Calcium requirements of physically active people[1–4]

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ABSTRACT   Dietary calcium and physical activity have been independently, but inconsistently, associated with the development of increased peak bone mass and reduced bone loss later in life. An examination of the literature points to important effects of dietary calcium on bone health. During the development of peak bone mass, calcium intakes of <1 g/d are associated with lower bone mineral density. At intakes approaching calcium requirements, physical activity is a more important predictor of bone mineral density than is calcium intake. In studies of postmenopausal women, calcium intakes of 1 g (25 mmol/d) appear to be necessary to effect a positive impact of exercise on bone mineral density in the spine. Calcium intakes recommended for protecting bone health appear to be adequate to protect against other disorders with an etiology that includes inadequate dietary calcium. Calcium requirements as modified by physical activity need to be determined for each population subgroup according to sex, age, race, and cultural environment. *Am J Clin Nutr* 2000;72(suppl):579S–84S.

KEY WORDS   Calcium requirements, physical activity, bone mineral content, bone mineral density, peak bone mass

INTRODUCTION

Calcium is an essential nutrient that is required structurally in bones and teeth, as an intracellular regulator, and as a cofactor for numerous proteins and enzymes. Because it is the dominant mineral in bone apatite, calcium is the only nutrient whose storage form serves a functional role.

Dietary calcium intakes by modern humans decreased, relative to those of early humans, when cultivated cereal grains displaced roots and tubers as staple foods (1). The fruit of a plant (including cereal grains) is the poorest accumulator of calcium from the soil. In the United States, dairy foods contribute ≈75% of dietary calcium. Unfortunately, calcium intake by many Americans, especially females, is less than that recommended by the National Academy of Science's Food and Nutrition Board's dietary reference intakes (**Table 1**) (2). It is difficult for individuals to meet these requirements without liberal consumption of dairy products. Those who choose to meet their calcium needs through fortified foods or supplements should be careful to also meet the needs of other nutrients supplied by dairy products.

A profile of the nutrients contributed by dairy products in the American diet is shown in **Table 2**. Manufacturers of fortified foods and supplements should consider packages of nutrients rather than a single nutrient when replacing a food group. In the case of calcium, inclusion of vitamin D, magnesium, and riboflavin might be important if the diet does not include 2–3 servings of dairy products per day. Supplement users might also benefit from additional zinc and iron.

Consumption of adequate dietary calcium reduces the risk of several disorders, the most studied of which is osteoporosis. A substantial body of evidence suggests that adequate calcium also protects against hypertension. Newer evidence suggests a role for dietary calcium in protecting against colorectal cancer. Adequate dietary calcium may also offer protection against kidney stones and lead poisoning.

Much evidence has been accumulated to support adequate calcium intake in health maintenance and disease prevention. Little is known, however, about specific calcium requirements across the life span for different physiologic states, races, and sexes, or how these requirements are modified by lifestyle choices. In this article, I evaluate the evidence of calcium requirements from several outcome measures, with a particular perspective on physical activity as a modifier of these requirements. Diet and exercise are 2 lifestyle choices that can be altered by individuals to benefit health. Additional research is needed to answer more fully the impact of dietary calcium and physical activity.

OSTEOPOROSIS

Osteoporosis is a major health problem affecting >25 million persons in the United States. Prevention of osteoporosis is the most cost-effective means of managing this disease. Two strategies to keep bone mass above the threshold for fracture are optimizing peak bone mass during bone growth and consolidation and reducing the subsequent rate of bone loss. Adequate dietary calcium is required to maximize the development of peak bone mass within an individual's genetic potential and to reduce bone resorption later in life. Less than 90% of the total-body bone mass in females is achieved by age 16.9 y, 95% by age 19.8 y, and 99% by age 26.2 y (4). Thus, the period for influencing optimization of peak bone mass by either calcium or exercise

TABLE 1
Adequate calcium intakes[1]

Group	Calcium
	mg/d
Infants	
Birth to 6 mo	210
6 mo to 1 y	270
Children	
1–3 y	500
4–8 y	800
Adolescents and young adults (9–18 y)	1300
Adults	
19–50 y	1000
>50 y	1200

[1] Adapted from reference 2.

declines rapidly after adolescence, and thereafter the goal is to reduce bone loss. Aside from hormone replacement therapy, diet and exercise are 2 important means of achieving this goal.

Calcium intake

Recently, Heaney (5) reviewed the literature relating calcium intake and bone health since 1975. Over all age ranges, 52 investigator-controlled calcium intervention studies have been conducted. All but 2 showed a positive effect of calcium intake on bone gain or retention or a reduced risk of fracture. These exceptions could be attributed to calcium intakes already at required levels in one study and subjects in perimenopause in the other study.

