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ORIGINAL COMMUNICATION

A 7-week reduction in salt intake does not contribute to markers of bone metabolism in young healthy subjects

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Background: Sodium intake increases urinary calcium excretion and may thus lead to negative calcium balance and bone loss.
Objective: We hypothesised that reducing sodium intake would reduce urinary calcium excretion and have a beneficial influence in bone metabolism.
Design: A total of 29 subjects, 14 males and 15 females, were divided into two study groups. One group (low-sodium group (LS)) reduced sodium intake for 7 weeks by substituting low- salt alternatives for the most important dietary sources of sodium. The other group, serving as a control group (C), was given the same food items in the form of normally salted alternatives. Fasting serum samples as well as 24-h urine samples were obtained in the beginning and at the end of the study. Urinary sodium, urinary calcium, urinary creatinine, serum calcium, serum phosphate, serum creatinine, serum parathyroid hormone (s-PTH), serum C-terminal telopeptides of Type-I collagen and serum bone alkaline phosphatase (s-B-ALP) were analysed.
Results: The LS group showed a significant decline ($P=0.001$) in urinary sodium/creatinine ratio without a significant effect on urinary calcium/creatinine ratio. In the LS group, s-PTH increased ($P=0.03$). The C group showed an increase in s-PTH ($P=0.05$) and in s-B-ALP, but no differences were observed between the study groups in the changes of serum markers of calcium and bone metabolism.
Conclusions: We have shown that reducing the sodium intake of young, healthy people with adequate calcium intake over a 7-week period does not affect the markers of bone metabolism.
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Introduction

Osteoporosis is considered one of the most serious public health problems worldwide. A commonly suggested strategy to diminish bone loss is to maintain positive calcium balance by consuming a diet containing adequate amounts of calcium and vitamin D. The positive relationship between urinary sodium and calcium excretion in humans has been established previously in both cross-sectional (Goulding, 1981; Law *et al*, 1988; Shortt *et al*, 1988; Chan *et al*, 1992; Matkovic *et al*, 1995; Dawson-Hughes *et al*, 1996; Itoh & Suyama, 1996; O'Brien *et al*, 1996; Jones *et al*, 1997) and experimental studies (Breslau *et al*, 1982; Goulding & Lim, 1983; Castenmiller *et al*, 1985; Law *et al*, 1988; McParland *et al*, 1989; Zarkadas *et al*, 1989; Chan *et al*, 1992; Evans *et al*, 1997). It could be hypothesised that sodium-induced urinary calcium loss decreases serum calcium concentration, which is compensated for increased excretion of parathyroid hormone (PTH) from the parathyroid glands (Evans *et al*, 1997). Therefore, high dietary salt intake, typical of Western diets, not only contributes to blood pressure but may also be deleterious for bone metabolism. Based on a longitudinal study of postmenopausal women, it has been concluded that

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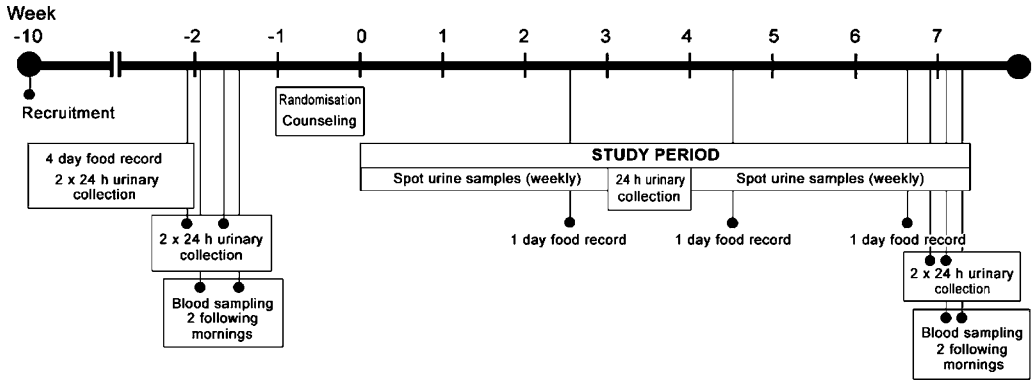

Figure 1 Study design.

a 50% reduction of salt intake is just as effective in preventing bone loss as a daily increase of 891 mg of dietary calcium (Devine *et al*, 1995).

