Calcium supplementation of the diet: justified by present evidence

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In a recent review Kanis and Passmore concluded that there was no case for supplementation of the diet with calcium for the prevention or treatment of osteoporosis.[1] We consider that present evidence, taken as a whole, points to a different conclusion. We consider the matter under five main headings: experimental calcium deficiency; human calcium requirements; the relation between calcium intake, bone mass, and bone loss; the relation between calcium and hip fracture; and the role of calcium in established osteoporosis.

Experimental evidence

The evidence that dietary calcium deficiency causes osteoporosis in adult animals was reviewed by one of us many years ago,[2] has seldom been challenged, and has often been confirmed[3-8]; deficiency may also cause osteomalacia in young animals.[9] Even the osteoporosis of experimental oophorectomy is itself dependent on calcium[4 5 8] and sodium intake.[10] Calcium deficiency causes osteoporosis in mammals because calcium continues to be lost in the faeces and urine even when calcium intake is restricted. The resulting negative calcium balance is met by bone resorption mediated by parathyroid hormone,[11] which maintains the all important concentration of ionised calcium in the plasma at the expense of the skeleton. The osteomalacia of vitamin D deficiency is not due to malabsorption of calcium but to loss of the calcaemic action of vitamin D on bone.[12] Thus calcium deficiency causes osteoporosis in adult animals; vitamin D deficiency causes osteomalacia.

Calcium requirements in humans

YOUNG ADULTS

The calcium requirement of human adults is generally defined as the intake at which calcium intake and output are equal, which is the same as the value at which net absorbed calcium and urinary calcium are equal. This value can be determined only by measuring calcium balance on different calcium intakes. The many hundreds of careful balance studies performed in Europe and the United States in the past 50 years converge on a mean calcium requirement of about 500-600 mg/day in normal young adults.[13 14] This is illustrated in figure 1, which shows that net absorbed calcium is equal to urinary calcium excretion when they are both about 150 mg/day. The intake required to provide this net absorption is about 550 mg/day, which is therefore the mean requirement. The allowance needed to meet this requirement in 95% of normal subjects is about 800-1000 mg.[15] Even in the widely quoted work of Malm on male Norwegian prisoners, some of whom took a year or more to "adapt" to low calcium intakes, the final computed mean calcium requirement was 420 mg/day.[16] To achieve this figure many of the subjects were in prolonged negative calcium balance and must therefore have lost significant amounts of bone before the final equilibration.

Even these calculations probably underestimate the true calcium requirement since they do not allow for dermal losses, recently estimated at 60 mg daily,[17] which must add several hundred milligrams to the requirement because fractional net absorption diminishes as intake increases (fig 1). Children, of course, need more calcium to ensure a positive calcium balance for bone growth, and even in the third decade of life a small positive calcium balance is still required.[18]

NORMAL POSTMENOPAUSAL WOMEN

Urinary calcium excretion rises at the menopause. This can be seen in a 24 hour urine sample but is much more apparent in the calcium:creatinine ratio after an overnight fast.[19] This is illustrated in figure 2, which shows calcium/creatinine values in premenopausal and postmenopausal women at 9 pm and 9 am (fasting).[20] There is no difference between the two groups in the evening but a clear difference in the morning, by which time the premenopausal women have adapted to their overnight fast while the postmenopausal women have failed to do so.

Since calcium absorption does not rise at the menopause—rather it appears to fall[21 22]—this extra urinary calcium must be drawn from bone.[21-23] This increased loss increases the requirement by at least 200 mg calcium daily[14 23 24] and increases the allowance correspondingly. A simultaneous fall in calcium absorption would increase the calcium requirement and allowance still further.[22] Associated with these events, there is an increase in bone turnover, which can be seen in the biochemical markers of bone formation and resorption,[19 25] in kinetic measurements,[26] and on histological examination of bone.[27 28] The crucial question, of course, is whether this increase in bone turnover is the cause or the result of the increased calcium losses from the body.

