Year 2000 dietary guidelines: new thoughts for a new millennium

The US Department of Agriculture and the US Department of Health and Human Services first developed the Dietary Guidelines for Americans in 1980 (1). The dietary guidelines provide advice to healthy Americans on food choices to promote healthy eating and reduce risk of disease. The dietary guidelines were updated in 1985, 1990, and 1995 to ensure their accurate interpretation of the most recent science (2–4). The 1990 National Nutrition Monitoring and Related Research Act mandates the review of the dietary guidelines every 5 y. The Dietary Guidelines Advisory Committee was appointed to review the current guidelines and make recommendations for changes for the 2000 edition.

This advisory committee will struggle with many issues while reviewing a myriad of new scientific data related to diet and health. For example, individual risk of chronic disease varies widely and is strongly influenced by genetics. This issue prompted the American Heart Association to remind health professionals in the Dietary Guidelines for Healthy Americans that “…population-wide guidelines do not address the specific needs of all individuals” (5). The current advisory committee must decide which recommendations need to be targeted to the population through the dietary guidelines and which should be promoted to individuals at risk through some other means. Another important issue that should be addressed is the value of segmenting the dietary guidelines by age (6). The 1995 Dietary Guidelines Advisory Committee “strongly” recommended the development of dietary guidelines for children (7). They suggested that a working group be charged with developing provisional guidelines for children and the supporting peer-reviewed documentation. The US Department of Agriculture targets dietary advice to children through its food guide pyramid for young children (8). In a similar context of age-segmented recommendations, a food guide pyramid was recommended for the elderly (9). The committee will need to rigorously review the available scientific data to develop valid and useful dietary guidelines for Americans.

The American Society for Nutritional Sciences–American Society for Clinical Nutrition Public Information Committee organized a symposium at Experimental Biology ‘99 (held in Washington, DC, on April 19, 1999) to discuss timely scientific issues that the current committee will likely be addressing. Time constraints prevented this symposium from including presentations on all of the controversial nutrition issues the committee will face. Two dietary issues were chosen to be examined more closely with presentations both pro and con. This provided an opportunity to illustrate that there are usually 2 sides to most issues. Sometimes the sides differ greatly and at other times there is only little disagreement.

The first of the issues examined was the need for a dietary guideline for calcium intake. The discussion is presented in the following 2 papers. The second issue, which focused on the current guideline for sodium intake, will be featured in an upcoming issue of the Journal.

Ultimately, the information contained in the dietary guidelines will be transferred to consumers through educational efforts, food and supplement marketing, feeding programs, and policy decisions. Recommendations from the advisory committee will need to be based on sound science rather than on a consensus to ensure that following the dietary guidelines provides benefit without harm (10–13). How the dietary guidelines may eventually be applied by consumers must also be considered in the determination of what is recommended and how those recommendations are presented to both health professionals and the general public. This issue was also addressed at the symposium.

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There should be a dietary guideline for calcium

Introduction

In theory, dietary guidelines are instruments of national nutritional policy rather than statements of nutrient requirements (1). Dietary guidelines are about diets rather than nutrients. There is, therefore, a built-in resistance to incorporating nutrient-specific issues into the guidelines. However, that line in the sand has already been crossed for 4 nutrients [alcohol, fat (and cholesterol), sugar, and sodium] and there are compelling, diet-specific reasons for adding calcium to that list or, perhaps, for substituting calcium for 1 of the 4.

I will summarize here, but not attempt to reargue, the importance of an adequate calcium intake. This point has been satisfactorily dealt with in several nutritional policy-related official statements (2–5) and recent reviews (6) and must be considered firmly established. There are still dissenters, of course, but their stance seems based either on a highly selective reading of the evidence or on premises or preferences that have little or no credible evidential base.

Multisystem involvement of calcium

Adequate calcium intakes have been convincingly shown to protect the skeleton (5), to lower blood pressure (7–9), to reduce the risk of colon cancer (10, 11), to lessen the symptoms of premenstrual syndrome (12), and to reduce the risk of renal stone formation (13, 14). The evidence is strong for both osteoporosis and hypertensive disorders. In the former, the size of the effect is large, whereas with the latter, the effect at a general population level is smaller (8).

The evidence is persuasive for the other disorders as well, but less massive than for osteoporosis and hypertension, and the size of the effect at the population level is still uncertain. For all the disorders concerned, optimum benefit occurs at intakes above both prevailing intakes and the dietary reference intakes of virtually every industrialized nation. These seemingly diverse effects of calcium have a largely dietary form than a biochemical basis (discussed below), which is in itself a reason for a dietary guideline for calcium.

