European Journal of Clinical Nutrition (1997) 51, 394–399

The effect of dietary sodium on calcium metabolism in premenopausal and postmenopausal women

CEL Evans, AY Chughtai, A Blumsohn, M Giles and R Eastell

Human Metabolism & Clinical Biochemistry, University of Sheffield, Northern General Hospital, Herries Road, Sheffield, S5 7AU, UK

Objective: To investigate the effects of high and low sodium diets on urinary calcium, bone turnover and calcium absorption in pre and postmenopausal women.
Design: Experimental, prospective and longitudinal study.
Setting: Samples were taken at the hospital and the diets were followed at home.
Subjects: Volunteers were recruited from the hospital and were either hospital staff or post-graduate students. No volunteers failed to complete the study but one was omitted from analysis due to lack of compliance.
Interventions: Eleven healthy premenopausal women aged 22–47 y and 11 healthy postmenopausal women ages 45–70 y followed a high (300 mmol/d) and a low (50 mmol/d) sodium diet for one week each. On the 7th day of each diet, blood and urine samples were taken.
Results: On the high sodium diet 24 h urinary sodium and calcium values relative to creatinine were significantly higher for all subjects ($P < 0.05$). Postmenopausal women on the high sodium diet had biochemical evidence of increased bone resorption in relation to the low sodium diet. However in premenopausal women there was no such change. Calcium absorption did not change significantly in either group.
Conclusions: It appears that postmenopausal, but not premenopausal, women respond to a high sodium diet by an increase in bone resorption which may lead to reduced bone density.
Sponsorship: Arthritis and Rheumatism Council Project Grant R44.
Descriptors: dietary sodium; urinary calcium; bone turnover; deoxypyridinoline; calcium absorption; strontium

Introduction

Osteoporosis is a painful and debilitating disease characterised by a decrease in bone density making bones susceptible to fracture with minimal trauma. Genetic, mechanical, hormonal, nutritional and behavioural risk factors have been identified as possible causes. As the proportion of our elderly population steadily increases there is likely to be a doubling in the number of hip, vertebral and Colles' fractures over the next 30 y which has considerable public health consequences, both socially and economically. It is therefore vital that women adopt a lifestyle that minimises their risk of osteoporosis. Diet is one of the few factors that can be changed and improved relatively easily unlike genetic factors, making it an important area of research. Recent research has centred mainly on the role of dietary calcium in bone metabolism but there is increasing evidence that other dietary factors such as sodium have a significant part to play. This paper explores the effects of sodium on calcium metabolism in pre and postmenopausal women to see if these two groups of women adapt differently to a sodium stress.

Dietary sodium increases urinary calcium leading to a temporary decrease in serum calcium and a consequent rise in PTH. This occurs because in the renal tubules, calcium transport is closely linked to sodium transport and factors that tend to reduce sodium transport at this site, such as high dietary sodium also tend to decrease calcium reabsorption and increase calcium excretion (Goldfarb, 1989). The increase in PTH in turn may, (a) increase renal calcium reabsorption (b) increase bone resorption and (c) increase renal 1-α hydroxylase activity resulting in increased production at 1,25 $(OH)_2$ (Figure 1).

The relationship between dietary sodium and calcium excretion has been approached in a number of ways. In population studies, dietary sodium appears to be only a very weak determinant of urinary calcium excretion (Kesteloot & Joosens, 1990). Other studies have shown quite strong correlations ($r = 0.57$) between sodium/creatinine and calcium/creatinine excretion in older and younger women following a diet unrestricted in calcium (Goulding, 1981) or fixed calcium diets of 400 mg (Sabto *et al*, 1984).

The optimum approach to examine this relationship is to alter the dietary sodium and observe the response in urinary calcium. The effect of taking sodium supplements, on the urinary excretion of calcium in postmenopausal women has been studied (Zarkadas *et al*, 1989; McParland *et al*, 1989). Urinary calcium excretion increases by approximately 0.6 mmol/100 mmol of sodium at the highest dose of sodium of 300 mmol. It is estimated that for a woman with a total body calcium value of 900 g, that this would be equivalent to an extra 1%/year rate of bone loss assuming that adaptation took place solely through an increase in bone resorption. Other studies involving postmenopausal women have shown a decrease in urinary sodium, calcium and hydroxyproline/creatinine when following a restricted sodium diet (Need *et al*, 1991).

