Determinants of Bone and Blood Lead Levels among Teenagers Living in Urban Areas with High Lead Exposure

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Although lead has been extensively studied in children, its sources and effects remain unclear in adolescents. This study examined the relation of blood and tibia bone lead levels to lead determinants. One hundred adolescents living in Mexico City and surrounding suburbs were studied. Blood lead was measured by atomic absorption spectroscopy, and tibia lead was measured by a K X-ray Fluorescence (KXRF) instrument. Blood lead ranged from 1.8 to 29.2 μg/dl, with a mean of 7.4 μg/dl. Bone lead ranged from <1 to 44.82 μg Pb/g bone mineral, with a mean of 4.28 μg Pb/g. Predictors of bone lead included higher traffic density near the home, mother’s smoking history, and time spent outdoors. Predictors of log-transformed blood lead included bone lead levels, male sex, use of lead-glazed ceramics, and living in Mexico City. Bone lead remained a significant predictor of blood lead after adjusting for covariates in a final multivariate regression model. In our final model, a rise in bone lead from the middle of the lowest quintile to the middle of the highest quintile (a difference of 21.6 μg Pb/g) was associated with an increase in blood lead of 1.2 μg/dl. Our data suggest that in addition to current sources of environmental lead exposure, bone lead accumulated over time constitutes a moderate source of circulating lead during adolescence and may account for some of the adverse health effects documented in recent studies.

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Lead remains one of Mexico City’s primary environmental pollutants despite increasing use of unleaded gasoline and recent regulations governing paint containing lead, lead-soldered cans, and lead-glazed ceramics (1). This is of concern because health effects related to lead are being documented at ever lower levels. Indeed, no threshold has been identified below which lead exposure can be considered safe (2,3).

Much investigation has focused on the effects of lead in children, but adolescents have been largely understudied. Because behavior patterns change with age, sources of lead exposure may vary in populations of different age distributions (1). Maternal lead levels transferred to the fetus via the placenta or to the breast-fed infant via breast milk and environmental exposures via hand–mouth behavior are responsible for the high exposure levels observed during early development. Subsequently, exposure from ambient lead diminishes and internal sources (mostly bone lead) may become more important as a chronic source of endogenous lead.

Adolescence is a uniquely important period in relation to lead effects because physical growth is at a rapid and final stage and therefore vulnerable to lead action (4). In addition, growth-induced bone turnover may also release lead stored in bone, thereby increasing the amount of lead dose and toxicity elsewhere in the body. Lead toxicity may worsen school performance during these years (2), which, in turn, may play an important role in determining a young adult’s subsequent pursuits. Of note in this regard is a recent study linking bone lead levels to an increase of antisocial, delinquent behavior in teenagers (5).

In this study, we describe the determinants of bone and blood lead and compare the influence of bone lead to environmental factors in determining blood lead levels in a group of adolescents living in the Mexico City area.

Materials and Methods

Study population. Using a cross-sectional study design, we examined a group of 100 adolescents between 11 and 21 years of age, selected from two sources. One group of 47 adolescent volunteers consisted of friends and relatives of employees of the American British Cowdray (ABC) Hospital; close to 50% of these participants attended private schools. The second sample consisted of 53 volunteers from a public high school located near the same hospital. The population was recruited from September 1995 to May 1996. All study participants were invited for bone and blood lead level testing and filled out a questionnaire developed by our research team to collect information on risk factors for lead exposure, as well as on nutrient intake. Study subjects signed an informed consent agreement and received the results with appropriate counseling.

Questionnaires. A questionnaire was used to collect information on general characteristics (age, sex, type of school, etc.) and known risk factors for lead exposure: occupational exposure, use of lead-glazed ceramics, use of eyeliner with kohl, exposure to lead paint, exposure to lead in air (location of the house in relation to traffic, nearby lead industries, and time spent outdoors). Questions about the use of lead-glazed ceramics were illustrated by photographs to help participants recall the type of pottery used at home.

More dietary calcium and caloric intake were measured using a food-frequency questionnaire based on the consumption of 128 food items in the last 12 months. This questionnaire has been validated in Mexican populations (6) and was developed following the approach suggested by Willet et al. (7).

