DOI: 10.1007/s00267-001-0007-1

Tijuana Childhood Lead Risk Assessment Revisited: Validating a GIS Model with Environmental Data

ELISABETH J. GONZALEZ
Orange County Health Care Agency
Divisions of Environmental Health and Disease Control & Epidemiology
2009 E. Edinger Ave.
Santa Ana, California 92705, USA

PATRICK G. PHAM
JONATHON E. ERICSON
Department of Environmental Analysis
University of California, Irvine
Irvine, California 92697-7070, USA

DEAN B. BAKER
Center for Occupational and Environmental Health
University of California, Irvine
Irvine, California 92612, USA

ABSTRACT / The objective of this research was to determine the spatial distributions of childhood lead poisoning and soil lead contamination in urban Tijuana. The Bocco–Sanchez model of point-source emissions was evaluated in terms of validity and reliability. We compared the model's predicted vulnerable populations with observed cases of childhood lead poisoning in Tijuana, identified fixed point sources in the field, and analyzed 76 soil samples from 14 sites. The soil lead results were compared to the blood lead analyses performed on Tijuana children whose blood lead levels were $\geq$10 μg/dL, who reported that they did not use lead-glazed ceramics for cooking or storing food ($n = 63$). Using GIS, predicted vs observed risk areas were assessed by examining spatial patterns, including the distribution of cases per designated risk area. Chi-square analysis of expected vs observed values did not differ significantly at the $p = 0.02$ level, showing that the model was strikingly accurate in predicting the distribution of subjects with elevated blood lead. Results reveal that while point sources are significant, other sources of lead exposure are also important. The relative public health risk from exposure to lead in an urban setting may be assessed by distinguishing among sources of exposure and associating concentrations to blood lead levels. The results represent an iterative approach in environmental health research by linking environmental and human biomarker lead concentrations and using these results to validate an environmental model of risk to lead exposure.

The worldwide distribution of lead (Settle and Patterson 1980; Ng and Patterson 1981), the numerous pathways of human exposure (EPA 1993), and well-documented toxicity of lead in biological systems (Goyer 1993) render lead as one of the most ubiquitous environmental toxins. The natural baseline of lead in pre-Industrial humans was at least 1000 times lower than Americans living in 1978 (Ericson and others 1979). For environmental management, the real issues of lead in an urban setting are its relative toxicity, location, concentration, and longevity in soil. Lead as a toxic agent affects a broad spectrum of biological systems including neurological, immune, renal, reproductive, sensory, and hematological (Goyer 1993). It affects individuals throughout developmental processes, particularly affecting vulnerable populations such as fetuses, children (Goyer 1996), pregnant and lactating mothers (Gulson and others 1998), and the elderly. Lead, which is stored and sequestered in bone, can be remobilized, particularly with osteoporosis, during extended bed rest, pregnancy, and lactation (Gulson and others 1998), as well as during periods of increased bone turnover, such as occurs during childhood, hyperthyroidism, and certain pathological states (Tsaih and others 1999). In addition, exposure to lead at relatively low levels is of particular concern during childhood due to its effects on cognitive development (Pirkle and others 1998). Geochemical factors (species of lead, particle size, and matrix in which the species is incorporated) control the availability of lead in soil or dust (Davis 1992). Nutritional status, intensity of hand-to-mouth behavior, differences in quantity and concentration of ingested lead, and variation in clearance rates of blood Pb by the kidneys determine the magnitude of individual variation in blood lead levels from environmental media (Mahaffey 1998).

KEY WORDS: Exposure assessment; Risk management; Soil lead; Model validation; Urban; GIS; Tijuana, Mexico

*Author to whom correspondence should be addressed; *email*: jeericso@uci.edu

Tijuana as a Study Area

Tijuana is a major urban area located in Baja California, Mexico, on the United States border near San Diego, California. Tijuana has an official population of 954,000 people [Instituto de Estadistica, Geografia e Informatica (INEGI) 1994] and an unofficial population of over 1.5 million. It is estimated that 52% of inhabitants of Tijuana cross into the United States for various purposes and that half of those who cross remain in the United States. In effect, the migration of Mexican citizens transfers their "lead poisoning problem" to U.S. jurisdiction. At the same time, potential sources of lead exposure in Tijuana derive in part from industrial development closely related to U.S. interests. Therefore, it is important to consider lead exposure as a transborder problem which, if unaddressed, will lead to adverse long-term economic, social, and health consequences on both sides of the border.