Studies in young men and women (Breslau *et al*, 1982) have shown that on high sodium diets of 250 mmols compared with low sodium diets of 10 mmols, urinary

Correspondence: Prof R Eastell
Received 26 April 1996; revised 3 March 1997; accepted 9 March 1997

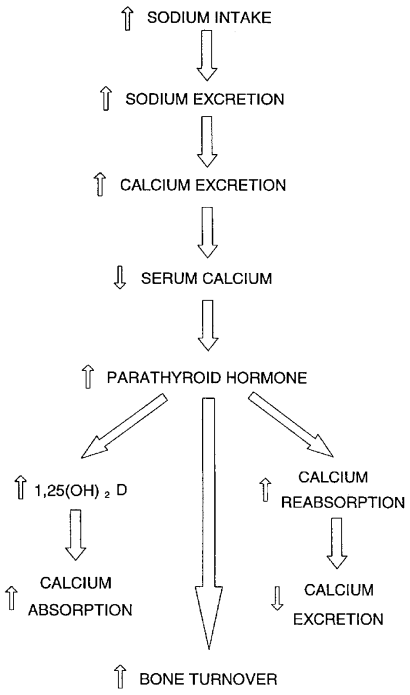

Figure 1 The effects of sodium on calcium metabolism.

sodium and calcium excretion increase but so does calcium absorption, showing that an increase in urinary calcium does not necessarily indicate bone loss. However, bone turnover was not measured. This study is the first to compare the differences in adaptation to a sodium stress in pre and postmenopausal women.

Methods

Informed consent was obtained from twelve healthy premenopausal women ages 22–47 y and eleven healthy postmenopausal women ages 45–70 recruited in Sheffield. Volunteers were excluded if they had taken oral contraceptives, hormone replacement therapy or drugs affecting calcium or sodium metabolism within the previous year, had fractured any bones or had any systemic disease. Bone mineral density (BMD) of the lumbar spine of all volunteers was between 75 and 125% of a normal healthy young adult measured by Luna + DPX. Vertebral fracture was excluded in all subjects by *X*-ray of the lumbar and thoracic spine.

The study spanned three weeks for each volunteer. During the first week volunteers ate a normal diet while completing a four day weighed intake to determine their habitual calcium intake. In the second and third week they ate a low salt diet containing 50 mmol of sodium and their usual amount of calcium as assessed by a 4 d weighed intake. On the last two days of each week they completed two 24 h urine collections and, also on the last day of each week, a calcium absorption test. In the second OR third week (randomised) they had 250 mmol of sodium supplements.

The low sodium diets were designed to be as close as possible to each volunteers normal diet. The sodium supplements provided for the high sodium diet consisted of 3 sachets of salt, each containing 1.2 g of sodium chloride, to sprinkle on food, 11 slow sodium tablets (manufactured by Ciba), each containing 0.6 g of sodium chloride, to have in-between meals and 3 Oxo cubes, each containing 1.5 g of sodium chloride, to make into three savoury drinks. These provided 60, 110 and 80 mmols of sodium respectively making a total of 250 mmol.

Each volunteer completed a 24 h urine collection on days 5 and 6 of the three different diets- habitual, low and high sodium. Urine was collected in a 2.5 L plastic bottle containing 2 g of boric acid and measured to the nearest 5 mls, then stored at -20°C in 5 ml sample tubes. Fasting blood samples were taken using serum tubes at the beginning of each hospital visit. The blood was centrifuged and stored at -70°C until assayed.

Calcium absorption was measured using the method validated by Blumsohn & Eastell (1995). 667 mg (2.5 mmols) of strontium chloride dissolved in milk was given to each volunteer straight after a standard breakfast (Blumsohn & Eastell, 1995). Blood tests were taken exactly 3 and 5 h after ingestion of the oral Sr tracer and the samples stored at -20°C.