Blood lead measurement. Venous blood samples were collected in lead-free tubes and analyzed using graphite furnace atomic absorption spectrophotometry (Perkin-Elmer 3000; Perkin-Elmer, Chelmsford, MA) at the trace metals laboratory of the ABC Hospital in Mexico City. External quality control was provided by the laboratory standardization program of the Wisconsin State Laboratory of Hygiene (Madison, WI). Our laboratory had adequate precision and accuracy during the study time [correlation = 0.98; mean difference 0.71 μg/dl; standard deviation (SD) = 0.68]. Blood lead measurements are reported in micrograms per deciliter (1 μg/dl = 0.0484 μmol/l).

Bone lead measurement. We used a 109Cd spot-source K X-ray fluorescence (KXRF) system to measure bone lead levels.

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The system was constructed at Harvard University and installed in our research facility. The details of its physical principles, technical specifications, and use in Mexico have been explained elsewhere (8,9). In brief, the instrument uses a $^{103}Cd$ gamma-ray source to provoke an emission of fluorescent photons from target tissue that are then detected, counted, and arrayed on a spectrum. The net lead signal is determined after subtraction of Compton background counts using a nonlinear least-squares algorithm. The lead fluorescence 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 unit of measurement is expressed in micromgrams of lead per gram of bone mineral (μg Pb/g). By normalizing the measurements to calcium counts, the measurement is rendered insensitive to variations in bone shape, size and density, overlying tissue thickness, and movement (10). Validation studies of the instrument indicate a fairly high degree of precision and accuracy of the point estimates in comparison to chemical analyses in studies of lead-dosed phantoms. Repeated measurements in 35 subjects in another study showed a high intraclass correlation of 0.84 and 0.82 for tibia and patella, respectively (11).

Because the instrument provides a continuous, unbiased point estimate that oscillates around the true bone lead value, negative point estimates are sometimes produced when the true bone lead level is close to zero. The instrument also provides an estimate of the uncertainty associated with each measurement that is derived from a goodness-of-fit calculation of the spectrum curves and is equivalent to a single standard deviation. Although a minimum detectable calculation limit of twice this value has been proposed for interpreting an individual’s bone lead estimate (12), retention of all point estimates makes better use of the data in epidemiological studies (13).

For the present study, 60-min measurements were taken from the left mid-tibia shaft after the region had been washed with a 50% solution of isopropyl alcohol. The energy line coefficients used to convert channel numbers into energy (eV) were obtained daily by measuring the K X-ray peak positions from a lead target. Once per week, the room housing the XRF instrument was cleaned with a high-energy particulate air filter vacuum cleaner. A blank phantom was then positioned and measured 20 consecutive times overnight as an additional calibration check. Analysis of means and standard deviations did not show a significant shift in accuracy or precision.

**Data analysis.** Univariate analysis was conducted on all variables to describe their distribution and check for outliers. Because blood lead level had a right-skewed distribution, we used the log-base e transformed variable (LBPb) for statistical analysis. The uncertainty measures of the tibia lead (TPb) determinations were used to verify the quality of the data; in accordance with previous studies, a TPb measurement was discarded.

### Table 1. Mean and standard deviation (SD) of blood and bone lead levels in relation to demographic and lifestyle factors in adolescents in Mexico City