Several calcium or dairy supplementation trials have been conducted in children. All of these trials have shown bone mineral content (BMC) or bone mineral density (BMD) to be increased in the supplemented group compared with the placebo-control group whether the calcium was from salts (6–9), fortified foods (10), or dairy products (11, 12). However, to maintain the advantage in bone mass, adequate dietary calcium must persist. When supplements were discontinued, 1-y follow-up comparisons showed no significant differences between the calcium-supplemented and the placebo-control groups (13, 14).

In adults, cross-sectional studies do not always show a positive influence on bone. Results of approximately three-fourths of the 86 observational studies reviewed by Heaney (5) were positive. Two meta-analyses of the literature showed significant positive correlations between calcium intake and bone mass (15, 16). Welten et al (15) analyzed 33 eligible studies in men and women aged 18–50 y. The correlation between calcium intake and bone mass for the cross-sectional studies was $r = 0.13$. From the 4 intervention studies, the authors concluded that calcium intakes of 1000 mg/d could prevent the loss of 1% of bone per year at all sites except the ulna. Strong evidence in support of lifelong adequate calcium intake comes from the association between increased BMD of the radius in postmenopausal women and the frequency of dairy consumption in childhood (17).

In a review of trials conducted in postmenopausal women, Dawson-Hughes (18) concluded that the radius was partially protected by 1 g Ca/d but that the spine was unresponsive in early menopause. Bone loss in the first 3 y of postmenopause is hormonally driven and is difficult to prevent without hormone replacement therapy. Beyond the perimenopausal years, calcium intakes of 800 mg/d protected the radius and at least partially protected the spine from loss. In late postmenopausal women who received 1 g Ca/d, loss in BMD of the femoral neck was also significantly less than in a placebo group (19).

Physical Activity

Weight-bearing exercise has also been associated with positive benefits to bone mass or BMD. However, this relation is not supported by the literature to the extent of dietary calcium and bone mass. Few exercise intervention studies have been performed. Most are cross-sectional and involve indirect measures of physical activity or persons already participating in certain activities. Most previous exercise studies are potentially biased because they are nonrandomized; others have inadequate power to show statistical significance or are compromised by previous physical activity and other confounding factors.

Bailey et al (20) reviewed studies examining the relation between physical activity and bone mineral acquisition in children and adolescents. The studies show mixed results. The strongest evidence that physical activity plays an important role in bone mineral accrual in growing children comes from unilateral control studies in which the physically active or dominant limb has a higher BMD or BMC than the opposite limb in the same individual.

Physical activity was reported to have a greater role in affecting BMD before puberty (21, 22). For example, the benefit of playing tennis or squash to BMC was twice as great if females started playing at or before compared with after the onset of menarche (23). In 10-y-old female gymnasts, total-body BMD was ≈5% higher than that in control subjects and annual gains were ≈2% greater (24). In a 3-y prospective study of 90 white children aged 6–14 y, prepubertal children in the highest quartile of weight-bearing physical activity had 4–7% greater rates of bone mineralization than did children in the lowest quartile of activity (21). In a 15-y prospective study of 84 males and 98 females studied from ages 13–28 y, physical activity was the strongest predictor of spine BMD in men at age 28 y (25). Weight was the strongest predictor in females. Similarly, in 581 children aged 7–9 y, physical activity, but not calcium intake, correlated significantly with radial BMD measured 14 y later in the same subjects (26). In 53 girls and 50 adolescent Canadian boys studied longitudinally, active boys and girls had 9% and 17%, respectively, greater total-body BMC compared with that in sedentary peers 1 y after achieving peak BMC velocity (27). A 10-mo, high-impact, strength-building

TABLE 2
Nutrients contributed by dairy products in the US food supply to the diets of women aged 19–50 y, based on 4-d food records[1]

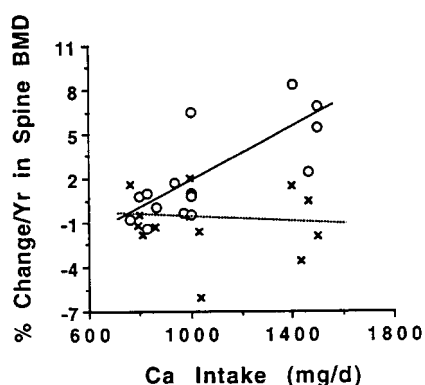| Nutrient	Percentage
	%
Calcium	64.0
Phosphorus	42.0
Riboflavin	41.6
Potassium	32.2
Protein	29.4
Magnesium	28.7
Vitamin B-12	41.4
Fat	25.5
Vitamin B-6	14.9
Energy	19.8
Vitamin A	21.8

[1] From reference 3.

