

Predictors of Plasma Lead Among Lithographic Print Shop Workers in Mexico City

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Background Plasma lead is considered a biological marker that reflects the fraction of lead in blood that is toxicologically available. We examined the relationship between plasma lead and other biomarkers of lead exposure in 69 lithographic print shop workers.

Methods Lead was measured in plasma and whole blood (by inductively coupled plasma-magnetic sector mass spectrometry), in bone (by ¹⁰⁹Cd X-ray fluorescence), and in hand wipes and occupational air samples. Personal hygiene habits at work were surveyed.

Results Mean age was 47 years and 86% (n = 59) were men. Mean lead levels were 0.3 µg/L in plasma, 11.9 µg/dL in blood, 46.7 µg/g in patella, and 27.6 µg/g in tibia. Taken together, two multivariate linear models explained 57% of variability in plasma lead levels. Predictors for the first model were lead in patella ($\beta = 0.006$), blood ($\beta = 0.008$), and hygiene index ($\beta = -0.11$). Predictors for the second model were lead in tibia ($\beta = 0.008$), blood ($\beta = 0.008$), and hygiene index ($\beta = -0.13$).

Conclusions This study demonstrates that accumulated bone stores and hygiene habits are both significant independent predictors of plasma lead levels in active workers at this print shop. Am. J. Ind. Med. 46:245–252, 2004. © 2004 Wiley-Liss, Inc.

KEY WORDS: plasma lead; bone lead; air lead; occupational exposure; Mexico

INTRODUCTION

Traditionally, deleterious effects of lead exposure on health have been assessed in occupational studies using blood lead level as a biological marker for recent exposure. These measurements are also used to formulate decisions on chelating treatment, assess industrial hygiene control, and evaluate the contribution of different exogenous and endogenous sources of lead exposure [Chavalitnitikul et al., 1984; Manton, 1985; Hodgkins et al., 1991; Ulenbelt et al., 1991; Kentner et al., 1994; Hernández-Avila et al., 1998].

However, beginning several decades ago, attempts have been made to measure plasma lead levels under the assumption that they reflected a more toxicologically active fraction of lead than did whole blood lead levels [Cavalleri et al., 1978]. Since that time, it has been shown that lead in plasma comprises only ~0.2–0.8% of whole blood lead levels in environmentally exposed subjects [Manton and Cook, 1984; Cake et al., 1996; Hernández-Avila et al., 1998;

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Smith et al., 1998, 2002]. Determination of plasma lead levels was not performed routinely in the past principally due to lack of sensitive instrumentation capable of detecting very low levels of lead in plasma and the enormous challenges posed by incidental contamination of plasma samples from environmental sources during collection and processing. Other factors that have confounded past investigations of plasma lead as a biomarker include sample contamination from hemolysis occurring during blood-sample collection and processing and use of anticoagulants (e.g., EDTA) that may have artifactually altered distribution of lead between plasma and cellular fraction [Smith et al., 1998].

Lead concentrations in air, hands, clothes, working surfaces, hair, and face as well as non-hygienic behaviors at the workplace have been identified as exogenous sources of exposure influencing lead levels in blood [Chavalitnitikul et al., 1984; Askin and Volkmann, 1997]. Lead accumulation in bone was long suspected as an internal source of exposure and measurements by more accurate methods have proven that bone lead levels are an important endogenous source of exposure [Gerhardsson et al., 1993; O'Flaherty, 1993; Smith et al., 1996]. In fact, up to 95% of total lead accumulated in the body of an adult is stored in the skeleton [Barry, 1975]. Bone lead can be measured with non-invasive K X-ray fluorescence (KXRF) techniques, providing a good marker of chronic exposure [Gerhardsson et al., 1993; Hu et al., 1995].

The majority of occupational studies have not provided a complete set of endogenous and exogenous lead exposure measurements. The purpose of this study was to assess the relationship between plasma lead and a variety of exogenous and endogenous sources of lead exposure in a group of active lithographic print shop workers in Mexico City.

