p = 0.04$). $HCO3^-$ and BE supported these changes by showing a significant fall from phase 1 to 3 (both: $p < 0.001$) and also from phase 2 to 3 ($HCO3^-$: $p = 0.003$, BE: $p = 0.02$). Nearly all bone resorption markers and acid-base parameters reached their baseline level in phase 4. We conclude that high sodium intake induces a low grade metabolic acidosis and thereby causes bone resorption.