FIGURE 1. Mean change in bone mineral density (BMD) in the lumbar spine as affected by calcium intake in exercising (○) and nonexercising (X) women. Reproduced from reference 33 with permission of the American Society for Bone and Mineral Research.

intervention in 9–10-y-old females showed that in children, in contrast with adults, bone area could be increased over that in control subjects in addition to total-body, spine, and femur BMD (28). Similarly, 30-min weight-bearing physical education lessons 3 times/wk increased femoral volumetric BMD by $1.14 \pm 0.33\%$/mo over 8 mo in prepubertal boys (29).

In adults, cross-sectional studies have shown that physical activity accounts for <25% of the variance in spine BMD. A retrospective study showed that participation in high-school sports could predict BMD of the hip in 204 young women aged 18–31 y, but spine, radius, and total-body BMD could still be influenced by current energy expenditure (30). Intervention studies showed only small changes in bone with resistance training. One of the few randomized exercise intervention studies was carried out by Snow-Harter et al (31) in premenopausal women. An 8-mo weight-training or jogging program resulted in a significant increase in lumbar spine BMD. Both weight training and jogging resulted in similar increases (1.2% compared with 1.3%, respectively) in BMD, whereas a slight decrease was observed in the control group.

Data are scarce with respect to exercise and bone health in women aged 36–50 y. More studies have been done in postmenopausal women with mixed results (discussed in "Calcium and physical activity interaction"). Despite the failure of some studies to show a positive impact on bone when it is mechanically loaded, total physical activity plays a positive role in bone health. Sinaki et al (32) found a positive correlation ($r = 0.24$, $P = 0.05$) between physical activity score and BMD in 68 postmenopausal women. Aside from improved BMD, the physically active have better muscle tone and posture. Better stability can lead to fewer falls, thereby reducing the risk of fracture independent of bone strength. Across all ages, promotion of a physically active lifestyle is more important than prescription of a specific exercise.

Calcium and physical activity interaction

Many lifestyle factors working in concert influence whether an individual meets and maintains his or her genetic potential for peak bone mass. In this review, I consider only a single interaction between one nutrient and physical activity. This is a narrow view of the influence of lifestyle factors on bone health, and the failure to consider even this single interaction has led to much confusion about the role of these factors on bone health.

A review by Specker (33) illustrates the importance of considering both calcium intake and physical activity in determining the effects of either on BMD. Data from 16 exercise intervention trials in peri- or postmenopausal women were compiled. The effects of calcium intake and exercise on spine and radius BMD are shown in **Figures 1** and **2**, respectively. In the spine, increasing calcium intake without the mechanical stimulus of exercise had only a modest effect. At calcium intakes <1 g/d, exercise intervention failed to improve spine BMD. It took both the stimulus of exercise and sufficient raw materials to mineralize bone. The effects of exercise and calcium on the radius were less impressive, but most training programs do not mechanically load the radius to a large extent. Generally, in exercise studies in which calcium intakes were <1 g/d or calcium intake was unknown, it should not be surprising that results of exercise intervention are unimpressive. Supporting epidemiologic evidence comes from a large study of 1075 elderly women and 690 elderly men (34). Osteoporosis was much less prevalent in the highest tertile of dietary calcium intake, body mass index, and quadriceps strength than in the lowest tertile (12% compared with 64% for women and 1.5% compared with 40% for men).

A positive interaction of exercise and calcium was also shown in younger subjects. In a longitudinal, prospective trial of 156 women aged 18–26 y, a positive correlation was seen between spine BMD and both the ratio of calcium to protein intake ($r = 0.200$, $P = 0.017$) and physical activity ($r = 0.175$, $P = 0.038$) (35). The authors used a multiple-regression model of changes in spine BMD to evaluate the interaction between calcium intake and physical activity. Assuming a constant protein intake of 65 g/d for a 25-y-old woman, the effects of varying calcium intake on a constant level of average physical activity and the effects of varying physical activity on the current recommended calcium intake for this age are shown in **Table 3**. The positive effect of physical activity on spine BMD could be augmented substantially with calcium intakes of ≥1 g/d.

