Impact of Breastfeeding on the Mobilization of Lead from Bone

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To evaluate the hypothesis that lactation stimulates lead release from bone to blood, the authors analyzed breastfeeding patterns and bone lead concentrations as determinants of blood lead levels among 425 lactating women in Mexico City for 7 months after delivery (1994–1995). The authors measured in vivo patella and tibia lead concentrations at 1 month postpartum using K x-ray fluorescence. Maternal blood samples and questionnaire information were collected at delivery and at 1, 4, and 7 months postpartum. Blood lead was analyzed using graphite furnace atomic absorption spectroscopy. Mean blood lead level at delivery was 8.4 µg/dl (range: 1.8–23.4). Mean cortical and trabecular lead levels were 10.6 µg/g (range: nondetectable to 76.5) and 15.3 µg/g (range: nondetectable to 85.9), respectively, reflecting a population with elevated and diverse past and current lead exposure. The association of bone lead and breastfeeding with blood lead was estimated using generalized estimating equations. Breastfeeding practices and maternal bone lead were important predictors of blood lead level. After adjustment for bone lead and environmental exposure, women who exclusively breastfed their infants had blood lead levels that were increased by 1.4 µg/dl and women who practiced mixed feeding had levels increased by 1.0 µg/dl, in relation to those who had stopped lactation. These results support the hypothesis that lactation is directly related to the amount of lead released from bone. Am J Epidemiol 2002;155:420–8.

The adverse effects of lead on human health have been widely documented. However, the distribution and mobilization of lead from different parts of the body has not been fully studied. This is an important topic, particularly with respect to physiologic states such as lactation, which is expected to result in mobilization of lead mineral tissue (1, 2). Lactation has been recognized as a powerful stimulus for bone resorption. It is estimated that during breastfeeding, 4 percent of bone mass is mobilized; thus, lead accumulated in bone from past exposures may be released into the bloodstream and excreted in breast milk, constituting an important source of lead exposure for the breastfed infant (3). Most of the research on lead mobilization has analyzed longitudinal changes in blood lead levels or in blood lead isotope ratios during pregnancy and lactation to estimate lead release from the maternal skeleton to the breastfed infant. However, to our knowledge, none of the published studies have incorporated bone lead measurements to account for the different lead burdens of study subjects (1, 2, 4).

Until recently, epidemiologic studies on the toxicologic significance of human bone lead stores required the use of invasive methods (2). However, in vivo K x-ray fluorescence (KXRF) now permits noninvasive determination of bone lead burden in the large groups of subjects necessary for this kind of research. In a longitudinal study, we examined breastfeeding practices and used KXRF-determined bone lead levels as predictors of blood lead levels among women in Mexico City. The women had no occupational history of lead exposure. However, this population lives in an area where leaded gasoline was used until 1997. In addition, there is a long tradition in Mexico of cooking and storing food in lead-glazed ceramic ware. This has resulted in an adult population with generally high bone lead burdens both from past years of environmental exposure to leaded gasoline and from continued dietary exposure (5, 6).

We evaluated the hypotheses that breastfeeding stimulates lead release from bone to blood and that greater intensity of milk production will be associated with a larger release of lead from bone, reflected as a higher concentration of lead in the bloodstream.

MATERIALS AND METHODS

We studied a cohort of 425 women who participated in a randomized trial designed to assess the effect of calcium supplementation (1,200 mg/day) on blood lead levels during...
lactation. Between January 1994 and June 1995, 2,910 potential study participants were interviewed at three maternity hospitals in Mexico City. Of these, 1,382 were found to be eligible for the trial. Exclusion criteria included: logistics that would interfere with data collection, such as living outside of Mexico City; no intention to breastfeed; factors that might modify calcium metabolism, such as a physician’s diagnosis of multiple fetuses, preeclampsia, or pregnancy-related hypertension disorders; psychiatric, kidney, or cardiac disease; gestational diabetes; history of repeated urinary infections; familial or personal history of kidney stone formation; seizure disorder requiring daily medication; and ingestion of corticosteroids. Women who gave birth to premature neonates (<37 weeks) were also excluded from the study.

