A longitudinal study of the effect of sodium and calcium intakes on regional bone density in postmenopausal women[1–3]

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ABSTRACT The influence of urinary sodium excretion and dietary calcium intake was examined in a 2-y longitudinal study of bone density in 124 women postmenopausal for > 10 y. Analysis of bone density changes showed that urinary sodium excretion was negatively correlated with changes in bone density at the intertrochanteric and total hip sites. Multiple-regression analysis of dietary calcium intake and urine sodium excretion on the change in bone density showed that both dietary calcium and urinary sodium excretion were significant determinants of the change in bone mass over 2 y at the hip and ankle sites. These data suggest that an effect of reducing bone loss equivalent to that achieved by a daily dietary increase of 891 mg (22 mmol) Ca can also be achieved by halving daily sodium excretion. No bone loss occurred at the total hip site at a calcium intake of 1768 mg/d (44 mmol/d) or a urine sodium excretion of 2110 mg/d (92 mmol/d). We report a significant effect of sodium excretion on bone loss in this population. *Am J Clin Nutr* 1995;62:740–5.

KEY WORDS Bone density, urinary sodium excretion, calcium

INTRODUCTION

A recent review article identifies osteoporosis as a massive community problem and suggests that research should focus on the causes of osteoporosis and ways for reducing or preventing its prevalence in the community (1). Many factors have been suggested as causes of osteoporosis, namely, lack of physical activity, genetic differences, dietary factors, smoking habits, and metabolic disease (2). Studies of dietary nutrient intake on the skeleton have focused on calcium as the major element contributing to bone mass; however, other nutrients (3, 4)—including sodium (5, 6)—have been reported to affect calcium balance. Furthermore, increased urinary sodium excretion produces an increase in urinary calcium excretion (5, 6) and raised biochemical markers of bone turnover. These effects can be reversed by salt restriction (7), suggesting a possible relation with bone loss. Recently, sodium loading in an animal model has shown a negative effect on bone density measured by dual-energy X-ray technology (8).

It has been suggested that the consumption of sodium should be reduced by choosing low-salt foods and using salt sparingly (9). These recommendations follow research that reports that populations with a low sodium intake (< 1150 mg/d, or 50 mmol/d) have been shown to have a low prevalence of hypertension and mortality associated with cerebrovascular disease (10). The current recommended dietary intake (RDI) for sodium of 920-2300 mg/d (40–100 mmol/d) (11) reflects guidelines set for sodium intake by the United States and other countries (10, 12). However, sodium intakes for adults estimated from 24-h urinary excretion data show current intakes of 2990–4600 mg/d (130–200 mmol/d) (10), which exceed the upper limit of the RDI for sodium.

To explore the effect of dietary sodium intake on bone density, we analyzed the dietary profiles of 196 subjects screened for a placebo-controlled trial of dietary calcium supplementation and an exercise intervention on bone density. Of these, 168 subjects were enrolled and 124 subjects completed the study, which was primarily designed to examine the effects of calcium supplementation on bone density over 2 y in postmenopausal women who were > 10 y past menopause (13). Habitual dietary sodium intake was not manipulated in this study. For this investigation 4-d weighed diet-record and biochemical data were available for correlation with longitudinal bone density values.

SUBJECTS AND METHODS

Subjects

Subjects were participants in a study of calcium supplementation with random assignment to receive additional calcium as milk powder or tablets, compared with placebo (13). Recruitment and exclusion criteria are detailed elsewhere (13). The 241 women who met eligibility criteria were invited to seminars and 196 consented to undergo a physical examination, biochemical analysis, and bone scanning, and to complete a health questionnaire and record their dietary intakes and physical activity. One hundred sixty-eight subjects entered the

