Continuing Education Module

Maternal Calcium Intake and Metabolism During Pregnancy and Lactation

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ABSTRACT

Calcium is an essential nutrient during pregnancy and lactation. Calcium contributes to bone development in the fetus and neonate and is considered a critical nutrient. Physiological changes in calcium metabolism occur during pregnancy and lactation. Some women may lose some of their bone density during pregnancy and/or lactation, and then regain it after the cessation of lactation. Implications for childbirth educators include content regarding the topic of calcium in their classes.

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INTRODUCTION

For all women, pregnancy and lactation are times of high calcium requirement. The average calcium demand of a developing fetus is 30 g by the end of gestation (Kovacs, 2001). Eighty percent of this calcium amount is acquired during the third trimester while the fetal skeleton is rapidly developing (Kovacs, 2001). The average calcium transfer to the fetus during pregnancy is 50 mg/day during the second trimester and 250 mg/day during the third trimester (Oliveri, Parisi, Zeni, & Mautalen, 2004). If the mother does not have adequate dietary calcium preconceptionally and during the pregnancy, significant maternal bone density could be lost, possibly putting her at risk for osteoporosis later in life. Additionally, low calcium intakes could lead to retardation of the fetal skeleton development and low

calcium concentrations in breast milk (Prentice, 2000).

Due to the growing interest in this topic, many studies have been conducted over the past 10 years regarding bone and mineral metabolism of calcium during pregnancy and lactation. In general, researchers have found that during pregnancy and lactation the mother's body physiologically adapts to the increased calcium needs of the growing fetus and newborn without the need for increased dietary calcium (Prentice, 2000). The recommended calcium intake in the United States for pregnant and lactating women is the same as for nonpregnant women: 1 g/day for women over the age of 19 years and 1.3 g/day for women 18 years old or younger (Simpson & Creehan, 2001; U.S. Department of Agriculture, 2004).

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**NORMAL BONE METABOLISM IN
NONPREGNANT WOMEN**

Comprehending the changes the body undergoes during pregnancy and lactation requires an understanding of normal bone metabolism. Bone formation and mineral metabolism involve the interaction between the parathyroid hormone (PTH), calcitonin, and vitamin D (see the Table). PTH maintains blood levels of ionized calcium, increases the rate of calcium phosphate release from bones, increases the conservation of calcium and the elimination of phosphate by the kidneys, and increases intestinal absorption of calcium by working through vitamin D. Calcitonin prevents the release of calcium from the bones and increases the kidney elimination of calcium, which lowers the overall calcium level in the blood. Vitamin D increases the absorption of calcium from the gut and increases the effects of PTH on the skeleton (Porth, 2002).

**PHYSIOLOGIC CHANGES IN CALCIUM
METABOLISM DURING PREGNANCY**

One of the earliest changes in calcium metabolism during pregnancy is a decrease in total serum calcium. This change is not physiologically significant though, and can be attributed to the decrease in serum albumin and the normal hemodilution that occurs during pregnancy. Levels of ionized calcium stay in the normal range. PTH levels fall into a low-normal range during the first trimester, but rise throughout the pregnancy to reach normal levels by the end of gestation. Calcitonin levels are increased throughout pregnancy, preventing extreme calcium loss from the maternal skeleton. However, some calcium from the mother's skeleton is used for fetal development and is not completely spared. The maternal skeleton returns to normal at the conclusion of breastfeeding if calcium intake

is adequate (Hreshchyshyn, Hopkins, Zylstra, & Anbar, 1988; Lopez, Gonzalez, Reyes, Campino, & Diaz, 1996). Other hormones including prolactin, estrogen, placental lactogen, and placental growth hormone create changes in the levels of 1,25-dihydroxyvitamin D, calcium absorption, and bone turnover rates (Kovacs, 2001; Oliveri et al., 2004).

The most adaptive change protecting the maternal skeleton from bone density loss may be the shift in levels of 1,25-dihydroxyvitamin D. This hormone rapidly increases during the first trimester and reaches its highest point during the third trimester (Oliveri et al., 2004). As the 1,25-dihydroxyvitamin D levels double, a 25% increase occurs in the amount of intestinal calcium absorbed (Kovacs, 2001). This increase can occur as early as 12 weeks gestation and, researchers hypothesize, the maternal skeleton starts absorbing more calcium early and stores it in preparation for when the fetus will need the calcium during skeletal development in the third trimester (Kovacs, 2001). This early storage of calcium in the maternal skeleton has been observed in some animal tests, but cannot be directly tested in humans. Urinary calcium excretion tends to increase across the course of pregnancy, from 12 weeks gestation onward, and can be associated with the increase in intestinal absorption, increased related filtered load of calcium, and the increased glomerular filtration rate that occurs during pregnancy (Kovacs, 2001).

