Calcium requirements in man: a critical review

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Summary
Many official bodies give advice on desirable intakes of calcium but no clear evidence of a calcium deficiency disease in otherwise normal people has ever been given. In Western countries the usual calcium intake is of the order of 800–1000 mg/day; in many developing countries figures of 300–500 mg/day are found. There is no evidence that people with such a low intake have any problems with bones or teeth. It seems likely that normal people can adapt to have a normal calcium balance on calcium intakes as low as 150–200 mg/day and that this adaptation is sufficient even in pregnancy and lactation. Inappropriate concern about calcium intake may divert attention and resources from more important nutritional problems.

Calcium is essential for the structure of bones and teeth, for the normal function of all cells and for many enzyme-controlled reactions. Changes in the concentration of calcium in the extracellular fluid lead to disturbances in the function of the nervous system, the heart and other muscles. While it is clear that calcium is an essential element can we define its minimal requirement in the human diet?

The identification of the minimal human requirement for any nutrient generally depends on finding the intake below which a deficiency disorder is likely to occur, but in the case of calcium no deficiency disorder has been recognized. The dietary intake of calcium per person varies greatly from 1000–1400 mg/day in Western countries to 200–500 mg/day in Japan and some developing countries (FAO/WHO Expert Group, 1962). In the West the calcium is derived mainly from milk, milk products and flour fortified with calcium. In developing countries, vegetables are the main source of calcium unless fish is an important constituent of the diet (Table 1). Many population studies of communities with different levels of calcium intake have been carried out without demonstrating any differences in the quality of bones or teeth (Walker, 1972). Further, it has been known for more than 30 years that with a low intake of calcium the proportion of calcium absorbed from the diet increases. For example Hegsted, Moscoso and Collazos (1952), using volunteers from among the inmates of a Peruvian prison, showed that intestinal absorption was adequate to maintain calcium balance even when the diet contained as little as 100–200 mg calcium. This 'adaptation', which takes about a week to be effective, does not occur in the absence of vitamin D. In 1953 Nicolaysen, Eeg-Larsen and Malm suggested that an endocrine factor, which they called the endogenous factor, was responsible. It is now clear that adaptation depends at least in part on the hormonal metabolite of vitamin D, 1,25-dihydroxycholecalciferol (Pento et al., 1977, Fig. 1).

<table>
<thead>
<tr>
<th>Source (per cent of total)</th>
<th>United Kingdom (urban)</th>
<th>United Kingdom (village)</th>
<th>Ivory Coast</th>
<th>India</th>
<th>Japan</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cereals</td>
<td>28</td>
<td>15</td>
<td>27</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>Starchy roots</td>
<td>2</td>
<td>1</td>
<td>37</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>Pulses and nuts</td>
<td>—</td>
<td>2</td>
<td>2</td>
<td>16</td>
<td>26</td>
</tr>
<tr>
<td>Vegetables</td>
<td>4</td>
<td>7</td>
<td>2</td>
<td>17</td>
<td>17</td>
</tr>
<tr>
<td>Fruit</td>
<td>2</td>
<td>3</td>
<td>—</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Meat and poultry</td>
<td>2</td>
<td>3</td>
<td>2</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Eggs</td>
<td>2</td>
<td>2</td>
<td>—</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Fish</td>
<td>1</td>
<td>—</td>
<td>57</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>Milk and products</td>
<td>58</td>
<td>64</td>
<td>—</td>
<td>34</td>
<td>9</td>
</tr>
</tbody>
</table>

Many early attempts to define the requirements of calcium were based on balance studies in which the intake of calcium was varied and the lowest intake consistent with a 'positive calcium balance' was regarded as a desirable amount. Thus in 1950 the British Medical Association recommended 800 mg calcium daily. Such studies frequently were too short to permit complete adaptation and also were subject to serious errors which are only now being fully appreciated (Isaksson and Sjögren, 1967). The limitations of the information derived from balance...
studies are underlined by the differences between conclusions from short-term balance studies and those based on studies of the bones and teeth in people whose habitual diet contains less than 400 mg/day. Such studies, mainly in developing countries, provide a natural opportunity for examining the long-term effects of a 'low' calcium intake.

Low calcium diet

\[ \downarrow \]

Fall in calcium absorption

\[ \downarrow \]

Fall in plasma calcium

\[ \downarrow \]

Increase in production of parathyroid hormone by parathyroid glands

\[ \downarrow \]

Increased production of 1,25-dihydroxycholecalciferol by the kidney

\[ \downarrow \]

Increase in calcium absorption

\[ \downarrow \]

Plasma calcium restored almost to previous value

**FIG. 1.** Probable mechanisms involved in adaptation to a low calcium diet. It is unlikely that the plasma calcium level falls outside the normal range.