MATERIALS AND METHODS

Subjects

A cross-sectional study was carried out between June 1996 and May 1997 among 209 workers of a lithographic print shop located in Mexico City. One hundred seventeen (56%) workers agreed to participate in the study, although only 90 (43%) fully completed the first phase of the study [Aguilar-Madrid et al., 1999]. Among participants and non-participants, no statistically significant differences were present with regard to age, sex, or on-the-job tenure [Aguilar-Madrid et al., 1999]. Of these 90 subjects, 69 (77%) were randomly selected and agreed to participate voluntarily in the study phase, which included collection of whole blood for blood and plasma lead determinations. Among 69 participants and 21 non-participants, differences were observed in age and years living in Mexico City, and there was borderline significance in differences of on-the-job tenure. The reduced number of participants was mainly the result of logistical constraints associated with collection and processing of

plasma lead samples. Methods and results (excluding plasma lead levels) employed in the larger study that included the entire sample have been published elsewhere [Aguilar-Madrid et al., 1999].

The lithographic print shop began operations in 1935 and the working process still in use there is described in detail by Aguilar-Madrid et al. [1999]. Until 1990, books were produced exclusively using lithography, a process involving smelting a lead alloy of tin and antimony at a temperature of 35°C to make linotypes. This smelting process generates fumes of respirable lead oxide particles. In 1990, a cold-type process was introduced that involves computers, laser printers, photography, and offset printing. In this way, use of metal alloy became less frequent. However, at the time of our study the company continued to utilize both processes.

Data Collection

A structured questionnaire that collected information on the following characteristics was used: (i) sociodemographics; (ii) life styles; (iii) hygiene practices at work; and (iv) sources of environmental exposure to lead. We developed a hygiene index based on nine yes/no questions related to personal hygiene at work. The questions were the following: (1) Do you wash your hands before eating?; (2) Do you wash your face before eating?; (3) Do you smoke in your work area?; (4) Do you eat in your work area?; (5) Do you use a work uniform?; (6) Do you change your work clothes after finishing work?; (7) Do you wash your work clothes?; (8) Do you have a special place to keep your work clothes?; and (9) Do you take a shower after finishing your work?

All questions were equally weighted. One point was assigned for good hygiene habits (i.e., for each yes response to hygiene-related questions) and points were added up for a possible score ranging from 0 to 9.

Air Lead Measurements

Beginning in June 1996, airborne lead concentrations were measured within the workers' breathing zone (personal samples) during a single workshift. For this purpose, cellular ester filters were affixed to high-flow SKC pumps to collect airborne particulate lead. The flow-meter was calibrated at the beginning of the study in accordance with the altitude of Mexico City (7,000 feet above sea level) by a Gilian primary digital calibrator available at the Environmental Analysis Laboratory of a Mexico City firm (Análisis Ambiental in the Mexico City Federal District). This procedure was followed in accordance with Official Mexican standards [STPS, 1994, NOM-010 and STPS, 1994, NOM-033].

Pumps were calibrated daily with a flow-meter (or secondary calibrator) before being attached to the belt of each employee, as well as at the end of the working day. Every 2 hr, each pump was checked to maintain constant flow at 2 L/min.

Total lead levels collected on the 37-mm diameter and 8- μm pore cellulose ester filters were analyzed together with five field blanks by NIOSH [1994] Method 7105 (atomic emission spectroscopy in the laboratories of the National Institute for Occupational Safety and Health (NIOSH) in Cincinnati, OH. Lead concentrations were reported in micrograms per cubic meter ($\mu\text{g}/\text{m}^3$).

Hand Lead Measurement

At the same time that we conducted the air monitoring we also collected hand lead samples for workers at the midpoint in the worker's day and before eating, according to the NIOSH [1994] Method 9100 (lead in surface wipes) as described by Aguilar-Madrid et al. [1999]. Workers were provided with two disposable towels dampened with water, benzalkonium chloride, lanolin, and benzoic acid. They were shown how to use the towels to clean fingers, palms, and backs of hands up to the wrist. Subjects were instructed to perform this for 1 min; one towel was used before and one towel was used after washing hands. Towels were stored in separate, lead-free receptacles. The participant's right hand was traced on paper to calculate lead concentration according to surface size (of both hands). Samples were sent to the NIOSH Laboratory for analysis and concentrations were reported in micrograms per square meter ($\mu\text{g}/\text{m}^2$).

