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Brief Communication

Past Occupational Exposure to Lead: Association Between Current Blood Lead and Bone Lead

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Kim Metheny, BS; Michael Tobin, PhD

ABSTRACT. Mobilization of lead from bone is known to increase with age. The authors performed the current study to determine whether there was an association between current blood lead and bone lead in workers with no current exposure but with significant past workplace exposure. The authors assessed 58 men, aged 40 to 76 years, who had earlier exposure to lead and determined both current blood lead levels and bone lead levels. At the time of the current assessment, the average blood lead level was 10.9 $\mu\text{g}/\text{dL}$ and tibia bone lead concentrations ranged from -12.5 to 223.3 . The authors divided workers into 3 groups by age (40–49, 50–59, and 60–76). Correlations between blood lead and bone lead were highest in the 2 oldest age groups (.49 and .75, respectively). Hierarchical regression analysis was significant for an interaction between bone lead and age in predicting blood lead (the combination of age and bone lead significantly predicted an increase in current blood lead levels). The results support the hypothesis that lead stored in bone is a significant source of blood lead later in life. Older workers with past occupational exposure may face a particular risk for recirculation of lead in blood with advancing age.

KEYWORDS: adults, blood, lead, occupational diseases

Neurotoxic effects of lead have been documented for more than a century. Although levels that constitute “safe” exposure (eg, permissible exposure limits) have been revised downward in the recent past, lead exposure continues to be widespread in the United States, with more than 1.4 million industrial workers having potential lead exposure.¹ In 1998, an adult lead registry in New Jersey reported that there were “23,456 reports of occupational lead toxicity (blood lead level $\geq 1.21 \mu\text{mol}/\text{L}$ or $25 \mu\text{g}/\text{dL}$) in 4,011 workers, involving 496 workplaces.”^{2(p1128)}

Blood lead levels (BLLs) estimate current circulating levels in the blood, but they do not measure cumulative exposure or body burden. Lead is well absorbed by the lungs and gastrointestinal tract and eventually deposited in bone. Body burden for lead increases through the lifespan, and 90% to

95% of lead is stored in bone.¹ The biological half-life of lead in bone is around 30 years.¹ Bone lead concentration—measured with x-ray fluorescence (XRF)—provides a non-invasive estimate of cumulative lead exposure. With age, bone demineralizes; stored lead is remobilized, enters the bloodstream, and may provide an unexpected hazard, even if initial exposures were thought to be low.³ Postmenopausal women with no history of occupational exposure have been found to have significantly higher BLL, suggesting remobilization of lead from bone through involutional osteoporosis.⁴ Bleeker et al⁵ examined bone and blood lead levels in smelter workers for whom information on past blood levels was available. Bone lead was correlated with age, years of employment, and measures of current and cumulative blood levels.

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We conducted this study to determine whether there was an association between current BLLs and bone lead levels in workers with no current exposure but with significant workplace exposure in the past. We were particularly interested in determining whether the increase in BLLs found in older women held true for older men with past workplace exposure. Our sample was a group of workers previously employed at battery plants in Eastern Pennsylvania.

METHODS

Participants

Between 1981 and 1984, 288 lead-exposed workers were evaluated as part of an epidemiological study of lead conducted at the University of Pittsburgh.⁶ Lead-exposed workers were members of District 10, United Steel Workers of America, and worked at 3 industrial plants in Eastern Pennsylvania that manufactured lead batteries for automobiles and motorcycles. Only workers with no previous known exposure to neurotoxins, except lead, were recruited. All were white, native English-speaking males. Study participants were randomly selected from company lists. At initial testing, workers' mean age was 35 (range = 21–60) and blood lead levels averaged 40 µg/dL ($SD = 13.2$); highest peak blood levels reached 90 µg/dL.

