The paper by van Asselt et al (1) in this issue of the Journal, like much good research, raises more questions than it answers. The authors examined the prevalence of mild vitamin B-12 deficiency in elderly Dutch participants in the European SENeca Study (Study in Europe on Nutrition and the Elderly, a Concerted Action). Based on high methylmalonic acid (MMA) concentrations and low to low-normal plasma vitamin B-12 concentrations (<260 pmol/L), an astounding 23% of apparently healthy, free-living Dutch elderly (74–80 y old) were diagnosed as having mild vitamin B-12 deficiency. This itself is an important finding in view of the fact that vitamin B-12 deficiency can lead to progressive neurologic disease and dementia.

In seeking reasons for this high prevalence of mild vitamin B-12 deficiency, the authors concentrated on 2 possible etiologies: insufficient dietary intake of vitamin B-12 and malabsorption of vitamin B-12 because of atrophic gastritis. However, of the 25 subjects in whom mild vitamin B-12 deficiency was diagnosed, only 1 had an insufficient dietary intake of vitamin B-12. Note that the Dutch recommended dietary allowance for vitamin B-12 is 2.5 μg, virtually the same as the newly revised US and Canadian recommendations of 2.4 μg (2). In the Dutch study, intake of vitamin B-12 was probably underestimated because of the dietary history checklist method that was used and because the amount of vitamin B-12 content is incomplete. Moreover, surprisingly little is known about the bioavailability of vitamin B-12 from different food matrices, such as dairy products.

Atrophic gastritis is a second possible explanation for the prevalence of vitamin B-12 deficiency in this Dutch population. As diagnosed by serum pepsinogen markers, atrophic gastritis was shown to have a prevalence of 31% in this elderly population, the same as that reported among elderly persons living on the East Coast of the United States, but much higher than the reported 10% of elderly Americans living in the Midwestern United States (3, 4). Malabsorption of vitamin B-12 in atrophic gastritis is caused by the inability to efficiently dissociate vitamin B-12 from food complexes and the subsequent binding of whatever free cobalamin is released from food complexes by intestinal bacteria. Intrinsic factor is not a problem in most elderly with atrophic gastritis. Despite the high prevalence of atrophic gastritis in the Dutch population, mild vitamin B-12 deficiency could be explained by atrophic gastritis in only 25% of cases. Thus, in the majority of cases of mild vitamin B-12 deficiency, no explanation was apparent.

In the Dutch population studied, severe atrophic gastritis, but not mild-to-moderate atrophic gastritis, was more frequent in the mildly deficient vitamin B-12 subjects than in the nondeficient subjects. However, malabsorption of food vitamin B-12 in atrophic gastritis is not an all-or-nothing phenomenon. Others have shown malabsorption of vitamin B-12 in milder forms of atrophic gastritis as well (5).

How did supplement use affect the results? Supplement use by US elderly is common; it is estimated that 30–40% of seniors take supplements. The prevalence of supplement use in Netherlands is much lower (14%) (1). Moreover, the amount of vitamin B-12 contained in the Dutch supplements is markedly lower than the amount found in the average US supplement (2 compared with 6 μg). In this study, elderly persons taking supplements had higher cobalamin concentrations but not necessarily lower MMA concentrations. In general, higher MMA concentrations are much more common in the elderly in Europe (~40%) than in the United States (~15%). It is probable that the greater use of vitamin B-12 supplements in higher doses and the availability of cereals that have been fortified with vitamin B-12 in the United States accounts for these differences in prevalence.

What are the main questions that this paper has raised that need further investigation? On the top of the list of important questions is the following: what is the clinical significance of an elevated MMA concentration? In this paper, the authors document anemia in only 8% of those with mild vitamin B-12 deficiency and make no statements about neurologic or cognitive function abnormalities. Second, if poor dietary intake and atrophic gastritis cannot explain mild cobalamin deficiency in elderly people in most cases, what does? Could bacterial overgrowth be bringing about much of the vitamin B-12 deficiency in the elderly? Is the metabolism of vitamin B-12 different in the elderly? Finally, it is obvious that we need better food tables for vitamin B-12 and that we need to learn much more about the bioavailability of vitamin B-12 from various food sources. It is possible that with future study the new recommended dietary allowances in the United States and Canada for vitamin B-12 will be found to be low. For now, the advice that the B Vitamin Panel gave for elderly people, to get their vitamin B-12 by eating foodstuffs fortified with the vitamin or by taking a vitamin B-12–containing supplement, seems warranted (6). The findings of the present study strongly support this view.

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