TABLE 1
Calcium intake and output variables

<table>
<thead>
<tr>
<th>Group</th>
<th>Calcium intake, mg/d</th>
<th>Urinary calcium output, $v_a$, mg/d</th>
<th>Net calcium absorption, $S_i$, mg/d</th>
<th>Percentage net calcium absorption, %</th>
<th>Estimated true calcium absorption, $v_a$, mg/d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Placebo</td>
<td>1115</td>
<td>154</td>
<td>174</td>
<td>15.6</td>
<td>20</td>
</tr>
<tr>
<td>Milk</td>
<td>2365</td>
<td>205</td>
<td>396</td>
<td>16.7</td>
<td>191</td>
</tr>
<tr>
<td>Calcium carbonate</td>
<td>2315</td>
<td>223</td>
<td>474</td>
<td>20.5</td>
<td>751</td>
</tr>
<tr>
<td>Calcium carbonate + vitamin D</td>
<td>2315</td>
<td>246</td>
<td>574</td>
<td>24.8</td>
<td>328</td>
</tr>
</tbody>
</table>

much better. Their conclusions were based on urinary calcium output measurements. Hall et al (2) reported that urinary calcium output ($v_a$, in mg Ca/d) in menopausal women can be described by the following relation:

$$v_a = 114 + 0.23S_i$$

where $S_i$ is net calcium absorption (intake minus fecal output). Transforming the Mortensen and Charles data from millimoles to milligrams of calcium, one can construct Table 1.

The true calcium absorption, $v_a$, was estimated on the assumption that the endogenous fecal calcium output in people is approximately the same as the urinary calcium output and increases at about the same rate with increased intake as the urinary calcium output (3).

The fractional true absorption values one can then estimate are inordinately low (1.8%, 8.1%, 10.8%, and 14.2%). On the other hand, if one estimates how much of the extra calcium was absorbed, one comes up with high values. For example, the CaCO$_3$ + vitamin D group consumed an extra 1200 mg Ca and, from the above table, would have absorbed an extra 400 mg ($574-174$ mg), or 30%. These contradictory calculations show how important it is to do careful balance and tracer experiments before inferring that a fairly insoluble salt like CaCO$_3$ (4, 5), even when ingested in divided doses, is absorbed as well as is calcium from milk.

Mortensen and Charles expressed surprise that addition of vitamin D led to increased urinary calcium excretion within 24 h and remained high thereafter for 3 d. In rats that have been fed a vitamin D-replete diet, injection of calcitriol raised duodenal calbindin D$_{9k}$ and calcium absorption within 4 h (6). Humans may therefore respond to additional vitamin D in the diet quite promptly. However, the added vitamin D was only 5 $\mu$g, one-half of what is routinely added to one quart (0.946 L) of homogenized milk in the United States. Added reason, therefore, to be cautious about bioavailability without direct experimentation.

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REFERENCES


Results of a short-term, low-calcium adaptation study should not be generalized to children with persistently low calcium intakes

Dear Sir:

O'Brien et al (1) evaluated fractional calcium absorption (FCA) of white girls aged 8–16 y by using a stable-isotope technique. Mean FCA decreased from 0.58 to 0.26 10 d after switching from a strictly controlled low- to high-calcium diet. Although the girls could enhance FCA on the low-calcium diet, the authors concluded that prolonged periods of low calcium intake would be detrimental for calcium retention and peak bone mass (1). This is the first short-term study in white children showing enhanced FCA (range: 44–75%) at a calcium intake of 7 mmol/d; the results were comparable with those of a recent study in Chinese children with a habitual calcium intake of 21.5 mmol/d and were lower than those of children with a calcium intake of 9 mmol/d (2).