From the original cohort, we were able to contact 89 individuals who agreed to come to Pittsburgh for reevaluation in connection with the current study. The mean age at current testing for the sample was 53.7 years (range = 40–76). Only workers with no ongoing exposure were included in the current analyses (ie, last exposure was at least 8 months before the current examination). Of the 89 workers, 59 had no ongoing lead exposure. We excluded 1 participant due to unusable XRF data. The final data pool consisted of 58 participants (M age = 55.7 years; range = 41–76). The average number of years since last exposure was 10 (range = 9 months to 17 years). At the current assessment, average BLL was 10.93 µg/dL ($SD = 5.1$; range = 3–25). Tibia lead concentrations for the 58 workers ranged from –12.58 to 223.33 ppm ($M = 67.78$). Because both systolic blood pressure and smoking have been associated with lead,^{7–10} we performed assessments to evaluate current blood pressure and smoking history. The Institutional Review Board of the University of Pittsburgh School of Medicine approved the protocol, and we evaluated participants only after receiving informed consent. We reimbursed participants for travel to the study site and for participation in the protocol.

Assessment of Blood Lead

To assess current BLL (µg/dL), a registered nurse drew one tube of blood (~6 mL). BLLs were determined using atomic absorption spectrophotometry conducted by Central Laboratory Service, Inc, an affiliate of the University of Pittsburgh Medical Center (detection limit = 3 µg/dL). The lab is certified for the analysis of lead in blood by the

Occupational Safety and Health Administration and the Centers for Disease Control and Prevention.

Assessment of Bone Lead

We used XRF to measure tibia bone lead. The participant sat in a plastic armchair, with one leg lightly restrained in a polystyrene jig. In XRF trace analysis, an excitation source of photons induces fluorescence of characteristic x-rays. We obtained bone lead concentration estimates with the conventional K-XRF technique, employing 88.025 keV from ¹⁰⁹Cd to induce Pb K-shell x-ray fluorescence, measured with a backscatter counting geometry. An excitation source of photons induces fluorescence emission of characteristic x-rays that are used to quantify the amount of trace element in the sample matrix. By recourse to calibration data, the ratios of individual Pb K x-ray areas to the “elastic scatter” peak (88.0 keV) provide bone lead estimates in units of µg Pb/g of bone mineral. We estimated bone lead concentrations from the lead K $\beta_{1,3}$ x-rays (84.94 and 84.45). A 30-minute tibia exposure resulted in an effective dose of 17 nanosievert (nSv), corresponding to 0.001% of the average natural environmental radiation dose (3 millisievert [mSv]). The University of Pittsburgh Radiation Safety Committee approved the XRF protocol.

This method of analysis, by taking bone density into consideration, provides measures of lead concentration independent of variation in that value.¹¹ To maintain quality control, a check standard (lucite-encased lead target) is run prior to participant measurement to calculate general instrument performance. The lead peak amplitudes and locations in the acquired spectrum are compared to control norms established from replicate counts of the standard described by Kanipe.¹² We used conservative criteria to set our minimum detectable concentration (MDC) values. We estimated instrumental MDC as 4.65, the standard deviation of replicate low-lead “blank” National Institute of Standards and Technology phantom measurements. For 16 replicates, we obtained an MDC of 14.7 µg Pb per gram plaster, corresponding to an MDC of 21.5 µg Pb per gram bone mineral. When the lead K x-ray signal is small, the masking effect of the Poisson-distributed background frequently produces negative bone lead concentrations. For the current study, only 3 participants had numbers in the negative range (–1 to –12), and we transformed these values to 0.

RESULTS

Association Between BLL and XRF in Lead Workers

We divided participants into 3 age groups: 40–49 years, $n = 19$; 50–59 years, $n = 21$; and 60–76 years, $n = 18$. We found little association between bone levels and blood levels in the youngest age group, and the correlation for the youngest group was not significant ($r = .35$, $p = .13$). The association in the 2 older age groups was greater: in workers aged 50–59 years, $r = .49$ ($p = .004$), and in workers