There has been a great deal of binational concern over the health effects associated with exposure to lead in children. However, very limited data exist on the exposure levels of children in Tijuana, B.C., who may be exposed to multiple lead sources during development. Exposure to lead can result from maternal transfer, both *in utero* and during lactation. Exposure can also result from environmental sources such as soil lead and dust (including that generated from the combustion of leaded gasoline and from lead-based paint), lead-soldered plumbing, and inadvertent occupational take-home exposure from family workers in radiator shops, auto shops, brass foundries, and battery and metal recycling. Exposure may be due to ingestion from low-temperature lead-leaching glazed pottery, canned and home-grown foods, candies, and lead-rich traditional Mexican home remedies.

Despite excellent medical and health care in Tijuana, which has experienced many successes in areas including the recent vaccination program, very few physicians have diagnosed and treated patients for clinical lead poisoning. Also, at present there is not a great awareness among government, industry and citizens of problems associated with lead exposure. Prior to 1998, there were no trained lead abatement officers and the certified analytical laboratory in the General Hospital, Tijuana, was not in existence. With increased industrialization, the lead problem can only worsen in the absence of a program encompassing surveillance, prevention, and abatement. Although lead additives to gasoline have been phased out, leaded gasoline continues to be available and appears to be a dominant fuel since it is less costly than unleaded alternatives. In effect, citizens of Tijuana have effectively used leaded gasoline for 20 years longer than U.S. consumers.

Importance of Soil Lead Studies

Exposure to residential soil containing lead levels between 50 and 150 μg/g has been shown to pose a significant risk to young children and pregnant mothers (Reagan and Silbergeld 1989). We hypothesized that a major source of lead exposure in Tijuana would be dust and soil from the local environment. Although most of the potential residential lead sources can be eliminated or greatly reduced, environmental lead most likely will present a long-term problem. This concern is due both to the residence time of lead in soil, which is 400 to 3000 years, depending on the soil type and environmental condition (Lansdown 1986), and to the finding that environmental soil lead appears to impact public health differentially compared to other sources of environmental lead exposure (Hemphill and others 1991). Specifically, studies of children exposed to lead in an urban setting and/or near a smelter reveal that these sources of exposure to lead in soil result in higher blood lead levels than does exposure from mining wastes, even when the soil lead concentrations are similar (Steele and others 1990). Further, accumulated soil lead in the urban environment has been shown to be a highly significant source of exposure for children. Mielke and others (1997) found "a response 12 orders of magnitude stronger for soils than for age of housing" when environmental concentrations were correlated with blood lead.

Within the urban environment, exposure to lead at levels which pose a risk are influenced by sociodemographic characteristics of the population, and a recent Washington, D.C., soil lead study found that higher blood lead concentrations were associated with lower education levels (Elhelu and others 1995). Another study focusing on the distribution of soil lead in Ottawa, Canada, resulting from transportation routes found a significant association with the prevalence of hyperactivity among school children (Ericson and Mishra 1991). Mexico City has provided the opportunity to study variant Pb exposures and their health effects due to increased levels of Pb observed in this urban population (Romieu and others 1994, Rothenberg and others 1995).