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study, 42 in each group. The interventions involved the ingestion of calcium tablets containing 1 g elemental calcium as calcium lactate-gluconate (Sandoz, Basle, Switzerland) with or without an exercise program consisting of 4 h of weight-bearing exercise each week. The 4-d average dietary intakes of sodium and calcium were adjusted for the sodium and calcium contents of the supplement, 407 mg (18 mmol) and 1000 mg (25 mmol), respectively. The third group took identical placebo tablets at bedtime. The 4-d average dietary intake of sodium was adjusted for the 407 mg (18 mmol) Na contained in the supplement supplied by the manufacturer. The group receiving skimmed milk powder was instructed to consume 208 mL (84 g) milk powder/d (Bonlac Foods, Melbourne, and Murray Goulburn Co-op, Brunswick, Australia), which contained 1 g elemental calcium as verified by our analysis. Subjects were asked to consume their supplement before going to bed. Because of the extra energy value of this supplement, subjects in this group were asked to reduce their energy intake by 1220 kJ by avoiding high-fat foods. The 4-d average dietary intakes of sodium and calcium were adjusted for the nutrient content of the milk-powder supplement, 353 mg/d (15 mmol/d) and 1087 mg/d (27 mmol), respectively. Entry into the four groups was by block randomization using sealed envelopes prepared before the study. The study was approved by the Human Rights Committee of the University of Western Australia. Informed consent was obtained from each subject. The data from this study are available for further analysis. The study population for this current investigation consists of data pooled from all four groups.

Compliance

All data were included in the analysis because compliance was high. Compliance with tablet consumption was checked by tablet counting and set at a consumption of 85% of tablets. Ninety-seven percent of the calcium group, 100% of the placebo group, and 100% of the calcium and exercise group were compliant. Milk-powder compliance was checked by weighing the returned packets. It was set at 85% of the possible total consumption. Eighty-eight percent of subjects were compliant.

Dietary and activity measurements

Over the duration of the study the women completed 4-d, weighed diet records administered by two trained nutritionists at baseline, 1 y, and 2 y. Habitual sodium intake was not manipulated in this study. At baseline, six subjects did not complete a weighed diet record. The remaining 190 baseline diet records and 130 and 127 diet records collected at 1 and 2 y, respectively, were analyzed by using DIET 1 nutrient calculation software (Xyris Software, Queensland, Australia). The program uses the NUTTAB 90 database, a nutritional database that uses chemical analysis of Australian foods. At each time point a 4-d average intake was calculated for calcium, phosphorus, sodium, energy, and protein. To estimate the calcium intake over the duration of the study, the 1-y and 2-y data were averaged. The change in calcium intake was estimated from baseline and 1-y data. During the study, dietary sodium intake was determined from the average of the 1-y and 2-y 24-h urine sodium collections and not from the 4-d weighed diet records because these did not detail discretionary use of sodium salt. The change in sodium intake was estimated from baseline and 1-y dietary data. One subject's average calcium intake was > 3 SDs outside the group's normal range and was excluded from the longitudinal analysis.

Each subject completed an activity record for a 7-d period every 6 mo. From these records the women's most active 2 h of the day were scored by using tables of metabolic equivalent activities (14) as a measure of aerobic activity to derive an activity score (MET). One metabolic equivalent was defined as the energy consumed per minute sitting at rest; activities were measured in relation to that standard. The values for an average 55-kg woman were used; the results quoted are therefore independent of body weight. To estimate the change in activity over the duration of the study, the baseline measurement was subtracted from the averaged 6-mo data.

Biochemical measurements

Each subject collected a 24-h urine sample, which was analyzed for sodium and calcium by using routine methods on a SMAC analyzer (Technicon Corp, Tarrytown, NY). The 24-h urinary sodium and calcium excretions were averaged over the 2-y period. The change in 24-h urinary sodium and calcium excretions was determined from baseline and 1-y data. Plasma alkaline phosphatase was measured by using routine methods on a SMAC analyzer. Serum calcitriol (1,25-dihydroxyvitamin D) was measured by using a column extraction technique followed by assay with calf thymus cytosol binding protein (15); the intra- and interassay CVs for the calcitriol assay were 14% and 20%, respectively. Serum intact parathyroid hormone (PTH) was measured by using an immunochemiluminometric method (16) and were 3.6% and 6.2%, respectively. The changes in plasma alkaline phosphatase, serum calcitriol, and serum intact PTH were determined from baseline and 1-y data.

