ABSTRACT. As part of a longitudinal study of the sources and developmental effects of current urban lead exposure, lead was measured in tap water from the homes of 249 infants, in 100 breast milk samples, and in 73 samples of the infant formula used by non-nursing mothers. Also, the blood lead levels of the infants who received these fluids were determined at birth and at 6 months of age. Among the infants who were breast fed, the lead content of their milks correlated very well with their 6-month blood lead levels ($r = .42, P = .0003$). The mean lead content of infant formulas and breast milk were not significantly different, nor was the blood lead of children fed one or the other. Lead levels in maternal milk correlated poorly with umbilical cord blood lead ($r = .18, P = .10$). Tap water and infant blood lead levels correlated minimally ($r = .11, P = .10$). Since milk represents much of the diet of young infants and because breast milk lead levels are stable, it is possible to relate blood lead and daily dosage in this population.
MATERIALS AND METHODS

As a result of a survey of umbilical cord blood lead concentrations from 11,837 consecutive births at the Boston Living-In Hospital between September, 1979 and April, 1981, 249 children were enrolled in a study of lead and infant development. Subjects were drawn equally from the highest, lowest, and centermost deciles of blood lead. To be eligible for enrollment, the child must also have been expected to reside within Greater Boston (inside Route 128) for the next 2 yr in an English-speaking household, and to be free of serious medical conditions. In every case, informed consent was obtained from the parents. In general, their mothers were white (87%) and well educated (mean maternal schooling = 14.5 yr). Most children (88%) lived with both parents, and the mean maternal age was 30 yr.

Specimens of milk were collected in the homes by the mothers, on two visits (1 and 6 months post-partum) with a new, pre-cleaned, acid-washed, polyethylene, 35-ml cylinder vial with a hinged cap. Brand name and infant diet information were obtained also. If the mother was nursing, she was asked to collect breast milk directly with the vial, manually expressing the milk without any pump or other cup; if formula was being used, the mother was asked to put into the vial milk of the same dilution she used in feeding. These samples were frozen and stored at -5°C until analysis. About 30% of the mothers were given a vial in a plastic bag so that they could later collect and freeze breast milk and bring it to the hospital on their next routine visit. Although nearly all of the nursing mothers provided samples, laboratory resources limited the number of milk samples tested. Each month a random portion of those collected were measured.

Milk samples were brought to room temperature, shaken, and sonicated. Duplicate aliquots of 200 µl were digested in a microwave oven with an acid mixture under vacuum. The residue was redissolved in dilute perchloric acid and the lead was measured by anodic stripping voltammetry (ESA, Bedford, MA, Model 2014). Contamination during sample collection, transport, and storage was measured using sham procedures and distilled water. Only 1.0 µg/L was attributable to this contamination.

Non-parametric statistical tests were employed for comparisons of groups and paired samples. Correlations were measured by distribution-free rank methods.

RESULTS

The lead content of all breast milk and formulas are shown in Table 1. Their ranges overlap, and the lead concentrations do not differ significantly as judged by non-parametric Wilcoxon tests of rank scores (P = .14) or median tests (P = .24). Furthermore, among the three brands of formula used in this survey (i.e., Similac, Enfamil, and Isomil), lead levels do not differ significantly, nor are lead levels related to iron enrichment or container type (i.e., bottle or can). However, formula lead levels were more variable than breast milk lead levels (F-score for inequality of variance = 2.42, P < .01). Formula lead levels correlate poorly with infant blood lead levels (r = .11, P > .5, N = 73).

The lead concentrations in breast milk collected at 1 month differ minimally from those collected at 6 months; the mean difference between 20 paired samples is 0.7 µg/dl (SE = .5). Non-parametric tests reveal no differences among these two breast milk categories. Thus, lead levels in milk appear stable from 1 to 6 months. Cord blood lead correlates poorly with breast milk lead (r = .18, P = .11).

Tap water lead concentrations average 6.1 µg/L (SE = 1.4) and do not correlate significantly with breast milk, formula, or blood lead.

