Women’s dietary calcium requirements are not increased by pregnancy or lactation\(^1,2\)

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Women are often advised to consume additional calcium during pregnancy and lactation. The assumption in the 10th edition of the *Recommended Dietary Allowances* is that calcium intake should be increased from 20 to 30 mmol (800 to 1200 mg)/d during both pregnancy and lactation \(^1\). The rationale is that in pregnancy this amount of calcium is needed for the mineralization of fetal bone, into which \(\approx 750\) mmol Ca is deposited at a rate of 5-6.25 mmol (200-250 mg)/d in the last trimester. However, early work with \(^{48}\)Ca in adolescent girls showed that at least some of the fetal demand for calcium was likely to be met from increased maternal absorption that started early in pregnancy \(^2\). During lactation, the recommended intake of calcium is increased by the same amount as in pregnancy to replace calcium secreted into breast milk, the upper limit of which is taken to be 7.5 mmol (300 mg)/d. In 1991 the equivalent recommendation in the United Kingdom was that additional dietary calcium was not required during pregnancy because mobilization of maternal bone supplied adequate calcium during the first 3 mo \(^3\). During lactation it was thought that although the spontaneous increase in maternal food intake would supply adequate calcium to replace that secreted into breast milk, there was insufficient evidence that maternal calcium metabolism is adapted during this period. Furthermore, data were available showing that bone density is diminished during the first 3 mo of lactation, so that intake should increase by 14.3 mmol (550 mg)/d.

In recent years more has been learned about the remarkable changes in calcium homeostasis that occur during pregnancy and lactation. Calcium absorption can now be measured more easily with stable calcium isotopes. Another development has been the availability of accurate methods of measuring bone mineral density and bone mineral content, including dual-energy X-ray absorptiometry (DXA) and quantitative computerized tomography, respectively; urinary and lumbar spine mineral content were measured by DXA and quantitative computerized tomography, respectively; urinary and breast milk calcium were assessed; and calciotropic hormones and biochemical markers of turnover were analyzed to explore possible mechanisms involved in the calcium changes. The investigators showed that the calcium required for fetal bone mineralization can be obtained by an increased efficiency of maternal calcium absorption in pregnancy, with no detectable mobilization of maternal bone for this purpose. Dietary calcium intake also increased. Even though urinary calcium was \(\approx 50\%\) higher in the third trimester, the \(\approx 15\) mmol (600 mg)/d absorbed at this time should still be adequate to supply the calcium needs of the fetus.

At its earliest investigation in lactation (2 mo postpartum), calcium absorption had returned to prepregnancy values and urinary calcium losses were less than half those at baseline. The source of the breast-milk calcium was predominantly maternal spinal trabecular bone, with the reduction in urinary calcium contributing to calcium retention. Between 1 and 2 wk postpartum and 5 mo after the resumption of menstruation, bone mineral density was significantly lower in the total body and arms. The density of trabecular bone of the spine fell between 2 wk and 2 mo of lactation but returned to prepregnancy values by 5 mo after menses resumed, although total-body bone mineral density had not. Although many dramatic changes in calciotropic hormones were observed during both pregnancy and lactation, none could definitively explain the shifts in calcium flux.

In the study by Ritchie et al \(^4\), a combination of several factors made the measures taken during late lactation somewhat difficult to interpret. Of the 14 subjects, 5 took oral contraceptives postpartum and 4 were only partially breast-feeding by 2 mo. These problems are to be expected in subjects who are recruited before conception. In the study by Laskey et al \(^5\) in this issue, bone changes during the first 3 mo of lactation were studied by DXA in 47 breast-feeding British women \(^5\). These authors also found a substantial decrease in whole-body bone mineral content and located this loss in the spine and femoral neck. This loss did

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not occur in a comparison group who had fed formula since parturition, indicating that the postpartum skeletal changes were not due to pregnancy per se. The number of breast-feeding women was intended to be sufficient to enable the exploration of factors influencing the magnitude of change in bone mineral. The greatest loss was observed in taller women and those with a higher breast-milk volume. The percentage of mineral lost at the femoral neck was greater in women who weighed less and had a smaller bone area. No other factors measured, including breast-milk calcium concentration and vitamin D–receptor genotype, were predictors of bone loss.

Both studies showed that calcium mobilization to the fetus and to breast milk was the result of changes in maternal metabolism, which apparently are not influenced by the amount of dietary calcium consumed. No outcomes in either study were related to calcium intake. In the British study the estimated calcium intake of the lactating women varied considerably, ranging from 11.2 to 57.2 mmol (440 to 2300 mg)/d. More work is needed, however, to confirm that postlactational bone mineral replacement is independent of calcium intake: the number of women who were not using oral contraceptives and who were predominantly breast-feeding throughout lactation in the US study was probably too small and the British study ended too soon, at 3 mo postpartum. It is also worth remembering that total bone mineral density had not recovered by 5 mo after resumption of menses in the American women. This finding should be investigated further. Also, the average calcium intake of both the British and American women was > 1300 mg/d, lending uncertainty to the generalizability of these results to women who consume less calcium. However, a daily supplement of 17.9 mmol (714 mg)/d for 1 y did not affect bone mineral content or breast-milk calcium of lactating Gambian women with a very-low dietary calcium intake (7.1 mmol/d, or 283 mg/d) (6). Epidemiologic data also support the complete replacement of lactation-induced bone loss because neither the number of children breast-fed nor the duration of breast-feeding are risk factors for loss of bone mineral or fractures in later life (7, 8).

These studies also support the recently revised recommended US-Canadian dietary guidelines that no increase in calcium intake is required for either pregnant or lactating women (9). A population subgroup about which there is remaining concern, however, is lactating adolescents. In one study, adolescent mothers were encouraged to increase their dietary calcium to 40.0 mmol (1600 mg)/d and at 16 wk postpartum this had prevented loss of bone mineral in the forearm compared with a group consuming 22.5 mmol (900 mg)/d (10). Laskey et al’s data (5) also imply that women breast-feeding more than one infant could lose more bone calcium and thus may be at greater risk of calcium deficit, especially if they are small. This implication requires confirmation. Although both of the studies in this issue (4, 5) attempted to identify some of the factors involved in the changes in maternal calcium metabolism, it is still unclear what causes the impressive physiologic alterations observed. More work in this area may elucidate additional factors involved in the regulation of bone metabolism. Finally, although most women may not need calcium supplements during pregnancy and lactation, this does not preclude advice about their need to consume dairy products. Dairy products are obviously excellent sources of the many nutrients that are needed in larger amounts by pregnant and lactating women.

REFERENCES