Bone measurement

Bone density in the lumbar spine (L1-L4), distal tibia and fibula (ankle), and the hip sites were assessed by using dual-energy X-ray technology on a QDR 1000 bone densitometer (Hologic, Waltham, MA). The CV at the lumbar spine was 1%. The hip site was defined as the area including the femoral neck, trochanter, and intertrochanter site and had a CV of 1.5%. The neck site was defined as a box 6-mm wide, traversing the femoral neck placed against the greater trochanter and had a CV of 2.3%. The trochanter site was a triangular region whose boundaries were defined as the lateral edge of the femoral neck region and a line connecting the midpoint of the femoral neck at the edge of the femoral neck area to the point where the edge of the femur changes curvature below the trochanter and had a CV of 2.2%. The intertrochanteric region was the remainder of the femur extending 10 mm below the lesser trochanter; the CV was 2.2%. The program for assessing the ankle site was modified from a program devised to measure distal radius and ulna bone density. A device to hold the foot still was manufactured and the tibia and fibula scanned in the postero-anterior plane. The area of interest, the ultra distal site, was defined as the area 6–26 mm proximal to the ankle joint space. The CV at this site in 15 young adults measured 6 mo apart was 1.8%. One subject's change in bone density at the trochanter site was > 3 SDs outside the group's normal range and was excluded from the longitudinal analysis.

Statistical analysis

Baseline and averaged values are reported as the mean ± SD. The regression line for the change in bone density over time was calculated by least-squares analysis for each individual completing three or more bone density estimations and was used to derive a measure of rate of change (loss: $mg \cdot cm^{-2} \cdot y^{-1}$). All P values are two-tailed. The statistical package used was SPSS for Windows (SPSS, Inc, Chicago). Statistical analysis was conducted by using Spearman's rank correlation coefficient and stepwise-multiple-regression analysis. Residuals were examined for normality.

RESULTS

Baseline values for the study population are shown in **Table 1.** Average dietary calcium intake, average 24-h urinary sodium and calcium excretions, and the changes in bone density for each site, in activity, in dietary sodium intake, and in urinary sodium and calcium for the longitudinal study are detailed in **Table 2.**

A significant positive correlation was found between the average dietary calcium intake with change in bone density at the total hip, intertrochanter, femoral neck, and ultra distal site of the ankle (**Table 3**). From this correlation it was possible to calculate the calcium intake required for no change in bone mass over the 2 y of the study. No bone loss was encountered at the intertrochanter, femoral neck, and total hip site at a daily total calcium intake of 1664 mg (41 mmol), 2042 mg (51 mmol) (**Table 4**), and 1768 mg (44 mmol), respectively (Table 4, **Figure 1**). Because a mean bone loss of 10.75 $mg \cdot cm^{-2} \cdot y^{-1}$ occurred at the ultra distal site of the ankle, the daily total calcium intake required to maintain no change in bone density at this site required gross extrapolation beyond the data points.

The average 24-h urine sodium excretion was significantly negatively correlated with the change in bone density at the total hip (Table 3, **Figure 2**) and the intertrochanter site (Table 3). From this correlation it was possible to calculate the urinary sodium excretion required for no change in bone mass over the 2 y of the study. No bone loss was encountered at a daily urinary sodium excretion of 2620 mg (114 mmol) at the intertrochanteric site (Table 4) and 2110 mg (92 mmol) at the total hip site (Table 4, Figure 2).

Multiple regression was performed with change in bone density at all sites as the dependent variable and average calcium intake, average urinary sodium excretion, weight, and change in activity as independent variables. The results of this analysis showed that urinary sodium excretion was a significant negative correlate of change in bone density at the total hip site and ultra distal site of the ankle after other factors were allowed for (**Table 5**).

In linear correlation, weight showed no consistent relation with change in bone density at hip and ankle sites but was significantly positively correlated with the lumbar spine ($r = 0.240$, $P < 0.005$) and when entered into the multiple-regression analysis was significant at the spine and neck sites (Table 5). By repeated-measures analysis of variance, weight did not change over the time of the study or with any of the treatments and was significantly positively correlated with average urinary sodium excretion ($r = 0.189$, $P < 0.05$).