Most of the trials on the effects of sodium on calcium and bone metabolism have been conducted in the setting of shift from a low-sodium (Breslau *et al*, 1982; Castenmiller *et al*, 1985; McParland *et al*, 1989; Chan *et al*, 1992; Evans *et al*, 1997; Lin *et al*, 2003) or normal-sodium (Goulding & Lim, 1983; Law *et al*, 1988; Zarkadas *et al*, 1989; Evans *et al*, 1997) to a high-sodium diet. To our knowledge, there is only one study in which the changes in urinary calcium excretion and bone resorption were observed after subjects had restricted their habitual salt intake (Need *et al*, 1991). The results were very promising, indicating that sodium restriction decreases calcium excretion and bone resorption in postmenopausal women. However, the duration of the study was only a few days, a fasting urinary specimen instead of 24-h collection was provided, and subjects with low habitual salt intake were excluded from the study. The aim of our study was to investigate whether a moderate reduction in salt intake over 7 weeks in young, healthy, free-living people affects calcium and bone metabolism in a positive manner.

Subjects and methods

Subjects

A total of 32 young (age 21–39 y), healthy subjects (14 males, 18 females) were recruited from the Viikki campus area of the University of Helsinki through advertisements. The study protocol, which was approved by the Ethical Committee of Faculty of Agriculture and Forestry of the University of Helsinki, was carefully explained to the volunteers and their informed written consent was obtained. Information on general health status, physical activity, use of alcohol, smoking and use of medication or hormones were obtained in a questionnaire. Exclusion criteria were any chronic diseases or medications known to affect calcium or bone metabolism. Nine of the women were using oral contraceptives. In the recruitment phase, the habitual diets of the subjects were assessed on the basis of food records over a 4-day period. Sodium and creatinine excretions were assessed on the basis of two 24-h urinary collections over the last 2 days of the dietary recording period. Two women dropped out before the beginning of the trial, one because of pregnancy and another because of lack of time. In addition, during the trial, one subject was not able to follow the study protocol and was excluded from the study. Eventually, 29 subjects (14 males, 15 females) completed the study successfully.

The study protocol

The study was conducted over 7 weeks during September and October 2000 (Figure 1). The subjects were divided into two groups, seven men in each, so that the mean sodium excretions were similar in both groups during the recruitment phase (Table 1). The low sodium (LS) group was aiming to reduce the sodium content of their diet to 80 mmol/day, while the control (C) group kept to their habitual diet. The subjects in the LS group got personal and individual dietary counselling on how to reduce their sodium intake. They were asked to consume low-sodium or sodium-free products provided by the project instead of the breads, spreads and cold cuts they normally consumed. A low-salt lunch was also served on weekdays. Low-sodium meat products were delivered on Fridays for preparation of low-salt meals at home during the weekend. The C group was given identical food items and lunch, but with a normal salt content. Otherwise, all the subjects were asked to follow their habitual dietary patterns. Compliance was checked by spot urine specimens collected weekly on a randomly selected day (data not shown), by one 24-h urine collection in the middle of the study and by ordering the subjects to keep one 24-h food record every 2 weeks during the study. Fasting serum samples were taken on two consecutive mornings in the beginning and at the end of the study period. All serum samples were taken anaerobically in vacutainer tubes. Urinary samples (24-h) were collected from each of the 2 days preceding the blood samplings. The serum and urine samples were stored immediately at -20°C until analysed.

Table 1 Background and dietary data of the two study groups[a]

