Low Calcium Intake Among African Americans: Effects on Bones and Body Weight

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ABSTRACT This review was performed to summarize and integrate the evidence relating calcium intake to health status in African Americans, with special attention to bone and fat. Despite lower average calcium intakes, African Americans typically have skeletons more massive than those of whites. This is the result of a relative resistance of the bony resorptive apparatus to parathyroid hormone, which forces better urinary conservation of calcium and, at some life stages, more efficient intestinal calcium absorption as well. This adaptation, however, has other costs and appears to contribute to a greater risk in African Americans for several chronic diseases, including cardiovascular disease and stroke, obesity, and the insulin resistance syndrome. Higher calcium intakes not only support the skeleton in African Americans, just as they do in whites, but reduce the disease burden for other chronic diseases as well. J. Nutr. 136: 1095–1098, 2006.

KEY WORDS: • calcium intake • calcium requirement • osteoporosis • fracture risk • obesity

Adequate dietary calcium intake is essential for the optimal function of many body systems, and there is a persuasive body of new evidence that low intakes contribute to, or aggravate, the disease burden of disorders as varied as osteoporosis, kidney stones, hypertension, colon cancer, and obesity (1,2). This new evidence reflects the emerging recognition of what can be called long-latency deficiency disease (3). Nutrition has largely operated around the premise of a single deficiency disease for each nutrient. Thus, vitamin C deficiency produced scurvy, thiamine deficiency beriberi, vitamin D deficiency rickets, and so on. For all these nutrients the latency period for disease was short, with dysfunction manifesting itself generally within a few weeks following withdrawal of the nutrient. This was unavoidable because otherwise early nutrition scientists would not have been able to make the connection between cause (deficiency) and effect (disease).

Because most nutrients are essential for optimal functioning of most cells, the notion of 1 disease per nutrient had to be considered implausible on its face. Nevertheless, operationally, it led to the eradication, in the developed nations, of most of the major nutritional deficiency scourges. The growing recognition of the involvement of nutrients in chronic diseases, distinct from the index disease classically associated with each nutrient, has led to a more comprehensive scheme for a generic nutrient (Fig. 1).

It is instructive to insert calcium into this scheme, as in Figure 2. The index mechanism here is inadequate absorption of calcium (or equivalently, excess excretory calcium loss), and the principal nonindex mechanisms are 1) binding of harmful digestive by-products in the gut lumen by unabsorbed calcium and 2) “off-loop” responses to the elevated production of parathyroid hormone (PTH), which is a regular concomitant of low calcium absorption (2). (“Off-loop” in this context means that the effects produced are not a part of the feedback loop regulating PTH secretion.) This background understanding helps clarify certain features of calcium nutriture in African Americans, discussed in what follows. Briefly, available evidence suggests that the balance between the index disease and nonindex diseases may be different for blacks and whites.

Calcium intake and bone health. As documented in the National Health and Nutrition Examination Survey (NHANES), African Americans have both lower calcium intakes than...
whites and higher bone mass (4,5). The seeming contradiction presented by these 2 observations has led some to conclude that calcium was irrelevant to bone health. However, this difference means only that the 2 races have different calcium requirements for skeletal health. The key question is whether within-group calcium intake is related to bone mass. The evidence in whites on this point is overwhelming (6). Fewer studies have been done in African Americans, but such evidence as is available (2) indicates that, just as for whites, for primates, and for most laboratory animals, adult bone mass is, indeed, a function of calcium intake.

The explanation for the seeming paradox is found in the threshold behavior of calcium nutrition, as depicted in Figure 3. Calcium, like iron and several other nutrients, functions as a "threshold nutrient," meaning that health status improves as intake rises up to some point, the "threshold," above which further increases in intake produce no further effect. Figure 3A depicts this relationship for calcium retention (or calcium balance, which is the determinant of bone mass in mature adults). The threshold, indicated by the asterisk in the figure, is the point below which there is some measurable change in skeletal status related to intake. Figure 3B contrasts a schematic intake–retention curve in African Americans with that of whites, indicating that, in African Americans, the threshold point is shifted to the left; that is, it occurs at a lower calcium intake. Figure 3B also contrasts hypothetical intakes for the 2 curves, 1 for African Americans and the other for whites, showing graphically how the same intake supports better calcium retention in blacks than in whites.