The number of reported Pb exposure sources in Tijuana, both residential and environmental, has represented a challenge to deconvolute major source contributions. Bocco and Sanchez (1997) identified a number of fixed point sources of lead contamination. This project was designed to test the Bocco and Sanchez

model, which identified vulnerable populations relative to fixed point sources of lead contamination in Tijuana. A fixed-point source was defined as an industrial site using lead as an input in processing, or one which could potentially emit lead as a by-product of processing, together with all major gas stations (Bocco and Sanchez 1997). The main objective of our research was to determine the spatial distributions of childhood lead poisoning and soil lead in urban Tijuana and to determine the degree of risk posed by point sources relative to other types of sources of lead exposure in the environment. Secondarily, we compare the predicted vulnerable populations identified by Bocco and Sanchez (1997) with observed cases of childhood lead poisoning in Tijuana.

Methods

Epidemiological Survey

The USEPA/UCI Tijuana Lead Project was supported by the U.S. Environmental Protection Agency and Centers for Disease Control, with permission of the Secretaria de Salud, Municipality of Tijuana, and 6 health care sectors.

A geographical, age-stratified epidemiological 3-year study was conducted to survey the blood lead concentration, residential lead use, and socioeconomic and demographic factors of 1719 Tijuana children ages 1.5–6.9 years. All research was approved by the Human Subjects in Research Committee of the University of California, Irvine (UCI HS No. 94-410). The sampling strategy was designed to achieve a representative sample of children living throughout Tijuana, while being able to examine associations between blood Pb concentration and sources of lead exposure. The mean age among subjects was 4.0 ± 1.5 years. All of the subjects were Hispanic, and 49.3% were female (Ericson and Baker 1999). The overall mean blood lead level in Tijuana was 6.2 ± 3.4 μg/dL. In accordance with CDC and Mexican guidelines, 9.8% of subjects had BLLs between 10.0 and 19.9, and 1% had levels from 20 to 43 μg/dL (the maximum level found in this study). In comparison, the CDC's NHANES III survey (the primary source for monitoring BLLs in the U.S. population) found that the mean BLL among 1–5 year olds was 2.7 μg/dL and that 4.4% had levels equal to or exceeding 10 μg/dL. We found that 10.8% of the children were classified as lead poisoned (≥10 μg/dL) as defined by Mexican law following CDC guidelines. These children were enlisted in the case management program. Venous blood samples were obtained from each child in the study following a proscribed protocol.

Table 1. Blood lead and soil lead survey results

	Mean	Range	SD
Soil lead (μg/g) (n = 76)	223	2–7870	982
Blood lead (μg/dL) (n = 63)	13.2	10–33	4.9
	Geometric mean		SD
Soil lead (μg/g) (n = 76)	37.5		1.6
Blood lead (μg/dL) (n = 63)	12.7		4.9

Samples were analyzed by the Public Health Laboratory, County of San Diego, in 1997 and by the Blood Lead Laboratory in the General Hospital, Tijuana, in 1998 using atomic absorption spectrometry and voltaic stripping, respectively. Both laboratories are registered with and perform regular quality control samples provided by the CDC—State of Wisconsin Certified Lead Testing Program.

Soil Lead Survey

In March 1997, a survey was undertaken to assess the soil lead concentration around point sources in Tijuana. A team from UCI was deployed to locate sites reported to be point source emitters (Bocco and Sanchez 1997) and to collect samples in proximity of the reported point sources. The point source sites which were the basis of the model were used as the sampling locations for the UCI field study. All sites which were in current operation and which were accessible for sampling were included. In the field, researchers determined the type of industry or source at each location before collecting samples. The team also had the opportunity to sample at apparent sources which had not been previously identified. A total of 76 samples from 14 sites was collected and analyzed. The blood and environmental lead results are presented in Table 1.

Soil Lead Analysis

The 76 samples depicted in Figure 1 were analyzed by Fayette Environmental Services in accordance with the protocol of Mielke and others (1983). The highest Pb content was found to be 7870 μg/g. The soil samples were observed to contain highly variable lead levels, and 57 samples contained less than 100 μg/g. The mean soil lead content was 223 μg/g. All of the 19 samples which contained lead concentrations between 100 and 7870 μg/g were collected from five point sources. These sources represented industrial emitters of lead and are depicted in Figure 2. Low soil lead levels contributed to the variability in this data set and question the legitimacy of many of the remaining purported fixed points as sources of contamination.