	Low sodium (7M + 7F)	P-*value change*	*Control (7M + 8F)*	P-*value change*	P-*value difference between groups*
Age (y)	30 ± 4.3		27 ± 5.9		0.08[b]
Height (cm)	170 ± 8.8		170 ± 9.9		0.98[c]
Weight (kg)	71 ± 7.8		66 ± 13.0		0.20[c]
BMI (kg/m^2)	24.7 ± 2.7		22.7 ± 2.7		0.05[c]
Energy intake (MJ/day)					
Recruitment phase	8.09 ± 2.0		9.16 ± 2.3		0.20[c]
During the study[d]	7.80 ± 1.4		8.28 ± 1.6		0.43[b]
Change	−0.29 ± 2.5	0.83[e]	−0.88 ± 1.6	0.07[e]	0.45[c]
Calcium intake (mg/day)					
Recruitment phase	954 ± 355		1153 ± 379		0.16[c]
During the study[d]	938 ± 505		993 ± 341		0.73[c]
Change	−16 ± 404	0.89[f]	−160 ± 369	0.12[f]	0.73[b]
Protein intake (g/day)					
Recruitment phase	73 ± 22		89 ± 30		0.12[c]
During the study[d]	79 ± 17		89 ± 18		0.13[c]
Change	5.3 ± 18.5	0.31[f]	−0.5 ± 17.2	0.91[f]	0.38[b]

[a]Mean ± s.d.
[b]Mann–Whitney *U*-test.
[c]Unpaired *t*-test.
[d]Average from three separate 24-h food records.
[e]Wilcoxon's signed rank test.
[f]Paired *t*-test.

Laboratory methods

Urinary sodium excretion was analysed by using ion-specific electrodes (Kone Microlyte Ion Selective Analyzer, Kone Corporation, Espoo, Finland). Urinary calcium and creatinine excretions were analysed with an automatic analysator (Konelab 20, Thermo Clinical Labsystems Oy, Espoo, Finland). Urinary sodium and calcium excretions in the recruitment phase, in the beginning and at the end of the study are averages of two consecutive collections. In the middle of the study, urinary sodium and calcium excretions are based on a single 24-h urine collection. Serum calcium, phosphate and creatinine concentrations were analysed with an automatic analysator (Konelab 20, Thermo Clinical Labsystems Oy, Espoo, Finland). Serum intact PTH (s-PTH) concentration was measured with an immunoradiometric assay (Nichols Institute, San Juan Capistrano, CA, USA). The intra-assay CV was 1% and interassay CV was 4%. The concentration of serum C-terminal telopeptides of Type-I collagen (s-CTX) was measured using enzyme immunoassay (Serum Cross Laps™ One Step ELISA, Osteometer BioTech A/S, Herlev, Denmark). The intra- and interassay CVs were less than 10%. Serum bone alkaline phosphatase (s-B-ALP) concentration was measured with an immunoassay (Alkphase-B®, Metra Biosystems, Mountain View, CA, USA). The intra- and interassay CVs were 4 and 6%, respectively. Serum calcium, phosphate, creatinine, s-CTX and s-B-ALP were measured from one sample in the beginning and at the end of the study. The other measurements from serum are averages of samplings from two consecutive days in the beginning and at the end of the study. All samples were analysed in duplicate.

Dietary analysis

To study the habitual diet of the subjects, food records were collected over 4 days period in the recruitment phase. During the intervention, the diet was monitored by three 24-h food records. Mean energy and nutrient intakes were estimated from the food record data using the Micro-Nutrica software package (1993, Social Insurance Institution, Helsinki, Finland). All food records were obtained and analysed by a dietitian. Because of the limited possibilities to measure dietary sodium intake on the basis of dietary records, urinary sodium excretion was used.

Statistical methods

Calculations of sample size were based on the unpublished findings of Lamberg-Allardt and Kärkkäinen, who noticed that s-PTH increased 15 ± 12 ng/l due to sodium load. All the data were analysed using SPSS 10.0 software package (SPSS Inc., Chicago). All variables were tested for normality using Shapiro–Wilk's normality test. Paired Student's *t*-test for normally distributed data or nonparametric Wilcoxon's signed rank test for skewed data was used to compare the changes in measured variables during the study in both study groups. Unpaired Student's *t*-test for normally distributed data or nonparametric Mann–Whitney *U*-test for skewed data was used to compare the background data and changes in dietary and serum variables between the groups. For the creatinine-adjusted urinary sodium and calcium excretions, logarithmic transformation was used to normalise the skewed data, and repeated-measures analysis of variance was used to study time and group effects. If time and group interaction became significant, *post hoc t*-tests

were performed. Pearson's correlation coefficient was calculated to determine the relationship between creatinine-adjusted sodium and calcium excretions. For all variables, a *P*-value of less than 0.05 was regarded as significant.