| Independent variables | Mean blood lead ± SD (μg/dl)* | Mean tibia lead ± SD (μg Pb/g) |
|-----------------------|-------------------------------|--------------------------------|
| Sex                   |                               |                                |
| Females 62            | 6.4 ± 3.2                     | 5.5 ± 8.6                      |
| Males 36              | 9.1 ± 5.5**                   | 3.8 ± 8.5                      |
| Age (years)           |                               |                                |
| 11–14                 | 5.4 ± 1.4                     | 3.8 ± 11                       |
| 15–16                 | 7.4 ± 4.2                     | 3.8 ± 8.3                      |
| 17                    | 7.5 ± 4.1                     | 5.5 ± 7.8                      |
| 18–21                 | 7.9 ± 5.2                     | 6.8 ± 8.3                      |
| Type of school        |                               |                                |
| Public                | 7.4 ± 4.1                     | 5.5 ± 7.8                      |
| Private               | 7.3 ± 4.1                     | 2.5 ± 10.9*                   |
| Place of birth        |                               |                                |
| Other city            | 6.2 ± 4.0                     | 3.1 ± 7.3                      |
| Mexico City           | 7.9 ± 4.4**                   | 5.7 ± 9.0                      |
| Currently living in   |                               |                                |
| Mexico City           |                               |                                |
| No                    | 3.8 ± 1.5                     | 3.7 ± 7.0                      |
| Yes                   | 7.7 ± 4.4**                   | 5.0 ± 8.7                      |
| Total months          |                               |                                |
| Living in Mexico City |                               |                                |
| <100                  | 7.3 ± 4.8                     | 4.5 ± 9.9                      |
| 101–192               | 6.7 ± 4.9                     | 1.6 ± 7.0                      |
| 193–204               | 8.0 ± 3.8                     | 5.4 ± 7.4                      |
| >204                  | 7.4 ± 3.7                     | 7.9 ± 8.0*                    |
| Lead industry close to home |         |                                |
| No                    | 7.2 ± 4.0                     | 5.2 ± 7.9                      |
| Yes                   | 7.7 ± 5.1                     | 3.9 ± 10.2                     |
| Traffic density       |                               |                                |
| Almost none           | 7.7 ± 6.4                     | 0.5 ± 8.1                      |
| Low                   | 6.6 ± 4.1                     | 4.2 ± 8.6                      |
| Medium                | 8.4 ± 3.9                     | 6.7 ± 7.5                      |
| High                  | 7.0 ± 2.7                     | 7.5 ± 8.0*                    |
| Most frequent type of transportation |     |                                |
| Car                   | 6.1 ± 2.8                     | 2.1 ± 9.3                      |
| Bus                   | 6.3 ± 1.9                     | 5.6 ± 8.1                      |
| Subway                | 7.8 ± 4.1                     | 6.3 ± 7.3                      |
| Public van            | 8.7 ± 6.0                     | 6.8 ± 8.1                      |
| None                  | 8.8 ± 6.3                     | 3.2 ± 10.5                     |
| Minutes spent outdoors/week |               |                                |
| 1–120                 | 6.3 ± 3.5                     | 3.8 ± 7.6                      |
| 121–360               | 7.6 ± 5.6                     | 4.4 ± 8.9                      |
| 361–2400              | 8.4 ± 5.2                     | 4.4 ± 8.5                      |
| >2400                 | 8.1 ± 3.2*                    | 9.4 ± 9.8*                     |
| Painted house in last 12 months |         |                                |
| No                    | 6.8 ± 3.5                     | 4.1 ± 8.5                      |
| Yes                   | 8.2 ± 5.3*                    | 5.9 ± 8.7                      |

*Significant differences between categories tested by analysis of variance after taking the logarithm of blood lead levels.

*Includes only females.

*p < 0.15; **p < 0.05.
if its associated uncertainty was >10 μg/g. The bivariate analysis consisted of analysis of variance tests of TPb and LBPb for each categorical variable. The ANOVA tests were also conducted on age-adjusted TPb. Finally, a multivariate model was created for each dependent variable (TPb and LBPb) by including all of the independent variables that reached a statistical significance of $p<0.15$ in the bivariate analyses. A backward elimination procedure was then applied that discarded variables that did not reach statistical significance at the $p<0.05$ level. All of the statistical analyses were conducted using STATA (7.0).

## Results

The population of 100 adolescents consisted of 62 females and 38 males. Their ages ranged from 11 to 21, with a mean age of 17 years. Eighty percent of study participants had blood lead levels <10 μg/dl, with a median of 6.1 μg/dl and a mean of 7.4 μg/dl. The point estimates of tibia lead levels ranged from -19.9 to 44.8 μg Pb/g, with a mean of 4.8 and a median of 5.3. A total of 25 participants had point estimates of bone lead at or below zero, and 26 had point estimates of bone lead >10 μg/g. Two subjects were eliminated from subsequent analyses. One had a bone lead measurement uncertainty estimate >10 μg/g, and the other did not provide the bone measurements. The median measurement uncertainty estimate for the remaining subjects was 4.5 μg Pb/g.

In bivariate analyses, older adolescents had higher mean TPb levels, with a mean difference of 6 μg separating ages 11–14 from ages 18–21 years (Table 1); however, the differences between age groups did not meet the $p<0.15$ cut off level for statistical significance. Significantly higher bone lead levels were associated with attendance at public school, being born in Mexico City, high traffic density, greater amounts of time spent outdoors, and having a mother who smoked (Table 1).

