TABLE 425-6 SIMPLE METHOD FOR CALCULATING DIETARY CALCIUM INTAKE

STEP 1: Estimate calcium intake from calcium-rich foods

Product	# of Servings/d	Estimated calcium/ serving, in mg	Calcium in mg
Milk (8 oz.)	_____	× 300	= _____
Yogurt (6 oz.)	_____	× 300	= _____
Cheese (1 oz. or 1 cubic in.)	_____	× 200	= _____
Fortified foods or juices	_____	× 80 to 1000	= _____
		Subtotal = _____	

STEP 2: Total from above + 250 mg for nondairy sources

= total dietary calcium TOTAL Calcium, in mg = _____

Source: Adapted from SM Krane, MF Holick, **Chap. 355**, in *Harrison's Principles of Internal Medicine,* 14th ed. New York, McGraw-Hill, 1998.

foods such as certain cereals, waffles, snacks, juices, and crackers. Some of these fortified foods contain as much calcium per serving as milk. Green leafy vegetables and nuts, particularly almonds, are also sources of calcium, although their bioavailability may be lower than with dairy products. Calcium intake from the diet can also be assessed **(Table 425-6)** and calculators are available at *NOF.org* or *NYSOPEP.org*.

If a calcium supplement is required, it should be taken in doses sufficient to supplement dietary intake to bring total intake to the required level (1000–1200 mg/d). Doses of supplements should be ≤600 mg at a time, because the calcium absorption fraction decreases at higher doses. Calcium supplements should be calculated on the basis of the elemental calcium content of the supplement, not the weight of the calcium salt. Calcium supplements containing carbonate are best taken with food because they require acid for solubility. Calcium citrate supplements can be taken at any time. To confirm bioavailability, calcium supplements can be placed in distilled vinegar. They should dissolve within 30 min.

Several controlled clinical trials of calcium, mostly plus vitamin D, have confirmed reductions in clinical fractures, including fractures of the hip (~20–30% risk reduction). All recent studies of pharmacologic agents have been conducted in the context of calcium replacement (± vitamin D). Thus, it is standard practice to ensure an adequate calcium and vitamin D intake in patients with osteoporosis whether they are receiving additional pharmacologic therapy or not. A systematic review confirmed a greater BMD response to antiresorptive therapy when calcium intake was adequate.

Although side effects from supplemental calcium are minimal (eructation and constipation mostly with carbonate salts), individuals with a history of kidney stones should have a 24-h urine calcium determination before starting increased calcium to avoid significant hypercalciuria. Many studies confirm a small but significant increase in the risk of renal stones with calcium supplements, but not dietary calcium. A recent analysis of published data has suggested that high intakes of calcium from supplements are associated with an increase in the risk of heart disease. This is an evolving story with additional studies that confirm or refute this finding. Because high calcium supplement intakes increase the risk of renal stones and confer no extra benefit to the skeleton, the recommendation that total intakes should be between 1000 and 1200 mg/d is reasonable.

VITAMIN D Vitamin D is synthesized in skin under the influence of heat and ultraviolet light **(Chap. 423)**. However, large segments of the population do not obtain sufficient vitamin D to maintain what is now considered an adequate supply [serum 25(OH)D consistently >75 μmol/L (30 ng/mL)]. Because vitamin D supplementation at doses that would achieve these serum levels is safe and inexpensive, the Institute of Medicine (based on obtaining a serum level of 20 ng/mL) recommends daily intakes of 200 IU for adults <50 years of age, 400 IU for those 50–70 years, and 600 IU for those >70 years. Multivitamin tablets usually contain 400 IU, and many calcium supplements also contain vitamin D. Some data suggest that higher doses (≥1000 IU) may be required in the elderly and chronically ill. The Institute of Medicine report suggests that it is safe to take up to 4000 IU/d. For those with osteoporosis or those at risk of osteoporosis, 1000–2000 IU/d can usually maintain serum 25(OH)D above 30 ng/mL.

OTHER NUTRIENTS Other nutrients such as salt, high animal protein intakes, and caffeine may have modest effects on calcium excretion or absorption. Adequate vitamin K status is required for optimal carboxylation of osteocalcin. States in which vitamin K nutrition or metabolism is impaired, such as with long-term warfarin therapy, have been associated with reduced bone mass. Research concerning cola intake is controversial but suggests a possible link to reduced bone mass through factors that are independent of caffeine. Although dark green leafy vegetables such as spinach and kale contain a fair amount of calcium, the high oxalate content reduces absorption of this calcium (but does not inhibit absorption of calcium from other food eaten simultaneously).

Magnesium is abundant in foods, and magnesium deficiency is quite rare in the absence of a serious chronic disease. Magnesium supplementation may be warranted in patients with inflammatory bowel disease, celiac disease, chemotherapy, severe diarrhea, malnutrition, or alcoholism. Dietary phytoestrogens, which are derived primarily from soy products and legumes (e.g., garbanzo beans [chickpeas] and lentils), exert some estrogenic activity but are insufficiently potent to justify their use in place of a pharmacologic agent in the treatment of osteoporosis.

Patients with hip fractures are often frail and relatively malnourished. Some data suggest an improved outcome in such patients when they are provided calorie and protein supplementation. Excessive protein intake can increase renal calcium excretion, but this can be corrected by an adequate calcium intake.

Exercise Exercise in young individuals increases the likelihood that they will attain the maximal genetically determined peak bone mass. Meta-analyses of studies performed in postmenopausal women indicate that weight-bearing exercise helps prevent bone loss but does not appear to result in substantial gain of bone mass. This beneficial effect wanes if exercise is discontinued. Most of the studies are short term, and a more substantial effect on bone mass is likely if exercise is continued over a long period. Exercise also has beneficial effects on neuromuscular function, and it improves coordination, balance, and strength, thereby reducing the risk of falling. A walking program is a practical way to start. Other activities, such as dancing, racquet sports, cross-country skiing, and use of gym equipment, are also recommended, depending on the patient's personal preference and general condition. Even women who cannot walk benefit from swimming or water exercises, not so much for the effects on bone, which are quite minimal, but because of effects on muscle. Exercise habits should be consistent, optimally at least three times a week.

PHARMACOLOGIC THERAPIES

Before the mid-1990s, estrogen treatment, either by itself or in concert with a progestin, was the primary therapeutic agent for prevention or treatment of osteoporosis. There are now a number of new medications approved for osteoporosis and more under development. Some are agents that specifically treat osteoporosis (bisphosphonates, calcitonin, denosumab, and teriparatide [1-34hPTH]); others, such as selective estrogen response modulators (SERMs) and, most recently, an estrogen/SERM combination medication, have broader effects. The availability of these drugs allows therapy to be tailored to the needs of an individual patient.

Estrogens A large body of clinical trial data indicates that various types of estrogens (conjugated equine estrogens, estradiol, estrone, esterified estrogens, ethinyl estradiol, and mestranol) reduce bone turnover, prevent bone loss, and induce small increases in bone mass of the spine, hip, and total body. The effects of estrogen are