TABLE 1
Baseline variables measured in postmenopausal women[1]

Variable	Value	
Weight (kg)	66 ± 10	[191]
Protein intake (g/d)	76 ± 16	[190]
Calcium intake (mg/d)	805 ± 320	[190]
Phosphorus (mg/d)	1269 ± 311	[190]
Sodium intake (mg/d)	1982 ± 580	[190]
Energy intake (kJ/d)	6830 ± 1457	[190]
Urinary sodium excretion (mg/d)	2783 ± 1081	[196]
Urinary calcium excretion (mg/d)	144 ± 71	[196]
Total hip bone density (mg/cm^2)	830 ± 120	[196]
Trochanter bone density (mg/cm^2)	620 ± 90	[196]
Intertrochanter bone density (mg/cm^2)	970 ± 150	[196]
Femoral neck bone density (mg/cm^2)	700 ± 100	[196]
Ultra distal ankle bone density (mg/cm^2)	620 ± 100	[196]
Lumbar spine bone density (mg/cm^2)	860 ± 150	[195]

[1] $\bar{x}$ ± SD; n in brackets.

TABLE 2
Average dietary and biochemical variables for year 1 and year 2 of the study, change in dietary and biochemical variables between baseline and year 1, and change in bone density and activity over the duration of the study measured in postmenopausal women[1]

Variable	Value	
Change in total hip bone density ($mg \cdot cm^{-2} \cdot y^{-1}$)	−0.99 ± 11.71	[134]
Change in trochanter bone density ($mg \cdot cm^{-2} \cdot y^{-1}$)	1.05 ± 10.97	[133]
Change in intertrochanter bone density ($mg \cdot cm^{-2} \cdot y^{-1}$)	−1.57 ± 15.54	[134]
Change in femoral neck bone density ($mg \cdot cm^{-2} \cdot y^{-1}$)	−1.84 ± 10.24	[134]
Change in ultra distal ankle bone density ($mg \cdot cm^{-2} \cdot y^{-1}$)	−10.75 ± 10.16	[134]
Change in lumbar spine bone density ($mg \cdot cm^{-2} \cdot y^{-1}$)	0.76 ± 11.36	[134]
Average dietary calcium intake (mg/d)	1407 ± 530	[123]
Average urinary sodium excretion (mg/d)	3049 ± 808	[127]
Average urinary calcium excretion (mg/d)	167 ± 68	[128]
Difference in urinary sodium excretion (mg/d)	234 ± 1256	[128]
Difference in urinary calcium excretion (mg/d)	19 ± 61	[133]
Difference in dietary sodium intake (mg/d)	187 ± 624	[130]
Change in activity (METS/d)	385 ± 46	[112]

[1] $\bar{x}$ ± SD; n in brackets. METS, metabolic equivalent activities.

TABLE 3
Correlation coefficient of predictors of the rate of change of bone density at the spine, hip, and ankle

	Average urinary sodium excretion	Average dietary calcium intake
Total hip	−0.192[1]	0.219[1]
Intertrochanter	−0.196[1]	0.195[1]
Trochanter	−0.132	0.172
Femoral neck	0.098	0.220[1]
Ultra distal ankle	−0.132	0.184[1]
Lumbar spine	−0.003	0.076

[1] $P < 0.05$.

TABLE 4
Regression of calcium intake (mg/d) and urinary sodium excretion (mg/d) on change in bone density ($mg \cdot cm^{-2} \cdot y^{-1}$) at hip sites in postmenopausal women (for statistically significant correlation in Table 3)

Change in total hip bone density = 0.005 × calcium intake − 8.84
Change in intertrochanter bone density = 0.007 × calcium intake − 11.65
Change in femoral neck bone density = 0.004 × calcium intake − 8.17
Change in total hip bone density = −0.003 × urinary sodium excretion + 6.33
Change in intertrochanter bone density = −0.003 × urinary sodium excretion + 7.86

There was no correlation between baseline measures of bone formation (alkaline phosphatase) or calcitropic hormones (PTH or calcitriol) and urinary sodium excretion (data not shown). Baseline urinary sodium excretion was significantly positively correlated with baseline urinary calcium excretion ($r = 0.227$, $P < 0.001$). The change in calcitriol, PTH, and alkaline phosphatase between baseline and 1 y was not correlated with change in urinary sodium excretion between baseline and 1 y.