Variables found to be associated with increased LBPb in bivariate analyses ($p<0.15$) were male sex, being born in Mexico City, currently living in Mexico City, greater amounts of time spent outdoors, past use and current use of lead-glazed ceramics, having painted the house in the past 12 months, current smoking, current alcohol consumption, use of eyeliner, and higher levels of lead in tibia bone (Table 1).

In the final multivariate model of TPb after backward elimination (Table 2), covariates included male sex, use of lead-glazed ceramics in the past, current use of lead-glazed ceramics, living in Mexico City, and tibia bone lead. Bone lead accounted for 4.1% of the variation in blood lead levels (in comparison with a total model adjusted $r^2$ of 0.39). We observed a moderate relation between tibia bone lead and blood lead levels (Fig. 1). A rise in bone lead from the middle of the lowest quintile to the middle of the highest quintile (a difference of 21.6 μg Pb/g) was associated with an increase in blood lead of 1.2 μg/dl.

Low calcium intake was marginally associated with blood lead levels. In comparison with the rest of the group, participants who had a calcium intake in the lower quartile (mean 623 mg/day) had higher blood lead levels (1.85 μg/dl; $p = 0.071$).

## Discussion

Our results show that bone and blood lead concentrations vary according to several environmental variables. That bone and blood lead did not correlate with the same environmental variables may be due, at least in part, to the distinct time aspects of these two biological markers of lead dose: bone lead mostly reflects cumulative absorption over years, whereas blood lead reflects relatively recent exposure. Thus, the correlation of bone lead, but not blood lead, with traffic density may be an indication of the recent decline in leaded gasoline combustion as a source of lead exposure in Mexico City (15). The association of bone lead (but not bone lead) with lead-glazed ceramic use confirms the status of lead-glazed ceramics as an important source of current lead exposure (7) and indicates the difficulty in capturing lifetime lead-glazed ceramic use by using a questionnaire.

Table 2. Multivariate regression model of tibia lead (n=98; adjusted $R^2 = 0.136$)

| Independent variables | Coefficient | SE  | p-Value | 95% Confidence interval |
|-----------------------|-------------|-----|---------|-------------------------|
| Traffic density       |             |     |         |                         |
| Almost none           | Reference   |     |         |                         |
| Light                 | 2.52        | 2.32| 0.280   | -2.08-7.12              |
| Medium                | 4.27        | 2.45| 0.085   | -0.59-9.14              |
| High                  | 5.60        | 2.99| 0.065   | -0.34-11.55             |
| Time spent outdoors (min/week) | 0.005 | 0.00 | 0.042 | 0.00-0.009 |
| Mother smokes         |             |     |         |                         |
| No                    | Reference   |     |         |                         |
| Yes                   | 5.01        | 1.86| 0.008   | 1.32-8.70               |
| Intercept             | -1.90       | 2.0 | 0.345   | -5.87-2.07              |

Table 3. Multivariate regression model of blood lead (n=92; adjusted $R^2 = 0.39$)

| Independent variables | Coefficient | SE  | p-value | 95% Confidence interval |
|-----------------------|-------------|-----|---------|-------------------------|
| Sex                   |             |     |         |                         |
| Female                | Reference   |     |         |                         |
| Male                  | 0.269       | 0.088| 0.003  | 0.098-0.449             |
| Tibia lead (μg Pb/g)  |             |     |         |                         |
| Never users           | Reference   |     |         |                         |
| Past users            | 0.009       | 0.004| 0.338  | 0.005-0.018             |
| Past users            | Reference   |     |         |                         |
| Current use of Pb-glazed ceramics (days/week) | 0.083 | 0.024 | 0.003 | 0.034-0.131 |
| Lives in Mexico City  |             |     |         |                         |
| No                    | Reference   |     |         |                         |
| Yes                   | 0.485       | 0.158| 0.003  | 0.171-0.798             |
| Intercept             | 1.043       | 0.154| 0.000  | 0.736-1.350             |

SE, standard error.
smoking parent, tobacco may be a very prevalent source of lead accumulation in children. Unlike other environmental sources of lead, the control of which requires governmental regulation, tobacco exposure in the home can be more easily controlled on an individual basis. Thus, from a public health perspective, it is important to provide parents and adolescents with information about this addi-
tional detrimental health effect of smoking.

