Calcium Intake and Availability From the Human Diet

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As outlined elsewhere in this issue, there are valid medical indications for increasing dietary calcium intake to prevent or ameliorate specific health conditions for selected, susceptible persons. The health community and the general public have become conscious of the need for augmenting the intake of oral calcium. Although the World Health Organization specifies a 400 mg to 500 mg daily calcium intake as adequate for adults, the 1980 RDA (recommended daily allowance) for adults in the United States was 800 mg, and a further increase to 1000 mg for women was contemplated for the revised RDAs. The majority of individuals, specifically adults, in this country consume less than their respective RDA level and much less than the amounts required to achieve target intake levels for calcium-responsive conditions. Dairy foods constitute the major source of dietary calcium in western diets, contributing 60% to 75%. Factors intrinsic to the chemical nature of dietary and supplemental calcium, and to the meal contexts in which it is consumed, influence the absorption and ultimate utilization of the mineral by the body. Calcium from plant foods is less available than that from animal sources. Calcium carbonate may produce less remodeling and strengthening of demineralized bone than dairy calcium. Milk sugar (lactose) is an important factor as it aids calcium absorption on the one hand, but it can prevent milk consumption in certain persons who lack sufficient levels of intestinal lactase enzyme and suffer symptoms of carbohydrate intolerance on consuming dairy products. Several dietary strategies related to dose, timing, combination with other foods, and the use in vitro or in vivo of exogenous microbial lactases have been designed to overcome symptomatic lactose intolerance. It is likely that approaches to an increase in calcium intake to target intake levels above 1 g daily will require a combination of increased dietary consumption and some specific supplementation with inorganic calcium sources. (CLIN NUTR 1986;5:167-76)

A focus on calcium intake and calcium's role in metabolism has burst forth in recent years. The comparison articles in this issue bear on that fact. The bottom line of the issue, as perceived by an increasingly calcium-conscious public, is on calcium *intake*, and it is to this theme and its obligate ramifications regarding selection of calcium sources and their biologic availability that this article is dedicated.

Evidence of the surging interest in calcium can be found on all sides today. At the recently concluded meetings of the Federation of American Societies for Experimental Biology (FASEB) in St. Louis in April 1986, between the combined physiology and nutrition societies, a total of 51 scientific papers on calcium metabolism were presented. It is widely admitted that the draft of the tenth edition of the recommended dietary allowances (RDA), aborted (deferred) in its presentation by a policy decision of the National Academy of Science, contained a recommendation for an increase in the recommended daily calcium intake above previously recommended levels for adult women. The industrial and commercial implications are obvious in the rush to adjust individual calcium intakes in the United States population. Inorganic sources of calcium, ranging from pulverized oyster shells to chewable antacid tablets (Tums) are being promoted. In the midst of the withdrawal of government subsidies to dairy farmers and the curbing of dairy herds in the United States, the milk industry is eager to capitalize on *calciomania* by educating the public to the virtues of dairy sources of calcium. The Carnation Company, for instance, is offering a free *Calcium Boosters* booklet, which recommends the addition of nonfat dry milk powder to other recipes and foods, including yogurt, a beverage itself based on milk solids. Recognition of some of the issues embodied in the accompanying features in this issue of CLINICAL NUTRITION by the medical community, by the pharmaceutical industry, by food and health-food industries, and by the public itself has led to an unprecedented consciousness about dietary calcium.

Indeed, as the preceding articles have illustrated, strong — but not incontrovertible — evidence can be mobilized for a role of dietary calcium in the prevention of bone loss, the genesis of essential hypertension and toxemia of pregnancy, and colonic neoplasia. However, logically considered, the amount of calcium *absorbed* and *retained by bone* may be important to the first of those issues, while the amount *absorbed* but *released by bone* (or never taken up into the skeleton) may influence arterial pressure, whereas the fraction of dietary calcium *never absorbed* and bathing the large bowel may determine its effects on the incidence of malignant tumors in the colon. This article will review dietary intake of calcium, food sources of the mineral, genetic and other

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Table I. Content of calcium in selected foods in western diets

Food item	mg/100 g portion
Milk and cheese	143
Cereal grains	97
Fish	65
Eggs	52
Green vegetables	36
Red meats	19
Tubers	12
Fruits and jams	12
Beverages	7
Fats and oils	4

intrinsic factors influencing calcium ingestion and metabolism, and certain extrinsic (dietary) factors relating the amount of calcium consumed to that absorbed and used by the body.

CALCIUM IN THE HUMAN BODY

Calcium is critically important for the structure and function of the human organism. The adult male is estimated to have 1.2 kg of calcium in his body, 99% of which is located in the skeletal system. It makes up the mineral base of the hydroxyapatite of bone.[1] In the soft tissues, specifically muscle (both skeletal and cardiac), ionic calcium is a critical determinant of excitation and contraction. To support the contractile state of these muscle groups, the level of calcium in extracellular fluid is tightly and homeostatically regulated within narrow concentration limits.