Over the first year of the study the change in urinary sodium was significantly positively correlated with the change in urinary calcium ($r = 0.452$, $P < 0.05$). At baseline, sodium intake was correlated with baseline urinary sodium excretion ($r = 0.31$, $P < 0.001$) and during the first year of the study the change in urinary sodium was significantly positively correlated with the change in total sodium intake ($r = 0.179$, $P < 0.05$).

The relation between dietary calcium intake and urinary sodium excretion, determined by using the regression equation for the change in bone density at the total hip site (Table 5) and setting that change to zero (ie, no bone loss), is shown in **Figure 3.** From nominal amounts of urinary sodium excretion, estimations of the amount of dietary calcium required to neutralize the negative effects can be made. For example, a sodium excretion of 1931 mg/d (84 mmol/d) with a dietary calcium intake of 1000 mg/d (25 mmol/d) would prevent bone loss.

DISCUSSION

Our data show that a high sodium intake measured as urinary sodium excretion is associated with an increased bone loss at the hip site. This is supported by a recent finding of the negative effect of salt loading on bone density in an animal model (8). To our knowledge no previous studies of the negative effect of dietary sodium on changes in longitudinal bone density in women have been published.

The multiple-regression analysis (Table 5) shows that urinary sodium excretion is a significant determinant of changes in total hip bone density even when the possible confounding factors of weight, calcium intake, and change in activity are included in the equation. This finding suggests that the higher the urinary sodium excretion the greater the bone loss. No change in bone density at the total hip site was achieved with an average urinary sodium excretion of ≈2100 mg/d (92 mmol/d). The importance of the sodium effect can be gauged from calculations based on the regression equations between dietary calcium or sodium excretion and change in bone density at the total hip site. This shows that halving a nominal urinary sodium excretion of 3450 mg/d to 1725 mg/d (150–75 mmol/d), which would have an effect on bone density equivalent to an increase in daily dietary calcium intake of 891 mg (22 mmol/d). This suggests a potentially powerful dietary effect of sodium on bone loss at the hip and perhaps the ultra distal ankle site. The lack of effect of either sodium or calcium at the spine site may have been due to the confounding effects of degenerative joint disease or aortic calcification, which are prevalent in this age group.

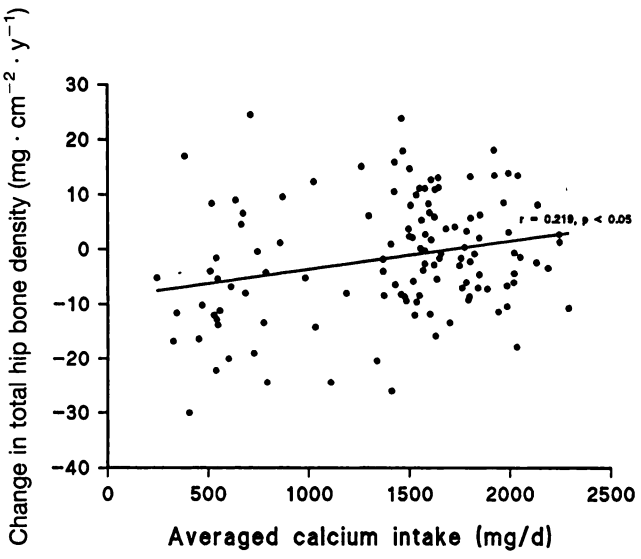

FIGURE 1. Correlation between the average calcium intake between year 1 and year 2 of the study and change in hip bone density ($y = 0.005x - 8.84$; $r = 0.219$; $P < 0.05$). No bone loss occurred at a daily total calcium intake of 1768 mg (44 mmol).