Urinary calcium was measured using Colourimetric-end point. O-cresolpthalein. IL test 181619-80 on instrument IL Monarch. Urinary sodium was measured using Flame photometry on instrument IL943. Urinary creatinine was measured using Colourimetric-rate. Jaffe reaction on IL monarch. Urinary Deoxypyridinoline was assayed in duplicate by HPLC (Colwell *et al*, 1993). All results for urinary sodium, calcium and deoxypyridinoline were expressed in relation to creatinine excretion (Bingham, 1992). Osteocalcin was assayed in duplicate by radioimmunoassay (RIA) using the Nichols Institute Human Osteocalcin RIA (Nichols Institute, San Juan Capistrano, CA). Intact parathyroid hormone was measured in duplicate by immunoradiometric assay (Allegro, Nichols Institute, San Juan Capistrano, CA).

A paired *t*-test was used to compare the low and high sodium diets in each group with respect to urinary sodium/creatinine, urinary calcium/creatinine, urinary deoxypyridinoline/creatinine, serum osteocalcin, PTH and strontium absorption. An unpaired *t*-test of the differences between the low and high sodium diets was used to investigate any differences between pre and post menopausal women with respect to the aforementioned measurements. A *P*-value of less than 0.05 was taken as significant.

Results

Twenty-two women out of the twenty-three recruited completed the study. One volunteer from the premenopausal group had no increase in urinary sodium/creatinine on the high sodium diet and therefore was considered a protocol violator as no changes in calcium metabolism would be seen if urinary sodium did not increase. Postmenopausal women were significantly older with a mean age of 57 y (s.d. 8.1) compared with 32 y (s.d. 8.9) in premenopausal women. Postmenopausal women were significantly shorter with a mean of 1.57 m (s.d. 0.07) compared with 1.64 m (s.d. 0.05) in premenopausal women. There was no significant difference in weight between the two groups. Postmenopausal women had a mean weight of 66.2 kg (s.d. 6.9) compared with 62.6 kg (s.d. 11.9) in premenopausal women. Calcium intake was not significantly different between the two groups. Premenopausal women had a mean intake of 861 mg (s.d. 427) and postmenopausal women had a mean intake of 741 mg (s.d. 172). Blood pressure was normal in all women and did not change significantly on the high or low sodium diet.

Urinary creatinine did not change significantly with diet

or between groups. For premenopausal women mean urinary creatinine was 8.05 mmol (s.d. 2.38) on the high sodium diet and 7.86 mmol (s.d. 1.65) on the low sodium diet. For postmenopausal women mean urinary creatinine was 7.91 (s.d. 2.56) on the high sodium diet and 8.45 (s.d. 2.27) on the low sodium diet.

Urinary sodium excretion of volunteers while following their habitual diet ranged between 50 and 200 mmol per day. Urinary sodium increased on the high sodium diet in all subjects (Figure 2). See Table 1 for the statistical results of the paired *t*-test. On the low sodium diet containing 50 mmol of sodium, mean daily urinary sodium excretion for the premenopausal women was 38.1 mmol (s.d. 3.8) and for the postmenopausal women was 60.6 mmol (s.d. 29.5). On the high sodium diet containing 300 mmol of sodium, mean daily urinary sodium excretion for the premenopausal women was 254.2 mmol (s.d. 85.0) and for the postmenopausal women was 285.0 mmol (s.d. 74.8). There were no significant differences between the two groups in urinary sodium excretion when expressed per mmols of creatinine. There was also no difference between the two groups of women in the change in urinary sodium excretion from low to high sodium diets (Table 1). Mean urinary sodium excretion appeared to reflect dietary sodium intake for all women.

Urinary calcium increased significantly in pre and postmenopausal groups of women when on the high sodium diet (Figure 3). On the low sodium diet mean urinary calcium for the premenopausal women was 3.17 mmol (s.d. 1.94) and for the postmenopausal women was 3.47 mmol (s.d. 1.63). On the high sodium diet mean urinary calcium for the premenopausal women was 4.05 mmol (s.d. 1.72) and for the postmenopausal women was 4.54 mmol (s.d. 2.04). When expressed per mmol of creatinine, in premenopausal women mean urinary calcium increased by 37% and in postmenopausal by 48%. There was no significant difference between pre and postmenopausal women in the change in urinary calcium between the two diets (Table 1).