It is widely recognized that the calcium ion plays an essential role as an intracellular second messenger and that it mediates processes as diverse as muscle contraction, interneuronal synaptic signal transmission, glandular secretion, cell division, and blood clotting. These biochemical functions of calcium are exceedingly well protected, first by intracellular calcium stores and by the sheer size of the extracellular nutrient reserve (the skeleton), and second by an elaborate endocrine control system (the parathyroid hormone–vitamin D axis and calcitonin). As a consequence of these protections, nutritional calcium deficiency virtually never compromises, or even threatens, the essential biochemical functions of the mineral. Calcium is unique among the nutrients in that deficiency relates not to impairment of its biochemical roles, but instead to 3 groups of effects that are a consequence of low intake: 1) reduction in the size of the calcium reserve, 2) reduction in the quantity of unabsorbed calcium in food residues, and 3) collateral effects on the other body systems of the regulatory apparatus that protects the organism from hypocalcemia.

The skeletal effect of dietary calcium is straightforward. Skeletal mass (ie, the size of the calcium nutrient reserve) is a direct function of intake up to age-specific thresholds, both during and after growth. It is now clear that contemporary calcium intakes support neither full realization of the genetic potential for skeletal mass nor its maintenance. Roughly 50 studies of investigator-controlled increases in calcium intake have been published, most of which were randomized controlled trials published since 1990 (6). All but 2 studies showed greater skeletal mass gain during growth, reduced bone loss with age, or reduced osteoporotic fracture risk. The sole exceptions among these studies were a supplementation trial in men in which the calcium intake of the control group was already high (nearly 1200 mg/d) (15) and a study confined to early postmenopausal women in whom bone loss is predominantly due to estrogen deficiency (16).

Complementing this primary evidence are ~80 observational studies testing the association of calcium intake with bone mass, bone loss, or fracture (6). It was shown elsewhere (17) that such observational studies are inherently weak, not only for the generally recognized reason that uncontrolled or unrecognized factors may produce or obscure associations between the variables of interest, but because the principal variable in this case, lifetime calcium intake, cannot be measured directly and must be estimated by dietary recall methods. The errors of such estimates have been abundantly documented (18, 19). Nevertheless, more than three-fourths of those studies showed a calcium benefit. In the face of the inaccuracies of the method, the fact that the results of most of these observational studies are positive emphasizes the strength of the association.

Most of the investigator-controlled studies used supplements as the source of calcium, but at least 8 used dairy sources; the results of all these studies were positive. Additionally, essentially all the observational studies involved natural food sources (principally dairy products) and the food sources produced effects comparable with those of supplements. Hence, no further distinction needs to be made between dietary and supplemental sources of calcium. Skeletal effects, at least, depend mainly on total calcium intake.

The effect of calcium intake on colon cancer risk has a different, but equally straightforward basis. In individuals with hereditary or acquired oncogenic factors predisposing to colon cancer, constituents of the chyme residue (ie, unabsorbed fatty acids and bile acids) act as cancer promoters by stimulating colonic mucosal proliferation and mitotic activity. Dietary calcium, precisely because it is poorly absorbed, is also a part of the food residue that reaches the colon. By forming calcium soaps with the fatty acids and salts with the bile acids, dietary calcium renders the fatty acids and bile acids inert; ie, calcium functions as an antipromoter. Calcium’s ability to do this depends on the relative quantities of the reactants in the food residue. With high-calcium diets there is an excess of calcium in the chyme and the promoters are fully complexed; with low-calcium diets the opposite is the case. (Incidentally, this imbalance with low calcium intakes is made worse by another dietary feature, the relatively high fat content of modern diets, which leads to a higher concentration of cancer promoters in the residue, ie, more unabsorbed fatty acids and bile acids.)

The mechanisms for protection in the hypertensive disorders and in premenstrual syndrome are less well understood, but appear to be related to the chronically high blood concentrations of parathyroid hormone, 1,25-dihydroxyvitamin D, or both in persons with low calcium intakes. These hormones, which evolved to sustain extracellular fluid $Ca^{2+}$ concentrations during periods of low environmental calcium availability, also increase cytosolic $Ca^{2+}$ con-
centrations; in sensitive tissues such as vascular smooth muscle, this effect thereby increases vascular tone.

