**Letters to the Editor**


doi:10.1093/ageing/afl158
Published electronically 17 January 2007

**Reply**

Vitamin B₁₂ deficiency is common in older people and is associated with cognitive impairment in the absence of anaemia or macrocytosis [1–3]. The prevalence of vitamin B₁₂ deficiency increases in the elderly, mainly due to malabsorption of food-bound vitamin B₁₂ due to atrophic gastritis that limits the ability of older people to release vitamin B₁₂ from dietary sources in meat, fish and dairy products. In reply to Dr Vargese, there was no association between alcohol consumption and vitamin B₁₂ status in the Banbury B₁₂ population study [1]. Thyroid function was not measured in our population study and so we cannot speculate on any such association [1]. However, the important finding of the Banbury B₁₂ study was that almost all cases of undiagnosed vitamin B₁₂ deficiency did not have anaemia or macrocytosis. The high prevalence of undiagnosed vitamin B₁₂ deficiency is relevant to clinical practice and suggests that it would be prudent to measure vitamin B₁₂ or holotranscobalamin, the metabolically active fraction of vitamin B₁₂, in older people presenting with symptoms suggestive of dementia or cognitive impairment. Correction of established vitamin B₁₂ deficiency in the early stages is appropriate, particularly among those with relevant symptoms. Nevertheless, it is unclear if correction of vitamin B₁₂ deficiency could attenuate the rate of cognitive decline in older people. Randomised evidence for the effects of 3 to 7 years of treatment with B vitamins on cognitive function should be available from ongoing trials of B vitamin supplementation for the prevention of cardiovascular disease in due course. The results of these trials are required before making any recommendation on the use of B-vitamins in patients with established cardiovascular disease for the prevention of dementia. Further large-scale randomised evidence of vitamin B₁₂ supplementation for the maintenance of cognitive function is required in older people in the absence of cardiovascular disease or dementia [4].

**Vitamin D supplementation and the prevention of fractures and falls**

**SIR**—The use of vitamin D supplementation in elderly patients to prevent fractures remains a controversial issue. Although the study by Law [1] did not show a reduction in non-vertebral fractures with vitamin D, its contribution to the argument against its efficacy in fracture prevention is questionable. There are major limitations of the study, which were not mentioned in its discussion. Importantly, it was not a double-blind, placebo-controlled study, which weakens the significance of its findings. Also, the mean duration of follow-up was only 10 months, and it is unclear as to whether this was taken into consideration in calculating the statistical power of the study. As it is known that vitamin D’s effects on bone resorption are relatively modest [2], it could not have been expected to see a significantly positive result from a trial with such a short mean duration. None of the currently approved treatments for osteoporosis in double-blind, placebo-controlled studies have prospectively shown a reduction in non vertebral fractures in anything <12 months treatment and follow-up [3].

Further studies, adequately powered and with robust methodologies, are required before this controversy can be finally put to rest.

**Conflicts of interests**

Conflict of Interest: None

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Table 1. Incidence of non-vertebral fractures and falls in participants allocated vitamin D and control over the second year of a randomised controlled trial (omitting fractures and falls occurring in the first 12 months)

<table>
<thead>
<tr>
<th></th>
<th>Vitamin D</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of participants</td>
<td>307</td>
<td>726</td>
</tr>
<tr>
<td>No. (%) of participants with at least one:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-vertebral fracture</td>
<td>9 (1.8%)</td>
<td>10 (1.4%)</td>
</tr>
<tr>
<td>Fall</td>
<td>128 (25%)</td>
<td>180 (25%)</td>
</tr>
</tbody>
</table>

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