From the women identified as eligible during the first interview, 629 (45 percent) agreed to participate in the study. These women completed a baseline evaluation, including a questionnaire that assessed known risk factors for environmental lead exposure. We also measured anthropometric variables and collected samples of blood and breast milk. Bone lead concentration was estimated with the KXRF instrument. Twelve of these women later decided not to breastfeed their children and thus were excluded from the study. Participants who completed the baseline evaluation (1 month postpartum) and started calcium supplementation were followed for 6 months. At 4 months postpartum, field personnel visited study participants at home ($n = 550$) and obtained blood and breast-milk samples and updated the questionnaire information regarding breastfeeding practices and use of lead-glazed ceramics, among other variables. At 7 months postpartum, 504 participants visited the research center, where information was updated and blood and breast-milk samples were collected. Figure 1 shows the sample sizes during the study period. Results regarding lead levels in breast milk are not yet available.

The research protocol was approved by the Human Subjects Committee of the National Institute of Public Health of Mexico and by the participating hospitals. All of the participating mothers received a detailed explanation of the study and of the procedures used, after which they signed an informed consent form. Women also received counseling on reduction of lead exposure.

**Lead measurements**

Blood lead was analyzed with a graphite furnace atomic absorption spectrophotometry instrument (PerkinElmer 3000; PerkinElmer Instruments, Wellesley, Massachusetts) at the metals laboratory of the American British Cowdray

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**FIGURE 1.** Sample size profile for participants in a study of bone lead, blood lead, and lactation, Mexico City, Mexico, 1994–1995.

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Hospital in Mexico City. The laboratory standardization program of the Wisconsin State Laboratory of Hygiene (Madison, Wisconsin) provided external quality control specimens varying from 2 µg/dl to 88 µg/dl. Our laboratory maintained acceptable precision and accuracy during the study period (correlation = 0.98; mean difference = 0.71 µg/dl, standard deviation 0.68).

Bone lead measurements lasting 30 minutes were taken of each subject’s midtibia shaft (representing cortical bone) and patella (trabecular bone) after the region had been washed with a 50 percent solution of isopropyl alcohol. We used a spot-source $^{109}$Cd KXRF instrument constructed at Harvard University and installed in a research facility at the American British Cowdray Hospital. The physical principles, technical specifications, validation, and use of this and other KXRF instruments have been described in detail elsewhere (7, 8). In brief, the instrument uses a cadmium $\gamma$-ray source to provoke the emission of fluorescent photons from target tissue which are then detected, counted, and arrayed on a spectrum (9). Net lead signal is determined after subtraction of Compton background counts by a linear least-squares algorithm. The lead fluorescent signal is then normalized to the elastic or coherently scattered $\gamma$-ray signal, which arises predominantly from the calcium and phosphorus present in bone mineral. The instrument also provides an estimate of the uncertainty associated with each measurement. For purposes of quality control, we excluded participants whose bone lead measurements had uncertainty estimates that were greater than 10 µg and 15 µg of lead per gram of bone mineral for tibia and patella, respectively ($n = 56$).

Previous studies have shown that the use of lead-glazed ceramic ware produced at low temperatures is a major source of lead exposure in this population (10). To estimate exposure from this source, women in the study were asked about the number of days in a week that they used this kind of ceramic ware for cooking. Visual aids were used to help study subjects distinguish between lead-glazed and nonlead-glazed ceramics. All participants received information regarding this risk and were advised not to use this type of ceramic ware. Information on the use of such ceramics was updated at every visit.

One of our hypotheses was that greater intensity of milk production would be associated with a larger release of lead from bone. We characterized this process using breastfeeding practices as a proxy variable. For this, we defined three categories: exclusive breastfeeding—that is, women who fed their children nothing but breast milk; partial breastfeeding—mothers who combined breast milk with liquids and other foods; and a third group of nonlactating women. This classification was updated at 4 and 7 months postpartum. At 1 month postpartum, all women were breastfeeding. Parity and previous lactation were also included in the model to account for prior periods of potentially accelerated bone lead mobilization (10).

**Statistical analysis**

Univariate and bivariate summary statistics and plots of distributions were examined for all variables. Data on blood lead were not normally distributed, so we logarithmically transformed this variable (natural logarithm) to normalize it.

The KXRF instrument used to estimate bone lead levels provides a continuous, unbiased point estimate that oscillates around the true bone lead value. Negative point estimates are sometimes produced when true bone lead values are close to zero. In our study, 13 percent of tibia estimates and 16 percent of patella estimates of lead concentration were negative. To avoid bias due to these negative values, we estimated mean tibia and patella lead levels using intervals regression (11). This technique allowed us to work with the observed data points and censored observations. For the analyses, we assumed as censored observations those measurements below a cutoff point of 5 µg of lead per gram of bone mineral. To assess the robustness of our findings in relation to the influence of the negative bone lead values, we reanalyzed the data after replacing the observed negative values with the new simulated positive values obtained assuming a uniform distribution in the interval (0, 5). For all analyses presented here, we used either values derived from the KXRF instrument or values obtained with the simulated data.

