



Lead Poisoning Among Pregnant Women in New York City: Risk Factors and Screening Practices

Susan Klitzman, Anu Sharma, Leze Nicaj,
Ramona Vitkevich, and Jessica Leighton

ABSTRACT *This article presents information on pregnant women with incident blood lead levels (BLLs) of 20 µg per deciliter or greater as reported to the New York City Department of Health between September 1996 and June 1999 (n = 33). Almost half of the women were diagnosed during their third trimester of pregnancy, often at their first prenatal visit. The median BLLs at incidence and at last report among women who were retested were 25 and 15 µg/dL, respectively, a 40% decline. The median incident BLL among newborns (n = 25) was 12 µg/dL. The BLLs were inversely associated with maternal age and length of time in the United States and directly associated with gestational age and pica behavior. Cases were more than twice as likely to be foreign-born women than all women who gave birth in New York City. Prenatal care facilities employing a policy of universal blood lead testing of all pregnant women at the time of their first visit reported disproportionate numbers of cases, accounting for 77% of cases yet only 11% of all births citywide. The findings suggest that (1) the promulgation of recent rules and guidelines for lead risk assessment and screening among pregnant women appears to have been effective in identifying cases that might not have otherwise come to light; (2) case management and environmental interventions were initiated promptly; (3) cases experienced, on average, significant BLL reductions over time; and (4) there is a need for additional public health interventions for pregnant women in urban, multicultural centers. While the data suggest that universal screening may increase case finding among high-risk, immigrant populations, further studies and surveillance are needed to determine systematically the most effective approach.*

INTRODUCTION

Among the most significant environmental health achievements of the last quarter century has been a major reduction in the blood lead levels (BLLs) of both children and adults in the United States. Population geometric mean BLLs declined over 80%—from 12.8 µg/dL to 2.3 µg/dL in the period between the late 1970s and early 1990s.¹ More recent blood lead surveillance data from 1999 indicate that the declines are continuing.² These reductions have been attributed to regulatory bans

All authors were affiliated with the New York City Department of Health at the time of their work on this project. Dr. Klitzman is Associate Professor at Hunter College in the City University of New York, Urban Health Program; Dr. Sharma is with North Central Bronx Hospital, Bronx, New York. Ms. Nicaj is with Breathnet, Inc., New York, New York.

Correspondence: Susan Klitzman, DrPH, Associate Professor, Hunter College in the City University of New York, Urban Public Health Program, 425 East 25th Street, New York, NY 10010. (E-mail: sklitzma@hunter.cuny.edu)

on lead in gasoline, solder from canned food, and interior house paints, all of which have served to reduce environmental lead levels in soil and air.¹

Despite overall population declines in BLLs, lead exposure continues to be a public health concern for at least two reasons. First, studies show toxic effects of lead at lower and lower levels.^{3,4} Second, despite overall population improvements, certain subgroups continue to be at risk for high-dose lead exposure and its toxic effects, including: occupationally exposed adults, preschool-aged children, pregnant women and their fetuses, persons who live near sites where lead has been improperly disposed, and persons who intentionally or unintentionally ingest or inhale lead-contaminated food or other products.⁵ In the past quarter century, lead poisoning prevention efforts have focused primarily on controlling and monitoring exposures relevant to the first two groups: occupationally exposed adults^{6,7} and young children living in homes with dust and paint chips from deteriorated lead-based paint.⁸

In the case of pregnant women and fetuses, it has been well established that lead causes decreased fertility^{9,10} and readily crosses the placental barrier, causing spontaneous abortion, stillbirth,¹¹⁻¹³ preterm delivery, and low birth weight.^{10,14} The current risk of lead exposure among pregnant women, however, is not well characterized. One frequently cited national statistic, based on an extrapolation of data from the late 1970s, estimated that, in the late 1980s, 11% of pregnant women had BLLs of 10 µg/dL and higher and 0.4% had BLLs of 20 µg/dL and higher.¹⁵ These estimates are not applicable today, however, since (as already noted) population BLLs have declined substantially during the intervening years.¹ Blood lead data collected from pregnant women in St. Louis, Missouri, in the late 1980s found that 27% and 6% had BLLs of 10 and 15 µg/dL or higher, respectively.¹⁶ Blood lead surveillance of women of childbearing age (18–45 years) in New York State, based on 1996 data, found that 2% of all women tested had blood lead levels of 10 µg/dL or higher; among those with BLLs of 10–24 µg/dL, 35% were pregnant.¹⁷ Relatively little is known, however, about the current prevalence, risk factors for, or sources of lead poisoning among pregnant women.