It has been debated whether or not a dietary deficiency of calcium can exist. Allen[2] has commented: "Because the intestine can adapt to low calcium intakes by increasing the fractional absorption of calcium, some investigators believe that calcium deficiency cannot be caused by a low dietary calcium intake, except in a situation of extreme calcium deficiency." A major focus of our thinking about bone mineralization, however, relates to vitamin D (i.e., rickets, osteomalacia) or to osteoporosis, which is considered a concomitant of aging. Whether limitation of skeletal mineral mass can exist as a consequence of insufficient dietary calcium remains in question. Some epidemiologic data from Yugoslavia, however, comparing bone health and mass in relatively early adulthood to midadulthood between groups with high and low life-long calcium intakes, adds credence to the notion of low dietary calcium limiting bone mineralization per se.[3]

Another comparison, this time between rural and urban South African Bantu children (the former group consuming an average of 125 mg of calcium per day and the latter 337 mg of calcium per day), revealed hypocalcemia and elevated alkaline phosphatase levels in the former group.[4] This degree of dietary deprivation appears to lead to both skeletal changes (alkaline phosphatase alterations) and maladjustment of the circulating pool of calcium (hypocalcemia).

SOURCES OF CALCIUM IN THE DIET

Cultural factors and culinary practices influence the habitual calcium intake of a population. Persons in rural regions of developing countries represent the extremes of calcium ingestion, with the Bantu of South Africa having a notably low consumption,[5] and Guatemalans and Ethiopians having the highest.[6] Occidental omnivores have intermediary values for daily calcium (see below).

Foods rich in calcium in western diets are listed in Table I.[7] As can be observed, dairy products (milk and cheese) have about 150 mg of calcium per 100 g. Cereal grains have over 90 mg per 100 g, but the bioavailability of that calcium may be reduced by the nonabsorbable residues in unrefined cereals, as discussed below. Fish, especially that containing edible bones (e.g., salmon, herring, sardines), is also a rich source of calcium in western diets. Both eggs and green vegetables have over 40 mg of calcium per 100 g.

Certain regional cuisines have foods of exceptionally high calcium content. This is true of the grain *teff*, which is consumed as the major staple food in much of rural Ethiopia. One-hundred grams of this grain, as consumed, would contribute 110 mg of calcium. The corn tortilla in post-Mayan cultures (Guatemala, southern Mexico) is prepared with lime-soaked corn, and lime water (calcium hydroxide solution) is added at the time of cooking. The traditional tortilla from this region contains in excess of 200 mg of calcium per 100 g of the foodstuff.[8] As reported by Belizán and Villar,[6] the estimated daily intake of calcium (per capita) for Guatemalans is 1100 mg and for Ethiopians is 1075 mg.

Hazell[7] has outlined, from data in the United Kingdom, the relative contribution of the foods and food groups to the total intake of calcium as around 950 mg for persons in the British Isles (Table II). As can be seen, dairy products constitute the major contributor, with over 60% of calcium coming from milk and cheeses. Cereal grains provided about one fourth of the total, and all other sources comprised the remaining 17%. Drinking water also contributes to daily calcium intake. In "hard water" areas especially, this is an appreciable source of the mineral, contributing up to 7% to 8% of the daily intake from foods and beverages.[9, 10]

A focus on calcium intake and calcium's role in metabolism has burst forth in recent years.

FACTORS RELATED TO THE UTILIZATION OF DIETARY NUTRIENTS

A few overall principles applicable to any nutrient form a conceptual framework for studying the specific situation of calcium in the diet. First, nutrients come to us from the environment in the form of foods and beverages. The intestinal tract is the usual port of entry for nutrients entering the body. Under certain circumstances, the intestine can *regulate*, in a homeostatic fashion, the passage of nutrients from food, via the gut, into the body by varying the efficiency of absorption (altering the "fractional absorption")* of a given nutrient.[11] Maximal absorption efficiencies are usually observed when an organism is deficient in the nutrient and/or the nutrient is present in low concentration in the food and hence in the intestinal lumen. Even with this maximal up-regulation of absorption, there are circumstances in which this does not fully compensate for the deficit and nutritional depletion can continue. Minimal absorptive efficiencies are usually seen when an organism is replete with the nutrient and when the nutrient is in high concentration in the intestine after a meal. Even in this situation, if the amounts ingested are excessive, an accumulation of the nutrient in the body, above the normal and desirable pool size, can result from this "flooding" of the system.

*Fractional absorption is the percentage expression derived from the ratio of the amount of nutrient absorbed/total amount of nutrient ingested × 100.

Two important points derive from these principles. The first is that it is not sufficient to assess the process of nutrient acquisition in a person simply by determining absorptive efficiency and assessing whether it is operating at a "high" or a "low" rate. Rather, the key issue is the *net uptake* of ingested nutrient by the body. This net uptake is the *product* of the total nutrient intake times its efficiency of absorption from that meal or diet. Suppose, for example, that the average, typical absorptive efficiency (fractional absorption) of a nutrient was 0.4 (40%). However, in a person with a chronically marginal intake of 10 mg, the absorption efficiency could be 90%. The net amount of that nutrient absorbed under this circumstance would be 9 mg. Alternatively, when 90 mg of the same nutrient is ingested in a meal by a healthy, well-nourished person, the fractional absorption is found to be 10%. Compared with the usual efficiency of 40%, this is clearly low. However, this does not represent a pathologic situation but, rather, an adaptive and regulatory one, as the net uptake is again 9 mg. It would not be unexpected that the usual dietary meal content of this nutrient was 22.5 mg, which, metabolized at the "normal" absorptive efficiency of 40%, would also provide a net uptake of 9 mg. Of course, if 90% and 10% represent the limits of physiologic adaptation for healthy persons, then chronic consumption of less than 10 mg, or more than 90 mg, will lead to imbalances in the body's economy. Age-related changes and/or intestinal or systemic pathologic conditions can alter these control mechanisms. Thus, a person who absorbs either 10% or 90% of a 22.5 mg intake of the nutrient in question has a high probability of being abnormal and would develop a nutrient imbalance over time.