Results

Background and dietary data

Mean age, height and weight in recruitment phase were similar in both groups, but BMI was slightly higher in the LS group compared to the C group (Table 1). Dietary records indicated no differences between the groups in mean energy, calcium and protein intakes in the recruitment phase or during the study. No significant dietary changes, except the reduced sodium intake in the LS group, occurred during the intervention.

Effect of a reduction in sodium chloride intake on urinary sodium excretion

Changes in urinary sodium/creatinine ratio in both groups during the study are shown in Figure 2. In the beginning of the study, the mean$\pm$s.d. daily urinary sodium excretion for the LS group was 145 ± 46 mmol (13.1 ± 6.3 mmol/mmol creatinine) and for the C group 139 ± 34 mmol (11.3 ± 4.6 mmol/mmol creatinine). There was no difference between the groups ($P=0.38$). In the middle and at the end of the study, the mean$\pm$s.d. daily urinary sodium excretion was significantly lower in the LS group compared to the C group ($P=0.003$ and $P<0.001$, respectively). In the LS group, there was a significant ($P<0.001$) decrease of 72 ± 39 mmol (7.9 ± 5.6 mmol/mmol creatinine) in daily urinary sodium excretion from the beginning to the end of the study. In the C group, the slight decrease of 12 ± 39 mmol (2.5 ± 4.7 mmol/mmol creatinine) in daily urinary sodium excretion was not significant ($P=0.102$). At the end of the study, the mean$\pm$s.d. daily urinary sodium excretions were 73 ± 22 mmol (5.2 ± 2.0 mmol/mmol creatinine) and 127 ± 40 mmol (8.8 ± 3.2 mmol/mmol creatinine) in the LS and C groups, respectively.

Effect of urinary sodium excretion on urinary calcium excretion

Changes in urinary calcium/creatinine ratio in both groups during the study are shown in Figure 3. Although calcium excretion tended to decline ($P_{\text{time(start–end)}}=0.06$, repeated measures analysis of variance), neither the time and group interaction nor changes within the groups were significant. The mean$\pm$s.d. daily urinary calcium excretions in the beginning of the study were 3.6 ± 1.5 mmol (0.30 ± 0.08 mmol/mmol creatinine) and 3.6 ± 1.2 mmol (0.27 ± 0.06 mmol/mmol creatinine) in the LS and C groups, respectively. There was a significant correlation between sodium/creatinine ratio and urinary calcium/creatinine ratio in the beginning of the study ($r=0.45$, $P=0.01$). At the end of the study, the mean$\pm$s.d. daily urinary calcium excretions were 3.6 ± 2.1 mmol (0.26 ± 0.11 mmol/mmol creatinine) and 3.8 ± 1.2 mmol (0.26 ± 0.07 mmol/mmol creatinine) in the LS and C groups, respectively. The correlation between sodium/creatinine ratio and urinary calcium/creatinine ratio became nonsignificant ($r=0.19$, $P=0.32$).

Effect of a reduction in urinary sodium excretion on s-PTH concentrations

There was a small increase in s-PTH level in both the LS and the C group (Table 2), but the change did not differ between groups.

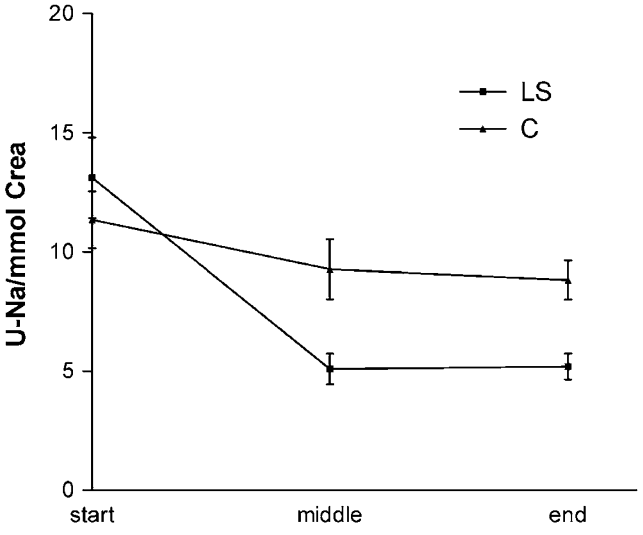

Figure 2 Urinary sodium/creatinine ratio (mean$\pm$s.e.m., mmol/mmol creatinine) during the study in the LS and the C group. Repeated measures analysis of variance: $p_{\text{time(start–end)}}<0.001$, $p_{\text{time(middle–end)}}=0.744$, $p_{\text{time*group(start–end)}}<0.001$, $p_{\text{time*group(middle–end)}}=0.785$.