<table>
<thead>
<tr>
<th>Sample Type</th>
<th>Number</th>
<th>Mean</th>
<th>SE</th>
<th>Range</th>
</tr>
</thead>
<tbody>
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<td>0-7.2</td>
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<tr>
<td>Formula milk</td>
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<td>2.3</td>
<td>0.3</td>
<td>0-17.8</td>
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<tr>
<td>Tap water</td>
<td>249</td>
<td>0.6</td>
<td>0.1</td>
<td>0-34</td>
</tr>
<tr>
<td>Umbilical cord blood</td>
<td>249</td>
<td>7.2</td>
<td>0.3</td>
<td>0-23</td>
</tr>
<tr>
<td>Infant blood (6-mo)</td>
<td>221</td>
<td>6.2</td>
<td>0.5</td>
<td>0-49</td>
</tr>
</tbody>
</table>

Table 1.—Lead Concentrations (µg/dl) in Milk, Water, and Blood Samples Obtained in Boston
Blood lead levels of 6-month-old children fed formula average somewhat lower than the blood lead levels of those who were nursed: 5.6 μg/dl (SE = 0.5) vs. 7.6 μg/dl (SE = 0.6), respectively (P = .32 by Wilcoxon rank-sum test, or P = .05 by Student’s t-test).

Blood lead levels at 6 months of age correlate very well with dietary lead intake among the children who were nursed (Spearman, rank r = 0.42, P = .0004). Figure 1 shows that the observed values and the best (i.e., minimizing the product moment) linear fit: blood lead = 4.5 μg/dl (SE = 1.0) + 0.9 μg/dl (SE = 4) x milk lead. This model yields an r² of 6%, P = .04. If the milk lead is expressed logarithmically, which is more appropriate given the non-normal, skewed pattern of milk lead, a better fit is obtained (r² = 10%, model P = .009): blood lead = 3.6 μg/dl (SE = 1.1) + 3.0 μg/dl (SE = 1.1) x Ln(1 + milk lead). The intercepts of these two models are equivalent, and they both predict nearly identical blood leads when milk are less than 6 μg/dl.

DISCUSSION

The lead content of milk and baby formulas has received considerable attention during the past decade. One issue has been the relative lead content of human breast milk compared with commercially available preparations. In this survey, commercially available formulas have lead levels similar to those of mothers’ milk. The blood lead levels of the infants who received commercial preparations were insignificantly lower than those who were nursed by their mothers.

The concentrations of lead we found in breast milk are about the same as those reported previously. A nationwide sampling by Dillon, Wilson, and Schäffer, nearly a decade ago, resulted in a mean of 2.6 μg/dl. In 1975, Lamm et al. reported that breast milk lead averaged 2 μg/dl for 7 mothers, whereas infant formula and evaporated milk had much higher levels. Ryu et al. found that some commercial formula from Iowa in 1976 had markedly more lead than did breast milk. In addition, infants fed canned formula had elevated blood lead levels.

The reduction in lead content of infant formulas is attributable to improved packaging methods. Earlier soldering methods resulted in splashes of molten lead entering the can and contact between the solder and food, but the increased use of bottled formula, more careful canning techniques, and different seam designs have resulted in lower dietary lead intakes among infants.

Currently, the lead content of home tap water in Boston does not contribute demonstrably to the lead intake from formulas. This may be contrasted to the situation in Scotland, for example, where higher levels of lead in drinking water, often above 100 μg/L, had been the predominant source of lead to bottle-fed infants. The median dietary lead levels of 10 μg/dl and blood lead of about 18 μg/dl in Glasgow were both much higher than our findings in Boston. Our data most resemble the very lowest lead categories in Glasgow. Furthermore, combining Glasgow and Boston...
data demonstrates the curvi-linear nature of the response of blood lead to dietary intake when considered over a range from 0.1 to 2.3 mg/kg.

Milk lead accounts for only 10% of the variance in 6-month blood lead levels. When environmental lead observations and personal maternal factors are also considered, 18% of the variance in cord blood lead is explained. The significant covariate-adjusted predictors of cord blood lead in this same population at birth include indoor dust lead levels, smoking tobacco, drinking coffee and alcohol, parity, and maternal age. Also, monthly mean cord blood lead correlated well (product-moment r = .76) with monthly variations in sales of gasoline lead and indoor air lead levels. If infant blood lead is adjusted for these environmental predictors, breast milk lead is still the strongest correlate of 6-month blood lead. Breast milk lead levels do not correlate significantly with any of these environmental or maternal factors and provide additional information about the child's lead exposure.

The absolute values and variances of lead in this population were so small that no health concerns were aroused.

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