Calcium, Magnesium, Phosphorus and Vitamin D Fortification of Complementary Foods

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ABSTRACT Provision of the bone minerals and vitamin D as fortificants in food or as dietary supplements designed for older infants and toddlers in Latin America is likely to be beneficial and safe. Currently available data are inadequate to establish the precise amounts of these nutrients that would be required for such a supplement. These amounts would vary according to the local base diet. However, reasonable estimates can be made on the basis of current dietary recommendations as well as existing data on bioavailability and customary intake. The strongest case can be made for calcium and vitamin D supplementation. Because excessive dietary calcium can reduce zinc absorption as a result of interactive effects within the intestine, an appropriate ratio of calcium to zinc should be used, even if this means adding zinc as a fortificant or supplement. Magnesium supplementation may be appropriate in some circumstances but it cannot be routinely advocated at present. It is unlikely that phosphorus supplementation is needed for most population groups because of the relatively high usual dietary phosphorus intakes, primarily from phosphate salts added to carbonated beverages and as food preservatives. J. Nutr. 133: 2994S–2999S, 2003.

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Provision of calcium, magnesium, phosphorus and vitamin D as part of complementary foods should be considered to incorporate an adequate supply of these nutrients into the context of the diet as a whole. Several factors must be considered in order to determine the appropriate amount and form of nutrients to provide in each case. These factors include the amount of the nutrient needed to prevent overt deficiency diseases, such as rickets, as well as the cost and form of the fortified nutrients that will yield the highest bioavailability. In addition, consideration should be given to the safety of adding these minerals, especially in terms of evaluating potentially adverse nutrient interactions such as those that can occur between calcium and iron.

There is a paucity of experimental evidence available from which to derive the daily recommended intakes for the amount of calcium needed by older infants and toddlers. Moreover, there are virtually no published data regarding phosphorus, vitamin D or magnesium requirements in this age group. Given the absence of experimental data specific to this age group, current recommended dietary reference intakes are usually interpolated from studies conducted on older children.

Calcium requirements in older infants and toddlers

Accretion. Determining dietary calcium requirements requires the evaluation of the total amount of calcium needed by the skeleton during the ages of interest. The classic method used to determine this is the analysis of cadavers for total calcium content. These analyses were mostly performed more than 50 y ago. Although the chemical analysis was likely to be accurate, uncertainties regarding the cause of death and the health status of the individuals involved, as well as the fact that very few infants were analyzed, render this an unreliable source of information on which to base dietary recommendations (1).

In the past 30 y, various radiological and densitometric methods have been used to determine calcium and bone accretion during childhood. These have ranged from the use of metacarpal morphometry in the early 1970s (2) to more recent analyses using techniques such as dual-energy X-ray absorptiometry (3).

In general, despite the disparity of techniques and popula-
tion studies, estimates of calcium accretion by the skeleton in childhood are reasonably consistent. Typically, mean calcium accretion is ~80 mg/d during the 1st y of life, rising slightly to ~100 mg/d during the 2nd y of life (4). There may be some differences in typical accretion rates of calcium in comparing breast-fed with formula-fed infants (5), with higher values occurring in formula-fed infants; however, these differences have not been well characterized and there is no evidence that they are functionally significant. Of interest is that the rate of calcium accretion (80–100 mg/d) in infancy appears to remain fairly consistent throughout early childhood; it probably does not increase substantially until after age 4–5 y and rises much more rapidly during puberty (3,6), when it can reach levels as high as 300–400 mg/d (7).

Intake. An Adequate Intake (AI) guideline, rather than a Recommended Dietary Allowance (RDA), for calcium intakes was set by the recent Dietary References Intake Panel of the Food and Nutrition Board of the National Academy of Sciences (7). The AI for infants was derived on the basis of expected usual calcium intakes of breast-fed infants during the 1st y of life. It is useful to review the AI of infants aged 6–12 mo and extend this information to what is known about the 2nd y of life.