Under primitive conditions, with a normally high calcium intake, parathyroid hormone secretion would have been episodic and confined largely to periods of fasting or famine. Under modern dietary conditions, however, parathyroid hormone secretion is continuously high. Presumably, a sensitive subset of the population with less redundancy in their control systems develops autonomic dysregulation as a consequence of this sustained exposure, much as fava beans unmask glucose-6-phosphate 1-dehydrogenase deficiency in certain persons of Mediterranean ancestry. Interestingly, this is a dietary, or foods issue, rather than just a calcium issue, because diets high in potassium and magnesium, among other nutrients, appear to potentiate the calcium effect. In the Dietary Approaches to Stop Hypertension (DASH) Study (8), the blood pressure benefit produced by the addition of nonfat dairy products was approximately twice as great as was reported for calcium supplements alone.

Finally, protection from kidney stones has a basis similar to protection from colon cancer. Unabsorbed dietary calcium forms complexes not only with fatty acids, but also with dietary oxalate, thereby preventing its absorption. Although oxalate of dietary origin normally accounts for less than one-fourth of the renal oxalate burden, any reduction in urinary oxalate will lower the risk of calcium stone formation. Furthermore, because urinary oxalate is a stronger risk factor for kidney stones than is urinary calcium, reduction in urinary oxalate excretion produces a net reduction in renal stone risk. (This effect of oral calcium has long been recognized and exploited in the management of the syndrome of intestinal hyperoxalosis, in which the intestinal hyperproduction of oxalate leads to massive kidney calcification and for which the standard therapy is large oral doses of calcium carbonate.)

It may be helpful to point out that all these disorders are multifactorial and that inadequate calcium intakes explain only a part of the respective problems. If there is any residual significant uncertainty in the scientific community about the importance of a high calcium intake, it may be precisely because of the multifactorial character of these disorders. One’s individual scientific experience with osteoporosis or hypertension, for example, may be so dominated by the effects of other equally real factors (eg, female hormones, fall patterns, or ethnicity in the case of osteoporosis) that calcium effects are pushed into the background. This is one of the reasons randomized controlled trials are so crucial. In addition to the strong causal inference they permit, they effectively factor out, for investigational purposes, the other important variables and thereby serve to establish the reality of the calcium effect, not as the sole cause of the disorders concerned, but as one of several.

Why a calcium guideline?

Contemporary diets typically contain less calcium than is needed to ensure the foregoing benefits. Moreover, the disorders concerned relate to several components of contemporary diets, not just to calcium. Because these problems transcend single-nutrient issues, they are fundamentally dietary problems, not nutrient problems. A calcium guideline is needed to round out the current dietary guidelines.

At a total diet level, it is worth recalling that the primitive human diet, the one that prevailed during the millennia of hominid evolution and to which our physiologies were adapted, had a high calcium density, estimated to be 2.9–3.3 mg Ca/kJ (70–80 mg Ca/100 kcal) from vegetable sources alone and substantially higher if, as was often the case, the diet included insect grubs or the bones of small prey or fish (20). In contrast with foods accessible to industrialized populat-
a “superstar mineral,” devoting 2 issues of Science Times to the topic in 1998 (23); and Newsweek magazine in its millennial medicine issue summarized calcium’s many benefits under the headline “The Little Mineral That Could” (24). Over the past 17 y of my own extensive interaction with science writers, the focus of their questions to me has shifted from whether we need calcium to how we can best get all we need. Thus, confusion is not a reason to postpone a calcium guideline. In fact, in the face of the public information now available, not having a calcium guideline could well be a source of confusion.

Moreover, adding a calcium guideline would complement 2 of the other guidelines, namely, the recommendations to consume a diet low in fat and a diet moderate in sugars. Failure on both of these counts either contributes to the problems of low calcium intake (eg, colon cancer) or itself further lowers calcium intake. On the other hand, “eating a diet rich in calcium” (or however a calcium guideline might be expressed) would complement the other guidelines and help improve the total diet. This is because most readily available high-calcium sources (dairy foods and vegetable greens) are either naturally low in fat and sugars or are widely available in low-fat varieties. At the same time, both are high in many other essential nutrients, thereby substantially enhancing overall diet quality (25, 26). Finally, adding a calcium guideline [or possibly substituting calcium for the problematic sodium guideline (27)] would help to encourage a rational calcium fortification policy, in accord with the Surgeon General’s 1988 report on nutrition and health (28).