In addition to better utilization of dietary calcium (and, indeed, the mechanism responsible for that better utilization), African Americans have been shown to have a relative resistance to the bone resorptive action of parathyroid hormone. As a consequence, at prevailing calcium intakes, African Americans have a higher circulating level of parathyroid hormone (in part because bony resorptive response to PTH is blunted and in part because calcium intake is low), lower urinary calcium loss, and, at some ages, higher calcium absorption as well. The evidence for these differences is documented elsewhere (2), and here it is useful only to note that Aloia et al. (7), in a study matching African American and white women for weight, found that African American women had lower excretion of bone resorption biomarkers despite a PTH level that was as high as or higher than those in whites. Similarly, Cosman et al. (8) showed that, with the same infused dose of parathyroid hormone, African Americans had a smaller rise in resorption biomarkers than did whites. It is this relative resistance that forces higher PTH secretion and leads to more efficient utilization of dietary calcium.

Calcium intake and body weight. Various epidemiologic and observational studies have shown a pattern of an inverse relationship between dietary calcium intake and risk of being obese or overweight (9–13). The associations described are highly consistent across study designs and databases as varied as NHANES, Continuing Survey of Food Intakes by Individuals (CSFII), the Quebec family study, the Heritage study, and the Coronary Artery Risk Development in Young Adults (CARDIA) study, to mention only some. Parikh and Yanovski, in their review of this issue, commented specifically on the high degree of consistency across the various lines of evidence (14).

For the most part, the major dietary calcium source in the studies cited has been dairy. The effects have been observed in men and women, black and white; however, in several of the studies, the associations appear to be stronger for women than for men. Additionally, there have been several randomized controlled trials involving, necessarily, fewer individuals (9,15–17). For the most part these controlled trials have shown greater weight reductions for high-calcium diets than low, and greater reductions for dairy products than for calcium supplements containing the same quantity of calcium (14–16). Also, the effect of calcium in these trials seems to be greatest in the context of a weight reduction regimen, and particularly in subjects who are obese at entry. These observations underscore the fact that calories still count. What they add up to is that a diet that provides a caloric deficit and a high calcium intake produces better weight loss than a diet with the same energy content that is low in calcium.
Nevertheless, not all studies have been positive. Barr et al. reviewed 17 randomized controlled trials of calcium supplementation for a skeletal endpoint and found only 1 that showed a weight effect in those given supplemental calcium (18). It should be noted that none of these trials included energy restriction, none restricted entry into trial to obese individuals, and most were too small to have the power to find an effect as small as calcium is likely to have (see below).

Actually the first of the controlled trials reporting an effect was a study in African American hypertensive men whose diets were augmented by 2 servings of yogurt per day (9). Not only was blood pressure reduced (the primary outcome measure of the study), but body fat decreased by 4.9 kg (15.2%). Subsequent trials have shown a shift in the components of body mass on diets high in dairy calcium, with relative protection of lean body mass and predominant weight loss occurring from the fat compartment, particularly around the waist (15,16). Additionally, in 1 of those trials (15), insulin responsiveness was measured and was found to be substantially improved in individuals with a high dairy calcium intake (but not in those supplemented with calcium carbonate). This finding is consistent with the observation of the 10-y prospective CARDIA study of young adults, black and white (13), in which conversion from normal health status to both obesity and hypertension was inversely related to dairy intake.

The mechanism behind the calcium effect on body weight is complex. One small portion of the effect is probably complexification of free fatty acids in the gut lumen by the higher level of dietary calcium, blocking their absorption (19). The caloric deficit that can be produced in this way, at the calcium intakes involved in the various trials, is small and not sufficient to explain the observed weight loss, but this phenomenon probably contributes to at least a small portion of the effect.

Another factor is the satiety effect of high calcium intake when it is in the form of dairy foods. At a population level, individuals with high dairy intakes do not have higher energy intakes than those with low calcium intakes (20,21). Clearly, therefore, dairy foods are substituting for other foods, lower in calcium content. At least 2 intervention studies have been reported in which dairy food intake was augmented over short periods of observation, without any formal attempt at reducing nondairy food intakes (22,23). In both studies, total energy intake went up slightly, but body weight did not change significantly in 1 and increased to only a small extent in the other. Moreover, the increase in reported energy intake was substantially less than the energy provided by the augmented intake of dairy foods. This fact, as well as data from diet diaries, indicates spontaneous substitution of dairy foods for other, lower calcium sources.

Finally, at a systemic level, low calcium intakes are well known to evoke increased secretion of parathyroid hormone. PTH acts on the kidney to reduce excess urinary calcium loss, on bone to increase resorption (thereby releasing calcium from the bony reserves), and on the gut to improve calcium absorption, through the endocrine action of vitamin D, that is, the synthesis of 1,25(OH)₂D in the kidney.

But the same 1,25(OH)₂D, as it turns out, acts in many other tissues as well. In a succession of studies in various genetically modified strains of mice, as well as in both mouse and human cell culture systems, Zemel and his colleagues have shown that 1,25(OH)₂D opens calcium channels in the cell membranes of several tissues (24), allowing calcium to flood into critical intracellular compartments. As a consequence, a variety of genes are switched on or off. In the adipocyte these include, most importantly, an increase in the expression of fatty acid synthase and a decrease in the expression of the enzymes responsible for lipolysis.