The outcome variable was change in blood lead level, which was measured at three different times for each study subject. The fact that observations were not independent had to be considered in the analyses to avoid bias in the estimation of the standard errors. Generalized estimating equations (12) take this correlation into account, so this method was used to assess the relation of bone lead levels and lactation with blood lead levels, controlling for other covariates during the study period. This technique also allowed us to adjust for independent variables that changed with time and were updated on every occasion, such as hematocrit level, feeding practices, and frequency of cooking with lead-glazed ceramic ware, as well as for independent variables that did not change with time, such as blood lead level at delivery, history of lactation, and parity. We performed two separate analyses: those including all women with at least one measurement during the study period ($n = 617$) and those confined to the subsample of women who completed follow-up and had no missing data ($n = 425$). Since the results were very similar, only results of the latter analyses are presented.

To explore potential nonlinear associations between bone lead levels and blood lead levels, we examined the relations between variables using generalized additive models (13). All statistical analyses were performed using Stata software, release 6.0 (Stata Corporation, College Station, Texas). The required graphics were generated in Microsoft Excel (Microsoft Corporation, Seattle, Washington).

**RESULTS**

The mean age of the 425 participant women was 24.8 years (standard deviation 5.3), and 2 percent of them were aged 16 years or younger at the time of delivery. Approximately 41 percent ($n = 176$) were primiparous. Of the 249 women with previous pregnancies, 99 percent ($n = 246$) had breastfed their previous infants. Smoking during pregnancy was reported by 4 percent of participants ($n =
We found no significant differences between women who completed baseline evaluations and those who declined to participate in the study with respect to age, blood lead level at delivery, education, parity, percentage of primiparity, or history of lactation (see table 1).

At 1 month postpartum, all women were breastfeeding their infants, although only 102 (24 percent) reported exclusive breastfeeding. As expected, these percentages changed during the study period: at 4 months, 6 percent (n = 24) of the women reported exclusive breastfeeding, 71 percent (n = 301) reported partial lactation, and 23 percent (n = 100) had interrupted lactation. At 7 months postpartum, no women were practicing exclusive breastfeeding; 56 percent reported partial breastfeeding (n = 240) and 44 percent (n = 185) had stopped breastfeeding.

Table 2 presents summary statistics for blood lead levels stratified by different categories of the main covariates involved in the analyses. Women who breastfed exclusively had higher blood lead levels than those who practiced partial breastfeeding: the latter women, in turn, had higher blood lead levels than those who stopped lactation. However, in these cross-sectional analyses, differences were statistically significant only at 7 months postpartum (table 2).

Primiparous women had slightly higher mean blood lead levels than those who had a previous pregnancy. In addition, women with a history of prior lactation had lower (p = 0.09) blood lead levels (9.1 µg/dl) than those who had never lactated before (9.7 µg/dl).

The mean bone lead concentration at 1 month postpartum was 10.6 µg lead/g bone mineral for tibial bone (11.6 µg lead/g after correcting for negative values) and 15.3 µg lead/g bone mineral for patellar bone (16.9 µg lead/g after correcting for negative values). We observed a statistically significant correlation (ρ = 0.4, p < 0.01) between patellar and tibial lead levels. To avoid collinearity, we decided not to include both biomarkers simultaneously in the regression models presented.

Mean blood lead levels decreased with time: Levels were 9.4 µg/dl (standard deviation 4.4), 8.9 µg/dl (standard deviation 4.0), and 7.9 µg/dl (standard deviation 3.3) at 1, 4, and 7 months postpartum, respectively (test for trend: p < 0.01). The longitudinal models proposed to explain these changes included breastfeeding practices as the main predictor. The estimates were also adjusted by bone lead levels, primiparity, frequency (number of days) of cooking with lead-glazed ceramic ware during the week before the interview, history of lactation (measured as the number of months for which mothers had breastfed their previous children), calcium supplement intake, and an indicator variable for the study period (table 3).

We found an association between breastfeeding practices and blood lead levels: The more intense the practice of lactation (i.e., exclusive breastfeeding), the higher the blood lead levels. The model that adjusted for patella lead concentration predicted an increase in blood lead levels of 12.7 percent (95 percent confidence interval (CI): 6.2, 19.6) for women who practiced partial lactation and an increase of 18.6 percent (95 percent CI: 7.1, 31.4) for women who practiced exclusive lactation in relation to those who stopped lactation. The model that included tibia lead showed the same trend (table 3).