Breast-feeding is the preferred feeding method for virtually all infants in the 1st y of life. Rickets or calcium deficiency should not occur in vitamin D–replete, breast-fed, full-term infants aged 7–12 mo who have an age-appropriate solid food intake that includes calcium-containing foods. Typical human milk intakes in infants aged 7–12 mo are 500–700 mL/d, with a calcium concentration of 200–220 mg/L. This leads to an estimated total calcium intake of ~130 mg/d from human milk (7,8). There are few studies on calcium intakes from solid food in this age group, but a value of 140 mg/d for formula-fed infants has been reported (5,9). Adding these two calcium intakes led to the published AI value of 270 mg/d for infants aged 7–12 mo (7). However, this value is far below the mean observed calcium intake of 703 mg/d for this age group that has been reported in dietary surveys in the United States (7). Undoubtedly, this difference reflects the use of adapted formulas and whole cow’s milk to provide 500–600 mg/d of calcium at usual intakes of older infants.

During the 2nd y of life, solid foods become a greater part of most children’s diets. From dietary survey data, mean observed intakes in this age group were 766 mg/d whereas the current recommended intake is 500 mg/d (7). At this age, however, most children in the U.S. survey were probably receiving whole cow’s milk as their milk source, leading to the relatively high calcium intake.

Although calcium intake may be low in Latin American children because of lower dairy product intake, this is not the case in Mexico. Murphy et al. (10) found adequate calcium intakes (mean 735 ± 199 mg/d). This is far greater than the mean calcium intake of <220 mg/d found in children in Egypt and Kenya. The higher calcium intake in the Mexican children likely was due to the consumption of both dairy products and lime-treated tortillas in parts of Latin America (10,11). Absorption of calcium from this diet may be low because of poor bioavailability of calcium from the lime-treated tortillas (12).

The relatively high calcium intakes reported by Murphy et al. (10) may not occur among the poorer populations of Latin America. Wyatt and Tejas (13) recently reported large economic differences related to the calcium intakes of children aged 4–6 y in southern Mexico. In the children in the poorest areas, mean calcium intake was only 272 mg/d, primarily from corn tortillas. This increased to more acceptable intakes of 625 mg/d in medium-income families that were due to the increased availability of dairy products in families with a higher socioeconomic status.

In Mexico City a mean calcium intake of 516 mg/d was reported for children aged 1–5 y (14). In this group, 25% of the children had calcium intakes of <361 mg/d, which was associated with higher blood lead levels. This contrasts with a 25th percentile value of 599 mg/d for children aged 1–3 y (649 mg/d for children aged 4–8 y) in the United States (7), suggesting a much greater prevalence of very low calcium intakes in Mexico compared with the United States.

Absorption. There is a general consensus that calcium is well absorbed from human milk, with values for net calcium retention of ~50% of intake (1,9). Calcium absorption values from infant formulas are highly variable because of the various carbohydrate, protein and mineral sources of these formulas. Although it is generally stated that calcium bioavailability is lower from formulas than from human milk, this may not always be the case. Early findings may have been related to the greater concentration of calcium in infant formulas than in human milk (1,7). In general, however, values of 30–40% absorption are typical for cow’s milk–based infant formulas or whole cow’s milk (15,16). The efficiency of calcium absorption from any food source is likely to depend more on the total amount added and its interaction with other food components such as vitamin D, phytate, oxalate and dietary fiber than on the relative solubility of the type of calcium salt added as a fortificant (17). However, if calcium intake is low, then each of these factors significantly affects the efficiency of calcium absorption (18).

Effects of deficiency. It is not easy to clinically identify deficiencies of bone minerals in most children unless they are severe enough to cause recurrent fractures or rickets. The most common disease associated with severe bone mineral deficiency in very small children is nutritional rickets. Although classically, nutritional rickets is caused by vitamin D deficiency, in some children very low calcium intakes may be partially or even primarily responsible for development of the disease (19,20).