Thus, low calcium intake produces a context in which the tendency of the adipocyte is to increase fat storage. This phenomenon, by itself, would not be sufficient to explain either obesity on low calcium intakes or weight loss on high because weight gain or weight loss requires positive or negative energy balance at a whole-body level. In 1 of their mouse models, Shi et al. observed an increased expression of uncoupling protein 2 in the animals fed a high-calcium diet (24), leading to increased consumption of metabolic fuel, that is, negative energy balance. There has been 1 report in humans that is suggestive of the same sort of phenomenon (25), but more work on this issue needs to be done.

Incidentally, this same effect of 1,25(OH)₂D, expressed not in adipocytes but in arteriolar smooth muscle, leads to increased vascular tone and, in otherwise susceptible individuals (such as those with sodium sensitivity), to hypertension. This effect of diet calcium is discussed in greater detail by McCarron, elsewhere in this supplement (26).

In our center at Creighton, we have studied 2 cohorts of women, 1 in the third decade of life (n = 348) and the other centered around age 50 (n = 216), and have found the same inverse relationship between calcium intake and either body weight or weight gain (27) as was reported for NHANES (10), CSFII (10), and the Quebec Family Study (11). In our study calcium intake accounted for only about 3% of the variability in weight; a fraction this small is not particularly surprising in a disorder that is as multifactorial as is obesity. Moreover, it is precisely this smallness that explains why the calcium effect had, for so long, been overlooked and why it could be missed in small trials of augmented calcium intake.

In our data the relationship in midlife women was such that, at prevailing calcium intakes (about 600 mg/d, i.e., 15 mmol/d), average weight gain was about 0.5 kg per year (28). By contrast, at currently recommended calcium intakes (in the range of 1000–1500 mg/d, that is, 25–37.5 mmol/d), average weight gain was zero.

It has been estimated that, at a population level (other things being equal), mean body weight would be about 6 kg less at currently recommended calcium intakes than at prevailing intakes. At a population level, such a difference is substantial and would produce large reductions in the fractions of the population above any given body mass index cutoff value, perhaps as much as an 80% reduction in risk of obesity, as

![FIGURE 4 Superimposition of the body's adaptive hormonal response (right axis) to varying calcium intakes on the skeletal intake-retention curve (left axis). (MCR = maximal calcium retention; MAR = minimal adaptive response.) The line for adaptive response is deliberately schematic, with response high at calcium intakes below total body need but dropping to low levels as intake approximates and exceeds need. (Copyright Robert P. Heaney, 2001. Used with permission.)](https://academic.oup.com/jn/article-abstract/136/4/1095/4664218)
reported from NHANES-III by Zemel (9) and as confirmed in analysis of our own data (28).

Because African Americans tend to have a higher risk of obesity, hypertension, and diabetes than do whites, it would seem particularly important for African Americans to increase their calcium intake. To this end, the National Medical Association has recently released an important consensus document, urging increased calcium (and specifically dairy) intake for all African Americans (29). This statement constitutes explicit recognition of the large body of science summarized very briefly in this review.

**Conclusion.** African Americans are able to adapt to prevailing low calcium intakes in a way that allows them to build and protect a skeleton that is as dense as or denser than those in whites. But the adaptive response [high circulating PTH and 1,25(OH)2D3] that permits this achievement has consequences for other body systems, contributing, for example, to the disease burden of hypertension, insulin resistance syndrome, and obesity. The optimal calcium intake for African Americans would be an intake that minimizes the need for an adaptive response and thereby reduces the signal that contributes to the burdens of these non-skeletal disorders.

It is important to note that, at the calcium intake that just reaches the threshold for bone, there is still considerable adaptation required, and that, therefore, a higher calcium intake would likely be beneficial for other body systems even if it produces no additional benefit for bone. This discordance between what is needed for bone health and for, for example, cardiovascular health or body weight, is depicted schematically in Figure 4. The size of the intake gap between the maximal calcium retention (i.e., the bony endpoint) and the minimization of the adaptive response is unknown but could easily be as large as 400–600 mg Ca/d (10–15 mmol/d). This is an important issue because the recommended calcium intake for both blacks and whites in the United States is pegged exclusively to a white skeletal endpoint (30). If the calcium requirement had been race-specific, that is, pegged to the intake required to amass and maintain the genetic program for bone in blacks, it almost certainly would not have been high enough to ensure optimal functioning of other body systems. The current adequate intake for calcium may well be correct for blacks, even though it is more than they need for skeletal health. Future policy determinations with respect to recommended intakes will have to take these racial and body system discordances into consideration.

**LITERATURE CITED**