Conclusion

In the final analysis, the desirability of a calcium guideline depends on pragmatic considerations: Will it help Americans consume a better overall diet? Will it help policymakers improve the nutrition of all Americans? My assessment of the situation leads me to answer “yes” to these questions. Whatever the final decision, we cannot lose sight of the need to improve calcium nutrition for the majority of Americans whose low calcium intakes place them at increased risk of osteoporosis, colon cancer, hypertension, and renalolithiasis.

Note added in proof

Recent reports have both added new disorders to the list of conditions associated with low calcium intake and shed new light on the general mechanism behind several of the known effects. Zemel et al (29), in an analysis of the third National Health and Nutrition Examination Survey database, showed that the risk of being obese increases 6-fold as one proceeds from the highest to the lowest quartile of calcium intake. Thys-Jacobs et al (30) recently reported an unprecedented reversal by calcium and vitamin D of polycystic ovary syndrome, a leading cause of infertility in women of childbearing years and a disorder not heretofore linked with the calcium economy. In both cell culture and transgenic mouse model systems, Zemel et al (29) showed that the high serum 1,25-dihydroxyvitamin D concentrations evoked by low calcium intake increase cytosolic free calcium ion concentrations in many tissues, and that, in the adipocyte, this change switches the cell from lipolysis to lipogenesis. In mice overexpressing the agouti gene, low calcium intake lowers core body temperature and increases body fat. This seemingly paradoxical effect of low calcium intakes on cytosolic [Ca\(^{2+}\)] was previously shown for platelets in patients with hypertension (31), as well as for smooth muscle cells. Presumably, it is partly responsible for increased vascular tone and thus contributes to hypertension. Thys-Jacobs et al (30) also explicitly propose that it is the effect of cytosolic [Ca\(^{2+}\)] on oocyte maturation that is the trigger for polycystic ovary syndrome in otherwise sensitive individuals.

Obesity is the most common dietary disorder in the United States today. To the extent that low calcium intakes contribute to the population burden of this disorder, a guideline for a high calcium diet make ever greater sense.

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Should there be a dietary guideline for calcium intake? No

In preparing this manuscript I took the privilege of using the minutes of the September 1998 meeting of the Dietary Guidelines Advisory Committee, which was held in Washington, DC (1). During that meeting, Cuberto Garza, in his introductory remarks to the committee, mentioned a list of 5 C’s: confusion, change, complexity, controversy, and challenge. These same words often came to mind when I was synthesizing the information for this article. The five C’s relative to calcium as given below provide a format for the discussion of why there should not be a specific guideline for calcium within the 2000 edition of the dietary guidelines:

1) confusion as to why there should be a separate guideline for calcium,

2) change in the recommended intakes within the guidelines,

3) complexity of the role of diet in explaining bone mass and fracture risk,

4) controversy surrounding fracture data, and

5) challenges in maximizing bone mass and preventing osteoporotic fractures.

Confusion as to why there should be a separate guideline for calcium

There is some confusion as to why there is a discussion about whether there should be a guideline specific for calcium because the dietary guidelines were not meant to be nutrient specific. The dietary guidelines describe food choices that will help the American people meet the recommended dietary allowances (RDAs) (2). The first of the 7 dietary guidelines in the 1995 edition is “Eat a variety of foods” (3). It is within this guideline that calcium appears. Within the main text of this one guideline, the words milk, calcium, vitamin D, and cheese are mentioned 18 times. In addition, a text box highlights the

recommended serving sizes of various dairy products and an additional box summarizes good sources of calcium other than dairy products. The food guide pyramid is also depicted within this guideline. Change in the recommended intakes within the guidelines

The food guide pyramid, developed by the US Department of Agriculture (USDA), is presented within the dietary guidelines. The food guide pyramid is an educational tool that recommends the kinds and amounts of foods to eat each day. The food groups and numbers of servings depicted in the food guide pyramid are based on recommendations contained within the RDAs, or dietary reference intakes (DRIs), and the dietary guidelines as well as on national survey data of food consumption patterns. To satisfy the 1989 RDA for calcium and to emphasize the need to eat a variety of foods, the current food guide pyramid contains dairy products as 1 of the 5 major food groups, with a recommendation to consume 2–3 servings of dairy products/d.

