Calcium and Osteoporosis[1]

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The Consensus Conference on Osteoporosis recommends that women consume 1000–1500 mg of calcium per day [see Spencer and Kramer (1)]. Since the average American woman apparently consumes 450–500 mg/d, she is being told either to increase her milk consumption by about a quart per day or its equivalent or to take calcium supplements. The timely note of Gordon and Vaughan (2) emphasizes that there are *no* data available to demonstrate that high calcium intakes do, in fact, help prevent osteoporosis.

Figure 1, drawn from the data summarized by Gallagher et al. (3) and the FAO food availability data (4), shows that, according to currently available information, hip fractures are more frequent in populations where dairy products are commonly consumed and calcium intakes are relatively high. Is there any possibility that this is causal relationship?

Since blacks are known to be less susceptible to osteoporosis than whites and small white women are especially susceptible, one must consider the possibility that the differences in incidence have a genetic basis. Although this has not been proven to be true of other chronic diseases, there appear to be no relevant data on this point. Since osteoporosis may have a very long generation time, probably the only way to evaluate this issue is to study immigrants from low- to high-incidence areas or the reverse. Such studies are urgently needed.

In the experience of most investigators (5) increasing protein in the diet raises urinary calcium excretion, although not all agree (6). The epidemiological data (fig. 2) show that hip fractures and protein intake are positively related, and obviously there is a positive correlation between calcium and protein consumption. Whether or not high protein intakes play a role in osteoporosis, most researchers would probably agree that it would be unwise to recommend increased protein consumption in populations where protein intakes are already high. Thus, if American women are being advised to increase their consumption of dairy products, they should probably also be advised to reduce their meat consumption proportionately. It seems unlikely that most Americans would find this acceptable.

It is now well known that, at least until menopause, body calcium is rather well controlled and protected and that this is accomplished primarily, if not entirely, by regulation of the circulating levels of 1,25-dihydroxy-cholecalciferol (calcitriol). Low calcium intakes raise circulating levels of calcitriol and improve the efficiency of calcium absorption, whereas high intakes depress calcitriol formation, and thus dietary calcium is inefficiently used. This almost certainly explains why calcium deficiency is so rare throughout the world wherever calcium intakes are low by American standards. The available evidence is consistent with what must be true—that the increased calcium needs associated with growth, pregnancy and lactation are met under most conditions by increased levels of calcitriol and, thus,

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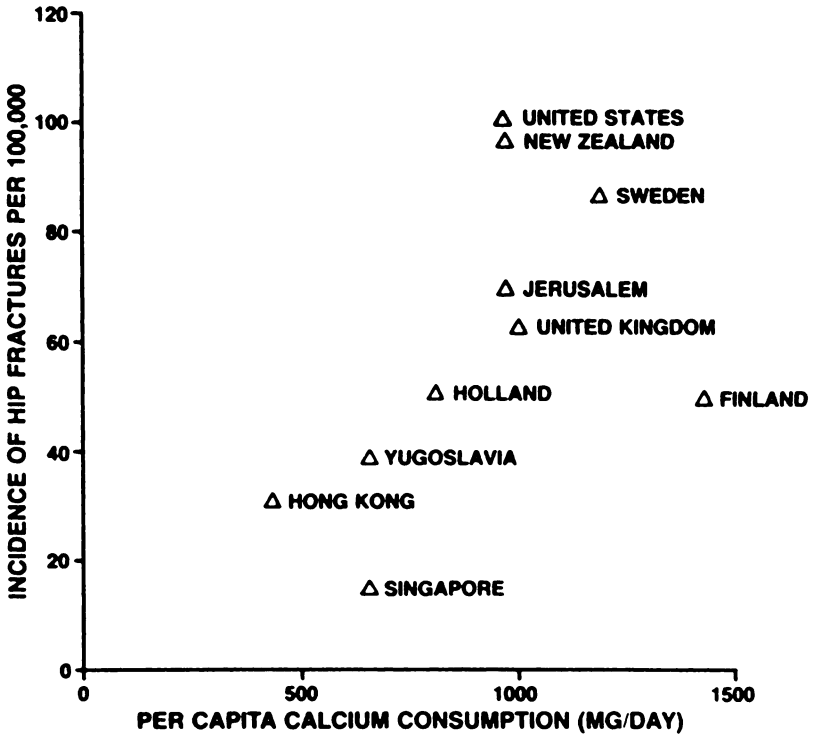

Fig. 1 Available calcium in the food supply compared with the incidence of hip fractures in females of several nations.

more efficient utilization of dietary calcium. People who are adapted to high calcium diets are, inevitably, rather inefficient utilizers of dietary calcium. Otherwise, such populations would have excessively large skeletons or suffer from calcium intoxication, and populations with relatively low intakes would be unable to form normal skeletons. If women in general required 800 mg calcium/d or more, calcium deficiency and osteoporosis would be rampant throughout the world.