Table II. Relative contribution of various foods to total dietary calcium intake in the United Kingdom*

Food item	Calcium contribution (mg/day)	% of total daily calcium**
Milk and cheese	572	60
Cereals	222	23
Green vegetables	40	4
Meat	28	3
Fruits and preserves	21	2
Root vegetables	21	2
Egg	14	1
Fish	13	1
Beverages	8	1
Fat and oils	3	1

*Modified after data from Hazell.[7] Expressed on per capita basis.

**Two percent of the calcium came from other sources not listed, including drinking water.

The second point is that nutrients are generally contained in a food matrix, not freely dissolved (although often, in beverages, nutrients are free in solution). Various mechanical and enzymatic (hydrolytic) processes are required to get the nutrients out of the food and into their elemental condition for absorption. Components in the foods containing the nutrient — or in other foods in the same meal — can interfere with the release of the free, absorbable nutrient. This impairs the availability of the nutrient for absorption. Conversely, natural dietary substances can enhance the liberation or preparation for absorption of a nutrient over the condition that would exist in the usual meal situation. This feature of dietary context affecting absorption has been conventionally termed "biologic availability."[12]

However, it has recently been increasingly appreciated — and a prime example is the nutrient in question here, *calcium* — that the mere fact of intestinal uptake and transport to the body is not tantamount to its ultimate utilization by the body. Dietary and environmental factors can produce a diversion of the nutrient from its usual physiologic role or can accelerate its loss from the organism. Thus, a *utilization* factor has been introduced in the comprehensive, modern definition of bioavailability.[13] As will be discussed, calcium has interactions with the diet beyond the limits of the gastrointestinal tract — interactions that contribute measurably to the net economy of this nutrient. In the context of both dietary supply and conscious supplementation with calcium, the whole range of factors discussed in this section must be borne in mind.

THE BIOLOGICAL AVAILABILITY OF DIETARY CALCIUM

Several comprehensive reviews of calcium absorption and bioavailability have recently appeared.[14, 15] Calcium absorption efficiency ranges from 30% to 60% in adults with daily intakes of 400 mg to 1000 mg.[16] The human intestine, however, can absorb 1.0 g of calcium per day from an intake of 7.5 g, a 13% fractional absorption.[17]

Intestinal absorption of calcium depends on the following two processes: (1) an active saturable (carrier-mediated, energy-dependent) transfer, dependent on vitamin D (1, 25-dihydroxyvitamin D hormone), localized to the upper gut, and (2) a nonsaturable, diffusional process, predominantly operating in the ileum.[14] Although kinetic data suggest that 80% of the calcium that is absorbed from a high-calcium meal is absorbed by the vitamin-D-dependent mechanism,[18] practical, experimental observations in postsurgical patients suggest that at least half of the calcium absorbed from ordinary repasts is taken up in the lower tract. A possible explanation is that it is necessary to liberate the meal calcium from its food matrices. Low gastric pH also helps to solubilize calcium, whereas hypochlorhydria conditions the precipitation of the mineral in the gastrointestinal tract.

Table III. Effect of orally ingested substances (dietary and pharmacologic) on calcium absorption

Depressing effect	Nil effect	Enhancing effect
Phytate	Phosphate	Lactose
Cellulose	Protein	Lysine
Sodium alginate	Ascorbic acid	Arginine
Oxalate	Citric acid	MCTs*
Alcohol		
Anticonvulsants		
Antacids		
Cortisol		
Tetracycline		

Composite from data provided by Allen[14] and Avioli.[23]
*MCT = Medium-chain triglyceride

Rapid growth, pregnancy, and lactation are normal physiologic conditions that enhance intestinal absorptive efficiency. Aging and menopause reduce absorption.[18, 19] Various intestinal diseases, especially those characterized by steatorrhea, produce calcium malabsorption.

A number of dietary factors have been tested with relation to the absorption of calcium in man. A synopsis of the findings for the interaction of calcium with other orally ingested substances is shown in Table III. The most important inhibitors of calcium absorption, in terms of the availability to human beings from food sources, appear to be plant constituents: phytate, dietary fiber, and oxalate. These compounds — alone, or in combination — limit the uptake of calcium to the point at which negative total-body calcium balance becomes manifest. The negative effect of bran on calcium balance was first shown during World War II by McCance and Widdowson.[20] Cummings et al.[21] recently replaced refined wheat products with unrefined grain in the diets of young men on a metabolic unit consuming over 1300 mg of calcium daily, increasing their dietary fiber intake from 22 g to 53 g daily. The result was negative balance. Oxalates also play an important role; Kelsay and colleagues[22] found a reduction in calcium balance of almost 200 mg in men consuming 1100 mg of calcium daily when fruits and vegetables were added as a source of some of the dietary calcium. Alcohol and other drugs can also exert a malabsorptive effect on calcium. Antacids probably hamper absorption through precipitation of calcium in the gut. Tetracycline chelates calcium. Anticonvulsants work by way of disruption of the metabolism of vitamin D.