Bone Lead Measurement

Approximately 8 months after the air and hand lead samples were collected, lead levels were measured in the patella and tibia bones in vivo using a KXRF instrument. Thirty-minute measurements were taken at the midshaft of the tibia and at the left patella after each region had been washed with a 50% solution of isopropyl alcohol. Bone lead measurements were made using a ^{109}Cd gamma ray KXRF instrument constructed at Harvard University and installed in a BRIMEX II Research Center at the ABC Hospital in Mexico City. This instrument utilizes a fixed gamma ray source to cause emission of fluorescent photons from the target tissue and that upon being detected, are transformed into a spectrum for quantitation. The physical principles, technical specifications, and steps needed for validation of the measurements have been described by Aro et al. [1994]. Results are reported as micrograms of lead per gram of bone mineral ($\mu\text{g Pb/g}$).

Plasma and Blood Lead Measurements

Nine months after sampling lead in the air and on the hands and 1 month after performing the bone lead measurements, blood and plasma samples were collected for lead measurements using ultra-clean methods [Hernández-Avila et al., 1998; Smith et al., 1998, 2002]. Whole blood and

plasma samples were taken by qualified personnel who followed a strict protocol to reduce contamination, using a class-100 HEPA-filtered air bench and who had rigorously cleaned sample collection and storage containers. For plasma lead sample collection, ~ 13 ml of whole blood was collected and plasma separated as previously described [Hernández-Avila et al., 1998; Smith et al., 1998, 2002]. For analysis of lead in whole blood, 3–5 ml of whole blood were deposited in a low-lead Vacutainer tube containing heparin as an anticoagulant. Following collection, blood and plasma samples were stored and shipped frozen to the Trace Metal Laboratory at the University of California Santa Cruz (UCSC), USA, for analysis by high resolution inductively coupled plasma mass spectrometry. Plasma lead results are reported as micrograms per liter ($\mu\text{g}/\text{L}$), while whole blood results are reported in units of $\mu\text{g}/\text{dL}$.

Data Analysis

Data were analyzed using STATA software, version 6.0. Exploratory information analysis was carried out to assess data quality and consistency as well as distribution of variables of interest. Plasma lead levels were log \hat{e} -transformed to normalize their distribution; subsequently, bivariate and multivariate analyses were carried out using univariate and multiple linear regression, respectively. Plasma lead/blood lead ratio was also evaluated as a dependent variable with univariate and bivariate analysis.

Diagnosis and analysis of model influence were performed using Studentized residuals [Montgomery and Peck, 1992], and extent of colinearity between patella lead and tibia lead was also assessed. Because colinearity existed between the two measures of lead in bone (tibia and patella), we constructed two models that assessed their predictive capacity independently.

RESULTS

Mean age of participants was 47 years (standard deviation (SD) = 12 years) ranging between 20 and 74 years of age; mean on-the-job tenure at the print shop was 8 years (SD = 10) ranging from 1 to 40 years, and 90% of participants reported use of lead-glazed ceramics to prepare their food. Mean plasma lead (PbP) content was 0.30 $\mu\text{g}/\text{L}$ (SD = 0.22) with a range of 0.05–1 $\mu\text{g}/\text{L}$. Mean for blood lead (PbB) was 11.9 $\mu\text{g}/\text{dl}$ (SD = 5.8) with a range of 3.5–30 $\mu\text{g}/\text{dL}$ (Table I). PbP/PbB ratio showed a mean of 0.24% (SD = 0.1) with a range of 0.06–0.5%. Nearly all workers (97%) consumed food in their work area. Although only 12% did wash their face before eating, 82% washed their hands before eating, and 82% of smokers routinely smoked at the workplace.