Increased bone resorption is widely assumed to be the primary event at the menopause, raising plasma and urinary calcium values and reducing parathyroid hormone and calcitriol production.[29] However, the increase in plasma calcium concentration at the menopause occurs wholly or mainly in the complexed fraction (due to a rise in plasma bicarbonate),[30] and neither parathyroid hormone nor serum 1,25-dihydroxyvitamin D concentrations appear to change at the menopause.[31 32] Moreover, the correlation between calcium absorption and urinary calcium excretion is positive,[33] whereas to satisfy the conventional hypothesis it should be negative. In addition, the rise in urinary calcium excretion at the menopause is greater than can be accounted for by the increase in ultrafilterable calcium,[34 35] which suggests the loss of a positive oestrogen effect on tubular reabsorption of calcium analogous to that on tubular reabsorption of sodium.[36] It is hard to see how a primary increase in bone resorption could cause this chain of events but easy to see how excess calcium loss could increase bone resorption. The fact that urinary hydroxyproline excretion is related to sodium intake[37 38] again suggests that bone resorption represents a response to a demand rather than a primary event.

Oestrogen administration lowers urinary calcium and hydroxyproline values and subsequently plasma alkaline phosphatase activity.[39] These effects are generally attributed to a direct effect of oestrogen on bone, an interpretation supported by the discovery of oestrogen receptors in bone cells[40] and compatible with the idea that oestrogen modulates the sensitivity of

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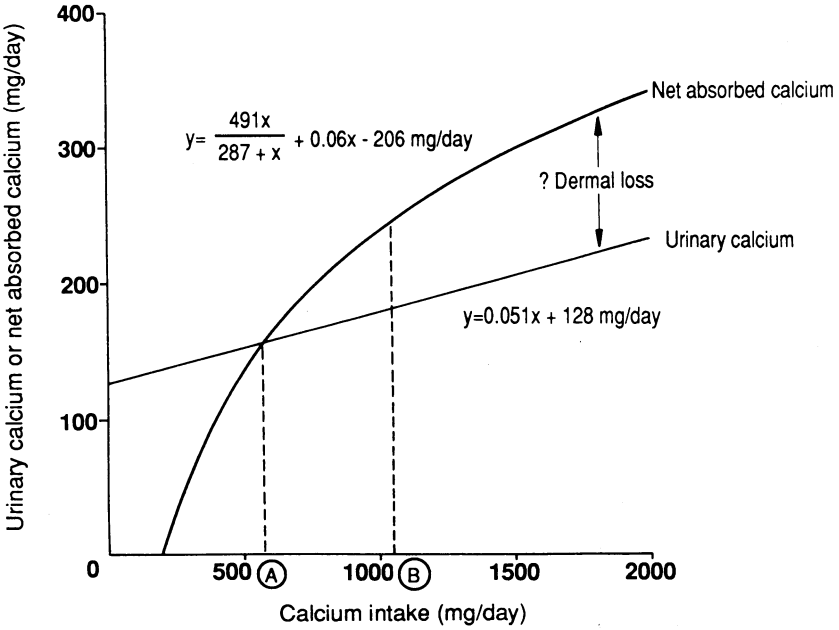

FIG 1—*Relation between calcium intake, net absorbed calcium, and urinary calcium calculated from 212 calcium balances in 85 normal subjects.[13] Absorbed and excreted calcium are equal, and balance zero, at an intake of 550 mg (A); this is the mean calcium requirement. When dermal losses are taken into account, however, true zero balance is not achieved until absorbed calcium reaches about 210 mg, raising the estimated requirement to about 1100 mg (B). The equations of the lines are shown; the final terms (5·1% and 6·0%) are not significantly different from each other*

bone to parathyroid hormone[41,42] or the calcium setpoint in the parathyroid glands.[43,44] There are also oestrogen receptors in the kidneys,[45] however, and the reduction in urinary calcium excretion could be due to a direct effect of oestrogen on tubular reabsorption of calcium. A reduction in urinary calcium values from, say, 5 to 3·5 mmol/day[46] reduces the mean calcium requirement by several hundred milligrams. To achieve the same effect by calcium supplementation requires an increase in calcium intake of the order of 1000 mg/day because the net absorption of calcium added to the average normal diet is only about 6-10% (see fig 1). This may help to explain why oestrogen treatment seems to be more "efficient" than calcium treatment.

Calcium intake and postmenopausal bone loss

The evidence that oestrogen treatment in adequate dosage inhibits postmenopausal bone loss is, of course, conclusive. The controversial issue is the role of dietary calcium intake and the value of calcium supplementation.