We observed a marginal inverse association between calcium intake and blood lead levels. However, we did not find a significant influence of dietary calcium on bone lead levels. This is in contrast to the inverse association reported between calcium intake and blood lead levels among children 1–11 years of age, who participated in the Second National Health and Nutrition Examination Survey (NHANES II) (19) and the protective effect we found in our study of bone and blood lead levels among lactating women (9). It is not possible to determine if this was due to the lack of a biological effect, the small sample size studied, or the difficulty in measuring adolescent dietary nutrients using a semi-quantitative food frequency questionnaire.

The significant association of bone lead with blood lead levels, even after adjusting for environmental variables, supports the hypothesis that endogenous lead in bone constitutes an important source of circulating lead in adolescents. As stated earlier, the cross-sectional nature of this study limits our ability to draw inferences on the directionality of the relationship between tibia and blood lead. We are not aware of any other investigations that have studied the relation between bone and blood lead levels in adolescents. Nevertheless, our results are similar to those described in other age groups.

Recently, in a study of blood lead levels during lactation among women living in Mexico City (9), we observed a significant relation between bone lead and blood lead after controlling for environmental sources of lead exposure. Similarly, Hu et al. (18) observed a significant influence of bone lead on blood lead after controlling for environmental lead among middle-aged to elderly men in the United States. The observed tibia lead levels in our study were lower than those observed for women of reproductive age in Mexico City (mean = 12.5 μg/g; SD = 11.6) (9) and for middle-aged to elderly men living in greater Boston, Massachusetts (geometric mean = 20.8 μg/g) (16). Our observations suggest that even at lower levels of accumulation and in adolescents, bone lead may serve as a significant contributor to circulating lead. It is possible that this effect
is enhanced by the rapid bone turnover that accompanies adolescent growth.

A comparison of our results with blood lead levels found in other studies in Mexico City is consistent with a temporal decline in environmental lead exposure among Mexican youngsters. Unleaded gasoline was introduced in Mexico beginning in 1990, and by 1997 all automobiles in Mexico City were required to use unleaded gasoline. In 1990, the mean blood lead level was 15.2 µg/dl in 1,553 children aged 5–16 years, and in 1992 the mean blood lead level of another sample of children 1–5 years old was 12.7 µg/dl, with more than 80% of the sample still exceeding 10 µg/dl (20). We observed a mean blood lead level of 7.4 µg/dl in the adolescents studied. Despite this progressive decline in blood lead levels, the influence on bone lead levels of traffic density in relation to each subject’s home and time spent outdoors clearly demonstrates the impact of previous air lead exposures on cumulative bone lead burden.

A comparison of our results with bone lead levels found in a recent study conducted in the Boston area among 500 students aged 13.4 to 18.9 years did not reveal large differences in mean levels of tibia bone lead (Fig. 2). In a study of 23 adolescents 18–21 years old (21), 66 young adults with a mean age of 20.5 years (22), and 169 students 13.5–19 years old (23), mean tibia lead levels were 3.0, 1.3, and 4.0 µg Pb/g, respectively. On the other hand, our study included some individuals with very high bone lead levels. Twenty six (26%) of the subjects in this study had bone lead levels >10 µg/g, whereas the percentages of subjects that had bone lead levels >10 µg/g of the previously mentioned studies were 0%, 3%, and 8%, respectively. Moreover, significant correlations were found between environmental factors and bone lead in our study, but not in the Boston studies, despite the use of similar techniques. The relatively small differences in mean bone lead levels may be due to general washout of lead in the skeleton during the rapid growth of the teenage years, a phenomenon predicted by kinetic modeling (24).

In conclusion, a number of environmental factors were found to be related to bone and blood lead levels in this group of Mexican adolescents, reflecting current and past sources of lead exposure. Although the lead levels themselves were not exceedingly high and within an order of magnitude to those observed in the United States, recent epidemiological studies indicate lead neurotoxicity at even low levels and should spur continued efforts at reducing exposure as much as possible. In addition to regulatory efforts to reduce environmental sources of exposure, there are various personal behaviors that can be changed, such as the use of lead-glazed ceramics and smoking. Finally, this study and others suggest that lead accumulated in bone from historical exposures may serve as an important endogenous source of circulating lead. Further research is needed to see if bone lead is a biological marker of dose that independently predicts toxicity. Measures may be needed to mitigate the effects of lead stored in bone as a secondary prevention strategy.

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