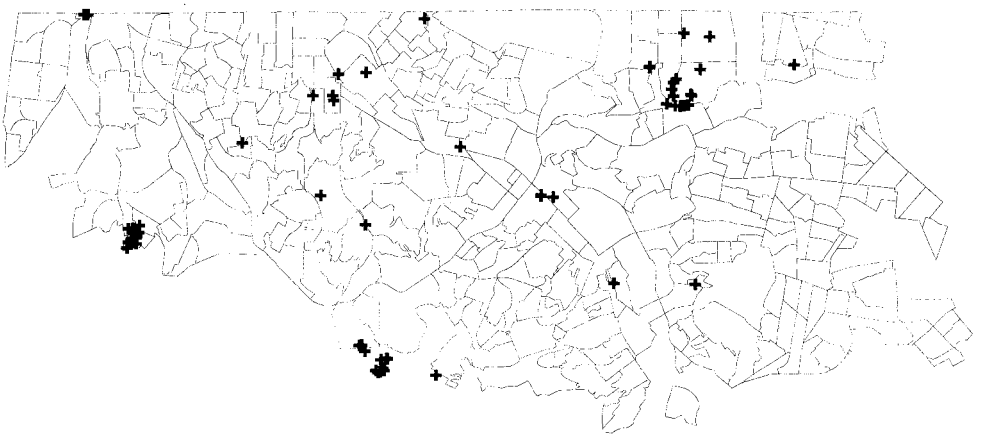

Figure 1. Distribution of 76 fixed point source soil lead samples collected from 14 sites in Tijuana.

Figure 2. Distribution of five point sources with soil samples containing 100 to 7870 μg/g soil lead ($n = 19$).

Reevaluation of the Bocco and Sanchez Fixed Point Emissions Model

Due to rapid industrialization during the latter half of the 20th century in Mexico, exposure to lead has become an increasingly significant public health problem (Romieu 1997). Along the United States–Mexico border, there has been limited information on blood lead levels until recently (Estaban and Hart 1998). In 1995, Bocco and Sanchez modeled the risk of lead exposure to susceptible children in Tijuana. Children aged 1 to 6 years old were the focus of their study, as they are the age group most susceptible to lead poisoning due to a high dietary rate of absorption and rapid growth compared to other age groups (Bocco and Sanchez 1997).

In 1996, the UCI investigators commenced to investigate the prevalence and possible causes of lead poisoning in Tijuana. Subjects aged 1.5–6.9 years were sampled citywide to achieve a representative sample of the exposed population. The frequency and distribution of cases in the city were recorded and analyzed by spatial analysis, with a case defined as any subject whose blood lead concentration was equal to or greater than 10 μg/dL, the CDC level of concern.

Based upon these analyses, the Bocco and Sanchez model of predicted risk was compared to the Tijuana field study data. Bocco and Sanchez' predictions, based upon fixed industrial lead point sources, assumed that distance would be the major determinant in lead contamination. Under these assumptions, the model as-