Experiments have shown that dietary phosphate, protein, ascorbic acid, and citric acid in normal to supradietary amounts do not alter the efficiency of calcium uptake in human subjects. Lactose has an enhancing effect (see below), as do the neutral amino acids lysine and arginine and medium-chain triglycerides. An extensive review of the dietary factors shaping calcium availability for intestinal uptake has been produced.[14, 23]

The public has become aware of the need for increased consumption of calcium.

Two issues of postabsorptive disposition of calcium merit consideration, as they bear on the utilization of consumed and absorbed calcium. High protein intakes increase the excretion of calcium in the urine.[24, 25] That this is modified by the type of phosphate in the most concentrated protein source in the western diet — *meat* — has been advanced.[26] Nonetheless, self-supplementation of protein by athletes and health-food faddists is common, and the overall effect on calcium economy caused by protein-induced hypercalciuria in these groups should be considered.

The other concern relates to the source of calcium and skeletal deposition of that mineral. Obviously, if one can demonstrate positive calcium balance in a person on a given oral regimen, it provides relative assurance that calcium is being deposited in *bone*. Studies in Nebraska, however, suggest that mere adhesion of new mineral to the bone is not enough to improve the tensile strength and fracture resistance. The remodeling of bone in a structurally appropriate manner to support weight-bearing stress is essential. Two sequential experiments by Recker and associates[27] and Recker and Heaney[28] suggest that milk calcium, but not calcium in a carbonate salt, leads to improved strength by stimulating remodeling of bone along with remineralization.

Older studies addressed various calcium sources and their absorption and utilization by human subjects.[29, 30] It was suggested that calcium from lactate was more absorbable than calcium in the gluconate form.[30] Recent interest in calcium supplements has already spawned clinical and laboratory experimentation concerning the bioavailability of specific forms and sources of calcium used in supplementation. Two such studies were presented at the recent FASEB meetings in St. Louis. Krzykowski et al.,[31] from Wisconsin, fed rats various calcium supplements or milk, finding minor differences in nonfat dry milk, calcium phosphate, oyster-shell calcium, calcium carbonate, calcium lactase, and chelated calcium as the calcium sources. In Nebraska, Kohls, Kies, and Fox[32] performed metabolic balance studies in human subjects, with different calcium supplement forms. No pronounced differences in apparent absorption were seen among milk, dolomite, calcium carbonate, calcium gluconate, calcium lactase, or oyster-shell calcium. Although the conclusions of these works will be judged better and appreciated with their publication in full in scientific journals, it is of note that research to guide human calcium supplementation is undergoing a renaissance.

MILK AND LACTOSE IN RELATION TO DIETARY CALCIUM

Milk, as noted, is an abundant source of calcium. A glass of cow's milk contains about 300 mg of calcium. Milk is also a rich source of riboflavin and contains more than 7 g of high-quality protein per glass. However, it is far from being "nature's perfect food" since it is only a variable source of other micronutrients. For instance, if we assume that a growing preschool child consumes 720 ml of milk daily, these three glasses would meet the child's entire dietary requirement with respect to calcium, phosphorus, and riboflavin.[33] However, it would take six glasses to meet the vitamin B_6 requirement, 20 glasses to meet the vitamin C requirement, and more than 80 glasses of unfortified milk to meet the preschool child's needs for iron and vitamin D. Cow's milk, unless contaminated with fecal bacteria, is virtually devoid of vitamin B_{12}. Of the trace elements, both copper and zinc exist in low concentrations in bovine milk. Obviously, after childhood the habitual diet contains a variety of other foods and beverages that complement the nutrient content of milk. However, to the extent that milk, cheeses, yogurt, chowders, and ice cream substitute for other sources of animal protein, a relative lowering of nutrient balance can be produced.

The principal carbohydrate in milk is lactose, a disaccharide that comprises glucose and galactose. As only these constituent simple sugars can be absorbed, lactose requires a membrane-bound intestinal enzyme (neutral lactase) for its digestion.[34] Milks from various mammalian species have different lactose concentrations, with human breast milk having 6.9 g per liter and cow's milk having 4.8 g per liter.[35] All healthy infants are well endowed with intestinal lactase, which persists during at least the first 2 years of life. Normally, as in any mammal, a genetically programmed involution of the enzyme occurs in human beings with the genotype of lactase nonpersistent.*[36] This can occur any time from age 2 as is common in Thai toddlers, to from 12 to 14 years, as seen in Finnish children.[34] The majority of the world's population is genetically lactase nonpersistent, and such persons have a reduction in their intestinal lactase activity levels to less than 5 IU per g of wet mucosal weight. The population groups susceptible to lactase nonpersistence are Oriental, South Asian, Sub-Saharan African, southern European, and native Americans.[37]

*The terms "lactase nonpersistent" and "lactase persistent" have recently been introduced as alternatives to the standard designations of "primary adult lactase deficient" and "lactase sufficient," which are considered to be excessively judgmental and inappropriate since the *majority* of the population normally has *low* lactase activity.