In summary, the extra demand for calcium from the growing fetus, especially during the third trimester, is physiologically compensated through changes in hormone levels. These hormonal changes lead to an increase in intestinal absorption, a decrease in renal calcium loss, and an increase in absorption from the maternal skeleton (intestinal calcium absorption becoming the primary source).

TABLE
Interactions Affecting Bone Formation and Mineral Metabolism

	Bone Metabolism Hormone Function
PTH	<ul style="list-style-type: none">• Maintains blood levels of ionized calcium• Increases rate of calcium phosphate release from bones• Increases the conservation of calcium and the elimination of phosphate by the kidneys• Works through vitamin D to increase intestinal absorption of calcium
Calcitonin	<ul style="list-style-type: none">• Prevents release of calcium from bones• Increases kidney elimination of calcium, lowering serum calcium levels
Vitamin D	<ul style="list-style-type: none">• Increases intestinal absorption of calcium• Increases effects of PTH on skeleton

MATERNAL BONE MASS DURING PREGNANCY

Despite the great interest in this topic, research regarding changes in maternal bone mass during pregnancy is limited because assessment techniques usually require the use of ionizing radiation that would be teratogenic to the fetus. Some studies have performed bone density measurements before pregnancy and postpartum or have used other techniques such as ultrasonography or single- or dual-photon absorptiometry (Prentice, 2000).

In one study, which measured bone density prior to and after pregnancy, bone mineral density decreased by approximately 3% in the lumbar spine and proximal femur (Black, Topping, Durham, Farquharson, & Fraser, 2000). However, other research supports that, while some bone loss does occur, new bone formation replaces it at equivalent or greater levels (Hreshchyshyn et al., 1988; Lopez et al., 1996). Researchers have also investigated alterations in serum markers of bone formation and urine markers of bone absorption. Based on the small amount of available data, it could be assumed that the bone-turnover rate increases throughout pregnancy, starting around 10 weeks gestation (Kovacs, 2001). Although women may lose approximately 3% of their bone density during pregnancy, they will regain normal density within a few months postpartum (Kovacs, 2001). However, this process may be delayed by breastfeeding.

A few examples exist in which the calcium balance between maternal skeleton and intestinal and renal conservation are not equal, possibly putting the mother at greater risk for bone density loss:

1. *Adolescent mothers.* Studies have shown that bone mass density decreases as much as 10% (testing in the forearm) in adolescent mothers during pregnancy and lactation, whereas this decrease did not occur in women over the age of 18 years (Oliveri et al., 2004). This loss can be offset by increasing the adolescent's dietary calcium intake (Oliveri et al., 2004).
2. *Multiple fetuses.* Studies have shown that mothers carrying twins had higher bone density loss than mothers carrying a single fetus (Oliveri et al., 2004).
3. *Heparin use.* Studies have shown that women who were using heparin injections to prevent deep-vein thromboses during pregnancy reported several cases of vertebral osteoporosis

(Oliveri et al., 2004). This condition may be due to the hypothesis that heparin inhibits the synthesis of 1,25-dihydroxyvitamin D. Without the compensation mechanism of increased intestinal calcium absorption with 1,25-dihydroxyvitamin D, a greater risk of bone density loss occurs (Oliveri et al., 2004).

4. *Cultures with low dietary calcium intake.* In a longitudinal study conducted by Zeni and colleagues (2003), researchers hypothesized that, if the mother's calcium intake was too low, the body would not completely compensate and a greater bone loss would occur. Bone turnover markers were measured between the second and third trimesters in 39 healthy, pregnant women. The researchers found that the level of the markers negatively correlated with calcium intake. This suggests that greater bone turnover occurs with lower dietary calcium intake (Zeni et al., 2003). One turnover marker, betaCTX, doubled in women with the recommended daily amount of calcium during the third trimester. For women who had half of the recommended amount of calcium, their turnover marker was eight times as high as a nonpregnant woman. Thus, the body's physiological response to calcium metabolism during pregnancy may not be enough to cover all the calcium needs of the fetus in women with low dietary calcium intake.

Any acute changes that may occur during pregnancy do not seem to have any long-term effects on calcium density or bone strength later in life. The few women who have low bone mineral density postpartum often have low bone density before conception due to other problems or medications (e.g., heparin, anticonvulsants, and corticosteroids). Osteoporosis during pregnancy cannot be blamed on the lack of dietary calcium or the metabolic needs of the developing fetus. Studies of women with osteoporosis were unable to link the disease with bone loss during pregnancy. Researchers found no difference in bone mineral density in elderly women who had children versus nulliparous women (Hillier et al., 2001; Paton et al., 2003). Focal, transient osteoporosis of the hip—a rare form of pregnancy osteoporosis—is not related to calcium content of the bone but, rather, to other conditions such as femoral venous stasis, ischemia, trauma, viral infection, marrow hypertrophy, immobilization, or fetal pressure on the obturator nerve (Kovacs, 2001).