The change in calcium recommendations between the 1989 RDA and the 1997 adequate intake (AI) (4) is shown in Figure 1. Across all age groups, the maximum recommended intake increased by only 100 mg/d, from 1200 mg/d in the 1989 RDA to 1300 mg/d in the 1997 AI. Because the food guide pyramid is currently designed for persons aged >2 y, the current recommended number of servings in each of the 5 major food groups, including the dairy group, should be sufficient to cover all age groups. If it is assumed that most of the calcium in the diet is obtained from dairy products, the calcium intake of a diet that includes 2–3 servings of dairy products/d could range from 570 to 1350 mg/d depending on the dairy product consumed [from a low of 285 mg Ca in a cup (240 mL) of buttermilk to a high of 450 mg in a serving of plain, nonfat yogurt]. This amount would increase by an additional 94 mg to 180 mg/d if 2 of the 3–5 servings of vegetables included a high-calcium source such as broccoli (47 mg Ca in 92 g or one-half cup) or kale (90 mg Ca in 65 g or one-half cup). Adding one more serving of dairy products per day, or encouraging the consumption of vegetables containing high amounts of calcium, should cover the additional 100-mg/d maximum increase in the recommended calcium intake. However, whether the number of servings within the food guide pyramid needs to be changed to satisfy the new AI for calcium will need to be determined by the USDA. It is not within the authority of the Dietary Guidelines Advisory Committee to change the food guide pyramid, although it is likely that they can make recommendations to the USDA.

Complexity of the role of diet in explaining bone mass and fracture risk

There is agreement among the scientific community that calcium is a critical nutrient for bone health. However, the amount of
calcium needed to optimize bone health and the role of calcium in bone mass and fracture risk is controversial. This controversy is likely due in part to the complexity of how various factors interact with each other in their effect on bone mass.

There are numerous examples of interactions among factors that may affect bone, most of which are just now being realized. Differences in loss of bone density at the femoral neck in postmenopausal women were shown to be associated with the vitamin D receptor genotype (5). Calcium intake, however, appears to modify the relation between bone loss and vitamin D receptor genotype so that a benefit of exercise in preventing loss of bone density only at calcium intakes greater than \( \approx 1000 \) mg/d. Another way of interpreting these data is that the benefit of a high calcium intake on preventing loss of bone density appears to occur only in the exercise groups.

If these interactions among individual factors are not considered in study designs or in the statistical analyses of data pertaining to bone density or fracture, estimates of the amount of variance attributed to these factors are not valid (11). Rather than being able to separate the amount of variation in bone mass within a population into individual factors whose variances sum to 100% as shown in Figure 5A (12), the relations are far more complex, with the individual factors interacting with each other in their influence on bone mass (Figure 5B). Therefore, part of the variation in bone mass within a population that is attributed to diet may be a function of how diet interacts with the specific genetic composition, physical activity patterns, or other characteristics specific to that population. The complexity of these interactions may be one of the reasons for inconsistent findings among various studies with regard to the role of diet in determining bone mass and fracture risk. The presence of interactions and lack of independence among the different factors may explain why some investigators reported that genetic effects may explain up to 75–80% of the variation in bone density (13, 14), whereas the same investigators reported that both physical activity (15) and diet (16) may each explain up to 40% of the variation in bone density. Eisman (17) recognized the possible influence of other factors on estimates of the variance attributed to genetic effects by stating that these estimates were made assuming that other known environmental and medical factors affecting bone density were controlled for.

The extent of the confusion, controversy, and complexity of the role of calcium intake in maximizing peak bone mass, preventing osteoporosis, and reducing fractures can be illustrated by the decision making process.

**Investigator 1**

YY Gene + yellow corn = yellow shanks

yy Gene + yellow corn = black shanks

**Conclusion:** 100% variation due to genetics

**Investigator 2**

YY Gene + yellow corn = yellow shanks

YY Gene + white corn = black shanks

**Conclusion:** 100% variation due to diet
sion of the Food and Nutrition Board (FNB) of the National Academy of Sciences to take a new approach to the RDAs. Rather than recommending one level of intake, or the RDA, the FNB agreed that 4 different recommendations would exist in the form of the dietary recommended intakes, or DRIs (4). The new DRIs consist of an estimated average requirement (EAR), a recommended dietary allowance (RDA), an adequate intake (AI), and a tolerable upper intake limit (UL). The FNB decided to set AIs rather than EARs for calcium on the basis of the following factors (4): “(1) uncertainties in the methods inherent in the balance studies that form the basis of the maximal retention model, (2) the lack of concordance between observational and experimental data (mean calcium intakes in the United States and Canada are much lower than are experimentally-derived values required to achieve maximal calcium retention), and (3) the lack of longitudinal data that could be used to verify the association of the experimentally derived calcium intakes for maximal retention with the rate and extent of long-term bone loss and its clinical sequelae, such as fracture.”