Univariate linear regression analyses were carried out to study the association between plasma lead and endogenous

TABLE I. Descriptive Statistics of Variables Under Study

Measurements	Mean	Median	SD	Range	n
Plasma lead ($\mu\text{g/L}$)	0.30	0.22	0.22	0.05–1.0	69
Plasma lead/blood lead ratio (%)	0.24	0.24	0.1	0.06–0.5	69
Blood lead ($\mu\text{g/dL}$)	11.9	11	5.8	3.56–30	69
Tibia lead ($\mu\text{g/g}$)	27.6	26.4	18.1	ND–73.8	67
Patella lead ($\mu\text{g/g}$)	46.8	38.2	29.3	ND–0.9	67
Air lead ($\mu\text{g/m}^3$)	0.9	0.3	1.6	ND–7.0	62
Pre-wash hand lead ($\mu\text{g/m}^2$)	8,530	842	22,100	27–159,000	63
Post-wash hand lead ($\mu\text{g/m}^2$)	225	86.5	320	3–1,460	62

Workers at a lithographic print shop in Mexico City, 1996–1997.

ND, non-detectable.

SD, standard deviation.

and exogenous predictors (Table II). Statistically significant associations were observed between plasma lead and blood lead, patella lead, tibia lead, age, education, and use of lead-glazed ceramics, but not with air lead, lead on hands, or hygiene index at work. Temporal differences in sampling between these latter factors may be the reason there was no significant relationship. Years living in Mexico City was marginally significant ($P = 0.06$). Blood lead ($\beta = 0.088$, $P < 0.001$) explained 49% of variability observed in plasma lead levels, whereas patella lead ($\beta = 0.013$, $P < 0.001$) and tibia lead ($\beta = 0.020$, $P < 0.001$) explained 25 and 24% of variability, respectively (Table II). Figure 1 shows the smoothed relationship between the natural log of plasma lead levels and lead levels in blood, tibia, and patella.

Table III shows results of two multiple linear regression models for plasma lead constructed separately with patella lead and tibia lead as main predictors and adjusting for both

TABLE II. Simple Linear Regression Analysis Between Plasma Lead Levels* ($\mu\text{g/L}$) and Endogenous and Exogenous Predictors

Predictors	Coefficient	SE ^a	R ²	P
Blood lead ($\mu\text{g/dL}$)	0.088	0.011	0.49	<0.001
Patella lead ($\mu\text{g/g}$)	0.013	0.003	0.25	<0.001
Tibia lead ($\mu\text{g/g}$)	0.020	0.004	0.24	<0.001
Air lead ($\mu\text{g/m}^3$)	0.046	0.06	–0.006	0.4
Age (years)	0.015	0.007	0.04	0.04
Living in Mexico City (years)	0.013	0.007	0.04	0.06
Education (years)	–0.073	0.027	0.08	0.01
Use of lead-glazed ceramics (yes/no)	0.78	0.275	0.09	0.006
Hygiene at work (0–9)	–0.028	0.086	–0.01	0.7
Pre-wash hand lead ($\mu\text{g/m}^2$)	0.016	0.042	–0.01	0.7
Post-wash hand lead ($\mu\text{g/m}^2$)	0.3	0.3	–0.02	0.9

Workers at a lithographic print shop in Mexico City, 1996–1997.

*In log_e scale.

^aSE, standard error.

blood lead and hygiene index; these models explain 57% of variability in plasma lead levels. In both models, coefficients for all predictors are similar.

We also observed that both patella lead and tibia lead show positive associations with plasma lead/blood lead ratio (Fig. 2). The hygiene index turned out to be a significant predictor of plasma lead after adjusting for blood lead and bone lead. For the patella lead model, the following results were observed: patella lead ($\beta = 0.00001$, $P = 0.009$), and hygiene index ($\beta = -0.0002$, $P = 0.05$), whereas for the tibia lead model, results were tibia lead ($\beta = 0.00002$, $P = 0.018$) and hygiene index ($\beta = -0.00030$, $P = 0.03$).