Ideally, a calcium intake level below which rickets is likely to occur could be identified. Unfortunately, it is not possible to do this with any certainty. Some suggestions regarding dangerously low intakes can be found in studies of calcium-deficiency rickets in Africa and Asia. For example, Egyptian children aged 1–2 y who had mean calcium intakes of 290 mg/d with adequate vitamin D status did not develop rickets (21). In contrast, in the same study 20 children mostly breast-fed with low vitamin D status and calcium intakes averaging 310 mg/d developed rickets. These results suggest that in the presence of adequate vitamin D, ~300 mg/d of calcium is enough to ensure that rickets will not occur. Among black South Africans, biochemical abnormalities associated with rickets occurred in children aged 7–12 y with calcium intakes of 125 mg/d but not in those with intakes of 337 mg/d. Neither the children with rickets nor the control children were vitamin D deficient (22).

There is strong support for the suggestion that a calcium intake of less than ~300 mg/d may pose a risk for the development of rickets notwithstanding normal vitamin D status (23). In Nigeria, children with calcium intakes of ~200 mg/d were reported to develop rickets despite normal vitamin D levels (24). Many children with similar dietary calcium intakes do not develop rickets in Nigeria. It is likely that crucial subtle differences in calcium bioavailability, genetic factors or other environmental conditions may contribute to the development of rickets at low calcium intakes (20).
Taken together, these data indicate that even without significant vitamin D deficiency, a calcium intake of ~300 mg/d is adequate to prevent rickets in most small children and infants below that, especially below ~200 mg/d, pose a substantial risk of rickets. The minimum intake of calcium to prevent rickets in children with low or marginal vitamin D status is not known but is likely much higher on the basis of substantially lower calcium absorption in that circumstance.

**Recommended intakes.** Given this confused data set, it should not be surprising that it has been relatively difficult to set clear guidelines for calcium intake in the 1st 2 y of life. In general, recommendations have been based either on usual dietary intakes or on balance data from older children. Some intake guidelines specify whether they were based on (although not necessarily only intended for) human milk–fed infants whereas others do not make this distinction. Currently in the United States, the calcium AI for infants aged 7–12 mo is 270 mg/d and for infants aged 12–36 mo is 500 mg/d. For infants on adapted cow’s milk formulas the recommendation is that mineral intake be increased because of the assumption that the calcium is not as bioavailable as that of human milk (7). A recommended intake of 335 mg/d is derived for infants ages 7–12 mo. In the United Kingdom, the recommended pattern of intakes is opposite that of the United States. The recommended calcium intake for infants aged 7–12 mo is 525 mg/d and for infants aged 12–23 mo it decreases to 350 mg/d (8).

Although it is lower than the United Kingdom value, there are no data to indicate that the Food and Nutrition Board’s AI of 270 mg/d for infants ages 7–12 mo leads to bone demineralization or rickets. Calcium intake of 130 mg from human milk and 50% calcium retention leads to a total of 65 mg/d of calcium retained from human milk. If the other 140 mg of dietary calcium comes from solid food, using an estimate for calcium retention of 20%–25% from solid food (7) leads to a calculated additional 28–35 mg/d of retained calcium. These totals are consistent with the 80–100 mg/d accretion value for calcium during infancy.

In the 2nd y of life, 20–30% of cow’s milk calcium is likely to be retained. Based on a cow’s milk calcium intake of 500 mg/d, milk intake alone would meet the 100-mg/d accretion requirement. However, for breast-fed children aged ≥12 mo with a human-milk intake of 400–500 mL/d, the net calcium intake from human milk might be only ~100 mg/d. Assuming 50% retention of calcium from human milk, then 50 mg/d of retained calcium would need to come from solid foods. This would require at least 250 mg/d of calcium (based on 20% retention), or a total calcium intake of 350 mg/d. This value is consistent with the United Kingdom recommendations and somewhat lower than the U.S. recommendations, which were based on 20% retention of dietary calcium.

From these calculations, it is apparent that infants who receive human milk without additional calcium sources and infants who are inappropriately weaned to juices or other noncalcium-containing beverages cannot readily meet the 100-mg/d calcium accretion level. It is not surprising then that rickets occurs in these infants in the 2nd y of life, especially when inadequate vitamin D status would lead to impaired calcium absorption below critical levels.