In 1993, comprehensive lead control regulations were promulgated in the state of New York, pursuant to the Lead Hazard Control Act of 1992. These regulations addressed, among other things, lead risk assessment and screening of pregnant women and universal reporting of blood lead test results.¹⁸ Pursuant to these regulations, in 1995, the New York State Department of Health (NYSDOH) and the American College of Obstetricians and Gynecologists (ACOG) District II, developed the *Lead Poisoning Prevention Guidelines for Prenatal Care Providers*.¹⁹ These guidelines directed health care providers to (1) provide anticipatory guidance on preventing lead poisoning during pregnancy; (2) conduct a risk assessment for current high-dose lead exposure at the initial prenatal visit and perform a blood lead test for those women found to be at current risk; (3) provide additional risk reduction counseling for women found to have a blood lead level of 10 µg/dL or higher (there are no clear guidelines regarding medical treatment for women with elevated lead levels during pregnancy); and (4) advise women at the postpartum visit about childhood lead poisoning prevention. These rules and guidelines in New York State provided an opportunity to learn more about the extent and nature of lead poisoning among pregnant women in New York City.

In September 1996, a 24-year-old woman (estimated gestational age [EGA] = 37 weeks) was reported to the New York City Department of Health (DOH) with a BLL of 53 µg/dL and a free erythrocyte protoporphyrin (FEP) level of 166 µg/dL.

She reported ingesting approximately a quart of dirt daily from her backyard for the past 3 months. Following this report, DOH initiated a surveillance program for women of childbearing age with newly identified BLLs of 20 µg/dL or higher. This report describes the surveillance and follow-up of incident cases of lead poisoning among pregnant women reported to DOH from September 1996 through June 1999.

METHODS

Data Collection

New York State regulations direct laboratories to report the results of all blood lead test results to state and local health authorities within 5 business days of the analysis.¹⁸ From these reports, blood lead test results on all women 15 years old and older were reviewed prospectively for the period from September 1996 through June 1999. For each female with a newly reported BLL of 20 µg/dL or higher (the definition of an incident case), follow-up was initiated within 1 business day for BLLs of 45 µg/dL and higher and within 1 business day for BLLs of 20–44 µg/dL. Pregnancy status was ascertained either from the patient or from her prenatal care (PNC) provider. Pregnant women were contacted to schedule an interview and home visit. Each woman was interviewed in her primary language regarding her work, reproductive, and personal and family lead exposure history. The home visit included an interview and visual inspection of possible sources of lead exposure. A colorimetric swab test was used to assess qualitatively the presence of lead in glazes used in ceramic ware. Consumable items, which were either reported or suspected to contain lead, were pulverized when necessary and sent to a laboratory for analysis.

At the time of the interview, each woman was counseled on the health effects of lead exposure to adults, fetuses, and children; possible ways to reduce exposure; proper nutrition; and the importance of seeking follow-up medical care for lead poisoning, prenatal care, and blood lead testing of children living in the household. The women were advised not to use or consume items known or thought to contain lead. Results of environmental sampling were provided to the women. PNC providers were also contacted to formulate a case management plan, to supply them with a copy of the NYSDOH/ACOG guidelines, and to ascertain their policies and practices concerning lead risk assessment and testing of pregnant woman. All subsequent blood lead testing was conducted by the PNC provider as part of the woman's follow-up care.

The DOH blood lead registry of all children tested and the registry of adults with elevated BLLs were monitored for subsequent blood lead tests on the woman and her baby. In addition, each of the reporting facilities was asked to notify DOH of the infant's date of birth and blood lead test results.

Data Analysis

Data obtained through the blood lead registry, interviews, and environmental sampling were entered onto a Microsoft Excel 2000 database. Data were analyzed to characterize maternal and fetal demographic characteristics, BLLs, and possible exposure sources. A paired *t* test was used to compare initial and subsequent blood lead tests for both mothers and newborns.