DISCUSSION

In this study, the most important endogenous predictors of plasma lead were blood lead, patella lead, and tibia lead and the best exogenous predictor was personal hygiene behavior. Likewise, the maximum value of plasma lead/blood lead ratio (i.e., 0.5%) was consistent with levels reported in other studies using similar methods [Manton and Cook, 1984; Schütz et al., 1996; Hernández-Avila et al., 1998; Smith et al., 1998, 2002].

The lack of association of air lead levels and blood and plasma lead in this study is not surprising given the temporal separation in collection of these samples, the relatively low air-lead levels, and the fact that we only collected one sample per worker. Other studies have observed that even workers exposed to high air lead concentrations can display low blood levels of this toxin, while persons exposed to low air lead concentrations can display high levels of lead in blood [Ulenbelt et al., 1991]. Based on these observations, we propose that oral ingestion and gastrointestinal uptake of lead may be an important and underestimated route of lead entry into the body of workers in this type of occupational setting.

In our study, three main exogenous sources were evaluated: a qualitative hygiene index, lead measured on hands, and air lead. Better hygiene behavior at work was associated with lower plasma lead levels (Table III). These

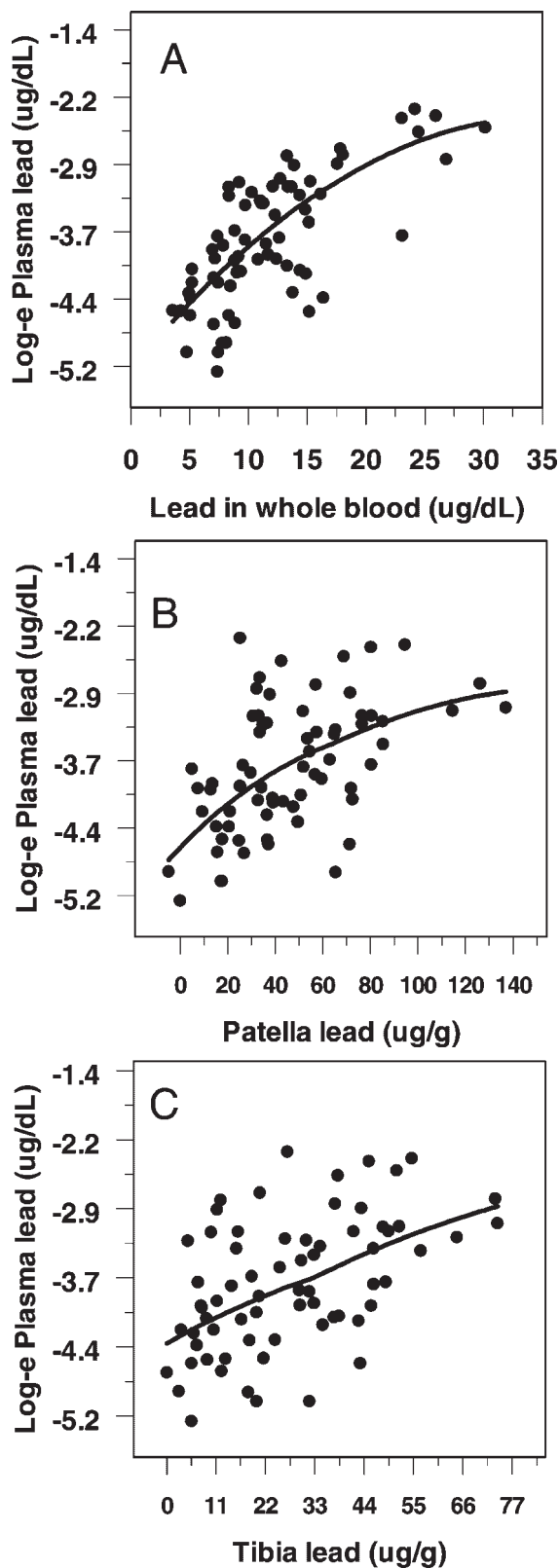


FIGURE 1. The relationships of plasma to other lead biomarkers, with superimposed smoothed plots (Lowess smoother, bandwidth = 0.8). (A) Blood lead. (B) Patella bone lead. (C) Tibia bone lead. Plasma lead was log-e transformed.