The evidence of a link between calcium intake and bone state in population studies is conflicting. Garn's studies in Central America showed no relation between calcium intake and metacarpal bone mass on a national basis.[47] The Matkovic study, on the other hand, found a significantly higher bone mass in a rural community with a high calcium intake than in a comparable community with a much lower calcium intake.[48]

Prospective studies of the relation between calcium intake and the rate of bone loss have also produced conflicting results. In one of these, however, all the subjects were given the same calcium supplement of 500 mg,[49] which would tend to obscure the effect of dietary calcium. In another, 38 of the 54 women in the "trial" were given calcitonin or oestrogen,[50] which would also obscure the effect of diet. A nine month study of 522 normal women showed a weak relation between calcium intake and bone loss[19]; another showed a relation between calcium intake and bone loss in the humerus but not the radius[51]; and another showed faster bone loss from the spine in 19 women with calcium intakes below 405 mg than in 19 with intakes over 777 mg.[52] Riggs found no correlation between calcium intake and bone loss in 106 premenopausal and postmenopausal women.[53] In an exercise study on 64 postmenopausal women, however, there was a significant inverse relation between calcium intake and the change in vertebral density.[54]

The evidence for a relation between bone density, bone loss, and estimated calcium intake in individuals and populations is therefore somewhat inconclusive. We think that a major reason for this is the large intrinsic error in diet histories and the even larger error arising from the assumption that current calcium intake is the same as average lifetime intake, or even remains constant over the time intervals between sequential bone mass measurements. Of perhaps equal importance are the confounding effects of other dietary constituents, notably phosphorus[23] (which may decrease net calcium absorption) and protein and sodium (both of which increase urinary calcium loss),[55,56] and, of course, the differences in obligatory calcium loss between individuals.

Calcium supplementation

The most widely quoted studies of calcium supplementation are those of Riis *et al* (on 14 women)[57] and Ettinger *et al*.[58] Both showed a failure of calcium to inhibit bone loss from the spine and distal forearm but an effect of calcium on bone loss from the proximal forearm and whole skeleton in women very close to the menopause. However, when a calcium supplement was added to an otherwise ineffective small dose of oestrogen vertebral bone loss was also inhibited.[58]

Most other studies have shown a positive effect of calcium on bone which has not always reached significance but has generally been intermediate between no treatment and oestrogen treatment. Figure 3 shows the cumulative changes in cortical area in the three groups of the Horsman study[59] and the corresponding data from the paper by Recker *et al*.[60] Both these studies showed an effect of calcium on bone loss which was intermediate between oestrogen treatment and no treatment. In a much larger study there was no change in mean forearm bone density in

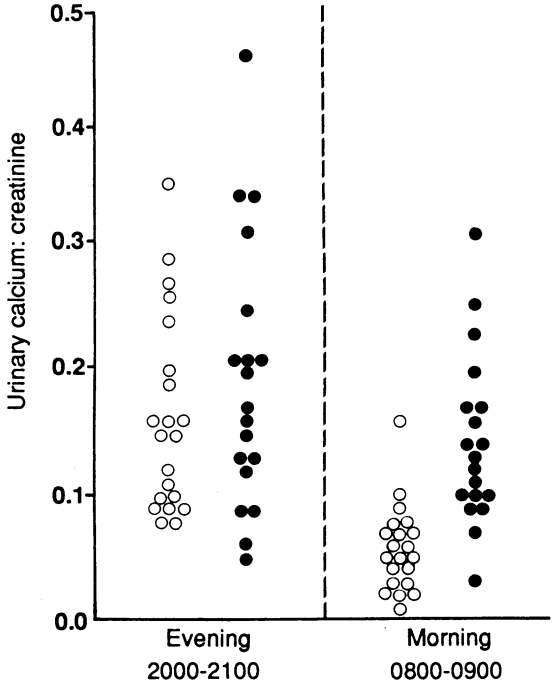

FIG 2—*Urinary calcium:creatinine ratios (mg/mg) in normal premenopausal (○) and postmenopausal (●) women. At 9 pm there is no difference between them but after a 12 hour fast the premenopausal women have lowered their calcium excretion and the postmenopausal women have not*

calcium supplemented women, although this was only just significantly different from the untreated controls.[61] Another study showed no loss of bone from the forearm in 24 calcium treated 70 year old women[62]; another showed an effect of calcium in 80 year old women.[63] The latest prospective study has shown a significant effect of calcium supplementation on bone loss in all 12 bone variables measured in 44 postmenopausal women compared with 38 controls.[64] Two very recent studies (published as abstracts only) have shown a positive effect of calcium in preventing bone loss.[65,66]