The interactions of various factors, both environmental and genetic, that affect bone mass are complex. These complex interactions likely explain much of the uncertainties in the estimates of calcium requirements.

Controversy surrounding fracture data

The role of high dietary calcium intake or consumption of dairy products in determining fracture risk is controversial. The results of retrospective studies are conflicting: some studies reported a protective effect (18, 19), whereas others reported no association between calcium intake and fractures (20, 21). Retrospective studies that collect dietary intake data after the fracture has occurred are subject to dietary recall bias (22), which often makes the results difficult to interpret. In addition, women who know they are at risk of fracture because of their family history may be more likely to consume more calcium than are women without a family history of fracture or osteoporosis. Prospective studies have the benefit of collecting dietary information before fractures occur, thereby reducing the possibility of recall bias.

Findings from the Nurses’ Health Study have generated a controversy over the role of dairy consumption in the risk of fracture (23). This 12-y prospective study of > 77 000 women found no benefit of increased dairy consumption, or increased calcium consumption, on the risk of fracture. Although not emphasized by the authors, a slight trend of increasing hip fracture risk with increasing dairy consumption was observed. Although these findings are receiving a significant amount of attention in the public press (24), a paper released 1 y later by the same group of investigators is not being referred to in the discussions surrounding this controversy (25). The more recent report examined the role of the vitamin D receptor gene in predicting hip fracture risk in a subset of the women from the Nurses’ Health Study for whom blood samples were available. In this report, hip fracture risk was higher in women with the BB genotype than in women with the bb genotype, with the risk being even greater for those women who had low calcium intakes. Because genotypes were available for only a subset of the women in the original report, it is difficult to interpret how these findings may have influenced the original results. Thus, at this time it is difficult to conclude whether high calcium intake is associated with fracture risk.

Two randomized trials of increased calcium and vitamin D intakes reported a reduction in the incidence of nonvertebral fractures among elderly persons whose mean calcium intakes were low at baseline (< 800 mg/d) (26, 27). It is not clear whether calcium alone would have led to the reduction in fracture risk or whether a similar effect would be observed in younger persons. However, additional evidence should be available soon. Ongoing studies such as the Women’s Health Initiative should provide evidence as to whether a high calcium intake with

FIGURE 4. Results of a meta-analysis showing the interaction between calcium intake and physical activity on mean percentage change in spine bone mineral density (BMD) in the exercise groups (■) and control groups (▲). Data are from reference 7 with permission.

FIGURE 5. It is often assumed that the variation in bone mass within a population can be attributed to individual factors (A). However, the variation in bone mass within the population is likely to be a function of how the different factors interact with each other (B). If these interactions occur, estimates of the amount of variance attributed to individual factors is not valid.
supplemental vitamin D prevents fracture. The Women’s Health Initiative is a large, randomized trial of ≈40,000 women, half of whom are receiving calcium and vitamin D and half of whom are receiving placebo. One of the main outcome variables in this large, clinical trial sponsored by the National Institutes of Health is fracture.

Additional studies sponsored by the National Institutes of Health involving randomized trials of increased calcium intake at various ages are also ongoing in an effort to determine the role and importance of increasing calcium intake in an effort to improve bone health. If there were already overwhelming evidence for a role of high calcium intakes in preventing fractures, such trials would be considered unethical and would be stopped.

**Challenges in maximizing bone mass and preventing osteoporotic fractures**

There is agreement among the scientific community that calcium is a critical nutrient for bone health. The controversy is in the amount of calcium needed to optimize bone health and prevent fractures and the extent to which that amount varies in the population. It is likely that the complexity among factors that affect bone have led to some of the inconsistent findings among studies and to the confusion and controversy surrounding the individual effects of these factors on bone. Determining whether and how these factors interact with each other has just begun, and defining such interactions represents a major challenge in bone research. The prevention of osteoporotic fractures is far more challenging than the simple addition of higher amounts of calcium to the diet. By including a dedicated dietary guideline focusing specifically on calcium, are we leading people to believe that they are doing enough about their health, rather than finding genuine solutions?

**Summary**

The following are the main reasons for not including a specific dietary guideline for calcium:

1. The dietary guidelines were not originally meant to be nutrient-specific recommendations, but rather general recommendations on food choices.

2. The current dietary guidelines already contain numerous recommendations regarding adequate calcium intake.

3. The relation between calcium intake and bone health is complex and poorly understood.

4. Including a specific dietary guideline for calcium may prematurely lead people to believe they are doing enough about their health.

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