TABLE III. Multiple Linear Regression Models for Plasma Lead Levels* (ng/ml)

Independent variables	Coefficients	SE ^a	95% CI
Model with patella ($R^2 = 0.57$)			
Hygiene index (0–9)	-0.11 ^b	0.06	-0.23, -0.005
Blood lead ($\mu\text{g/dL}$)	0.008 ^c	0.001	0.006, 0.01
Patella lead ($\mu\text{g/g}$)	0.006 ^d	0.002	0.002, 0.01
Model with tibia ($R^2 = 0.57$)			
Hygiene index (0–9)	-0.13 ^d	0.06	-0.25, -0.02
Blood lead ($\mu\text{g/dL}$)	0.008 ^c	0.001	0.006, 0.01
Tibia lead ($\mu\text{g/g}$)	0.008 ^b	0.004	-0.0004, 0.01

Workers at a lithographic print shop in Mexico City, 1996–1997.

*Log scale; 95% confidence interval.

^aSE, standard error.

^b $P > 0.05$.

^c $P < 0.001$.

^d $P > 0.001 < 0.05$.

findings have been observed in other studies where blood lead was used as a dependent variable [Ulenbelt et al., 1990, 1991; Chuang et al., 1999]. In our study population, lead measurement on hands did not explain plasma lead variability. This may be because blood sampling was not carried out simultaneously with sampling of lead on hands, and that only one (possibly non-representative) hand lead measurement was taken.

The negative association found in this study between plasma lead levels and the hygiene index (Table III) suggests that oral exposure and gastrointestinal uptake of lead was the predominant source of lead exposure in these subjects. Other studies have found similar results, but with blood lead levels as the marker of exposure [Ulenbelt et al., 1990; Askin and Volkmann, 1997; Chuang et al., 1999], further substantiating the importance of the hand–mouth mechanism in the occupational environment as a route of lead entry into the body through the digestive tract. Also, respiration of larger particles ($>2\text{--}3\ \mu\text{m}$) would be coughed up and swallowed leading to oral intake of airborne lead. We found that potential routes of lead exposure were numerous. None of the active workers used personal protective equipment such as gloves or respirators, and the printing facility had no methods for reducing lead levels in the air. Moreover, workers had no special place to eat, and 97% consumed food in the work area. In addition, 88% did not wash their faces before eating and 82% of smokers smoked at their workplaces. Although 82% reported they washed their hands before eating, this did not eliminate all metal from their hands, as proven by median lead levels of $86.5\ \mu\text{g}/\text{m}^2$ found in post-wash samples. We found reduced levels of exposure to lead with higher levels of education because more education was associated with better jobs in the lithographic print shop.

Few studies have attempted to quantitatively assess lead exposure through the digestive tract by different methods.