Blood lead levels varied with time and according to breastfeeding practices (figure 2). Adjusted geometric mean values for the women who exclusively breastfed their infants were 8.7 µg/dl (95 percent CI: 7.9, 9.5) and 8.7 µg/dl (95 percent CI: 7.8, 9.7) at 1 and 4 months postpartum, respectively. For the group that practiced partial breastfeeding, the adjusted geometric means were 8.2 µg/dl (95 percent CI: 7.7, 8.8), 8.3 µg/dl (95 percent CI: 7.7, 8.8), and 7.7 µg/dl (95 percent CI: 7.2, 8.2) at 1, 4, and 7 months postpartum, respectively. For the group of women who stopped lactation, the adjusted means were 7.3 µg/dl (95 percent CI: 6.8, 7.9) and 6.8 µg/dl (95 percent CI: 6.3, 7.3) at 4 and 7

### Table 1. Characteristics of participants and nonparticipants in a study of blood lead levels and lactation (among women with complete baseline information), Mexico City, Mexico, 1994–1995

<table>
<thead>
<tr>
<th>Maternal characteristic</th>
<th>Participants* (n = 425)</th>
<th>Nonparticipants (n = 981)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD†</td>
<td>No. with missing data</td>
</tr>
<tr>
<td>Maternal age (years)</td>
<td>24.8</td>
<td>5.3</td>
<td>0</td>
</tr>
<tr>
<td>Blood lead level at delivery (µg/dl)</td>
<td>8.4</td>
<td>4.0</td>
<td>3</td>
</tr>
<tr>
<td>Time living in Mexico City (years)</td>
<td>21.3</td>
<td>8.7</td>
<td>0</td>
</tr>
<tr>
<td>Education (years)</td>
<td>9.2</td>
<td>3.2</td>
<td>0</td>
</tr>
<tr>
<td>No. of pregnancies§</td>
<td>2.1</td>
<td>1.3</td>
<td>0</td>
</tr>
<tr>
<td>Primiparity (%)</td>
<td>41</td>
<td>0</td>
<td>47</td>
</tr>
<tr>
<td>Previous breastfeeding (%)</td>
<td>58</td>
<td>0</td>
<td>51</td>
</tr>
<tr>
<td>Use of lead-glazed ceramics at the beginning of the study (%)</td>
<td>49</td>
<td>0</td>
<td>44</td>
</tr>
</tbody>
</table>

* Participants were women who had data from all three lead measurements and complete data on all variables included in this analysis: maternal age, time living in Mexico City, education, number of pregnancies, history of lactation, use of lead-glazed ceramics, breastfeeding practices, bone lead concentration, and use of calcium supplements.
† SD, standard deviation.
‡ Kruskal-Wallis equality of population rank test.
§ Including the index pregnancy.
¶ Test of equality of two sample proportions.
months postpartum. Figure 2 shows the geometric means of blood lead levels at delivery for all participants, stratified by breastfeeding practices at 1 month postpartum. At this time, all groups had a similar blood lead level: The unadjusted geometric means were 7.6 µg/dl, 7.6 µg/dl, and 7.2 µg/dl for the groups who practiced exclusive and partial breastfeeding and for the 12 women who decided not to breastfeed their children, respectively. Differences were not significant. For comparison, figure 2 includes the corresponding values for the three groups.

Table 3 displays the parameter estimates for the breastfeeding practices in relation to those of women who stopped lactation (reference category). The parameter estimates represent the multiplicative effect on the predicted geometric mean blood lead levels. It also shows that tibia and patella lead concentrations were positively associated with blood lead levels. Our model estimates that a 10-µg lead/g bone mineral increment in patella and tibia bone lead levels would increase blood lead levels by 6.1 percent (95 percent CI: 4.2, 8.1) and 8.1 percent (95 percent CI: 5.2, 11.1), respectively. When we corrected for the negative bone lead values, these estimations became stronger: 7.4 percent (95 percent CI: 5.1, 9.7) and 9.0 percent (95 percent CI: 5.8, 12.3) for patella lead and tibia lead, respectively.