**Safety of calcium fortification.** The addition of mineral salts as fortificants in foods poses the potential risk of reducing the bioavailability of the native minerals in the food by changing their intestinal solubility or by competing for uptake at absorption sites that may occur between minerals with similar physical and chemical properties. The potential for such mineral–mineral interactions, such as between calcium and iron or calcium and zinc, has been studied primarily in relation to single mineral supplements to the diet rather than as fortificants in foods.

Calcium supplements decrease the absorption of iron contained within food when they are coingested or taken close together (25,26). However, in the context of whole diets, a series of human and animal studies has shown at least partial adaptation to this effect without prolonged inhibition of iron absorption or the development of abnormalities of iron status (27,28). In two randomized studies, there were no detrimental effects on iron absorption or status in infants fed calcium- and phosphorus-fortified formulas to age 9 mo (29), or in children fed calcium-fortified breakfast cereal (30). Thus, to date, no evidence indicates that calcium intake should be restricted to enhance iron status (30,31).

A negative effect of high intakes of calcium and phosphorus (even from milk) on zinc absorption has been demonstrated in adults (32,33) and term (38) infants fed human milk compared with formula, the latter having a higher calcium (and zinc) content. The ratio of calcium to zinc that could affect such an interaction ranged from ~20:1 (by weight) (36) to ≥50:1 (33). The interaction of calcium and zinc may be of particular relevance to infants and children, for whom rapid growth including catch-up growth, or recurrent diarrhea or infections may result in a greater need for zinc.

If the diet is only marginally adequate in zinc as a result of limited selection of zinc-containing foods with reasonable bioavailability (such as those low in phytate content or of animal origin), a high calcium intake may further compromise zinc status in infants and children at risk of marginal zinc status, such as premature infants, children recovering from growth failure and children with persistent diarrhea. Because 29% of the zinc content of the body is found in the bone mineral and as part of enzymes involved in bone turnover and collagen degradation, zinc status is an important factor for bone health (39). For the aforementioned target populations, optimal dietary ratios of calcium to zinc should be determined. In addition, there should be close monitoring of the effects of calcium supplementation on zinc absorption or status in a supplemented population.

In contrast to the above examples of antagonistic mineral–mineral interactions, the competition between lead and calcium for absorption is such that increased calcium intake may ameliorate lead toxicity. In Mexico City an inverse relationship was recently reported between calcium intake and blood lead levels in 200 children aged 13 mo to 5 y (14).

**Fortification recommendations.** Provision of additional calcium by fortification of foods commonly fed to older infants and toddlers has become increasingly common. The rationale for this is related to optimizing peak bone mass as well as preventing rickets. Many children in Latin America likely have low milk calcium intakes and need supplemental calcium from solid foods. It is reasonable for a fortified food to provide 100–200 mg/d of calcium. This could be provided as 100 mg of calcium per 30-g serving of complementary food. This amount is safe, would effectively help prevent calcium deficiency conditions and could be readily incorporated into the products.
Magnesium requirements in older infants and toddlers

Accretion and balance. Few data are relevant to magnesium requirements in children of this age. Analysis of cadaver data has led to an estimate of ~10 mg/d of magnesium accretion by the skeleton in older infants and toddlers. Taking childhood as a whole, a net magnesium accretion of ~4 mg/d has been calculated (7).

The amount of dietary magnesium needed to maintain this level of accretion is poorly defined. Human milk provides 20–30 mg/d of magnesium depending on intake level. Solid foods provided to older infants may typically provide ~55 mg/d of magnesium. Based on these data, a magnesium intake of 75 mg/d was set as the AI for infants ages 7–12 mo in the United States (7). For children ages 1–2 y, data from older children was interpolated to younger age groups (41) and an RDA of 80 mg/d was established by the Food and Nutrition Board (7).