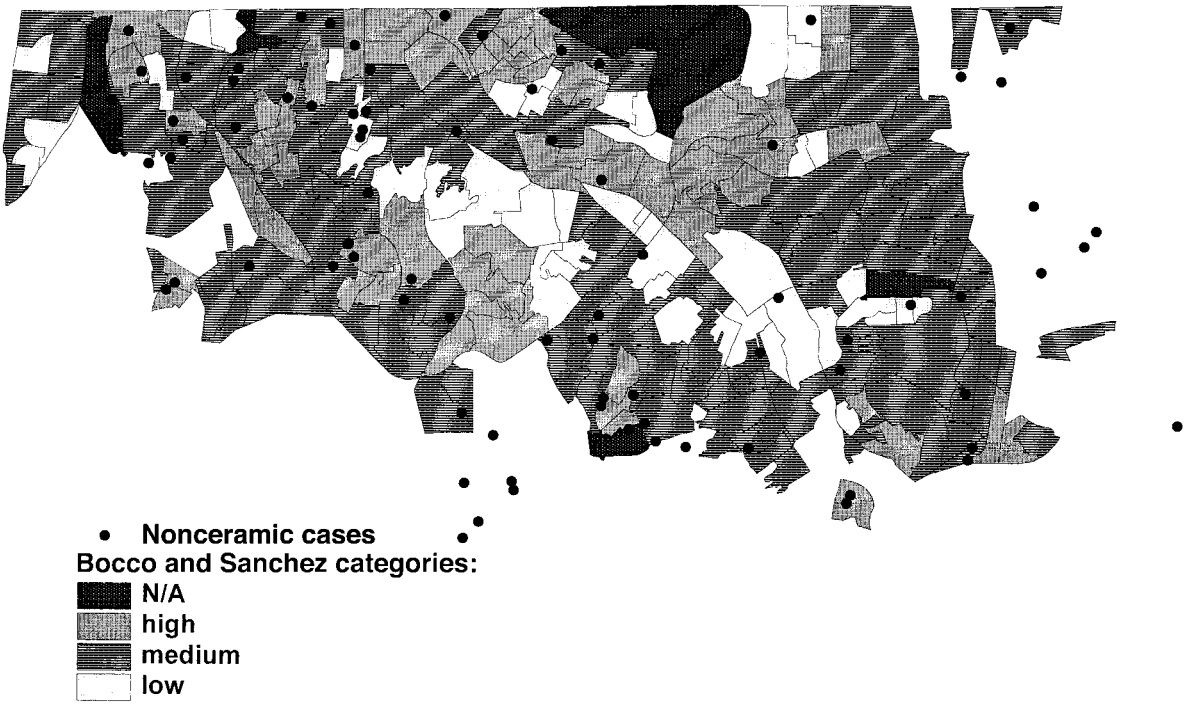

Figure 3. Comparison of risk areas between Bocco and Sanchez model and UCI study data.

Table 2. Expected vs observed cases: Bocco and Sanchez model and UCI study data

Risk class	No. non-ceramic-using subjects sampled	Bocco and Sanchez model: No. cases expected	UCI study: No. cases observed
Low	158	10	9
Medium	563	36	36
High	267	17	18

sessed the risk to lead exposure as directly proportional to the radial distance from purported lead point sources. Hence, industrial lead sources sited near census tracts would result in a population with greater vulnerability to lead exposure than those located at a greater distance from the site. Census tracts located at a greater distance from point sources would receive minimal contamination because they were assumed to be buffered from lead exposure.

The Bocco and Sanchez model was recreated using a digitized census map obtained from the Instituto de Estadistica, Geografia e Informatica (INEGI), identical to that used in the model. A geographic information system (GIS) database was used in identifying point source locations. Census tracts were organized based on Bocco and Sanchez' (1997) classification, and labels (high, medium, low, or N/A) were assigned to each tract, as depicted in Figure 3, which also depicts the distribution of nonceramic-using subjects with a blood lead level $\geq$10μg/dL.

Analysis and Results

The Bocco and Sanchez model of potential risk for lead exposure based on fixed point sources in Tijuana was strikingly accurate in predicting the distribution of cases within the designated risk areas, as observed in the UCI field and epidemiological studies. Comparison was made between predicted risk and observed frequency of elevated blood lead in subjects. To remove the principal confounding variable in this study, only the subjects with a blood lead level $\geq$10 μg/dL who reported that they did not use lead-glazed ceramics for cooking or storing food (n = 63) were considered in