In postmenopausal women there was a significant increase in urinary deoxypyridinoline/creatinine on the high sodium diet ($P = 0.024$) of 27% (Table 1). No such increase was seen in the premenopausal women where the difference between the low and high sodium diet was not significant (Figure 4). The difference between pre- and postmenopausal women in the change in deoxypyridinoline

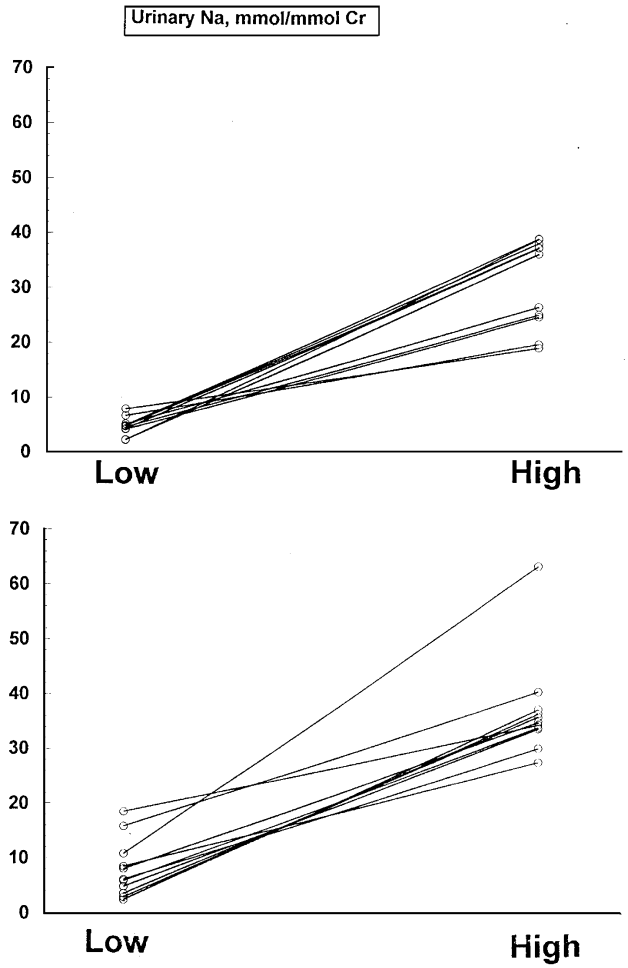

Figure 2 Changes in urinary sodium in eleven premenopausal women (upper panel) and eleven postmenopausal women (lower panel) on low (50 mmol) and high (300 mmol) sodium diets.

Table 1 Mean levels of urinary sodium, calcium and deoxypyridinoline/creatinine, serum osteocalcin, PTH and Strontium in pre and postmenopausal women on habitual, low and high sodium diet. The *P* values of the paired tests are comparisons between low and high sodium diets. The unpaired *t*-test of the difference (high minus low) between pre and postmenopausal women

	Level of sodium intake	*Mean (s.d.) premenopausal women*	*P value paired t-test (95% CI)*	*Mean (s.d.) postmenopausal women*	*P value paired t-test (95% CI)*	*Unpaired t-test of difference (95% CI)*
Urinary Sodium/Creatinine, mmol/mmol	habitual	15.3 (10.6)		14.2 (4.7)		
	low	4.77 (1.64)	$P < 0.001$	7.95 (5.25)	$P < 0.001$	$P = 0.476$
	high	31.3 (8.02)	(20.3, 32.7)	37.4 (9.34)	(23.0, 36.0)	(−11.3, 5.48)
Urinary Calcium/Creatinine, mmol/mmol	habitual	0.51 (0.23)		0.52 (0.21)		
	low	0.38 (0.2)	$P = 0.005$	0.42 (0.15)	$P = 0.01$	$P = 0.406$
	high	0.52 (0.2)	(0.051, 0.221)	0.62 (0.33)	(0.058, 0.339)	(−2.17, 0.091)
Urinary Deoxypyridinoline/ Creatinine, nmol/mmol	habitual	8.90 (4.88)		16.1 (3.67)		
	low	11.8 (4.92)	$P = 0.59$	14.3 (3.45)	$P = 0.024$	$P = 0.037$
	high	10.9 (5.77)	(−4.28, 2.57)	18.2 (7.04)	(0.622, 7.19)	(−9.2, 0.317)
Serum Osteocalcin, μg/L	habitual	6.17 (3.39)		8.12 (2.85)		
	low	6.58 (3.69)	$P = 0.024$	8.67 (3.04)	$P = 0.664$	$P = 0.421$
	high	6.06 (3.39)	(−0.85, −0.076)	8.53 (3.02)	(−0.885, 0.589)	(−1.12, 0.488)
Parathyroid hormone (PTH), ng/L	habitual	25.8 (11.5)		32.9 (11.1)		
	low	35.3 (10.8)	$P = 0.085$	29.5 (6.8)	$P = 0.127$	$P = 0.02$
	high	28.8 (7.34)	(−14.9, 1.16)	32.4 (8.79)	(−0.974, 6.69)	(−17.7, −1.71)
Strontium Absorption % administered dose/L of plasma at 5 hours	habitual	1.11 (0.34)		1.14 (0.31)		
	low	1.22 (0.34)	$P = 0.922$	1.10 (0.27)	$P = 0.874$	NS
	high	1.22 (0.25)	(−0.12, 0.132)	1.11 (0.29)	(−0.086, 0.10)	