Deficiency conditions. Although severe magnesium depletion has numerous readily apparent cardiovascular and neurological symptoms, it is much more difficult to identify early or marginal deficiency (42). The inability to adequately form bone related to vitamin D and parathyroid insufficiency as well as lack of magnesium for bone mineralization may be among the most important results of magnesium deficiency in small children. Serum magnesium cannot be directly related to magnesium status in children of any age and, thus, reliable information on the incidence of magnesium deficiency in children is not available.

Intake. Green leafy vegetables, unpolished grains and nuts are high in magnesium. Dairy products, meat and eggs contain lower amounts of magnesium. Although magnesium intakes may be slightly below optimal values, a severe deficiency in small children is uncommon. Murphy et al. (10) found mean intakes of about 250 mg/d in toddlers in Mexico, consistent with values in the United States. Similar values were seen in Kenya and Egypt, although the calcium intake was low in these countries. The RDA of 80 mg/d for children ages 1–3 y is at the first percentile level of U.S. intakes (7). Although excess magnesium consumed as a supplement can undoubtedly lead to diarrhea (43) and, at very high levels, to neurological and cardiac toxicity (44), this is very unlikely to occur with food fortification strategies providing relatively small amounts of magnesium.

Recommendations. Magnesium does not appear to be severely limiting in most diets in Latin America. However, provision of calcium supplementation of foods without magnesium is highly controversial. A ratio of 4:1 or lower (by weight) of calcium to magnesium has been advocated (45). Although the overall risk and benefit of this addition cannot be evaluated at this time, the addition of 40–60 mg/d of magnesium to a supplement (as a food fortificant) is very unlikely to have side effects. Until further data are available demonstrating low magnesium intakes or inadequate magnesium status, the addition of magnesium to a complementary food supplement would be a safe but not a necessary step and cannot yet be advocated. Further research is needed to identify the magnesium needs of small children.

Phosphorus requirements in older infants and toddlers

As with magnesium, few data are relevant to the phosphorus needs of weaning infants and toddlers. Estimates of phosphorus accretion suggest that 50–60 mg/d is accepted during the second year of life. Based on dietary and balance data, the AI of 275 mg/d was set for phosphorus for infants aged 7–12 mo (200 mg from solid foods and 75 mg from human milk) (7). For children aged 1–3 y, an RDA of 460 mg/d was established based on a factorial approach using tissue accretion to set an Estimated Average Requirement.

Intake. Principal dietary sources of phosphorus in older infants and toddlers include cow’s milk (or infant formula), meat and some soda beverages. Because of the ubiquity of these foods, intakes of phosphorus are generally high relative to requirements even for toddlers. Despite calcium intakes of <220 mg/d in Africa, the intake of phosphorus in infants was >500 mg/d. The intake of phosphorus also exceeded the calcium intake in toddlers in Mexico (10). The functional consequence of these high intakes, especially in the presence of low calcium intakes, remains a topic of considerable controversy (46). High phosphorus intakes have been suggested to contribute to hypocalcemia and fractures in children, but further controlled studies are needed to evaluate these relationships (47). Establishment of dietary phosphorus intake recommendations on the basis of an optimal calcium-to-phosphorus ratio, usually ~1.5–2:1 on a molar basis, is also an unproven but common practice.

Recommendations. It is unlikely that the diets of most weaning children need to be fortified with phosphorus. Phosphorus intake could be limiting in breast-fed older infants with very restricted solid food intake. When complementary food is expected to be the sole source of nutrients other than human milk, phosphorus should be included because it is a necessary component for bone and tissue growth. If phosphorus fortification of complementary foods is undertaken, 75–100 mg/d would be reasonable based on the limited available data and the limited potential usefulness of maintaining an appropriate calcium-to-phosphorus ratio. However, fortification of phosphorus should only be undertaken where clearly needed, as most children are more likely to have excess than inadequate phosphorus intake.