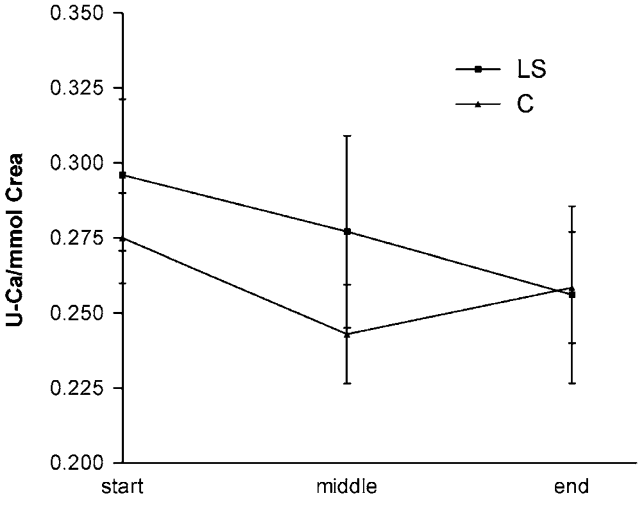

Figure 3 Urinary calcium/creatinine ratio (mean$\pm$s.e.m., mmol/mmol creatinine) during the study in the LS and the C group. Repeated measures analysis of variance: $p_{\text{time(start–end)}}=0.06$, $p_{\text{time(middle–end)}}=0.905$, $p_{\text{time*group(start–end)}}=0.408$, $p_{\text{time*group(middle–end)}}=0.108$.

Table 2 Serum markers of calcium and bone metabolism in the beginning and at the end of the study[a]

	Low sodium (7M + 7F)	P-*value change*	*Control (7M + 8F)*	P-*value change*	P-*value difference between groups*
Calcium (mol/l)					
Beginning	2.36 ± 0.1		2.41 ± 0.1		0.38[b]
End	2.40 ± 0.1		2.41 ± 0.1		0.76[c]
Change	0.03 ± 0.09	0.18[d]	−0.05 ± 0.2	0.90[e]	0.47[c]
Phosphate (mol/l)					
Beginning	1.14 ± 0.2		1.17 ± 0.3		0.73[c]
End	1.18 ± 0.1		1.15 ± 0.4		0.51[b]
Change	0.04 ± 0.1	0.24[e]	0.01 ± 0.4	0.57[d]	0.66[c]
Creatinine (μmol/l)					
Beginning	100.4 ± 15.8		101.9 ± 20.5		0.83[c]
End	101.4 ± 14.6		111.7 ± 28.6		0.24[c]
Change	1.0 ± 10.1	0.73[e]	9.8 ± 24.3	0.14[e]	0.63[b]
PTH (ng/l)					
Beginning	23.3 ± 8.7		23.1 ± 10.9		0.97[c]
End	28.3 ± 8.8		27.3 ± 16.2		0.41[b]
Change	5.1 ± 7.9	0.03[e]	4.2 ± 7.6	0.05[d]	0.77[c]
CTX (pmol/l)					
Beginning	3917 ± 1461		4222 ± 2089		0.60[c]
End	3526 ± 1460		3826 ± 2051		0.66[c]
Change	−391 ± 1362	0.32[e]	−395 ± 1551	0.34[e]	0.87[b]
B-ALP (U/l)					
Beginning	17.1 ± 4.8		16.6 ± 6.9		0.81[c]
End	18.8 ± 5.7		19.5 ± 9.6		0.54[b]
Change	1.7 ± 4.2	0.16[d]	2.9 ± 3.6	0.002[d]	0.19[b]

[a]Mean ± s.d.
[b]Mann–Whitney *U*-test.
[c]Unpaired *t*-test.
[d]Wilcoxon's signed rank test.
[e]Paired *t*-test.