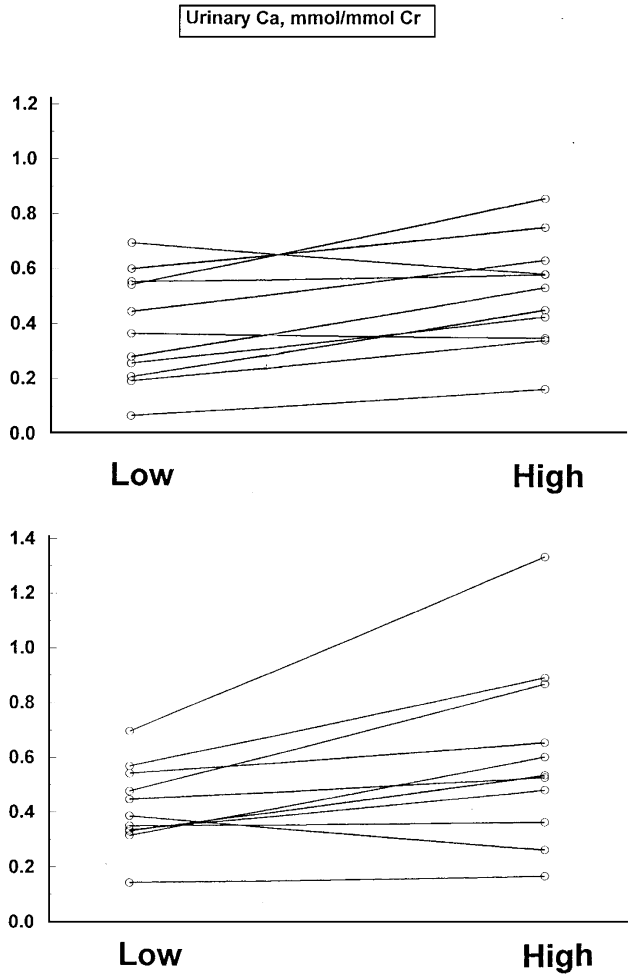

Figure 3 Changes in urinary calcium in eleven premenopausal women (upper panel) and eleven postmenopausal women (lower panel) on low (50 mmol) and high (300 mmol) sodium diets.