the comparison, and subjects considered in this analysis were those residing within the census tract boundaries of the Bocco and Sanchez model ($n = 988$). These 63 cases represented 6.4% of the overall sample, which reported no use of lead-glazed ceramics. The distribution of cases per risk area and frequency of cases based on total number of subjects per area were examined (Table 2). Chi-square analysis of expected vs observed values did not significantly differ at the $p = 0.02$ level. The census tracts which were expected to pose a high risk for lead exposure were observed to have the highest percentage of cases (13.7%) after adjusting for subjects sampled per risk group. The 63 cases classified as lead poisoned accounted for 6.4% of the sampled population not exposed to lead-glazed ceramics. This proportion was approximately equal to the 6.6% of cases found in the medium-risk group. Only 2.5% of those sampled in the low-risk areas were classified as cases. Distribution of cases in the low-, medium-, and high-risk areas adjusted for subjects sampled per risk category was 2:5:11, respectively. Without further investigation, it is not possible to state the source of the discrepancies between the model's purported lead point sources and the sites that were available to sample in the field. In terms of the UCI field study, sources that could not be validated or sampled may have been inaccessible for monitoring, no longer in operation, moved to another site, or misrepresented as producers or emitters of lead.

The results of the Bocco/Sanchez analysis state that fixed lead sources contribute locally to exposure of the population, estimated to be 14,116 exposed inhabitants (defined as "children, fertile women, and low-income groups") (Bocco and Sanchez 1997). This technique appears to underestimate the magnitude of childhood lead poisoning by over 60% compared to values estimated by the UCI Principal Investigators to be 9.3% of 400,000 children, ages 2–6, or 37,200 children including ceramic users (Ericson and Baker 1999). Researchers Bocco and Sanchez did not control for the use of lead-glazed ceramics or other potential confounding sources. Limiting the estimate to children not affected by the principal confounder (ceramic use) in this study, there are a predicted 20,440 non-ceramic users with a blood lead level $\geq 10 \mu g/dL$ in Tijuana. Thus, the risk from soil lead may be far more significant and widespread in the urban environment than accounted for by the point sources identified in the model.

Conclusions

In this study, an environmental model (Bocco and Sanchez 1997) was shown to be reliable in terms of predicting areas of low, medium and high risk to lead exposure. The underrepresentation in prediction of the number of lead-poisoned children may be due to unreliable information as to point source emitters of lead, a flaw in the model's assumption that risk is directly related to radial distance from a point source, or the omission of other significant sources of environmental lead in an urban environment. The results of the blood lead analyses reveal that many case management subjects who are not exposed to lead-glazed ceramics also do not live in proximity to a point source. We conclude that although point sources of lead remain a highly significant variable, they are not sufficient in terms of predicting the risk of exposure to lead in the urban environment. The risk for environmental lead exposure is not evenly distributed throughout Tijuana. Although industrial emissions (represented as fixed point sources of lead) represent local contamination problems, children in Tijuana are exposed to lead in soil from a variety of environmental sources, including soil lead residue from combustion of leaded gasoline. Nonetheless, the Bocco and Sanchez model was strikingly accurate in predicting the spatial distribution of subjects with blood lead levels $\geq 10 \mu g/dL$. Urban populations, particularly urban poor, will continue to be exposed to this significant source. We have known for some time that the effects of ubiquitous lead exposure on the health of urban populations remains a long-term problem. Thus, the task of limiting exposure to soil lead sources becomes more than just an end in itself. The foregoing study represents an iterative approach to prioritizing environmental health screening. The accuracy and usefulness of modeling relative risk to environmental lead at the census tract level have been demonstrated (Bocco and Sanchez 1997); linking environmental concentrations with blood lead results has also been shown to reveal a more accurate determination of potential risk to lead exposure in an urban environment.

Acknowledgments

We thank Haiou Yang, Ph.D., and Stacey Kojaku for project support. We are grateful to the student volunteers of E164L (Spring 1995), and E100 for field assistance. This research was supported by USEPA collaborative agreement CR824353-01-0 (Ericson), UC MEXUS, UCI and Global Peace and Conflict Studies, and UCI Center for Occupational and Environmental Health.

Literature Cited

Bocco, G., and R. Sanchez. 1997. Identifying potential impact of lead contamination using a geographic information system. *Environmental Management* 21(1):133–138.

Davis, A., R. V. Michael, and P. D. Bergstrom. 1992. Bioavailability of arsenic and lead in soils from the Butte, Montana, mining district. *Environmental Science and Technology* 26(3): 461–468.