Calcium intake and hip fracture

Ultimately bone mass per se is less important than fractures, and in preventing fractures the evidence for a calcium effect is strong. Matkovic *et al* showed a 60-75% lower incidence of hip fractures in women from a community whose dietary calcium was about 1000 mg/day than in women with half that intake.[47] More recently, in a prospective study Holbrook *et al* found a 60% lower hip fracture rate in both men and women in the upper tertile of calcium intake (over 765 mg/day) than in those in the lower tertile (below 470 mg/day).[67] In a Hong Kong study there was a significant inverse relation between hip fractures and calcium intake.[68] In a British study there was a relation between calcium intake and hip fractures in men but not in women.[69] Of particular interest is a recent study showing a lower incidence of hip fractures in subjects treated with a thiazide diuretic than in those not treated with a thiazide diuretic.[70] Taken in conjunction with other studies showing increased bone mass or bone density in thiazide treated women[71] this finding must support the view that calcium deficiency is an important factor in the development of osteoporosis since the calcium sparing action of thiazides is renal in origin and unlikely to be due to a direct action on bone.

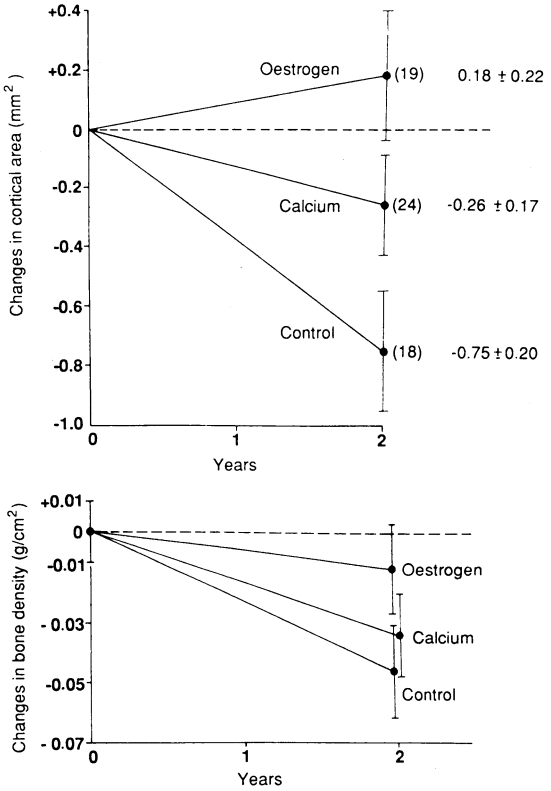

FIG 3—*Top: cumulative changes in mean metacarpal cortical area in controlled trial of calcium and oestrogen supplementation in postmenopausal women.*[59] *Bottom: cumulative changes in mean bone density in distal forearm in placebo treated, hormone treated, and calcium treated postmenopausal women.*[60] *Error bars are 1 SE*

Established osteoporosis

The potential role of calcium deficiency is very apparent in postmenopausal women with severe osteoporosis who present with vertebral compression fractures. These patients have a higher fasting obligatory calcium loss than normal postmenopausal women, a higher urinary hydroxyproline concentration, and, commonly, a significantly reduced absorption of calcium.[72] The urinary hydroxyproline concentration is positively related to the urinary calcium concentration and inversely related to calcium absorption[72] and can be reduced into the normal postmenopausal range by administering calcium supplements or calcitriol, or both.[73-76] This response to intervention suggests that the high bone resorption in these cases is the result rather than the cause of the low absorption and high excretion of calcium. There is also some evidence of an absolute or relative impairment of bone formation in some of these patients,[27,77] which is perhaps causally related to their low adrenal androgen concentrations.[78] This may be regarded as an additional risk factor which delays the restoration of bone destroyed in response to the calcium demand.