**Calcium requirements in adults**

The earliest estimates of the calcium needs of adults were based on balance studies; the figures proposed varied between 126 and 1020 mg/day in men and between 550 and 1067 mg/day in women (Malm, 1958). The earliest recommendations of official bodies were based on such figures. Thus, in 1953 the Canadian Council on Nutrition advised that an intake of 800 mg/day for men of 82 kg was a 'nutritional floor beneath which maintenance of the health of the people cannot be assumed'. The Nutrition Committee of the British Medical Association felt that in adults 800 mg/day was 'sufficient to establish and maintain a good nutritional state in representative individuals of population groups'.

However, detailed studies of the dimensions, composition and density of bones from communities with a low calcium intake, such as those of South African Negroes (Walker, 1972), have not shown impairment in any respect. In other words such diets are not associated with the development of osteoporosis. Furthermore the incidence of dental caries is substantially lower in 'underprivileged' populations with a low calcium intake than in Western countries. While many factors contribute to this difference it is clear that good teeth are compatible with a habitually low intake of calcium (Sheiham, 1967). In addition to its role in bones and teeth, calcium is involved in many enzymic reactions such as blood coagulation. While plasma calcium levels in populations with a low calcium intake are slightly lower than in the inhabitants of Western countries there is no evidence that any of these reactions are defective.

It seems clear that a calcium intake in the region of 300–500 mg/day causes no disability. Indeed the work of Hegsted et al. (1952) implies that normal people can adapt to a calcium intake lower than 200 mg/day. Calcium balance is impaired in total starvation (Fromm, Litvak and Degrossi, 1970; Kumar, Steen and McGeown, 1972), in patients with intestinal malabsorption or vitamin D deficiency and after partial gastrectomy (Paterson, 1975a). In normal adults, however, a disorder of calcium deficiency has never been described.

In this respect man appears to differ from experimental animals in which osteoporosis can undoubtedly be induced with a calcium-deficient diet (Stewart, 1975). This difference may be more apparent than real; no natural diet which is adequate in other respects is likely to contain so little calcium that intestinal adaptation cannot compensate. The possibility that patients with symptomatic osteoporosis are a special group, for whom the calcium intake is inadequate and dietary calcium supplements are indicated, has been repeatedly explored. Heaney (1971) advised that because osteoporotic patients conserve calcium poorly, 'a high calcium intake should be the cornerstone of any therapeutic regimen'. While calcium supplements cause a slight increase in plasma calcium levels and a fall in plasma parathyroid hormone levels, there is no evidence that symptoms are relieved, fractures prevented or that bone mass is increased (Shapiro et al., 1975; Spencer et al., 1976; Riggs et al., 1976). Large doses of calcium have been given by intravenous infusion but even this has not given consistent evidence of increased bone mass (Jensen et al., 1973; Dudl et al., 1973).

**Calcium requirements of growing children**

Children in Western countries with good nutrition usually grow faster and are ultimately taller than children from less-privileged communities. Although there are many differences in the diets of these two groups it seems unlikely that the inequality of the calcium intake contributes to the differences in growth (Walker, 1972), and the thickness of cortical bone is not reduced on the lower calcium intake. Table 2 shows some of the results of a study of different populations in Surinam. That study also showed that the cortical bone of Javanese school children taking about 30 ml milk daily was the same as that of Javanese children at boarding school taking 400 ml milk daily.
TABLE 2. Thickness of the metacarpal cortex and the dietary intake of calcium in white American and urban Creole children in Surinam (Luyken and Luykoning, 1969)

<table>
<thead>
<tr>
<th>Calcium intake (mg/day)</th>
<th>Americans</th>
<th>Creoles</th>
</tr>
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<tbody>
<tr>
<td>Metacarpal cortex (mm)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boys, age 6 years</td>
<td>2.6 ± 0.4 (18)</td>
<td>2.7 ± 0.3 (35)</td>
</tr>
<tr>
<td>Boys, age 9 years</td>
<td>3.2 ± 0.5 (20)</td>
<td>3.4 ± 0.5 (22)</td>
</tr>
<tr>
<td>Girls, age 6 years</td>
<td>2.4 ± 0.3 (17)</td>
<td>2.9 ± 0.4 (35)</td>
</tr>
<tr>
<td>Girls, age 9 years</td>
<td>3.2 ± 0.7 (21)</td>
<td>3.7 ± 0.5 (25)</td>
</tr>
</tbody>
</table>

Serious malnutrition, malabsorption or vitamin D deficiency in children leads not only to impaired growth but also to a reduction in skeletal mass. Thus Adams and Berridge (1969) found that children with kwashiorkor had a thinner metacarpal cortex than did normal children of the same age. However it seems clear that 'calcium deficiency' does not occur in healthy children whose diet is adequate in respect to nutrients other than calcium.