Elhelu, M. A., and others. 1995. Lead in inner-city soil and its possible contribution to children's blood lead. *Archives of Environmental Health* 50:2.

Ericson, J. E., and D. B. Baker. 1999. Childhood lead assessment in Tijuana, Baja California, Mexico. Report to the U.S. Environmental Protection Agency, U.S. Centers for Disease Control and Prevention, XVI Ayuntamiento of City of Tijuana.

Ericson, J. E., and S. I. Mishra. 1990. Soil lead concentrations and prevalence of hyperactive behavior among school children in Ottawa, Canada. *Environmental International* 16:247–256.

Ericson, J. E., H. Shirahata, and C. C. Patterson. 1979. Skeletal concentrations of lead in ancient Peruvians. *New England Journal of Medicine* 300:946–951.

EPA. 1993. Report of the NHANESII, EPA. Research Triangle Park, NC.

Goyer, R. A. 1993. Lead toxicity: Current concerns. *Environmental Health Perspectives* 100:177–187.

Goyer, R. A. 1996. Results of lead research: Prenatal exposure and neurological consequences. *Environmental Health Perspectives* 104(10):1050–1054.

Gulson, B. L., C. W. Jameson, K. R. Mahaffey, K. J. Mizon, N. Patison, A. J. Law, M. J. Korsch, and M. A. Salter. 1998. Relationships of lead in breast milk to lead in blood, urine, and diet of the infant and mother. *Environmental Health Perspectives* 106.

Hemphill, D. H. 1990. *In* D. H. Hemphill and C. R. Cothern (eds.) Trace substances in environmental health. XXIV. Proceedings of a conference held in Cincinnati, Ohio, USA, 8–12 July, 324 pp.

Instituto de Estadistica, Geografia e Informatica (INEGI). 1994. Population statistics for Tijuana. Mexicali, B.C., Mexico.

Lansdown, R., and W. Yule. Lead toxicity. Johns Hopkins University Press, Baltimore, MD.

Mahaffey, K. R. 1998. Predicting blood lead concentrations from lead in environmental media. *Environmental Health Perspectives* 106:Suppl. 6.

Mielke, H. W., J. C. Anderson, K. J. Berry, P. W. Mielke, Jr., and R. L. Chaney. 1983. Lead concentrations in inner city soils as a factor in the child lead problem. *American Journal of Public Health* 73:1366–1369.

Mielke, H. W., D. Dugas, P. W. Mielke, K. S. Smith, S. L. Smith, and C. R. Gonzales. 1997. Associations between soil lead and childhood blood lead in urban New Orleans and rural Lafourche parish of Louisiana. *Environmental Health Perspectives* 105:950–954.

Ng, A., and C. C. Patterson. 1981. Natural concentrations of lead in ancient arctic and antarctic ice. *Geochemical Cosmochimica Acta* 45:2109–2121.

Pirkle, J. L., R. B. Kaufmann, D. J. Brody, T. Hickman, E. W. Gunter, and D. C. Paschal. 1998. Exposure of the U.S. population to lead, 1991–1994. *Environmental Health Perspectives* 106(11):745–750.

Reagan, P. L., and E. K. Silbergeld. 1989. Establishing a health-based standard for lead in residential soils, Vol. 50:2. Lead Coalition, St. Paul, MN.

Rothenberg, S. J., S. Karchmer, L. Schnaas, E. Perroni, F. Zea, and J. F. Alba. 1995. Changes in serial blood lead levels during pregnancy. *Environmental Health Perspectives* 102:876–880.

Settle, D., and C. C. Patterson. 1980. Lead in albacore: Quick source to lead pollution in Americans. *Science* 207:1167–1176.

Steele, M. J., B. D. Beck, B. L. Murphy, and H. S. Strauss. 1990. Assessing the contribution from lead in mining wastes to blood lead. *Regulatory Toxicology and Pharmacology* 11:158–190.