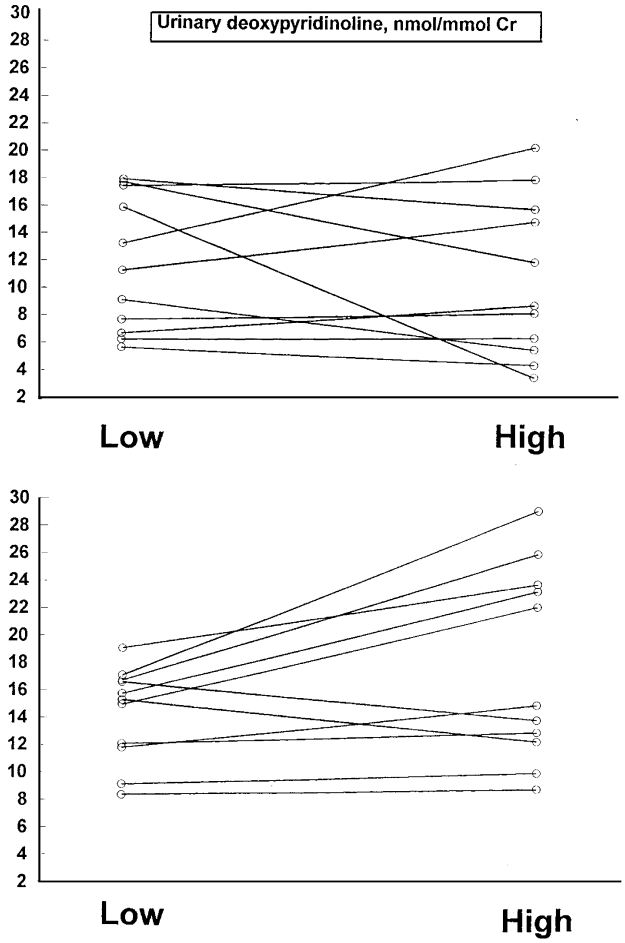

Figure 4 Changes in urinary deoxypyridinoline in eleven premenopausal women (upper panel) and eleven postmenopausal women (lower panel) on low (50 mmol) and high (300 mmol) sodium diets.

from a low to a high sodium diet was significant ($P = 0.037$).

In premenopausal women, serum osteocalcin decreased significantly on the high sodium diet by 8% from 6.58 μg/L to 6.06 μg/L ($P = 0.024$). However in postmenopausal women the difference was not significant (8.67 μg/L to 8.53 μg/L on the low and high sodium diet respectively). The change in osteocalcin in response to the change in dietary sodium was not significantly different in postmenopausal compared to premenopausal women (Table 1). Serum parathyroid hormone levels did not change significantly in either group, however, there was a significant difference between pre and postmenopausal women (Table 1). Strontium absorption did not change significantly in either group of women (Table 1).

Discussion

Postmenopausal women showed a significant mean increase in bone resorption of 27% (as measured by urinary deoxypyridinoline excretion) while following the high sodium diet compared with a low sodium diet. However, bone resorption in premenopausal women did not change significantly with a change in dietary sodium. It appears that postmenopausal women adapt differently, at least in the short term, to a sodium stress. Although there was no significant difference between levels of crosslinks in pre and postmenopausal women on the low sodium diet, on the high sodium dit (and also on the habitual diet) urinary crosslinks excretion, and therefore bone turnover, in postmenopausal women was significantly higher. Similar changes in hydroxyproline excretion have been seen in older women who had an average age of 67 y (McParland *et al*, 1989). There may be a gradual increase in bone turnover as a woman increases in age in response to a sodium stress or an increase in sodium sensitivity over time. There is no evidence that sodium intake increases with age and so it appears older women are more sensitive to a sodium load. A number of studies have shown that moderate increases in dietary sodium chloride, within the range of usual salt intakes, increase urinary calcium excretion. It can be seen from Table 2 that our results are similar to those obtained in previous studies.

Urinary sodium (not expressed per mmol of creatinine) was higher in the postmenopausal group on the low and the high sodium diet but these differences were removed when urinary sodium was expressed as mmol/mmol of creatinine. For total urinary sodium the differences were relatively small and unlikely to be of physiological significance. If the differences between urinary sodium and calcium are separated to look at the changes between the low sodium and habitual diet (50–110 mmol sodium) and habitual and the high sodium diet (110–260 mmol sodium) the change in mmol of urinary calcium per 100 mmol urinary sodium is 10 times higher when looking at lower levels of sodium

Table 2 Change in urinary calcium/100 mmol sodium in this study compared with previous studies

Reference	*Subjects*	*Change in diet Na (mmol)*	*Change in Ca/100 mmol*
Present study	Premenopausal women	50–300	+0.5
	Postmenopausal women		+0.7
Zarkadas *et al*, 1989	Postmenopausal women	usual +102	+0.6
Breslau *et al*, 1982	Young women and men	10–250	+0.6
Breslau *et al*, 1985	Postmenopausal and osteoporotic women	10–250	+0.5
Shortt *et al*, 1988	Young women	43–217	+0.7
McParland *et al*, 1989	Postmenopausal women	70–170	+0.6

(1.3 mmol calcium/100 mmol of sodium compared with 0.11 mmol calcium/100 mmol of sodium). The change in urinary crosslinks is less pronounced but still present with the change in crosslinks at lower levels of sodium (low-habitual diet) being just over double the change from habitual to high sodium diet. As sodium intake increases to higher levels, there appears to be proportionally smaller increases in urinary calcium.

