CORRESPONDENCE

We may return unduly long letters to the author for shortening so that we can offer readers as wide a selection as possible. We receive so many letters each week that we have to omit some of them. Letters should be typed with double spacing between lines and must be signed personally by all their authors, who should include their degrees. Letters critical of a paper may be sent to the authors of the paper so that their reply may appear in the same issue.

Correspondents should present their references in the Vancouver style (see examples in these columns). In particular, the names and initials of all authors must be given unless there are more than six, when only the first three should be given, followed by et al; and the first and last page numbers of articles and chapters should be included.

Non-hormonal treatment of osteoporosis

SIR,—Dr A St J Dixon (25 March, p 999) implies that insufficient calcium intake is responsible for loss of bone mineral and that dietary supplementation of calcium will prevent it. He gives little evidence, however, to support this view.

As discussed in our recent review,[1] and in subsequent correspondence, there is no good evidence that oral calcium supplements can prevent the commonest forms of osteoporosis—that is, postmenopausal and senile. Furthermore, contrary to the statement that the failure of Western society to consume enough calcium must inevitably lead to a loss of bone mineral, osteoporosis is in fact more common in societies with a high calcium intake than in those with a low calcium intake.[2]

The claim that microcrystalline hydroxyapatite protects patients with rheumatoid arthritis having corticosteroid treatment from the usual accelerated loss of bone is not fully substantiated by his own study.[3] Compared with controls the patients treated with microcrystalline hydroxyapatite had a small but significant reduction in loss of radial bone density when calculated as bone mineral content/bone width, but not when calculated as percentage of initial density. Since there was no difference in loss of ulnar bone density between treated patients and controls, it is extremely difficult to draw any meaningful conclusions.

The second study quoted using microcrystalline hydroxyapatite was a trial of treatments for a rare cause of osteoporosis—primary biliary cirrhosis.[4] The findings were that microcrystalline hydroxyapatite reversed the bone loss which is normally seen in this condition. These findings, however, were based on metacarpal morphometry using Garn's index,[5] which has several shortcomings as an assessment of bone mass.[6] In any event, no information on the treatment of other types of osteoporosis can be derived from the study. Certainly, accurate assessments of bone mass by total body calcium measurements using neutron activation analysis have again shown that large oral calcium supplements are unable to stop bone loss in women with postmenopausal osteoporosis.[7]

Fluoride treatment seems to have some effect on osteoporosis by stimulating bone formation. It is not fully established, however, whether the bone thus formed has normal strength and can give the mechanical advantage required in this condition. Riggs *et al*[8] suggested that fluoride treatment can reduce vertebral fracture rates in women with postmenopausal osteoporosis, although it was not as effective as oestrogen in this respect. It would be preferable, however, to see the results of a study where treatment allocations were randomised before drawing firm conclusions. A place for fluoride in the treatment of osteoporosis needs to be established, but the fact that about 40% of patients treated with the necessary doses of fluoride had adverse reactions[8] is always likely to limit its use.

At a time when osteoporosis is, if anything, on the increase[9] we think that it is most regrettable that the myth of calcium supplementation alone being effective in either the prevention or treatment of osteoporosis should continue to be potentiated.

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1 Stevenson JC, Whitehead MI. Postmenopausal osteoporosis. *Br Med J* 1982;**285**:585-8.
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3 Nilsen KH, Jaysen MIV, Dixon A St J. Microcrystalline calcium hydroxyapatite compound in corticosteroid-treated rheumatoid patients: a controlled study. *Br Med J* 1978;ii:1124.
4 Epstein O, Kato Y, Dick R, Sherlock S. Vitamin D, hydroxyapatite, and calcium gluconate in treatment of cortical bone thinning in postmenopausal women with primary biliary cirrhosis. *Am J Clin Nutr* 1982; **36**:426-50.
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8 Riggs BL, Seeman E, Hodgson SF, Taves DR, O'Fallon WM. Effect of the fluoride/calcium regimen on vertebral fracture occurrence in postmenopausal osteoporosis. *N Engl J Med* 1982;**306**: 446-50.
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SIR,—The leading article by Dr Allan St J Dixon (26 March, p 999) reiterates two notions about bone loss that were popular two decades ago. The first is that adult bone loss is a relatively recent phenomenon among Western peoples on a diet rather high in animal protein, say 60 g/day. The second is that it is associated with a low calcium intake and can be prevented by calcium intakes of 1 g/day and more.

From national and international studies conducted over the last two decades we have learnt that adult "osteoporosis" is international and by no means restricted to women in the Western world.[1] It exists in American Indians and Japanese, Jamaicans and Eskimos, Guatemalan Indians and Yugoslavians, Chinese and Greeks, and in American blacks.[2] Indeed, there is no population not characterised by bone loss after the age of 40, and all