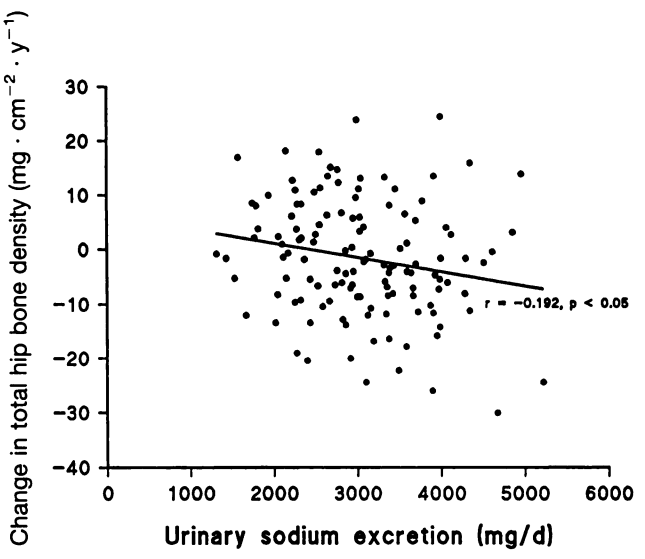

FIGURE 2. Correlation between the average urinary sodium excretion between year 1 and year 2 of the study and change in hip bone density ($y = -0.003x + 6.33$; $r = -0.192$; $P < 0.05$). No bone loss occurred at a daily urinary sodium excretion of 2110 mg (92 mmol).

TABLE 5
Multiple-regression analysis of the rate of change in bone density with weight (kg), change in metabolic equivalent activity (METS/d), average calcium consumption (mg/d), and average urinary sodium excretion (mg/d)[1]

Change in bone density	Regression coefficient	SE of the regression coefficient	Standardized regression coefficient	Significance of F value
Total hip site				
+ Average dietary calcium	$\times 5.1 \times 10^{-3}$	1.9×10^{-3}	0.25	0.006
− Average urinary sodium excretion	$\times 2.7 \times 10^{-3}$	1.2×10^{-3}	−0.20	0.002
+ Constant	× 0.1	4.8		
Intertrochanter site				
+ Average dietary calcium	$\times 7.6 \times 10^{-3}$	2.7×10^{-3}	0.26	0.006
− Constant	× 11.7	4.0		
Femoral neck site				
− Weight	$\times 2.7 \times 10^{-1}$	1.0×10^{-1}	−0.24	0.004
+ Average dietary calcium	$\times 3.8 \times 10^{-3}$	1.6×10^{-3}	0.22	0.001
+ Constant	× 10.6	7.4		
Ultra distal ankle site				
+ Average dietary calcium	$\times 3.5 \times 10^{-3}$	1.7×10^{-3}	0.19	0.035
− Average urinary sodium excretion	$\times 2.2 \times 10^{-3}$	1.1×10^{-3}	−0.18	0.016
− Constant	× 9.1	4.4		
Lumbar spine site				
+ Weight	$\times 3.8 \times 10^{-1}$	1.3×10^{-1}	0.27	0.005
− Constant	× 23.2	8.6		

[1] R = 0.33, 0.26, 0.35, 0.27, and 0.27 for total hip site, intertrochanter site, femoral neck site, ultra distal ankle site, and lumbar spine site, respectively.

The mechanism of the bone loss seen with higher urinary sodium excretion is likely to be related to higher rates of bone resorption because of increased calcium loss in the urine (5–7, 17). However, we were unable to find any correlation between baseline measures of alkaline phosphatase or calcitropic hormones and urinary sodium excretion. The physiologic mechanism by which sodium excretion increases urinary calcium excretion has been demonstrated in this and other studies but the negative effect on change in bone density has not been illustrated in humans.

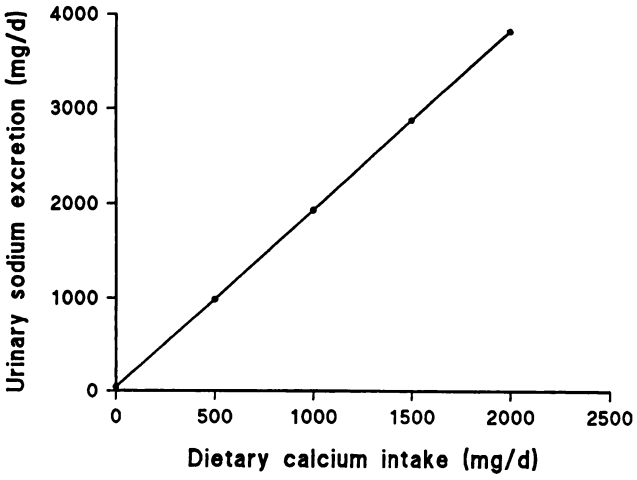

FIGURE 3. Relation between sodium excretion and dietary calcium intake to change in total hip bone density gained from the multiple-regression analysis (Table 5). The equation of the line for no change in total hip bone density = 0.0051 (dietary calcium) − 0.0027 (urinary sodium excretion) + 0.1.