Absorption of calcium and bioavailability are important.

Certain select ethnic groups bear a mutation of the lactase gene that enable them to maintain high lactase activity throughout life (lactase-sufficient). In the United States, persons of Germanic, Scandanavian, and Anglo-Saxon heritage are generally more than 90% lactase persistent. Moreover, as the gene for lactase persistence is autosomal dominant,[38] ethnic intermarriage has resulted in the extension of this condition to some members of nonwhite groups. However, among Mexican-Americans, Central Americans, Philippinos, Haitians, and Indo-Chinese — the "new" ethnic immigrants — and among Orientals, Italo-Americans, Sephardic Jews, and Afro-Americans living in the United States, the lactase-nonpersistent gene has predominated.

The consequences for milk consumption of lactase genotypes are complex. What is clear is that the residual lactase in lactase nonpersistent persons is capable of digesting only a few grams of oral lactose from a meal. Amounts of lactose beyond this are incompletely hydrolyzed and reach the colon, where the sugar is hydrolyzed by intestinal bacteria. This portion of sugar fermented in the colon is the basis for gas formation and the liberation of osmotically active derivatives of the sugars (short-chain fatty acids); this, in turn, can produce the subjective experience of symptoms, such as belching, flatulence, cramping pains, abdominal distension, and diarrhea, the so-called syndrome of *lactose intolerance*.[39]

Confusion has reigned regarding the importance of lactose intolerance as a dietary factor. This derives from the *clinical* diagnostic criterion applied by the medical profession, one which relates poorly — if at all — to the response to usual dietary quantities of milk, cheese, ice cream, etc. This criterion was the person's subjective symptom response to 50 g of lactose in a concentrated aqueous solution during a conventional "lactose-tolerance test" (50 g of lactose is the amount in a liter of cow's milk). In practice, persons consume a glass of milk (12 g of lactose) and perhaps an additional amount of lactose in a portion of a dairy product, such as, cheese, ice cream, cottage cheese, or milk in cereal (6 g lactose) in a typical meal. More relevant to a person's ability and willingness to include dairy items in the diet would be their experience after consuming 12 to 18 g of lactose as food. Newcomer[40] summarized the scientific literature in which lactase-nonpersistent persons were given 12 g of milk (the equivalent of a glass). He found about 20% of persons who were lactose maldigesters to actually be intolerant when tested with the 12 g dose. This can be viewed from various perspectives. First, 80% of persons with low lactase can eat *dietary* amounts of lactose-containing foods at will, without symptoms. Second, 20% of the affected symptomatic persons, on the other hand, is a substantial number of persons who might be restricting milk and dairy products (hence limiting calcium consumption). Third, some forms of remedial action on a dietary basis have been devised for this particular group.

Perhaps the simplest maneuver to reduce symptoms of lactose intolerance is to combine milk with solid foods. Evidence suggests that slowing down the transit of lactose through the gut is an effective way to reduce colonic fermentation of the sugar.[41, 42] Psyllium hydrophilic colloid, a natural fiber from psyllium seeds, used conventionally as a bulk laxative, had been shown to diminish lactose fermentation and improve lactose tolerance in highly susceptible Indo-Chinese lactose-maldigesting persons;[43] the mechanistic bases for this effect remain to be clarified.

A variety of microbial organisms elaborate β-galactosidase enzymes capable of hydrolyzing the bond uniting galactose to glucose in lactose. They have been commercialized in various industrialized nations.[44, 45] The application of this principle to the treatment of lactose intolerance has been joined in three ways. The first was to incubate

Table IV. Recommended daily allowances for calcium by age and sex for the United States (National Academy of Sciences, 1980)

Population age	Calcium intake (mg/day)
Infants	
0.0-0.5 yr	360
0.5-1.0 yr	540
Children	
1-3 yr	800
4-6 yr	800
7-10 yr	800
Males	
11-14 yr	1200
15-18 yr	1200
19-22 yr	800
23-50 yr	800
+51 yr	800
Females	
11-14 yr	1200
15-18 yr	1200
19-22 yr	800
23-50 yr	800
+51 yr	800
Pregnant woman	1200
Lactating woman	1200

Adapted from the ninth edition of the *Recommended Dietary Allowances*, 1980.[63]

milk *in vitro* with minute quantities of semipurified enzyme.[46] It was found that this process virtually eliminated symptoms of lactose intolerance in lactose-sensitive maldigester persons.[47, 48] Stable preparations for home use in one's own refrigerator are on the market. A slightly sweeter taste, caused by the free monosaccharides, is imparted with total hydrolysis; at 70% hydrolysis, the original flavor of milk is unaltered, but therapeutic effects against intolerance are maintained.[44] Such a processed fluid milk is available commercially in North America.

Although the rigors of gastric acidity and gastrointestinal proteolysis would be expected to inactivate any orally administered microbial lactases, such is not the case. Rosado et al.[49] showed that a β-galactosidase from *Kluyveromyces lactis* resisted in vivo destruction and effectively assisted the digestion of lactose when used as "enzyme-replacement therapy" orally in lactose-maldigesting persons when consumed with a meal. It is also feasible to use this in vivo replacement approach to treat lactose maldigestion in young children.[50, 51] The enzymatic effect persists, even when milk is consumed in a meal with solid foods.[52] This approach adds convenience to the use of enzymes, since milk consumed away from home can easily be treated. However, four times as much enzyme as needed for preincubation is required.

