



Enhanced zinc utilization during lactation may reduce maternal and infant zinc depletion^{1,2}

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About one-half of the globe's population is predicted to be at risk of inadequate zinc intakes (1). This estimate is based on a comparison between the requirement for absorbable zinc and the amount actually available from the food supply. The quantity of zinc available for absorption can be predicted from food balance sheets for the nation in question and the estimated bioavailability of zinc as determined by the presence of dietary factors that enhance (eg, protein) or inhibit (eg, phytate and calcium) zinc absorption. Homeostatic adjustments in zinc absorption or endogenous excretion due to increased needs are not considered when estimating the risk of zinc depletion. If these homeostatic adjustments improve the utilization of dietary zinc in times of increased need, the risk of insufficient zinc intakes worldwide would be lower.

Zinc requirements vary during the life cycle, and peak needs coincide with infantile and adolescent growth spurts, pregnancy, and lactation. On the basis of studies done primarily in experimental animals (2), it is assumed that homeostatic adjustments up-regulate zinc absorption and possibly reduce endogenous fecal excretion during periods of increased need. However, few studies have been done to determine whether similar homeostatic adjustments occur in humans. Because growing, pregnant, and lactating rats have larger rates of zinc accrual than do humans, changes in humans may be met by small changes in intake.

In individuals consuming diets lacking in highly available, zinc-rich, animal foods, the risk of zinc depletion increases with need if no homeostatic adjustments occur. For example, pregnant and lactating women need to absorb ≈ 1.5 – 2.0 mg more Zn/d than do nonpregnant, nonlactating women (3). If no homeostatic adjustments in zinc absorption or endogenous excretion occur to meet this need, pregnant and lactating women normally absorbing 20% of their zinc intake would need to double their intake to meet the peak demand. This is virtually impossible in populations with limited food supplies. If the efficiency of dietary zinc utilization, ie, the net absorption as a percentage of intake, does not improve, the risk of zinc depletion will be very high.

In this issue of the Journal, Sian et al (4) address the efficiency of dietary zinc utilization in lactating women consuming marginal-zinc diets. Eighteen women living in rural northeast China who were exclusively breast-feeding their infants were studied during their second month of lactation. The mean zinc intake of these women was low, 7.6 mg/d, but the amount potentially available for absorption was good because the phytate content of the diet was also low (923 mg/d); the molar ratio of phytate to

zinc averaged 11.7. Furthermore, the food supply was not extremely limited. The intake of staple foods and eggs by these lactating women was greater, and consequently, zinc intake was 50% greater, than in the nonpregnant, nonlactating women. Although the lactating women consumed more zinc than did the nonpregnant, nonlactating women, they utilized it more efficiently than did the nonpregnant, nonlactating women studied previously. Fractional zinc absorption averaged 0.53 in lactating women compared with 0.31 in the nonpregnant, nonlactating women. This 70% higher fractional absorption in addition to a 50% increase in zinc intake increased total zinc absorption from 1.6 to 4.0 mg/d. Endogenous fecal zinc losses also declined relative to the amount of zinc absorbed. These homeostatic adjustments permitted the women to achieve zinc balance while secreting 2.0 mg Zn/d in breast milk. The net utilization of zinc in the lactating women, estimated from the net zinc absorption divided by dietary zinc, increased from 6% to 30%. These data provide convincing evidence that lactating women who consume marginal-zinc diets can achieve zinc balance if the zinc they consume can be utilized efficiently.

Others have also reported higher fractional zinc absorptions in lactating women (5–8). The absolute values vary more than 2-fold, from a low of ≈ 0.3 in the United States and in Rio de Janeiro to a high of 0.7 in the Amazon Valley. This difference may reflect differences in the methods used, in the amount and availability of dietary zinc, and in the quantity of zinc secreted in breast milk. Only one of these studies was longitudinal, with reported measurements of fractional zinc absorption made before conception and during lactation (5). Of the 9 women who were not taking supplemental iron, fractional zinc absorption doubled from 0.15 to 0.31. Taken together, the results of these studies show that intestinal mucosal cells can sense the increased zinc demands of lactation and alter zinc uptake and retention. Also, the capacity to change zinc absorption appears to be robust, especially in women with marginal intakes.

Little is known about how zinc absorption and endogenous excretion are regulated in general, and even less is known about


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how regulation is altered to meet the needs during lactation. It was thought previously that fractional zinc absorption is dictated primarily by variation in zinc intakes, whereas endogenous losses reflect intake and tissue status (9). The data from the studies in Chinese lactating and nonlactating women (4, 10) suggest that the physiologic changes associated with lactation alter both intestinal zinc absorption and endogenous excretion. Hormonal signals probably play a role; however, it is not known which hormones are involved and what their target tissues are. The response to the hormonal signal is also unknown. One possible scenario may involve up-regulation of metal transcription factor 1, which induces the expression of genes for zinc transporter proteins that facilitate zinc transport across the basolateral membrane of the mucosal cell (11).

Lactation performance and infant growth were not studied in the Chinese women (4). Another study of 1956 pregnant and lactating Chinese women with similar, low zinc intakes, however, failed to find an association between maternal zinc intakes and infant gains in height or weight from birth (12). This finding suggests that lactation performance was maintained sufficiently to support infant growth when maternal zinc intakes were low. This situation may not be optimal, however. Infant growth and maternal zinc balance may be relatively insensitive indicators of zinc status. Also, it is not known how well milk zinc output is sustained for the extended periods of lactation common in this population. An overall decline in milk zinc concentrations as lactation progresses is physiologic and predictable (13). Little is known about the effect of low maternal zinc intakes on milk zinc concentrations as a result of long-term breast-feeding. Data from animals fed marginal zinc intakes suggest that zinc secretion is correspondingly decreased in milk (14). A comparison of milk zinc concentrations between women in developing countries and well-nourished women from the United States showed that zinc concentrations tend to be lower in the women from developing countries than in women from the United States at comparable times postpartum (13). These observations support the hypothesis that there is a dietary zinc threshold below which maternal homeostatic adjustments do not maintain milk zinc concentrations. Longitudinal trials of zinc supplementation in developing countries are needed to determine whether changes in maternal zinc intake alter total breast-milk zinc output.

In summary, the outstanding, careful study of zinc homeostasis in Chinese lactating women with marginal intakes (4) showed that the risk of maternal or infant zinc depletion, or both, decreased

with improved absorption and reduced endogenous excretion. Whether this increased efficiency in zinc utilization can be sustained throughout long-term lactation to support optimal infant growth and development is unknown. However, it likely reduces the prevalence of severe deficiency over the short term. 

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