Studies in adults showed that several weeks (3) or even months (4) were needed for the body to fully adapt to a change in calcium intake in terms of calcium homeostasis, hormonal changes, and FCA. No data on baseline FCA, vitamin D status, and bone markers corresponding to the habitual calcium intake of the girls were reported in the present study; it was unknown whether the girls had fully adapted to a change in the experimental calcium diet. Furthermore, before each calcium kinetic test, only a 10-d interval was allowed for adapting to a new calcium diet, so it was uncertain whether they had reached a new steady state of calcium metabolism. If the children had been put on experimental diets with sufficient time for full
adaptation to occur [≥4 wk in a recent study (3)], different results might have been obtained.

Although FCA during the low-calcium diet was elevated, the estimated true calcium absorption and calcium balance were remarked to be low relative to that during the high-calcium diet. In fact, their estimated values were derived from oversimplified calculations based on very brief experimental periods, and an assumption that endogenous calcium loss was unchanged across low- to high-calcium diets. Most importantly, unless a new steady state of calcium metabolism has been reached, the extent of FCA and urinary and endogenous calcium excretion in response to a change in the calcium diet is uncertain.

In the early parts of their article the authors stressed that the calcium diets were under experimental conditions, the study periods were short, and the sample size was small. It was premature for the authors to imply in the conclusion that prolonged periods of low calcium intake would adversely affect calcium retention and peak bone mass in most children. One has to be cautious in generalizing the results from this short-term study with a limited sample size (n = 11) to the population as a whole. Studies of the effects of persistently low-calcium diets on calcium retention and bone deposition of children need to be conducted in individuals habituated to low calcium intakes before any relevant, meaningful statements can be made (5, 6). It is particularly important for nutritionists and health professionals in Asia to realize that most calcium studies related to requirements and bone mass in childhood were mostly conducted in whites, whose dietary patterns, body compositions, growth and development, and to some extent hormonal status, are different from those of Asian children.

In the Discussion, the authors remarked that populations habituated to low calcium intakes have increased fracture risks, and that children who supplemented their low-calcium diets with calcium carbonate were benefited by enhancing their bone mineral mass. In fact, emerging epidemiologic data comparing the East and West revealed that Asians, although they have lower bone mass and lower calcium intakes, have an incidence of hip fracture that is only half of that of white US women (7). On the other hand, a recent follow-up study in white twins (8) and two follow-up studies in Chinese children (9; WTK Lee, SSF Leung, DMY Leung, JCY Cheng, unpublished data, 1996) revealed that the lasting effects of enhanced bone mineral from three well-controlled calcium trials disappeared after withdrawal of calcium supplements. Therefore, modest increases in bone mineral resulting from calcium supplementation may be a transient effect on bone turnover with exposure to a high-calcium load (10). Well-designed calcium supplementation trials coupled with a careful calcium kinetic study and sufficient sample size are warranted to study calcium requirements in children with prolonged periods of low calcium intakes.

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Letters to the Editor

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REFERENCES


Reply to WTK Lee

Dear Sir:

I would like to reply to the comments made by Lee regarding our study on the effects of low-calcium diets in children (1). Our study was designed to address the response of calcium absorptive efficiency to acute periods of low calcium intake in white children. We found that during short periods of inadequate calcium intake, urinary calcium excretion decreased significantly, and the calcium absorption efficiency increased significantly to 57%, a value comparable with those that Lee et al (2) reported in younger Chinese children consuming low calcium intakes.

One concern raised was that perhaps a 4-wk or longer study period would alter the study findings, as evidenced by prior data in adult white women (3, 4). Although these studies by Dawson-Hughes et al did follow hormonal and whole-body retention of a calcium radiotracer for an 8-wk period, the whole-body retention of calcium was significantly increased by week 1 and remained fairly constant despite alterations in calcitropic hormones over this time interval (3, 4). Furthermore, given the significant decrease in urinary calcium excretion in the girls from our study after 10 d of a low-calcium diet, we feel that it is unlikely that urinary calcium excretion would have decreased substantially if the study interval had been extended.

Although we did not provide baseline data on the habitual calcium absorption efficiency in these children, our prior data from a similar group of 51 healthy white children with