It appears that postmenopausal women adapt to a high sodium diet by replacing the increased urinary excretion of calcium with increased bone resorption. Premenopausal women, on the other hand, do not appear to replace the increase in urinary calcium with an increase in bone resorption which forces us to conclude that there is an increase in gut absorption. Attempts to measure changes in calcium absorption using strontium did not show any change with changes in dietary sodium in either pre or postmenopausal women. However, measuring strontium absorption is known to have a large standard error (Blumsohn, 1995). The results from the calcium absorption measurements showed that in postmenopausal women calcium absorption did not change with dietary sodium intake at all. However, in premenopausal women there was an increase in calcium absorption on both the low and the high sodium diet. This could have been due to a slight decrease in calcium intake on the prepared diets despite precautions taken to avoid this happening.

Bone resorption increases with the menopause but the independent effect of ageing is not clear. There is some evidence that calcium intake declines by about 10% in women between 35 and 75 y of age (Blumsohn, 1995) but this is due to a reduction in overall caloric intake, and calcium density of the diet does not appear to decline with age. Ireland & Fordtran (1973) found that elderly women were less able to adapt to a low calcium diet and calcium absorption in elderly subjects on a low calcium diet (300 mg/d) was lower than in younger subjects. This may be because the calcitriol response is blunted. Thus the increase in calcium absorption required in response to a sodium stress could be blunted in the elderly.

PTH levels did not tend to be higher on the high sodium diet as would have been expected. These results seem to contradict what would be expected to happen in response to a sodium stress. One explanation to this is that PTH responds to the sodium stress but the blood tests were taken early in the morning when the volunteer had not eaten for at least 10 h. Other studies have also shown how difficult it is to produce consistent results for PTH while following a high sodium diet (Zarkadas *et al*, 1989; Breslau *et al*, 1982; Chan *et al*, 1992). It is possible that PTH is significantly raised only in the first 3 d (McCarron *et al*, 1981).

Mean serum osteocalcin was higher in the postmenopausal women that in the premenopausal women for all the diets as would be expected (Eastell *et al*, 1991). A surprising observation was the decrease in osteocalcin in premenopausal women in response to increased sodium intake. The reason for this is not clear.

Conclusions

It is clear that in women of all ages an increase in dietary sodium results in an increase in urinary calcium excretion. This relationship is probably not linear, but within the range of habitual dietary intakes, namely 70–250 mmol/d in the UK, an increase of 100 mmol.d results in an increase in urinary calcium excretion of 0.5–0.7 mmol/d. If this amount of calcium came only from the skeleton, then this would result in an additional rate of bone loss of 1%/year. An increase in calcium absorption may be the adaptive mechanism in young adults but an increase in bone resorption may be more important in the elderly. Maladaptation to a sodium stress may be important in the development of postmenopausal osteoporosis. A low sodium diet results in a decrease in bone resorption and may be an appropriate preventative measure.

These results could have important consequences on dietary information given to women at risk of osteoporosis. If this difference in adaptation continued over the long term it would inevitably lead to bone loss over the years. There is evidence that high levels of urinary sodium excretion are associated with an increase in bone loss at the hip site (Devine *et al*, 1995) in women who are at least 10 y past the menopause.

Daily sodium intake for women in the UK varies between 90 and 440 mmol (DOH, 1991) and the UK estimated value of the mean intake in women is 132 mmol (Gregory *et al*, 1990). Healthy women can maintain sodium balance on much lower intakes so there is no risk of deficiency and no cause for concern in recommending that women reduce their sodium intake—other benefits of a low salt diet are well documented. It seems sensible therefore that postmenopausal women at risk of osteoporosis should be advised to reduce their salt intake.

References

Bingham SA, Murphy J, Waller E, Runswick SA, Neal G, Evans D & Cummings JH (1992): Para-Amino benzoic acid in the assessment of completeness of 24-hr urine collections from hospital outpatients and the effect of impaired renal function. *Eur. J. Clin. Nutr.* **46**, 131–135.