Calcium supplements are widely used in managing clinical osteoporosis, but we know of only one controlled trial in which they have been randomly compared with no treatment[79]; the effect of calcium was positive. When calcium has been used as a placebo against which other treatments have been compared, the placebo treated controls have shown no significant loss of bone in at least three studies.[80-82] In two large clinical studies without random allocation calcium supplemented patients lost less bone or suffered fewer fractures than untreated patients.[83,84] In a study in which calcium supplements were given alone to osteoporotic patients with normal calcium absorption and combined with small doses of calcitriol in those with calcium malabsorption the small loss of bone was not significant with either treatment.[85]

Conclusions

Although an observer can always find fault with virtually every publication which does not match his or her model of the relevant physiology, we consider that the evidence taken as a whole points to an important role for calcium in the genesis and management of postmenopausal osteoporosis. Calcium supplementation may not be as effective as oestrogen in preventing early postmenopausal bone loss but this may simply be a matter of dose, timing of administration, and possibly even compliance. Alternatively, there may be some fundamental difference between the pathogenesis of the exponential loss of bone immediately after the menopause and the subsequent linear age related component.[86] This issue should be resolved before universal oestrogen administration can be recommended.

Although there have certainly been some negative results with calcium, particularly in respect of trabecular bone, it might be more profitable to explore further the calcium model[87] than to abandon it. In the future, bone densitometry will identify the menopausal women at risk for osteoporosis who require preventive treatment with calcium or oestrogen, or both. Until then all postmenopausal women should be advised to ingest more calcium than they ingested before the menopause, preferably taking part at least of this extra calcium at night. Kanis and Passmore made the point for us admirably (though perhaps not very scientifically) in their unexpected conclusion: "Even if calcium were only 1% more effective than placebo in reducing the incidence of fractures it would prevent more than 1000 fractures

a year in Britain." The evidence suggests that a significant component of the osteoporosis which affects so many postmenopausal women in the West is attributable to a relative or absolute inadequacy of calcium intake and hence is potentially and easily preventable.

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After the Asylums

What does community care mean now?

Trish Groves

Relatives and friends provide the bulk of community care for about two million chronically ill and disabled people under 65 in England and Wales.[1] Nobody knows how many care for people with mental illness nor how many find that job difficult, though the experiences of voluntary organisations such as the National Schizophrenia Fellowship give some idea. The fellowship has over 6000 members and 150 local groups and in 1988 received more than 5000 appeals for help and advice (table I) from schizophrenic people and their relatives.[2]

One of the hardest things about caring for mentally ill relatives and friends at home is that it is often a forced choice (box). A young patient with chronic schizophrenia may have only two other options on leaving hospital—a hostel or bed and breakfast accommodation. Middle aged patients may have even less choice if their parents are old and their siblings are too busy with careers and children. Even when living at home is the first choice, it may not be good for patients or relatives: in schizophrenia it can worsen prognosis[3] and can hasten relapse if it engenders an emotionally charged atmosphere.[4] Relatives may become anxious and depressed and shunned by embarrassed friends,[5] so that they have to turn to strangers for help.

"He has spent half of the 13 years of his illness in and out of hospital and prison for minor offences, set fire to his mother's house, and attempted suicide three times in one year. The third time, the hospital tried to send him home on a bus with his pills. He is seen once a week by a psychiatric nurse who administers his antipsychotic injections. The brief visit is the only support he and his mother receive. He refuses to go to a day centre because he thinks that people are staring at him.

'He was discharged into a community care system that turned out to be me,' says his mother."—One schizophrenic patient's story, reported in *The Times* on 12 July 1989.

TABLE I—*Number of calls to the National Schizophrenia Fellowship in 1988 according to topics of concern*

Deficiencies identified	No of calls
General advice	3066
Community care and treatment	678
Family support	546
Accommodation	315
Social services	282
Obtaining benefits	198
Legal inquiries	153
Hospital care	84
Total	5322

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Hostels can be happy and healthy places to live. But how many are there, and how many more do we need?

The government recognises these stresses and expects social services authorities to arrange support for these informal carers. But it also states that its first aim in reorganising community care is to enable more ill and disabled people to live at home whenever possible.[1] For many severely mentally ill people living at home will not be possible unless they and their carers can get help during crises as well as in the long term. This means that there must be properly funded emergency clinics, out of hours social work teams, respite care beds, and special family therapy[6,7] in addition to less formal support. And for those who cannot cope alone or live with relatives there must be suitable accommodation.

NHS provision

The exact number of hospital beds for people with long term mental illness is not known. Perhaps this deficiency is predictable because data on admissions, bed use, and discharges are notoriously inaccurate in