older than 60, $r = .75$ ($p < .001$). We conducted a hierarchical regression analysis modeling bone lead (divided into 3 groups—low, medium, and high—on the basis of equal distribution of the data), BLL, age, smoking, systolic blood pressure, and the interaction between bone lead and age. For this analysis, BLL was the dependent variable. We entered age, systolic blood pressure, and smoking (average number of cigarettes per day) at the first step. Bone lead was entered on the second step, and the age $\times$ bone lead interaction term was entered on the third step. In the first step, age, smoking, and blood pressure were not significant predictors and accounted for only 5% of the variance ($\Delta F[3, 54] = 1.0$, $p = .35$). In the second step of the model, bone level added significantly to the model, accounting for 32% of the variance ($\Delta F[4, 53] = 19.6$, $p = .001$). In the third step, adding the interaction term—age $\times$ bone lead—significantly increased the prediction ($\Delta F[5, 52] = 6.47$, $p = .01$). The overall model predicted 41% of the variance. Thus, the results show that in addition to the simple effects of bone lead on BLL, the combination of age and bone lead significantly predicted an increase in current BLL.

COMMENT

We examined the association between BLL and bone lead in a sample of workers with past occupational exposure to lead who now range in years from middle to old age. The workers had not worked with lead, on average, for the preceding 10 years. The findings indicate a strong association between bone lead and blood lead, especially in workers in the older age groups. These results suggest that lead influx from bone is an important source of circulating lead. The data support prior findings indicating a remobilization of lead with increasing age.

Most researchers who have looked at the association between blood lead and bone lead have studied women or community-exposed samples. Lead release from bone has been shown to increase in response to pregnancy, lactation, and menopause, particularly among postmenopausal women not using estrogen.^{3,4,10,13,14} Several investigators have focused on environmentally exposed samples that included men. Kosnett et al¹⁵ found a reduction in bone lead in older, nonoccupationally exposed men and women, suggesting increasing bone turnover with age. In a more recent study of 656 community-exposed men, Elmarsafawy et al¹⁶ looked at occupational status—blue collar versus white collar—and found higher rates of bone lead in those men who had worked in primarily blue-collar jobs. However, blood levels were low and not associated with occupational status. Furthermore, the authors did not examine the effect of age. Bleeker et al⁵ assessed several measures of past lead exposure (eg, bone lead, current blood lead, integrated blood lead index) in workers with occupational exposure and found the strongest associations between blood lead and bone lead in workers who had been employed in earlier years when occupational standards were less stringent ($r = .17$). However,

these workers had a mean age of 46 years, and changes across the lifespan were not considered.

In this current sample, we did not find a significant association between current BLL and either blood pressure or smoking. Other researchers have noted an increase in systolic blood pressure in workers with past exposure to organic and inorganic lead,^{7,8} and positively associated cigarette smoking with blood lead.⁹ The lack of an association with cigarettes may be due to the small number of smokers ($n = 14$). In the regression model, the association between blood pressure and BLL was in the expected direction, but the significance level failed to reach the conventional cutoff ($p = .097$) and may reflect a lack of power for this and the other covariates.

A drawback to the current study is the availability of only a current BLL and lack of information on BLL for the workers at the time of their prior employment. This data was not available due to de-identification of records. Four persons in the current sample had levels above 20: one was aged 58 and the other 3 were older than 68. Of these 4 participants, none had worked with lead on the job for > 10 years and none reported exposure from hobbies or in the home. As none of the other participants had worked with lead for at least 8 months prior to testing, the detectable levels that we noted in this study are probably linked to the remobilization of lead from past stores.

The findings of relatively high BLLs in older individuals who had not worked with lead for more than a decade support the hypothesis that circulating BLL in older workers may be a surrogate marker for past exposure. Older workers with prior exposures to lead in the workplace may face an additional neurotoxic hazard due to lead remobilization long after exposure has ended.

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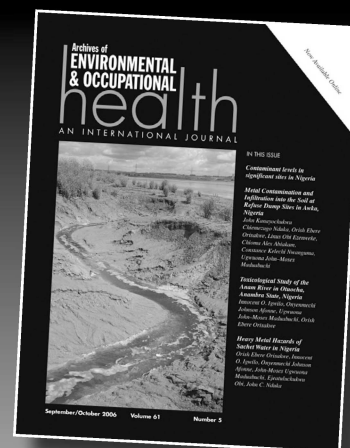
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