These data demonstrate that increasing dietary calcium protects against bone loss at the hip and ankle sites and that the change in calcium intake is positively correlated with the change in bone density. This suggests that the lower the calcium intake the greater the bone loss and that calcium balance at the hip is achieved with a calcium intake of ≈1700 mg/d (42 mmol/d). This calcium intake, necessary to achieve calcium balance, is valid for this study population, which had a mean (± SD) sodium intake over the 2 y of the study, measured by urinary sodium excretion, of 3049 ± 808 mg/d (132 ± 35 mmol/d). A lower required calcium intake would have achieved calcium balance if sodium intakes were within the RDI (11). Use of the relation between urinary sodium excretion and dietary calcium intake that results in no change in total hip bone density (Figure 3), calcium balance at an intake of 1198 mg (30 mmol/d) Ca/d would be achieved if sodium excretion was reduced to reflect an intake corresponding to the upper limit of the RDI for sodium (2300 mg/d, or 100 mmol/d). This calcium intake would be more achievable by individuals and bring intakes of both sodium and calcium in line with recommended values (11).

RDIs and recommended dietary allowances (RDAs) for calcium are currently not being achieved by elderly women in Australia (18) and the US (19). The recommendation for elderly women to further increase their calcium intake to 1500 mg/d (38 mmol/d), because declining concentrations of calcitriol reduce calcium absorption (19), is supported by these results. Reductions in bone loss could be achieved if individuals *1*) increased their recommended intakes of calcium (19), and *2*) followed current guidelines encouraging a reduction in sodium intake (9, 12).

Difficulties associated with measuring dietary sodium intake are evident in the low correlation with urinary sodium excre-

tion. This is because discretionary use of sodium (ie, sodium used in food preparation, in cooking, and at the table) may vary from 25% to 45% among individuals (10) and subjects would find reporting discretionary sodium use difficult. Nutritional databases may vary considerably in sodium contents of processed and convenience foods because the content is dependent on manufacturers' preparation, processing, and cooking techniques. Therefore, urinary sodium excretion is used as a more accurate and definitive measure of actual sodium intake in this investigation (10). The weak positive correlation between the change in dietary sodium intake and urinary sodium excretion between baseline and 1 y demonstrates that an increase in sodium intake would be associated with higher urinary sodium excretion.

Previous data suggest that urinary calcium excretion reflects urinary sodium excretion rather than the reverse (7, 20). The correlation between the change in urinary sodium and calcium is usually sodium driven (7, 20). Further, the apparent lack of effect of calcium intake on bone mass reported in other studies (21–23) may have been due to the confounding effects of a high sodium intake, which increases the filtered sodium load and reduces the tubular reabsorption of calcium. To compensate for this calcium leak, individuals with a high sodium intake may have increased calcitriol concentrations to enhance intestinal calcium absorption. Breslau (24) found a rise in calcitriol concentrations and gut calcium absorption in young normal adults with sodium-induced hypercalciuria, whereas McParland et al (17) did not find such an increase in elderly women . Thus, the increase in calcium absorption is not seen in postmenopausal women and may be due to an impaired ability to synthesize calcitriol (13). Nordin et al (20) reported an increase in fasting urinary sodium at the menopause, which increases urine fasting calcium excretion and influences calcium homeostasis. The lack of adaptation of the intestine and kidneys to high sodium intakes in elderly women needs to be considered when interpreting rises in urine calcium excretion.

In conclusion, the relation of increased urinary sodium excretion to increase bone loss has been illustrated. The promotion of dietary guidelines to prevent osteoporosis focuses on increased consumption of dairy products and other calcium-rich foods. We suggest that dietary information encouraging postmenopausal women to increase their consumption of calcium-rich foods should be coupled with messages to reduce their sodium intake, promoting a twofold dietary strategy for prevention of osteoporosis. A reduction in sodium-rich foods may enhance the effect of the calcium intake by preventing raised concentrations of urinary calcium. Our results suggest that further studies looking primarily at sodium intake reduction and its impact on bone density are justified.

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