Until contrary evidence is available, the possibility must be considered that a lifelong adaptation to a high calcium diet with a continual suppression of calcitriol formation and inefficient utilization of dietary calcium may eventually impair the ability of the body to utilize dietary calcium and to conserve body calcium.

It is also possible that a high osmotic load on the kidney, due mainly to a high protein intake or other characteristics of an "affluent diet," might eventually impair the ability of the kidney to regulate calcitriol levels.

As Gordon and Vaughan (2) have noted, the evidence implicating estrogen deficiency as a primary factor in osteoporosis is strong. Whether differences in rates of estrogen loss could explain the epidemiological relationships and, if so, what factors are involved, needs much more study. Physical activity is also strongly implicated and may be related to estrogen levels. Lane et al. (7) found that vertebral bone density is elevated in female athletes. However, excessive physical activity, resulting in amenorrhea and estrogen deficiency, accentuates bone loss (8, 9). A more sedentary life-style in the more affluent societies might explain, at least in part, the epidemiologic findings.

It should be noted that large, physically active women can be expected to consume more food and generally more calcium than small, sedentary women. Thus, it would not be surprising if surveys in this country, for example, might show less osteoporosis associated with higher calcium intakes. This should not be interpreted to mean that calcium is protective unless activity and body size are accounted for.

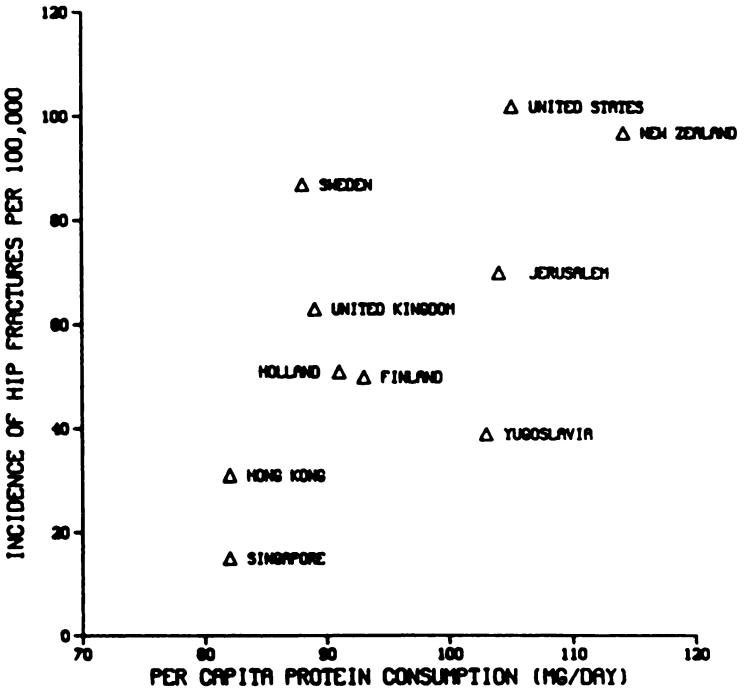

Fig. 2 Available protein in the food supply compared with the incidence of hip fractures in females of several nations.

True calcium deficiency would, of course, be expected to produce osteoporosis or something similar. This has been produced in animals fed a very low calcium diet (10, 11). Such data, however, are not necessarily relevant. The issue is not whether calcium is an essential nutrient; the issue is how much calcium is required to maintain health and whether or not calcium intake is related to the development of osteoporosis.

As figures 1 and 2 demonstrate, the currently available data, although limited, suggest that osteoporosis, like several other major chronic diseases, is largely a disease of affluent, western cultures. Diet must be suspected as a factor, but the causal agent is not known. Much more data from populations where calcium intakes are known to differ substantially using a variety of techniques now available are urgently needed. Since the susceptible populations apparently consume more, rather than less calcium, the epidemiological data do not implicate calcium deficiency as a cause. It seems quite clear that we do not understand the etiology of osteoporosis; the epidemiological data need an explanation, and something is wrong when current explanations are inconsistent with general experience.

It is dangerous to ignore the epidemiological data. The first rule in formulating public health policy should be the assurance that the recommendations are not detrimental. It will be embarrassing enough if the current calcium hype is simply useless; it will be immeasurably worse if the recommendations are actually detrimental to health.

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