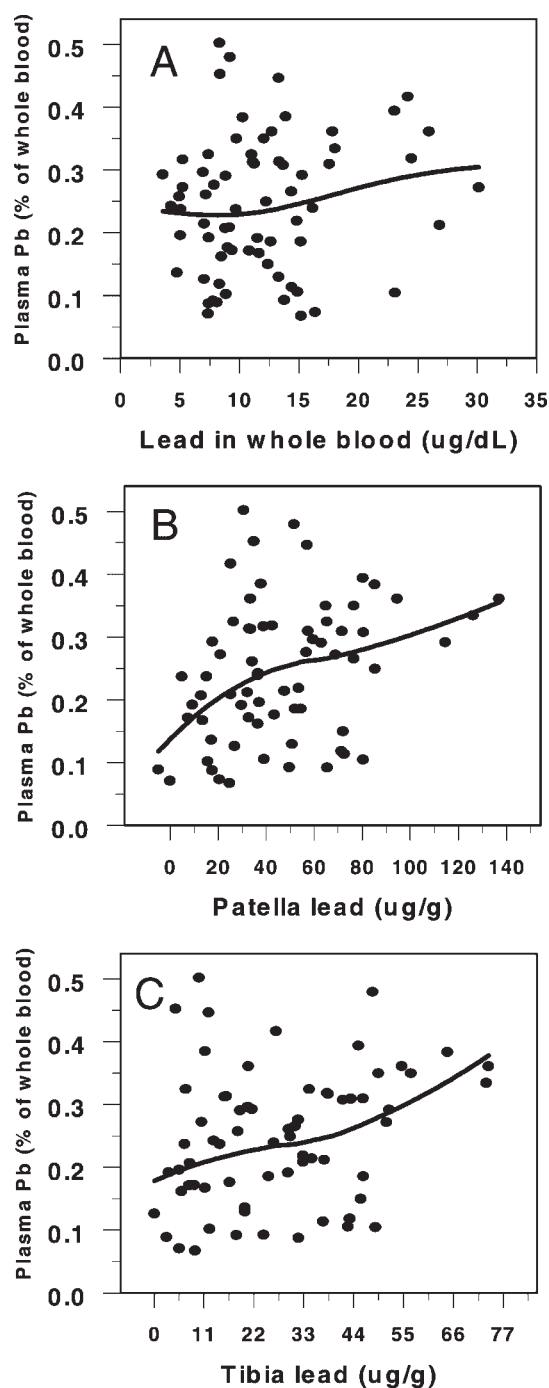


FIGURE 2. The relationships of plasma-to-whole-blood lead concentration ratio to other lead biomarkers, with superimposed smoothed plots (Lowess smoother, bandwidth = 0.8). (A) Blood lead. (B) Patella bone lead. (C) Tibia bone lead.

This lack of interest is perhaps due to the fact that only 8–10% of total lead ingested by an adult is absorbed [Tola et al., 1973], and the fact that until recently there were no reliable methods to measure exposure from hands, clothes, and working surfaces.

Quantitative measurements of lead concentrations have been carried out on workplace surfaces, and the clothes, hands, mouth, and face of workers [Lichtenwalner, 1992]. Qualitative methods, including questions related to personal hygiene habits at work have also been used [Chavalitnitikul et al., 1984; Far et al., 1993; Chuang et al., 1999]. Chavalitnitikul et al. [1984] showed, using simple linear log models, that lead concentrations on face, hands, working surfaces, and air are significant predictors of blood lead ($P < 0.0001$). Far et al. [1993] indicated that in a population of Malay origin, the magnitude of blood lead variance explained by lead on hands and mouth was 40%, probably because this population eats exclusively with their hands rather than with utensils. Recently, in a longitudinal study with a 7-year follow-up Chuang et al. [1999] concluded that smoking and eating at the workplace contribute significantly to blood lead levels. These results coincide with our findings, indicating an association between hygiene conditions at work and blood and plasma lead levels.

The partitioning of lead between plasma and blood cellular fraction balance is determined largely by lead-binding affinity and lead-binding capacity of each sub-compartment, e.g., red blood cells (RBC) and plasma. From a kinetics standpoint, however, the lead must first go into the plasma before it can get to the RBCs. Our data suggest that plasma has lower-capacity binding compared to the cellular fraction (e.g., RBCs). They also suggest that erythrocytes may store lead in a temporary and limited way. Our study results support this idea because lead in RBCs behaves as an endogenous predictor of plasma lead. Lead in RBCs was also associated with bone lead levels, as observed in other studies. However, the percent variability of plasma lead explained by blood lead in this study (47%) is notably lower than that reported by Hernández-Avila et al. [1998] (95%). This is probably because that study used a controlled population that had no occupational exposure. It also measured plasma, blood and bone lead at the same point in time, and lead levels were determined mostly by endogenous sources. Our study, on the other hand, measures a more variable occupational exposure as an exogenous source of lead exposure. In the study by Cake et al. [1996] done in a population occupationally exposed to lead, blood lead explained 67% of plasma lead variability, a percentage more similar to that reported in this study.