Yogurt, contrary to conventional opinion, contains lactose. In fact, it has 5% lactose, the same concentration of the sugar as in cow's milk. It has long been known, however, that the symptoms of lactose intolerance do not occur with this fermented product. Kolars et al.[53] determined the basis for this tolerance of yogurt by showing that hydrolysis "autodigestion" of yogurt lactose is produced by β-galactosidases liberated by gastric disruption of the culture bacteria. In pasteurized yogurt, in which bacteria are not viable, no assisted lactose digestion occurs, and symptoms of lactose intolerance are produced.[54] The effectiveness of intrinsic bacterial enzymes in yogurt has been confirmed in studies in other laboratories.[55] Thus, a first therapeutic approximation for promoting acceptance of a lactose-containing food or beverage by a lactose-sensitive maldigesting person, thus augmenting dietary calcium sources, can be found in the aforementioned strategies, alone or in combination with one another.

As a number of the treatments discussed here involve the hydrolysis of lactose before it approaches the intestinal membrane, commentary on the role of lactose as a promoter of calcium absorption by the diffusional (lower intestinal) mechanism is warranted. The bases for the observation of an enhancing role on calcium uptake by dietary lactose — even in lactase-deficient experimental animals — has been thoroughly reviewed.[14, 15] Studies of human subjects relating lactose-containing and lactose-free beverages (aqueous solutions or cow's milk) to calcium absorption and lactase status have been reported in children[56-58] and adults,[59-62] with inconsistent and conflicting results. In infants, lactose was found to increase the apparent absorption of calcium. In lactase-deficient children, 9 to 16 years of age, studied with metabolic balance techniques, calcium absorption was greater from a diet that contained lactose-hydrolyzed milk, compared with the lactose-free diet with extra calcium.[58]

A number of dietary factors have been tested with relation to the absorption of calcium.

Kocian, Skala, and Bakos[60] and Cochet et al.[61] found reduced calcium absorption in lactose-maldigesting adults who consumed a test dose of calcium with lactose. Tremaine et al.[62], however, found no difference between the absorption of calcium when they compared within-subject values with lactose-containing and lactose-hydrolyzed *milk* in either persons who were lactose maldigester or lactose digester. However, they did note that despite equivalent circulating 25-OH vitamin D levels, the efficiency of calcium absorption with both beverages was higher in lactase-nonpersistent persons than in lactase-persistent persons. They attribute this provisionally to a lower habitual calcium intake among the low-lactase persons who customarily avoided milk and dairy products. Thus, it would seem that, on balance, the efficiency of calcium uptake is not severely compromised by the consumption of milk with the lactose hydrolyzed to preclude this option as an approach to the augmentation of calcium in the human diet.

DIETARY ALLOWANCES OF CALCIUM AS A "NUTRIENT"

The furor that has arisen over the circumstances surrounding the decision not to publish the tenth edition of the *Recommended Dietary Allowances* has brought into sharper focus the distinction between a dietary substance as a "nutrient" (that is, a chemical to "fill the body's tank" for adequate status of the nutrient-dependent functions of normal metabolism) and a dietary substance as a "health promoter," or for use in nutrient-responsive pathologic conditions, i.e., taken in quantities above the nutrient requirement to repair damage (such as bone loss) or to prevent damage (such as hypertension or cancer). Under this heading, we are to consider the former role of calcium, i.e., as a *nutrient*.

Over the years, expert scientists have contributed estimations of the safe or recommendable daily dietary intakes of nutrients to assure that most of the healthy population would receive what their individual members required to support adequate nutriture. In the United States this has been the Recommended Dietary Allowances Committee of the National Academy of Science's Food and Nutrition Board. The most recent RDA book, the ninth edition,[63] was published in 1980. The calcium recommendations are presented in Table IV. Earlier, in 1974, an estimate of safe intakes meant to embrace the world's population was produced as the *Handbook of Human Nutrient Requirements*[64] by diverse committees of the United Nation's Food and Agriculture Organization and the World Health Organization. Their calcium recommendations are presented in Table V. Obvious differences are the greater detail in population categories in the former manual, and the generally lower recommendations for calcium intake produced by the latter body, with a worldwide focus. An adequate calcium intake of 500 mg for adult women, is projected by the *Handbook*, while the 1980 RDA recommends 800 mg for American women.