After adjustment for different covariates in the multivariate model, primiparous women tended to have higher blood lead levels than their multiparous counterparts: Their estimated geometric means were 8.7 µg/dl and 8.2 µg/dl (p = 0.1), respectively. A history of lactation was negatively related to blood lead levels, although this relation was not statistically significant (table 3). Within the full model, we also tested the interaction between bone lead and current breastfeeding practices. The association between patella lead and blood lead was higher for women with partial...
FIGURE 2. Adjusted geometric mean values for blood lead level according to breastfeeding practices (exclusive breastfeeding, partial breastfeeding, or no breastfeeding). Mexico City, Mexico, 1994–1995. Data were adjusted with generalized estimating equations for patella lead level, frequency of cooking with lead-glazed ceramics, history of lactation, and calcium supplement intake. The asterisk (*) indicates the unadjusted geometric mean for women who had stopped breastfeeding at baseline (1 month postpartum); these women (n = 12) were excluded from the subsequent analysis. The dagger (†) indicates the unadjusted geometric mean at delivery for the study participants: 7.6 µg/dl for both breastfeeding groups.

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lactation than for those who stopped lactation, and it was increased among women who breastfed exclusively. Regression coefficients for a 10-µg lead/g bone mineral increment were 0.053 for women who stopped lactation, 0.061 for the group that practiced partial lactation, and 0.067 for women who breastfed exclusively.

We also adjusted for changes in hematocrit level over the study period and for blood lead levels at delivery to ensure that differences in blood lead during lactation were not due to differences at baseline, even though (as we mentioned above) these values were very similar. The relation between breastfeeding practices and blood lead became slightly more apparent and remained highly significant (data not shown).

We reanalyzed the data using the information from all women with at least one follow-up measurement during the study period. Results remained very similar (data not shown).

The analysis using generalized additive models (figure 3) suggested a nonlinear relation between the fitted function for patella lead and log-scaled blood lead levels. Although the range of observations was limited, the smoothed plot seemed fairly linear until 15 µg lead/g bone mineral, after which the slope of the curve appeared to increase to approximately 30 µg lead/g bone mineral. A similar relation was observed for tibia lead (graph not shown). To account for this pattern, we included both a quadratic term and a cubic term in the regression models, but this made virtually no difference in the breastfeeding coefficients (data not shown).

We also explored the shape of this relation, categorizing bone lead levels in quintiles (figure 4). Since the coefficients of the rest of the covariates remained very similar to those of the model that assumed linearity, we decided to present results from the most parsimonious model.

**DISCUSSION**

Our study showed that breastfeeding is an important determinant of blood lead levels during the postpartum period. In comparison with women who stopped breastfeeding, women who continued to practice exclusive lactation showed a mean increase in blood lead concentrations of 19 percent. Similarly, participants who practiced partial lactation showed a 13 percent increase compared with non-breastfeeding women. This association is probably due to the increased bone resorption and mineral mobilization that occurs during lactation (3, 14).

We also documented that bone lead levels are important predictors of blood lead levels. Our model estimated that, after adjusting for type of lactation, parity, calcium supplement intake, and frequency of cooking with lead-glazed ceramics, an increase of 10 µg lead/g bone mineral in patella lead is associated with a 6 percent increase in blood lead levels.

Our results are similar than those reported by Gulson et al. (15, 16), who studied bone lead mobilization in a cohort of Australian women (n = 22) with low blood lead levels but varying bone lead burdens. By measuring lead isotopes, these authors documented that mobilization of bone lead was significantly higher during the postpartum period than during pregnancy. They observed that the percentage of bone lead mobilized showed a fairly linear increase during pregnancy and remained high but basically constant during the postpartum period, despite the duration of lactation. The

![Figure 3](image.png)

**FIGURE 3.** Generalized additive model-adjusted function for log-scaled blood lead levels and patella lead concentrations among lactating women, Mexico City, Mexico, 1994–1995. Data were adjusted for months from delivery, breastfeeding practices, frequency of cooking with lead-glazed ceramics, history of lactation, and primiparity. The dashed lines represent 95% pointwise confidence intervals. ND, nondetectable.
small sample size may have made it impossible to detect differences in bone lead mobilized in relation to duration or type of breastfeeding practices.

