

Fractures, calcium, and the modern diet¹

D Mark Hegsted

ABSTRACT Although high calcium intakes have long been recommended to prevent osteoporosis, there is little evidence that high calcium intakes effectively prevent fractures. Osteoporotic fractures are, like coronary artery disease, largely a disease of Western societies. Recent evidence that the statins that block the mevalonate pathway, lower serum cholesterol concentrations, and improve cardiovascular disease risk also prevent fractures, together with the increasing evidence that diets high in fruit and vegetables are beneficial in preventing fractures, suggest common dietary etiologic factors. Further research in this area should answer the long-standing question: Why do populations who consume low-calcium diets have fewer fractures than do Western societies who consume high-calcium diets? *Am J Clin Nutr* 2001;74:571–3.

KEY WORDS Osteoporosis, fractures, calcium, statins, low-fat diets

Hip fractures are a major health problem of the elderly and occur in ≈1.5 million Americans annually; this number will presumably increase as the population ages (1). Efforts at prevention have centered on increasing calcium intakes, and the recommended calcium intakes (2, 3) are now so high that it is difficult, if not impossible, to devise practical diets that meet these recommendations. These recommendations are based largely on calcium balance studies and the effect of calcium supplements on bone mineral content.

Calcium requirements for adults have been defined as the amount of dietary calcium required to yield an equilibrium between intake and excretion, ie, zero calcium balance. At this intake the body neither gains nor loses calcium. However, unless the intake of any nutrient is below some critical level or at levels high enough to be toxic, the body necessarily adapts to the usual intake. The time required to adapt to a new intake varies greatly with different nutrients and it may take considerable time—weeks or months—for the body to adapt to a new calcium intake, as was shown by Malm (4) many years ago. Such long-term studies are exceedingly rare but, unless the balance study is long enough to allow adaptation to occur, the amount of calcium required to produce balance in any individual is simply the amount of calcium that individual usually eats. The calcium intake of much of the world's population is low by American standards yet these populations develop and perform well without obvious signs of deficiency and are in calcium balance, as must be expected, at approximately their usual low intake (5, 6). In contrast, calcium consumption by most Americans is rela-

tively high and they require more calcium to achieve balance. Thus, it is ironic that as we encourage people to consume more calcium, their calcium requirement—as defined by calcium balance—increases, with no end in sight.

Various population studies within the United States (7–10) and elsewhere (11–13) that studied the relation between estimated dietary calcium intakes and fractures have failed to show that high calcium intakes protect against fractures. Such data, of course, suffer from the well-known inaccuracies of all measures of dietary intake. We know from numerous studies that compared reported intakes with actual energy expenditures that most people underestimate their intakes (14–17). It is unclear, however, whether the intake from all food sources is equally underestimated. Hence, it is not certain whether adjustment for energy intake improves the utility of such data. One might suppose, however, that self-reported intakes of any kind can distinguish between milk drinkers and those who consume little milk.

The utility of these data are also compromised because individuals vary in their susceptibility to all diseases. All diseases, including osteoporosis (18, 19), have a genetic component that may make it difficult to identify important environmental factors. When only a small proportion of the population is susceptible, a comparison of intakes and disease in the total population may be misleading. The intake of nonsusceptible individuals is not relevant to the question of whether high calcium intakes benefit those who have or might develop osteoporosis. Thus, these kinds of dietary studies may obscure the utility of calcium intake for the minority who are susceptible. Nevertheless, the long-standing recommendations to increase calcium intakes appear to have had little or no effect on the prevalence of osteoporosis or fractures in the United States.

The studies of the effect of calcium supplements on bone mineral content appear more promising. Many but not all studies have shown that calcium supplements cause a modest increase in bone mineral content that would appear to be helpful (20). The question, of course, is whether such changes have a significant effect on the fracture rate; this is still unclear. Many of these studies were compromised because vitamin D was administered simultaneously with calcium; administration of vitamin D supplements

¹Reprints not available. Address correspondence to DM Hegsted, Fox Hill Village, #428, 10 Longwood Drive, Westwood, MA 02090. E-mail: dmhegsted@aol.com.

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alone has been reported to inhibit bone loss in northerly climates, such as Boston (21). The results of a study in England, however, do not support this conclusion, suggesting that small changes in the bone mineral content may have minimal effects on fractures (22).

Over many years, various authors have expressed their doubts about the utility of high calcium intakes for the treatment or prevention of osteoporosis or fractures (23–25). Kanis (26) recently reviewed the evidence and concluded, among other things, that “There are no adequately controlled studies to show whether increased calcium intake has an effect on skeletal consolidation or subsequent fracture risk before or after longitudinal growth has ceased.” Kanis also concluded that “There is little justification for a global policy to reduce fracture rates by targeting calcium to the postmenopausal population, or indeed to elderly men.” He does believe that calcium supplements “decrease bone loss...in short term studies” but that there is “uncertainty as to the long term effects on bone mineral and therefore on fracture frequency.”


Worldwide data raise serious questions about the relation between calcium intake and fractures. A large proportion of the worlds’ population consumes low-calcium diets and, although quantitative data on the fracture rate in such populations are limited, it is obvious that these populations do not have excessive rates of fractures as would be expected if calcium requirements were far above their usual intake (27–31). Good data on the association of fracture rates with calcium intakes are available from Japan and clearly show that Japanese women have both less bone mineral and far fewer fractures than do American women (32, 33). It seems obvious that whatever the importance of calcium intake and bone mineral content may be, other important factors must be involved in determining the susceptibility to fractures.

Recent studies showed that the use of β -hydroxy- β -methylglutaryl coenzyme A reductase inhibitors (ie, the statins), which are widely used to lower serum cholesterol, promotes bone formation and is associated with a marked reduction in fractures (34–37). These drugs, which block the mevalonate pathway, were also shown to promote bone formation in mice (38). It appears remarkable that only a few weeks of statin treatment reduced the fracture rate.

The fact that the same drugs may be useful in controlling fractures and coronary artery disease calls to mind that osteoporotic fracture rates around the world are roughly correlated with the incidence of coronary artery disease (28). Countries in which a Western diet is consumed—primarily the United States, Canada, and northern Europe—have high rates of both coronary artery disease and fractures, suggesting that these may share some etiologic dietary factors. In addition, the international epidemiologic data on coronary artery disease stimulated the study of the relation between diet and coronary artery disease. Such data have had little effect on osteoporosis research. Although saturated fat and cholesterol appear to be the primary dietary factors affecting atherosclerosis, their involvement in the etiology of osteoporosis is problematic because a block in the mevalonate pathway may have many effects (39). It was reported that atherogenic high-fat diets reduce bone mineralization in mice (40). Diets high in fat, however, are necessarily low in other components. It may also be relevant to note that bisphosphonates, which are currently used to treat bone loss in osteoporosis, also modulate metabolic intermediates of cholesterol synthesis (39).

Western diets are also high in protein, especially animal protein. The international epidemiologic data show an association

between protein consumption and osteoporotic fractures (28, 41–43). The aciduria caused by such diets promotes urinary calcium loss. There are now considerable data indicating that high intakes of fruit and vegetables protect against fractures (44–46). Sebastian et al (47) reported that potassium supplements alone improve mineral balance, increase bone formation, and might explain the effect of diets high in fruit and vegetables.

The convergence of data indicating that similar dietary practices are beneficial for both coronary artery disease and fractures together with the effects of the statins should stimulate research to resolve the long-standing enigma: Why do populations who consume low-calcium diets have fewer fractures than do those with high intakes? 

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