Lead accumulation in bone occurs differently in trabecular bone (patella) than in cortical bone (tibia). Studies show that there is higher lead accumulation in trabecular bone during times of active lead exposure [Gerhardsson et al., 1993; Cake et al., 1996; Hernández-Avila et al., 1998], probably due to increased blood perfusion and bone turnover and a higher rate of lead accumulation by this type of bone. Higher lead accumulation in trabecular bone was also observed in this study (Table I), in which it behaved as a predictor of endogenous exposure for plasma lead. In adults,

approximately 25% of trabecular bone is reabsorbed and replaced each year. Consequently, toxicokinetics of lead accumulated in this kind of bone is more accelerated, whereas, due to the protective function of compact bones, only 3% of cortical bone is reabsorbed and replaced each year [García, 1997]. Bone coefficients in both models are very similar, probably due to a greater lack of precision in lead measurement in patella compared to lead measurement in tibia.

Several studies suggest that chronic exposure to lead derived from bone storage has negative repercussions. Hu et al. [1994] point out that an increase in patella lead levels of 37 $\mu\text{g/g}$ observed in construction workers are associated with a decrease of 11 g/L in hemoglobin even when blood lead concentrations were low (8.3 $\mu\text{g/dL}$, mean). Accumulation in bone is also associated with hypertension [Hu et al., 1996; Korrick et al., 1999; Lee et al., 2001; Rothenberg et al., 2002; Glenn et al., 2003] and reduced weight at birth [González-Cossío et al., 1997]. Therefore, the contribution of bone-stored lead is important. Gulson et al. [1995], Smith et al. [1996], and Rothenberg et al. [2000] indicate that the contribution of bone lead to blood lead levels of women of childbearing age reaches 45–70%. Thus, this dynamic lead not only affects the health of male and female workers, but probably that of their offspring as well.

This study explored possible predictors of the plasma lead/blood lead ratio. We observed that hygiene index and patella lead explained 13% of variability of this ratio, while the same index and tibia lead explained 10%.

One limitation of this study was that only one measurement of different endogenous and exogenous sources was made. In addition, measurements of lead on hands and in the air were not carried out simultaneously with those of plasma lead and blood lead. Another limitation was that retired workers did not participate in the study. They might have had higher bone lead levels and different behavior regarding blood lead and plasma lead levels because they would have been exposed principally to endogenous sources of bone lead. Likewise, assessment of oral exposure is a very complex task and the methods employed are inaccurate; thus, we must take into account that the real contribution of lead absorbed through this route to plasma lead levels may be underestimated. Finally, the fact that lead measured in air was not associated with plasma lead is probably due to the lack of information with regard to the proportion of lead that may be inhaled.

Lead intake through the digestive tract among workers of this print shop is perhaps the most important route of lead exposure. This is due to, among other factors, poor hygiene habits and deficient control measures for industrial hygiene, which favor contamination of hands, clothes, and working surfaces. This conclusion is based in part on the fact that very low air lead concentrations were recorded in this study (Table I).

Some studies have shown that implementation of educational programs for workers has helped lower their blood lead levels, as in the program developed and assessed by Chuang et al. [1999]. Similarly, application of programs for the effective use of personal protection equipment (gas masks) has also had a favorable impact [Lee et al., 1993]. However, the most important procedure to reduce contamination of working surfaces and tools, uniforms, and body parts (hands and face) is application of environmental control measures in lead particle-emitting sources. Kentner et al. [1994] showed that this, together with improvements in worker hygiene behaviors and a medical surveillance program, diminishes mean blood lead levels from 48.92 to 22.99 $\mu\text{g/dL}$. This study also showed that as workers improve their personal hygiene behaviors, plasma lead levels decrease. For this reason, permanent informative and training programs for workers on adequate working practices and healthy personal hygiene behaviors should be a fundamental objective in preventive programs for workers who are occupationally exposed to lead.

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