Effect of a reduction in urinary sodium excretion on markers of calcium and bone mineral metabolism

Serum calcium, phosphate and creatinine remained unchanged in both groups during the study (Table 1). Bone resorption, indicated by s-CTX concentration, did not change significantly in either group (Table 2). Bone formation, indicated by s-B-ALP concentration, did not change in the LS group, but the C group showed a significant increase in s-B-ALP concentration during the study. However, there were no significant differences in changes in s-CTX or s-B-ALP between the two study groups.

Discussion

The Finnish mean daily salt intake of 9. 9 g (171 mmol) for male subjects and 6.8 g (117 mmol) for female subjects exceeds the recommended 5 g/day (Reinivuo *et al*, 2003). Presuming that true sodium intake is slightly higher than urinary output, we conclude that the habitual salt intake of the subjects in this study was slightly below the Finnish average. However, the reduction in urinary sodium excretion due to the dietary intervention was very successful. By substituting the products altogether contributing 50% of the Finnish sodium intake for low-salt alternatives and by giving dietary counselling, we were actually able to go below the level of sodium excretion that we aimed at, 80 mmol/day, and which was reached in earlier studies of the same duration (for review, see Cutler *et al*, 1997).

On the basis of several earlier studies, there is an increase of approximately 1 mmol of urinary calcium for every 100 mmol of urinary sodium (for review, see Nordin *et al*, 1993). Therefore, it could be postulated that a reduction in salt intake should be of benefit in the prevention of bone loss because it would decrease the urinary excretion of calcium and preserve bone. The correlation of $r = 0.45$ between urinary sodium/creatinine and calcium/creatinine we found in the beginning of our dietary intervention is at the same level as previously shown in population studies (Goulding, 1981; Law *et al*, 1988; Itoh & Suyama 1996; Jones *et al*, 1997). The correlation disappeared by the end of the study, while both calcium intake and excretion remained constant. The relationship between sodium and calcium excretion is stronger in high calcium intakes compared to low intakes (Dawson-Hughes *et al*, 1996). In the subjects of this study, habitual calcium intake was well above the recommended 800 mg/day. However, for reasons unknown, one apparently healthy subject in the LS group showed a rise in calcium excretion. By excluding this subject, calcium/creatinine excretion would have reduced significantly in the LS group, but the correlation between urinary sodium/creatinine and calcium/creatinine would have remained nonsignificant at the end of the study (data not shown).

In theory, sodium-induced calciuria could lead to temporary reduction in serum calcium concentration that triggers secretion of PTH from the parathyroid glands. PTH acts in several ways on restoring serum calcium concentration. In the kidneys, PTH increases calcium reabsorption and hydroxylation of 25-hydroxyvitamin D to its active metabolite, 1,25-dihydroxyvitamin D, required for active calcium absorption from the intestine. In bone, PTH increases turnover and thus releases calcium from bone. However, there is only one experimental study demonstrating an increase in PTH secretion due to sodium loading in young people (Breslau *et al*, 1982), while in other studies no changes in PTH have been observed (Chan *et al*, 1992; Evans *et al*, 1997; Ginty *et al*, 1998). In our present study, s-PTH concentration increased in both LS and C groups, and the increase in bone formation marker was significant in the C group. Despite the rise in PTH, serum calcium and phosphate remained unchanged in both groups during the study. Although unexpected, our findings are not unique among studies in this field. Negative association between urine sodium/creatinine ratio and s-PTH level has been found in a cross-sectional study of premenopausal women (Chan *et al*, 1994). Evans *et al* (1997) noticed that concentrations of s-PTH and markers of bone metabolism were at their highest in premenopausal women while they were consuming low-sodium diet. Bone resorption, indicated by urinary deoxypyridinoline (DPD), increased due to sodium load only in postmenopausal women, and significant differences between pre- and postmenopausal women were seen in s-PTH and urinary-DPD. This is in agreement with other studies, indicating that there are differences in the metabolic responses to sodium between the young and the elderly (Breslau *et al*, 1985; Itoh *et al*, 1999). Although the association between sodium intake and bone resorption in young subjects has been found in several studies (Goulding, 1981; Goulding & Lim, 1983; Chan *et al* 1992; Itoh & Suyama, 1996; Jones *et al*, 1997), opposite findings also exist (Castenmiller *et al*, 1985; Ginty *et al*, 1998).