Vitamin D requirements in older infants and toddlers

Sources. Vitamin D is required for calcium absorption and is also involved in maintaining bone mineral homeostasis and regulating renal calcium excretion. Severe vitamin D deficiency is the primary etiology of most cases of nutritional rickets. Vitamin D is primarily derived naturally from cutaneous synthesis with exposure to ultraviolet radiation from sunlight. For populations with limited sun exposure because of environment, clothing or housing conditions, vitamin D is usually available in fortified milk because only a few foods, such as liver, contain significant amounts. All cow’s milk in the United States is fortified with vitamin D at a level of 2.5 μg/240 mL (100 IU/240 mL). Relatively few other foods are fortified with vitamin D, although many breakfast cereals provide ≥1 μg/serving. Relatively little vitamin D is present in human milk except when high dose supplementation of the lactating mother’s diet occurs (48).

Prevalence and causes of deficiency. There are few data regarding vitamin D intake, serum levels of 25-hydroxvitamina D or vitamin D–deficient rickets incidence in Latin America. It is likely that dietary intake is relatively low for most small children. Murphy et al. (10) reported mean intakes...
of 0.53 ± 0.45 μg/d of vitamin D among school children in Mexico.

Most vitamin D in the Latin America population is likely the result of sunshine exposure and resultant UV-B conversion in the skin of provitamin D to vitamin D. Vitamin D status and sunshine exposure cannot be assumed to be adequate throughout the Americas. In Ushuaia in Southern Argentina, plasma 25-hydroxyvitamin D concentrations (the best measure of vitamin D status) were suboptimal in both light- and dark-skinned children in winter (49). Approximately 70% of children had plasma 25-hydroxyvitamin D concentrations <30 nmol/L during the winter. Vitamin D status was greater in light-skinned than in dark-skinned children only during the summer. Increased urbanization with resultant decreased time spent outdoors and increased air pollution may also contribute to lower sunshine exposure in children. In general, skin absorption of UV-B is related to melanin and is therefore lower in darker-skinned populations. In Houston Texas, we found lower plasma 25-hydroxyvitamin D concentrations at all seasons in Mexican-American children than in Caucasian children (50). There was no functional effect of this difference because both groups had similar rates of calcium absorption and bone mineral content. Two of the 16 Mexican-American girls had 25-hydroxyvitamin D concentrations < 40 nmol/L and no concentration was <30 nmol/L.

The importance of fortification of vitamin D through the diet is increasingly recognized for breast-fed infants. In many cases the vitamin D deficiency will be subclinical but identifiable by a plasma biochemical profile of low plasma 25-hydroxyvitamin D (<30 nmol/L), high parathyroid hormone and low or subnormal calcium (52). Infants identified with such a biochemical profile normalized their status when supplemented with vitamin D for 1 mo with no signs of hypercalcinemia (51). Data from a European study demonstrated greater preadolescent bone mass in children who received vitamin D fortification in infancy than in children who did not (52).

Although rickets is not widely reported in Latin America, it may be present in high risk areas. A much higher incidence of rickets was reported in southern than in northern areas of Argentina, associated with an up to 52% incidence of very low (<20 nmol/L) 25-hydroxyvitamin D concentrations in southern Argentina compared with a 9% incidence in Buenos Aires (53).

In the United States there has been a dramatic recent increase in the incidence of rickets, primarily in African-American infants and toddlers (54). Many of these were breast-fed and essentially none had received supplemental vitamin D. This problem may worsen in the United States and Canada as the frequency of breast-feeding beyond age 1 y increases. Other factors increasing risk of rickets such as less time outdoors and increased sunscreen use, may make vitamin D–deficient rickets become more prevalent. It is of note that at least in the United States, rickets occurs in children of all socioeconomic classes.

**Recommendations.** The routine provision of vitamin D supplementation for all breast-fed infants is controversial. Although rickets is apparently uncommon in Latin America, increased breast-feeding, urbanization, the presence of air pollution, use of sunscreens and lack of cow’s milk–vitamin D fortification place infants and small children at risk for inadequate vitamin D status. It is reasonable to consider adding a small amount of vitamin D to any complementary food if this is technically feasible. A level of 1–2 μg/d would be consistent with other fortification strategies. This level would be safe based on the Tolerable Upper Intake Level of 25 μg/d established by the Food and Nutrition Board for infants aged <12 mo (7).

**LITERATURE CITED**