The overriding reality, with respect to actual daily calcium intakes by segments of the United States population has been that survey data consistently find the majority of individuals consuming less than the RDA level of the mineral. Dairy products have consistently supplied about 75% of the calcium in the diet in the United States. Calculations by the United States Department of Agriculture suggest that from 1909 to 1913 per capita calcium intake was 800 mg, from 1947 to 1949 it was close to 1000 mg, and by 1979 it had receded to 900 mg.[65] However, these *per capita* data might be excessively determined by the abundant calcium consumption of the young men in the sample. Avioli[66] has interpreted the calcium-intake findings of the 1971 to 1974 United States National Health and Examination Survey. It found that men 35 to 44 years of age had an average intake of 700 mg of calcium daily, while women in the same age bracket consumed only 500 mg. The only groups in the NHANES to meet their respective RDAs for calcium were females up to 11 years of age, and males up to 25 years. Thereafter, the means for both sexes fell below the recommended intake levels. The earlier falling-off in calcium intake for girls may explain some of their bone mineral problems in later life, for the calcium intakes remain low for the female population. For instance, when Albanese[67] had the diets of older women supplemented with calcium, he found their baseline spontaneous dietary calcium intake to be 400 mg; supplementation regimens raised it to the desired 1000 mg.

INTAKE ALLOWANCES FOR "CALCIUM-RESPONSIVE" CONDITIONS

The evidence is inconclusive for a consistent and universal role of *enhanced* calcium intakes in benefiting established metabolic bone disease, preventing essential hypertension or toxemia, or warding off colon cancer, although certain subgroups of the population probably are susceptible to beneficial effects in all three regards, as indicated by the companion papers. Without being overly optimistic and uncritical, let us operationally define these and other conditions of a similar nature, uncovered through future investigation, as "*calcium-responsive*." The differential point is that prescription of the mineral is for purposes other than maintaining the person's nutriture, with respect to calcium. The mechanism could be to produce positive calcium balance, as in the case of bone mineral loss; to produce a put-through of calcium into the bloodstream and extracellular fluids, as in the case of blood-pressure-related conditions; or to increase the exposure of the large intestine to calcium, that is a greater *colonic* put-through of the mineral. Although data with which to fix "therapeutic" or "effective" levels are scant, the same basic concept as pertains with nutrient allowance must be operative with calcium-responsive conditions, namely, increase the intake level of calcium from what it is to what it *should be*. More specifically than in the case of the RDAs, it is assumed that the susceptible population's current calcium intake level (or form of oral ingestion) is suboptimal. The *target* levels will be established as experience is gained in the domains discussed in the accompanying articles, and it is of little service here to insist on one or another milligram (or gram) figure. This is a conceptual prelude to the final element of this review on strategies to attain greater calcium intakes. Whatever the prescribed level of calcium for a calcium-responsive state, some behavioral approach to consuming more of the mineral must be followed, just as to ensure compliance with a generous nutritional allowance.

Table V. Recommended safe levels of calcium intake from the WHO/FAO expert group*

Population	Calcium intake (mg/day)
Infants 1 yr	500-600
Children 1-3 yr	400-500
Children 4-6 yr	400-500
Children 7-9 yr	400-500
Adolescents 10-12 yr	600-700
Adolescents 13-15 yr	600-700
Adolescents 16-19 yr	500-600
Adult man 20 yr and above	400-500
Adult woman 20 yr and above	400-500
Pregnant woman	1000-1200
Lactating woman	1000-1200

*Adapted from FAO/WHO *Handbook of Human Nutritional Requirements*.[64]

STRATEGIES FOR ACHIEVING RELATIVELY HIGHER ORAL CALCIUM INTAKES

The inevitable consequence of the foregoing discussion is that, be it for nutritional or therapeutic reasons, many persons will be found by their physicians or counselor, or by an evaluation of their own diets in terms of the RDA, present or projected, to be *wanting* in calcium intake. The operational goal can be simply stated in a strategy for increasing one's oral calcium intake, namely, as filling the *gap*. This is as follows:

Nutritional paradigm
- — My actual intake (estimate) for calcium is 650 mg.
- — My dietary requirement (perceived for me) is 1000 mg.
- — My gap to be filled is 350 mg.

* * *

Table VI. Strategy options for increasing the amount of ingested calcium

From dietary sources	From inorganic sources
Increase dairy product consumption	Add a vitamin-mineral supplement
Use calcium-fortified milk	Add commercial calcium-specific supplements:
Increase consumption of natural high-calcium foods (e.g., traditional tortilla, teff, bony fish)	Oyster-shell calcium
	Calcium carbonate chewable tablets (Tums)
	Calcium carbonate
	Calcium dibasic phosphate
	Chelated calcium
	Calcium lactate
	Calcium gluconate
	Calcium glucobinate

Therapeutic paradigm
— My actual intake (estimate) for calcium is 750 mg.
— As part of a regimen to treat osteoporosis, my physician wants me to consume 1800 mg.
— My gap to be filled is 1050 mg.

What are the considerations and what are the alternative strategies for individuals to comfortably and consistently comply with the recommendations in our two situations and close the gap?

The following two basic strategies for the increment of one's calcium consumption exist: (1) adding more calcium in the form of calcium-rich foods (dietary) and (2) adding an inorganic supplement. Since, in the United States as in Great Britain, milk and dairy products are the richest sources of calcium (140 mg/100 g), increasing the consumption of these dairy products is a simple solution. If a dietary deficit in calcium intake to be filled is 350 mg, the addition of a little more than a glass of milk to the diet would close the gap. This approach — adding milk, cheeses, or yogurt — could be extended to the limits of an individual's dietary adaptation (and economic capacity). However, lactose intolerance could limit the dairy route for calcium enhancement in the diet. In such an instance, the use of exogenous microbial β-galactosidase enzymes, either as preincubation for in vitro hydrolysis or directly added to milk (or taken in the form of pills) with the meal for in vivo assisted digestion, will eliminate symptoms from nondigested lactose reaching the colon.[49-52] Also, yogurt, with its "autodigestion" of lactose by the culture bacteria in the gastrointestinal tract, is another factor promoting tolerance of this food by lactase-nonpersistent individuals. It must be remembered, however, that one glass of milk contains 170 kcal. If another food is not sacrificed, an undesirable weight gain might ensue. To date, health concerns about saturated fat and cholesterol content have detracted from milk consumption. Low-fat milk partially redresses the concern of excess calories and of lipids.