Blumsohn A & Eastell R (1995): Measurement of intestinal calcium absorption by using stable strontium: authors reply. *Clin. Sci.* **88**, 243–244.

Breslau NA, McGuire JL, Zerwekh JE & Pak CYC (1982): The role of dietary sodium on renal excretion and intestinal absorption of calcium and on vitamin D metabolism. *J. Clin. Endocrinol. Metab.* **55**, 369–373.

Breslau NA, Sakhaee K & Pak CYC (1985): Impaired adaptation to salt-induced urinary calcium losses in post menopausal osteoporosis. *Trans. Assoc. Am. Physic.* **98**, 107–115.

Chan ELP, Ho CS, MacDonald D, Ho SC, Chan TYK & Swaminathan R (1992): Interrelationships between urinary sodium, calcium hydroxyproproline and serum PTH in healthy subjects. *Acta Endorcrinol.* **127**, 242–245.

Colwell R, Russell RGG & Eastell R (1993): Factors affecting the assay of urinary 3-hydroxypyridinium cross-links of collagen as markers of bone resorption. *Eur. J. Clin. Invest.* **23**, 341–349.

Devine A, Criddle RA, Dick IM, Kerr DA & Prince RL (1995): A Longitudinal study of the effect of sodium and calcium intakes on regional bone density in postmenopausal women. *Am. J. Clin. Nutr.* **62**, 740–745.

DOH (1991): *Dietary Reference Values for Food Energy and Nutrients for the United Kingdom.* London: HMSO.

Eastell R, Yergey AL, Vieira NE, Cedel SL, Kumar R & Riggs BL (1991): Interrelationship among vitamin D metabolism, true calcium absorption, parathyroid function, and age in women: Evidence of an age-related intestinal resistance to 1,25-Dihydroxyvitamin D action. *J. Bone Miner. Res.* **6** (2): 125–132.

Goldfarb S (1989): Dietary factors in the causation of negative-calcium balance in osteoporosis. A CPC Series. *Cases in Metabolic Bone Dis.* **3** (4): 1–8.

Goulding A (1981): Fasting urinary sodium/creatinine in relation to calcium/creatinine and hydroxyproline/creatinine in a general population of women. *NZ Med. J.* **93**, 294–297.

Gregory J, Foster K, Tyler H & Wiseman M (1990): *The Dietary and Nutritional Survey of British Adults.* London: HMSO.

Ireland P & Fordtran JS (1973): Effect of dietary calcium and age on jejunal calcium absorption in humans studied by intestinal perfusion. *J. Clin. Invest.* **52**, 2672–2681.

Kesteloot H & Joossens JV (1990): The relationship between dietary intake and urinary excretion of sodium, potassium, calcium and magnesium: Belgian International University research on Nutrition and Health. *J. Hum. Hypertension* **4**, 527–533.

McCarron DA, Rankin LI, Bennett WM, Krutzik S, McClung MR & Luft FC (1981): Urinary calcium excretion at extremes of sodium intake in normal man. *Am. J. Nephrol.* **1**, 84–90.

McParland BE, Goulding A & Campbell AJ (1989): Dietary salt affects biochemical markers of resorption and formation of bone in elderly women. *BMJ* **299**, 834–835.

Need AG, Morris HA, Cleghorn DB, De Nichilo D, Horowitz M & Nordin BEC (1991): Effect of Salt Restriction on Urine Hydroxyproline Excretion in Postmenopausal Women. *Arch. Intern. Med.* **151**, 757–759.

Sabto J, Powell MJ, Breidahl MJ & Gurr FW (1984): Influence of urinary sodium on calcium excretion in normal individuals. *Med. J. Australia* **140**, 354–356.

Shortt C, Madden A, Flynn A & Morrissey PA (1988): Influence of dietary sodium intake on urinary calcium excretion in selected Irish individuals. *Eur. J. Clin. Nutr.* **42**, 595–603.

Zarkadas M, Gougeon-Reyburn R, Marliss EB, Block E & Alton-Mackey M (1989): Sodium chloride supplementation and urinary calcium excretion in postmenopausal women. *Am. J. Clin. Nutr.* **50**, 1088–1094.