However, in contrast to our data and those reported by Gulson et al. (15, 16), a recent study by Osterloh et al. (17) reported no relation between blood lead levels and changes in bone density (used as a surrogate for bone lead mobilization) in a cohort of lactating women (n = 58). Participants in this study were observed from 2 weeks postpartum to 6 months postpartum. They had low blood lead levels (mean values of 2.52 µg/dl and 2.33 µg/dl at 2 weeks and 6 months postpartum, respectively), and the authors did not assess external environmental exposure. However, Osterloh et al. analyzed changes in bone density by subtracting the initial measurement from the final one, instead of using the longitudinal structure of the data. Their approach may have obscured a small underlying association. It is likely that by collapsing the entire study period to one point, they may have underestimated the real variability of bone loss, especially if a high rate of resorption was followed by a high rate of bone deposition, attenuating the potential relation between bone resorption and blood lead levels (18).

Unlike the case in these cohorts, blood lead levels in our study were higher and had greater variability (ranging from 1.8 µg/dl to 30 µg/dl during the study period). After adjusting for bone lead burden and for external environmental exposures such as the use of lead-glazed ceramic ware for cooking, we found a significant relation between breastfeeding and blood lead levels. Furthermore, in the study by Osterloh et al. (17), bone lead burden was not measured, making it impossible to distinguish between a real lack of a breastfeeding effect on bone lead mobilization and the null results that would be expected from a study population with a low bone lead burden. Moreover, it is notable that Osterloh et al. reported that blood lead levels were elevated during the first 6 weeks postpartum and that this change was positively and significantly associated with decreases in vertebral bone density (17). This would seem to suggest that bone lead mobilization occurred in the early stages of lactation, as we documented in our study.

As was expected, in our final models, parity and cumulative history of lactation (measured as the number of months for which the mothers had breastfed their previous children) were inversely associated with blood lead levels, although only the former factor was marginally significant (p = 0.1). In our study, most women with a previous pregnancy (99 percent) reported having breastfed before.

Several limitations of our study must be addressed. First, we evaluated the effect, during lactation, of bone lead on blood lead instead of on plasma lead, which is likely to be the most significant toxicologic fraction. It is possible that the observed association would have been stronger had we measured plasma lead (4, 19). Second, we had neither information on changes in bone density nor information on bone remodeling biomarkers, so we could not directly evaluate the hypothesis that lactation increases bone lead mobilization. We assumed that increases in blood lead were directly due to the mobilization of lead from bone. Our assumptions were based on the known physiology of bone during pregnancy and lactation and on the fact that the main environmental lead sources, such as the practice of cooking with lead-glazed ceramics, were accounted for and that changes in bone lead levels during pregnancy and lactation could not be explained by dietary lead (20).

During the study period, our research team actively informed the study participants about the risk of cooking with lead-glazed ceramic ware. This may explain the decrease in its use (from 35 percent to 15 percent) and may have changed the lead exposure patterns of the participants. This change was considered in the longitudinal analysis, so it probably does not explain our results. However, given our study design, we were not able to determine whether the observed increase in blood lead was due to mobilization of bone lead or to increased absorption of dietary lead. To address this issue, we evaluated the interaction between use of lead-glazed ceramics (the major source of dietary lead in this population (21)) and breastfeeding practices; it was not significant, which supports the hypothesis that the source of lead was the mineral tissue.

This study investigated a relatively large group of women with considerably higher bone lead burdens (the arithmetic means were 15.3 µg lead/g bone mineral and 10.6 µg lead/g bone mineral for the patella and tibia, respectively; using the correction for negative values, these statistics were 16.9 and 11.6, respectively) and greater variability in bone lead levels than a previous study from the United States (22). In addition, the study population had relatively low current environmental exposure, making the contribution of endogenous lead sources more apparent.

Women with higher bone lead levels may mobilize more lead to the bloodstream during pregnancy and lactation than those with lower lead burdens. This may increase exposure of the fetus and the breastfed infant to lead during critical periods of the development of the central nervous system, which in turn may be associated with potential deficits in neurobehavioral development. The mobilization of lead may

**FIGURE 4.** Geometric mean blood lead levels versus patella lead levels (categorized by quintiles) among lactating women, Mexico City, Mexico, 1994–1995. ND, nondetectable.
also have toxicologic consequences for the mother; once it is present in the bloodstream, it may be redistributed to target organs (3).

Breastfeeding should be promoted, as it offers clear protection against infant morbidity (diarrhea and respiratory infections) and mortality and improves cognitive development (23) in developing countries as well as in developed countries (24, 25). By no means do our results suggest that alternative infant feeding methods should be sought in populations with a high lead burden. It has been shown that lead levels in infant formula are greater than concentrations of lead in breast milk (26). Our data underscore the relevance of searching for and implementing procedures that reduce lead exposure from endogenous sources as well as environmental sources.

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