Pregnancy and lactation

It might be supposed that calcium deficiency would be likely when the demands for calcium are high, that is, during late pregnancy and lactation. The fetus at term contains about 30 g calcium, most of which is accumulated during the third trimester. Lactation calls for 150–300 mg calcium daily and the total needed over six months may be as much as 50 g. In 1962 an expert committee convened jointly by FAO and WHO was of the opinion that increased intestinal absorption could not compensate fully for these excessive calcium demands and that a negative calcium balance would be likely to occur.

Calcium balance studies have shown that most women are in positive balance in late pregnancy but there is no relationship between the amount of the positive balance and the calcium intake (Fig. 2). The wide variety of figures obtained probably reflects the intrinsic unreliability of the method (Isaksson and Sjögren, 1967) but the mean 'retention' is approximately that called for by the fetal demand. One study (Shenolikar, 1970) was carried out in India on women taking between 260 and 420 mg calcium daily. The calcium retention is equal to the fetal need and shows that adaptation, i.e. increased intestinal absorption, can compensate even for such a low intake.

An alternative approach to the question of whether calcium depletion occurs in pregnancy or lactation is provided by examination of the bones. Since 99% of the body's calcium is in bone, calcium depletion should be accompanied by a reduction in skeletal mass. The total body calcium, estimated by photon absorptiometry of the forearms, appeared to be unchanged in one study of thirteen women in Denmark (Christiansen, Rødbro and Heiniold, 1976). Atkinson and West (1970) used similar techniques on the lower end of the femur during lactation. They suggested that lactation was accompanied by a loss of calcium from the skeleton but the loss was small and within the error of the method used. On the other hand, many studies of large populations have shown that high parity and prolonged lactation have no effect on skeletal dimensions (Walker, 1972; Garn, 1970). This is as true of populations such as South African Negroes with a low calcium intake, as of those on a Western diet.

Official advice about calcium intake in pregnancy and lactation varies. One FAO/WHO report in 1963 commented on the 'ravages of calcium depletion' resulting from pregnancy and lactation in under-privileged communities. Another report (WHO Expert Committee, 1965) was more cautious and emphasized that 'the areas of ignorance relating to nutrition in pregnancy and lactation are extremely large, and it is impossible as a rule to state didactically that this or that particular form of dietary change will produce a specific benefit'. The earlier discussion implies that calcium supplements in pregnancy or lactation serve no useful purpose since, in normal women, the intestinal absorption of calcium adapts to compensate for a dietary intake of as little as 260 mg per day. As in non-pregnant adults such a low figure is most unlikely to occur in diets which are otherwise satisfactory.

Calcium deficiency and rickets

It has sometimes been suggested that calcium deficiency is important in the pathogenesis of rickets...
in children with severe dietary deficiencies but plentiful exposure to sunlight (Taylor, 1976). Rickets undoubtedly occurs in such children in Brazil and the Punjab but there is no evidence from any other study, human or animal, that calcium deficiency causes rickets. One possible explanation of the disorder in such cases is that these patients had a diet so limited that they had a deficiency, not only of vitamin D, but also of its precursor 7-dehydrocholesterol. In this way, sunshine would be ineffective in promoting vitamin D synthesis (Paterson, 1975b).

Conclusions

No calcium deficiency syndrome has ever been described from normal people and this is true even in growing children and lactating mothers. Adaptation in the intestine means that a normal person can adjust his calcium absorption to compensate for a calcium intake much lower than is likely to be found in any diet which is otherwise adequate. Despite these considerations which have been recognized previously (Walker, 1972; Garn, 1970), high figures for a recommended intake of calcium are given by many official bodies (Table 3).

Such official recommendations may have disadvantages. On a world scale it seems possible that efforts to ‘correct’ an ‘inadequate’ calcium intake may divert attention from the prevention of important disorders such as protein-energy malnutrition, xerophthalmia, rickets and iron deficiency anaemia. In Britain many dieticians and some obstetricians advise an increased milk intake in pregnancy. While such advice probably does not harm it almost certainly does no good. Furthermore two public health measures are of questionable value. School-children aged 5–7 years are still given one third of a pint (190 ml) of milk daily. This may be justified by the protein or energy content of the milk (although school meals should meet these needs) but cannot be justified by a necessity to provide calcium. Under the terms of the Bread and Flour Regulations (1963) calcium carbonate is added to all flour, other than oatmeal, at a rate of 1 g per 319 g flour. This addition, which is not used in other countries, has the effect of increasing the mean dietary intake of calcium in Britain from 781 to 1024 mg/day (FAO/WHO Expert Group, 1962). Since 781 mg/day is more than twice the habitual intake in countries with no known calcium deficiency disease, it seems unlikely that the fortification of flour serves any useful purpose. At a time when, as a nation, we are looking critically at priorities the author suggests that these two public health measures need further examination.

Acknowledgment

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References