Resorption and formation are tightly coupled in the bone-remodelling cycle. However, only a few studies have been carried out on the relationship between sodium intake and bone formation, and the results are quite conflicting. Two population-based studies of postmenopausal women (Devine *et al*, 1995; Dawson-Hughes *et al*, 1996) and experimental studies of postmenopausal (Evans *et al*, 1997) and premenopausal (Ginty *et al*, 1998) women did not demonstrate any relationship between sodium excretion and bone formation. High sodium intake has resulted in decreased bone formation in premenopausal but not in postmenopausal women (Evans *et al*, 1997). When the effect of three sodium levels on bone turnover was compared between middle-aged, overweight subjects consuming the DASH diet or a low calcium controI diet, salt restriction decreased bone formation only in the subjects consuming the DASH diet and had no effect on bone resorption in either group (Lin *et al*, 2003). In a crossover study of 10 postmenopausal women, bone formation as well as the marker of the activity of PTH, urinary cAMP, and serum 1, 25-dihydroxyvitamin D has even increased due to sodium supplementation (McParland *et al*, 1989).

Studies on direct effects of sodium on bone are overshadowed by methodological problems. On the basis of a 2-y longitudinal study on postmenopausal women, sodium excretion, assessed by two separate 24-h urine collections, was a determinant of the change in bone mass (Devine *et al*, 1995). It was concluded that no bone loss occurs when urinary sodium excretion is less than 92 mmol/day. In other population studies, no association between sodium and bone density has been found, but sodium intake has been assessed by a single urine specimen (Matkovic *et al*, 1995; Dawson-Hughes *et al*, 1996; Jones *et al*, 1997, 2001; Carbone *et al*, 2003) or by a dietary recall (Greendale *et al*, 1994).

In our study, we wanted to answer the following question: If reduced sodium intake diminishes calcium excretion, would the conserved calcium be beneficial to bone? To our surprise, in spite of the significant decrease in sodium excretion, we did not establish significant decrease in calcium excretion. Instead, we report a small increase of PTH in both LS and C groups and an increase of bone formation, indicated by s-B-ALP, in the C group. No significant changes in bone resporption, indicated by s-CTX, were found in either group. Because PTH increases bone turnover in general, it is not uncommon that bone formation also increases, and intermittent administration of PTH is known to increase bone formation more than it increases resorption, thus increasing bone mass (for review, see Dempster *et al*, 1993). Why bone formation increased in the C group is not clear. Unexpected results may partially be due to some limitations of our study. The effects of a reduction in sodium excretion on calcium metabolism are more striking in subjects with high habitual sodium excretion than in subjects with low habitual sodium excretion (Need *et al*, 1991). Subjects of our study were recruited from the campus area, and thus were highly educated, scientifically oriented and health conscious, which is likely to explain their moderate sodium intake. Furthermore, we did not determine the vitamin D status of our subjects. The study was started in early autumn, when the vitamin D status of the subjects should have been at its highest. However, serum 25-hydroxyvitamin D concentration is still high in October and should not affect s-PTH concentration (Lamberg-Allardt *et al*, 1983; Rapuri *et al*, 2002). Whatever the reason for the rise in PTH, it might have increased calcium absorption, which would have masked the effects of reduced sodium excretion. The study design also permitted some undetected dietary changes, despite the fact that the subjects were asked to change their dietary habits as little as possible, with the exception of sodium intake for the LS group. Finally, there were unexpectedly large interindividual variations in measured variables, which reduced the estimated statistical power and complicated the interpretation of results. That could have been avoided by screening the subjects for salt sensitivity or by increasing sample size.

In conclusion, a 7-week reduction of sodium intake in young healthy people with moderate sodium intake and adequate calcium intake did not have any positive effects on the markers of bone metabolism. Longer controlled trials with larger sample size, including multiple urinary collections and measurements of bone density, are needed to confirm these findings.

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