In a study of 3 age groups of women (25–30, 40–45, and 60–65 y), both high physical activity and high calcium intake were associated with a higher total-body BMC and larger femoral and radial shafts; although no significant interaction
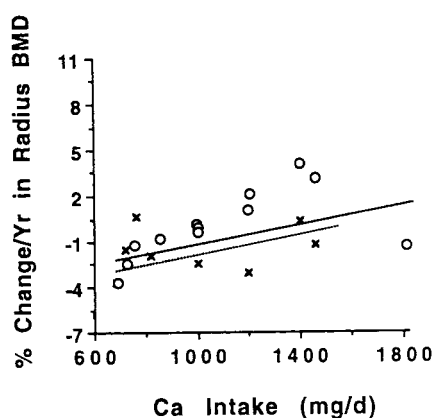

FIGURE 2. Mean change in bone mineral density (BMD) in the distal radius as affected by calcium intake in exercising (○) and nonexercising (X) women. Reproduced from reference 33 with permission of the American Society for Bone and Mineral Research.

TABLE 3
Estimated effect of calcium intake and physical activity on percentage change in spine bone mineral density (BMD) for a 25-y-old female consuming 65 g protein/d[1]

Physical activity (counts/h)[2]	Calcium intake	Change in spine BMD
	mg/d	%
Constant at 5	220	−1.05
Constant at 5	700	3.39
Constant at 5	1000	6.17
Constant at 5	1400	9.87
Constant at 5	2106	16.41
3	Contrast at 800	0.26
4	Contrast at 800	2.29
5	Contrast at 800	4.32
6	Contrast at 800	6.34
7	Contrast at 800	8.37

[1] Adapted from reference 35.

[2] Arbitrary units measured over 4 d with an accelerameter.

was found (36). Femoral neck BMD was 5% higher in physically active than sedentary groups and the association strengthened with age. Other prospective and cross-sectional studies have confirmed the positive role of regular exercise and adequate calcium intake as well as avoidance of smoking in young men and women (37, 38).

The interaction between physical activity and calcium intake is less conclusive in growing children. One large study of 1359 Dutch boys and girls aged 7–11 y reported a positive effect of high physical activity but no association between calcium intake and BMC (39). Another study (40) hypothesized that a calcium intake of up to 1 g/d is helpful but that higher intakes afford no additional advantage in improving BMD. In contrast, dietary calcium along with bone area, lean body mass, body fat, and skeletal age were the strongest predictors of total-body and radius BMD (41). Energy expenditure was significantly related to these measures of bone health when highest and lowest quartiles were compared.

Given the likelihood that stimulation due to exercise may require adequate calcium intakes to realize a positive change in bone, recent randomized exercise intervention trials have evaluated the effect of exercise in addition to calcium supplementation. Lohman et al (42) reported a positive effect of 18 mo of resistance exercise training on femoral trochanter and spine BMD in 56 women aged 28–39 y who were taking a supplement of 500 mg Ca in addition to their diet, which averaged 1023 mg Ca/d. Similarly, in 168 postmenopausal women who were randomly assigned to 1 of 4 groups (placebo, calcium from milk powder, calcium supplements, and supplements plus weight-bearing exercise), exercise in addition to calcium was beneficial in preventing bone loss at the tibia and hip (43). Calcium plus exercise was more beneficial than calcium alone at the femoral neck.

HYPERTENSION

High blood pressure affects >50 million Americans. Forty percent of whites and 50% of African Americans are vulnerable to hypertension. Other high-risk groups include the elderly, men (until middle age, when the risk becomes greater among women), and residents of the southeastern United States.

Because some subgroups are more vulnerable to hypertension than are others, it is likely that some subgroups will benefit from calcium supplementation more than others.

The effect of calcium supplementation on the total population was evaluated in a meta-analysis of 33 randomized, controlled trials of calcium supplementation among 2412 men and women (44). Only a modest decrease of 1.27 mm Hg ($P = 0.01$) systolic blood pressure and no significant change in diastolic blood pressure were found. However, in one vulnerable subgroup—pregnant women—a meta-analysis of 14 randomized trials showed more impressive decreases in both systolic (−5.40 mm Hg; $P < 0.0001$) and diastolic (−3.44 mm Hg; $P < 0.0001$) blood pressure as a result of calcium supplementation (45). In addition, the odds ratios for the calcium-supplemented and the placebo-controlled groups were 0.30 for hypertension and 0.38 for preeclampsia. In African American adolescents, a dose-response relation was found between calcium intake and blood pressure reduction (46). McCarron et al (47) proposed that a set point of 700–800 mg/d exists for dietary calcium intake and that the risk of hypertension increases at a greater rate at intakes below this amount. Thus, individuals consuming sufficient calcium to protect bones should be protected against the risk of hypertension, which is associated with inadequate dietary calcium. However, the effects of age, sex, race, ethnicity, and socioeconomic status on such a set point are unknown.