CALCIUM INTAKE AND HYPERTENSIVE DISORDERS

Several studies have examined whether calcium supplements would help reduce the incidence of pregnancy-induced hypertension (PIH) and preeclampsia. This idea first developed because of a 1980 epidemiological study conducted in Guatemala where scientists noticed that the Mayan Indians exhibited high calcium intakes and low incidences of preeclampsia and eclampsia (Atallah, Hofmeyr, & Duley, 2004).

Eclampsia is more common in countries where the dietary calcium intake is low (Patterson, 1984). Additionally, observational studies in the U.S. and Canada have shown that women who have lower calcium intakes exhibit a greater incidence of PIH (Patterson, 1984). The physiological basis behind this hypothesis is that low calcium intake stimulates PTH production, which increases the intracellular calcium levels. This leads to smooth-muscle vessel contraction and hypertension. Calcium supplements would reduce the intracellular calcium and relax the vessels (Atallah et al., 2004).

Atallah and colleagues (2004) conducted a review of research on the effects of calcium supplementation in PIH and preeclampsia. They found that, among 11 studies, 10 demonstrated a reduction in the incidence of hypertension with additional dietary calcium. Calcium supplements seemed to reduce the risk of PIH and preeclampsia in women who were at high risk for developing gestational hypertension. This reduction occurred among teenagers; women over 35 years old; African Americans; women with multiple gestation, preexisting diabetes, hypertension, renal disease, or obesity; and women with a personal or family history of PIH or preeclampsia (Atallah et al., 2004; Simpson & Creehan, 2001). Calcium supplementation also reduced the incidence of PIH and preeclampsia in women with low dietary calcium intake (Atallah et al., 2004).

One study that did not find a difference in hypertension with the use of calcium was a large research study conducted in the U.S. with 4,589 healthy, pregnant women (Levine et al., 1997). In this particular study, women were given either 2 g/day of supplemental calcium or a placebo, starting anywhere from 13 to 21 weeks gestation. The researchers found the systolic and diastolic blood pressures were similar in each group, and the increase in calcium did not reduce any of the conse-

quences of PIH and preeclampsia (e.g., preterm deliveries, small-for-gestational-age infants, or infant fatalities). Additionally, the frequency of eclampsia and HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count) was almost equal in both groups (Levine et al., 1997).

Because Levine and colleagues' (1997) large study did not find positive results, many health-care providers are still hesitant to recommend calcium supplementation to all expectant mothers. However, it should be noted that the U.S. study included women who were at low risk for gestational hypertension. Also, all of the women, even the control group, received some calcium in their regular prenatal vitamins (Atallah et al., 2004). This may support that calcium supplements only show results when the daily intake is lower than the amount that would provide maximum benefit (Zeisel, 2000). Women who are at high risk for gestational hypertension and those with low dietary calcium intakes may benefit from a calcium supplement during pregnancy (Atallah et al., 2004). However, more research is needed to test these hypotheses and to determine the ideal dosage of the supplement.

Belizan and colleagues (1997) conducted another related study to determine whether calcium supplementation during pregnancy could prevent childhood hypertension. The researchers believed that later blood pressure could be programmed during fetal development. The study included 591 children, ages 5 to 9 years old, whose mothers either took 2 g/day calcium supplement or a placebo. The researchers found that the systolic blood pressure was lower in the calcium group by an average of 1.4 mmHg. The most significant blood pressure changes were found in overweight children who had body mass indexes above 17.5. Their systolic blood pressures dropped an average of 5.8 mmHg.

CALCIUM DURING LACTATION

Calcium concentration of breast milk is independent of maternal dietary calcium intake (Oliveri et al., 2004). Concentrations do not change with calcium supplementation, even in women with low dietary calcium intake (Prentice, 2000). However, although no dietary calcium changes need to be made during lactation, several observational studies have shown that the amount of calcium the mother consumes during pregnancy may affect breast-milk concentration (Prentice, 2000).

The daily transfer of calcium from the mother to her infant during lactation ranges from 250 mg to

300 mg and has been observed to be as high as 1,000 mg/day. With a summation of this transfer, the mother could lose 25 g to 30 g of calcium over a three-month period of lactation, representing approximately 3% of her body's calcium stores. This has been found to increase steadily if the mother continues to breastfeed, with a 6% total body calcium loss after six months of lactation (Kovacs, 2001; Oliveri et al., 2004). However, previous research has supported that bone remineralization occurs at equivalent or higher rates after lactation is complete (Hreshchynshyn et al., 1988; Lopez et al., 1996).

