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].

Letters to the Editor


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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Letters to the Editor

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Reply

Sir—As Dr Zeimer states, our randomised controlled trial of vitamin D supplementation was not double-blind [1]. However, a double-blind design is more important when the trial outcome is a subjective one (such as well-being or pain relief); its absence should not have been critical in our trial where the outcome measures (fractures and falls) were more objective. While the mean duration of follow-up in our trial was only 10 months, 1,233 participants continued in the trial for > 12 months (up to 20 months) and Table 1 shows that there was no reduction in non-vertebral fractures or falls in participants allocated vitamin D even in the second year. Also, one would expect any effect of vitamin D in preventing non-vertebral fractures to be evident within the first year because most of the effect on bone density is attained within the first year. A two-year randomised trial of vitamin D in elderly women showed that femoral neck bone density was on average 1.7% greater in women allocated vitamin D than placebo by the end of the first year, and only 2.3% greater by the end of the second year [2] (most of the effect was therefore evident within the first year). Another such trial produced similar results [3]. Vitamin D might also prevent fractures by reducing the risk of a fall, and while our trial showed no reduction in falls, it is recognised that people with very low serum 25(OH) vitamin D concentration have impaired muscle function and power that predispose to falling [4], and a study of vitamin D supplementation in such women showed substantial improvement in muscle function and power after only 6 months, with much of the improvement apparent after 3 months [4]. Moreover, in the two trials of vitamin D supplementation (with or without calcium) showing the greatest reduction in incidence of non-vertebral fractures in treated patients, the proportional reduction in fractures in the first year was similar to that during the second and third year of the two trials [3, 5]. We conclude therefore that the failure of our trial to show the effect of vitamin D in preventing fractures was not attributable to short duration; rather, that it supports the results of larger recently published trials in showing no protective effect of vitamin D.

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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Video phone diagnosis of ‘funny turns’

SIR—We wish to highlight the usefulness of video phone technology in the diagnosis of ‘fits, faints and funny turns’ in the elderly. Recently, an elderly lady, known to have cerebrovascular disease, was admitted with a possible seizure. After a brief stay, she was discharged and reviewed in our outpatient clinic. Here, she was accompanied by her son who had recorded two further ‘funny turns’ on his video phone. These clips demonstrated seizures, and we were thus