Hypertension is a strong risk factor for stroke. In the Nurse's Health Study, women in the highest quintile of calcium intake had an adjusted relative risk of ischemic stroke of 0.69 compared with women in the lowest quintile (48).

OTHER DISORDERS

The relation between calcium intake and 2 other disorders, colorectal cancer and kidney stones, was examined in several studies and deserves brief mention here.

Colorectal cancer

There is strong evidence that calcium supplementation can reduce colorectal cancer in experimental animals. Epidemiologic evidence in humans supports a protective effect of higher calcium consumption, which is associated with a reduced risk of colorectal cancer. It has been hypothesized that calcium normalizes the rate and distribution of proliferating cells within the colon crypts, possibly by binding with bile acids (which are possible promoters of colorectal cancer) or by direct induction of terminal differentiation of the colorectal epithelial cells.

The first full-scale, randomized, double-blind, placebo-controlled clinical trial of calcium intake and colorectal cancer, conducted in 193 patients with sporadic adenomas, showed that calcium supplementation normalized the distribution of proliferating cells without affecting the proliferation rate (49). Further, calcium supplementation at 2 g/d was more effective than 1 g/d. The authors interpreted these results to support the hypothesis that calcium directly induces terminal differentiation. In a trial providing 1200 mg Ca/d through dairy products to subjects with a history of polypectomy for colonic adenomatous polyps, proliferation of colonic epithelial cells was normalized by treatment (50).

Two other randomized, clinical trials in subjects at high risk of colorectal cancer failed to show a benefit of calcium supplementation (51, 52). In the first study, only cell proliferation

index was measured, not distributional index, because the authors sought to test the hypothesis that calcium acts through binding of bile acids, which remained unchanged with calcium supplementation. In the second study, although rectal mucosal proliferation was not decreased (52), recurrance of adenomas was reduced by calcium supplementation (53).

Kidney stones

Most kidney stones are composed of calcium oxalate. Epidemiologic evidence shows that calcium intake is negatively correlated with incidence of kidney stones (54), perhaps by decreasing absorption of oxalates from diet through the formation of insoluble salts. The negative relation between dietary calcium and kidney stones is surprising given that hypercalciuria is a risk factor for calcium oxalate stones.

This seeming contradiction may be explained by a retrospective, cross-sectional study of 282 patients with calcium oxalate kidney stones (55). These authors found that dietary salt, as assessed by urinary sodium excretion rather than dietary calcium, was the principal dietary factor influencing urinary calcium excretion. The relation between dietary calcium and prevention of kidney stones was recently reviewed by Heller (56). He points out that there have been no clinical trials of the effect of calcium supplementation or restriction on incidence of kidney stones.

NEEDED RESEARCH

Many questions remain unanswered in the quest to understand calcium requirements and physical activity with respect to age, sex, race, and cultural environment. Ideal studies must be randomized for both calcium and exercise intervention to prevent treatment bias and to understand the independent variables. Failure to impose an intervention of calcium supplementation leads to inadequate assessment of usual calcium intake. Diet records or recalls and food-frequency questionnaires suffer from errors due to subjects' inability to estimate portion sizes, memory gaps, and the inability to reflect wide fluctuations in day-to-day intake. Failure to quantitate the exercise intervention makes it difficult to interpret results. In studies of changes in BMD, the stress of the exercise on the bone of interest should be quantitated.

Several other aspects of study design should be emphasized. In particular, a sufficient sample size is essential to achieve the necessary statistical power to detect differences. The duration of the exercise intervention also should be sufficient. For a study of changes in BMD or BMC, the study should span several remodeling cycles because of the lag between bone resorption and bone formation. Initial status of calcium or physical activity should be factored into the analysis because the effect of treatment is likely to be greater if status is initially low.

The most important needed research is to refine the understanding of calcium requirements as a function of physical activity according to various age, sex, and racial groups. Such research will require, at a minimum, 2×2 factorial designs for each population subgroup. Outcome measures can include fracture incidence, ratio of BMD to BMC, blood pressure, and cardiovascular reactivity. Other important questions to answer include the following:

1) What type, duration, and intensity of exercise should be recommended? What is the effect of cyclic exercise training for individuals who begin, stop, and begin again their commitment to exercise training?

2) What is the effect of time on efficacy when supplements are taken? What is the bioavailability of calcium sources not yet measured?

It is likely that additional research of this nature will support calcium intakes and exercise that are higher than current levels. How then can people be convinced to change their behavior? This question is the most difficult to answer. Behavior change will probably require a multifaceted approach. Research is needed to identify barriers to choosing a well-balanced diet and regular exercise. Research aimed at changing the environment and attitudes of those who influence the targeted group is also needed.

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