Bony fish could be another source of calcium to close modest calcium gaps on the order of 300 to 400 mg. This would require an average of two additional servings per day. The same calorie burden, as with milk, must be considered so as not to produce overweight. The fish, moreover, should also supply the other micronutrients in the food(s) for which it is being substituted. Recent interest in the unsaturated fatty acids of certain marine fish in terms of better cardiovascular health may collaterally encourage health-conscious persons to "kill two birds with one stone," using fish as the source of calcium and beneficial lipids.

The terms "lactose nonpersistent" and "lactose persistent" have recently been introduced.

Although impractical at the present time because of the lack of industrial production in the United States, traditional high-calcium foods from other countries (namely, Ethiopian *teff* or Mayan-style corn tortillas) could conceivably be produced and marketed in North America to increase the *dietary* options for enhanced calcium intake. The addition of six 30 g tortillas to the diet would close a 400 to 500 mg calcium-intake gap.

Calcium consumption can be increased by the addition of calcium-rich foods and an inorganic supplement to the diet.

Calcium fortification, that is, adding calcium to foods, is an approach that has the advantage of not requiring any behavioral modification in the introduction of a new food or beverage to one's menu and of getting more calcium within one's current energy intake scheme. Milk, by virtue of its flavor characteristics and ability to take more calcium into suspension, is a prime focus of efforts to promote calcium-enriched products. The *addition* of one glass of milk (240 ml), fortified to a level of 500 mg per 8-ounce glass, will close a dietary calcium deficit of that magnitude. *Substituting* a glass of calcium-fortified milk for the customary regular cow's milk by a habitual milk drinker will increase overall daily calcium consumption by about half that amount. One such "super milk" product being marketed is (CalciMilk, LactAid). Both calcium-enriched and lactose-hydrolyzed, it is a product that can be universally tolerated (except by persons with intolerance to other components of milk, such as protein allergy). However, the potential boost in absorptive efficiency may be lost by the lactose reduction. Calcium-fortified milks, with intact lactose content, will be on the market soon. Also, fruit drinks have been found to be successful vehicles for serious calcium fortification, and other foods are being explored. The food and beverage approach has psychological appeal, since no *medication* image or "sick role" is created in the user of this calcium-supplementation strategy.

Larger gaps, of up to 1 g or more of calcium, are not likely to be addressed, except with some recourse to inorganic calcium supplements. This can either be applied after an effort at dietary enhancement (above) or completely on the basis of pills and powders. One source is a vitamin-mineral pill. However, the quantities of calcium being considered are beyond the capacity for one tablet, if other micronutrients are also to be incorporated. Pill-size considerations and the density of elemental calcium in the compound used are indeed major issues with inorganic forms.[23] The following six commonly used calcium supplements, cited by Avioli[23] are (in ascending order for calcium content as a per cent of weight): the glucobionate (6.5%), the gluconate (9%), the lactate (13%), chelated calcium (20%), dibasic phosphate (23.3%), and the carbonate (40%). Thus, in a 1 g tablet of carbonate, one would receive 400 mg of calcium, whereas only 65 mg would be provided by the glucobionate. However, as doubts have been raised as to the effectiveness of calcium carbonate, vis-a-vis other sources, to support bone remodeling and strengthening, the density of packing calcium into a pill cannot be the only consideration in supplementation.

Compounded pills of oyster-shell calcium have also been marketed and have achieved wide acceptance. The possibility of incidental contamination by potentially toxic metals, such as cadmium and mercury, should be a consideration if long-term (lifelong) use of oyster-derived products is to be undertaken with this source. Also, in pregnant women, the fetal exposure to toxic minerals can be a deterrent to the use of oyster-shell sources of calcium.

CONCLUSION

To meet effectively the demands of a daily calcium intake of 800 mg or more, either to satisfy the nutritional dietary allowances or to take additional calcium to address a calcium-responsive condition is not a simple matter. Knowledge of the food sources of calcium in the diet is necessary to optimize the efficiency of that route. After calcium intake from foods and beverages is maximized, commercial inorganic sources of calcium may have to be invoked to meet the target level of intake. It is clear that any barrier to the consumption of milk and dairy products because of lactase nonpersistence can be overcome with dietary modifications. A concern with the ultimate utilization of the various calcium sources, specifically in promoting bone mineralization, is warranted, and it would appear that certain sources are preferable to others for remodeling of bone. More research on the long-term efficacy of various combined strategies to increase individual calcium intake is needed to carry forth the goal of greater consumption of the mineral, targeted by both the medical profession and the public at large, in an age of increasing calcium consciousness.

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