PHYSIOLOGICAL CHANGES IN CALCIUM METABOLISM DURING LACTATION

During lactation, PTH, serum calcium, ionized calcium, and urinary calcium excretion levels return to normal pre-pregnancy ranges (Oliveri et al., 2004). Calcitonin levels fall to normal within six weeks postpartum (Kovacs, 2001). Hormone levels of 1,25-dihydroxyvitamin D, which are important to maintaining calcium levels during pregnancy, return to normal within a few days of birth and remain in the normal range throughout lactation (Kovacs, 2001). As a result, no increase in intestinal absorption of calcium occurs to compensate for the loss to the neonate. Without this mechanism, the primary source of extra calcium becomes the maternal skeleton.

PTH-related protein (PTHrP), a hormone produced by the mammary glands and possibly influenced by higher prolactin levels, is increased significantly during lactation (Oliveri et al., 2004). The role of PTHrP is not completely understood; however, some believe the hormone somewhat influences the absorption of calcium from the maternal skeleton and suppresses PTH levels (Kovacs, 2001). Higher PTHrP levels correlate to loss of bone density mass in lactating women (Kovacs, 2001). Prolactin and estrogen influence calcium and bone metabolism, and lower levels of estrogen during lactation may lead to the increase in bone turnover rates (Oliveri et al., 2004). Studies have shown that women whose menses returned sooner and, therefore, experience the return of estrogen sooner, had a smaller loss of bone density during lactation than those with longer amenorrhea (Kovacs, 2001).

MATERNAL BONE MASS DURING LACTATION

Due to the influence of PTHrP and estrogen and to the lack of 1,25-dihydroxyvitamin D to compensate

for calcium transfer to the neonate, the maternal skeleton experiences a loss of bone density. The loss of bone mass seems to be a normal consequence of breastfeeding, and no additional dietary calcium supplements can prevent its occurrence (Kovacs, 2001).

Sowers and colleagues (1993) conducted a study that monitored the bone density of the femur and lumbar spine of 98 healthy women. Bone density was checked using x-ray densitometry at two, four, and six months postpartum, and 12 months after the women stopped breastfeeding. Women who breastfed for six months or more had an average bone mineral density loss of 5.1% from their lumbar spine and 4.8% from the femur. Women who did not breastfeed, or breastfed only for a few weeks after birth, lost no bone mineral density at either bone site. However, 12 months after weaning, the women's bone density returned to baseline levels.

Other studies have shown that, if a mother breastfeeds for up to 12 months, she is at risk for a 10% bone mineral density loss (Oliveri et al., 2004). This loss seems to be a natural part of lactation. All bone losses are regained 3–6 months after weaning, regardless of how much was lost, at a rate of 0.5–2% per month (Kovacs, 2001). How bone loss is restored is not completely understood, but it may be influenced by the return of estrogen and a decrease in PTHrP (Oliveri et al., 2004).

CALCIUM RECOMMENDATIONS

Maternal calcium loss during pregnancy and lactation is a normal part of fetal and neonatal development. During pregnancy, the woman's body physiologically compensates by increasing intestinal absorption, decreasing renal calcium loss, and reabsorbing some calcium from the maternal skeleton. During lactation, the maternal skeleton loses a small percentage of bone density mass to compensate for calcium loss in the breast milk, but it is wholly regained after weaning. Some studies have shown that calcium supplements will help lower the risk of PIH and preeclampsia. However, other health professionals have warned that the high amount of calcium given in these hypertension studies, in conjunction with the high intestinal absorption rate, could lead to hypercalcemia, urinary tract infections, and nephrolithiasis (Prentice, 2000). Additionally, they warn that a drastic increase in calcium intake may reduce the absorption of other minerals such as iron, magnesium, and zinc, which are also important to maternal and fetal health (Prentice,

1994). Therefore, pregnant and lactating women should follow the U.S. Department of Agriculture Food and Nutrition Information Center's recommendation of 1.3 g/day for women under 18 years old and 1 g/day for those aged 19 years or older (U.S. Department of Agriculture, 2004).

IMPLICATIONS FOR CHILDBIRTH EDUCATORS

Dietary teaching should focus on educating women about national dietary recommendations, including calcium intake, prior to pregnancy. Childbirth educators and other health-care providers can reinforce this information in their contacts with pregnant women. Given the importance of calcium to the developing fetus and the need for adequate stores, at least a brief discussion of the importance of calcium should be addressed early in pregnancy or early in the sequence of childbirth education classes. Ideally, discussions about the importance of calcium should be integrated with other nutrition information. It also seems logical to include content related to calcium reabsorption and bone rebuilding when breastfeeding is discussed to counter the adage, "a tooth for each child." The Web site Babycenter.com includes a particularly nice discussion of calcium and pregnancy and can be recommended to families who enroll in childbirth classes.

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