RESULTS

Demographic Characteristics

Thirty-three pregnant women with BLLs of 20 µg/dL or higher (including the index case) were reported to DOH from September 25, 1996, to June 30, 1999. The demographic characteristics of the population are summarized in Table 1. The me-

TABLE 1. Pregnant women with blood-lead levels ≥ 20 µg/dL, New York City, September 1996–June 1999

	Number	Percentage
Age		
Under 20	3	9
20–24	15	45
25–29	9	27
30–34	3	9
35–39	1	3
≥ 40	2	6
Mean 25 years		
Median 24 years		
Range 15–43 years		
Nationality (country of birth)		
Mexican	19	57
American (US born)	3	9
Belizean	2	6
Jamaican	2	6
Pakistani	2	6
Other, unknown*	5	15
Years in United States among foreign-born women: median = 3.5 years, range 1 month–20 years		
Borough of residence		
Bronx	16	48
Brooklyn	8	24
Queens	6	18
Manhattan	3	9
Staten Island	0	0
Year of diagnosis		
1996 (September–December)	4	12
1997	6	18
1998	20	60
1999 (January–June)	3	9
Estimated gestational age at diagnosis (in weeks)		
1st	6	18
2nd	11	33
3rd	14	42
Unknown	2	6
Median 25.4 weeks		
Range 6–39 weeks		

*Other includes one woman each from Bangladesh, Dominican Republic, Haiti, and Guyana.

dian age at initial diagnosis was 24 years (range 15 to 43 years). Of the individuals, 90% were foreign born, with the majority from Mexico (57%). The median length of time in the United States among the foreign born was 6 years (range 1 month to 20 years). The median EGA at diagnosis was 25 weeks; 42% were diagnosed during the third trimester of pregnancy.

Blood Lead Levels

Incident and follow up blood lead test results on pregnant women are summarized in Table 2. The median incident BLL of all women was 26 $\mu\text{g/dL}$, with two thirds in the 20–29 $\mu\text{g/dL}$ range. The three women with the highest BLLs (53, 56, and 130 $\mu\text{g/dL}$) received chelation therapy. Two (including the index case) were chelated during their pregnancies; the third woman and her baby were chelated immediately following delivery.

The number of follow-up blood lead tests and the time interval between tests varied widely among the women. There were 28 women (85%) who had a second blood lead test (median number of days between the first and second tests was 29). Among the 25 of these women who did not receive chelation therapy, 22 (88%) experienced a decline between the time of the incident and first follow-up blood lead tests. Almost half these follow-up BLLs were below the case definition of 20 $\mu\text{g/dL}$. The average decline was 7 $\mu\text{g/dL}$, from 28 to 21 $\mu\text{g/dL}$ ($P < .001$), a 25%

TABLE 2. Incident (≥ 20 $\mu\text{g/dL}$) and follow-up blood lead levels (BLLs) among pregnant women, New York City, 1996–1999

Blood lead levels, $\mu\text{g/dL}$	Blood lead test			
	All women, Test 1 (n = 33)	Women with second lead test and not chelated (n = 25)*		
		Test 1	Test 2†	Last test‡
<20	—	—	13	18
20–39	27	22	9	7
≥ 40	6	3	3	0
Median	26	25	19	15
Mean	32	28	21	16
Range	20–130	20–51	5–43	5–49
Median number of days since last test	—	—	29 days	65 days
Range	—	—	3–267 (First–second)	12–777§ (First–last)
Mean change since first BLL	—	—	7 $\mu\text{g/dL}$ $t = 5.58$ $P < .001$	12 $\mu\text{g/dL}$ $t = 9.9$ $P < .001$

*Excludes three women who received chelation therapy with CaEdTa; two who had miscarriages, and three others for whom no follow up BLLs were reported.

†No subsequent test results were reported for 9 of the 13 women with BLLs <20 $\mu\text{g/dL}$ at the time of the second test.

‡Includes all women who had two or more blood lead tests. The last test represents the second test for 14 women, the third test for 1 woman, and the fourth or more test for 10 women.

§Includes one woman who had two pregnancies during the study period; altogether, her blood lead levels were monitored for 2.1 years.

reduction. Overall, among the 28 women who received at least two blood lead tests, 71% had BLLs below 20 µg/dL at the time of their last reported blood lead test. There was some tendency for the last reported BLL to be lower and to show a greater decline among women whose last reported test occurred after delivery compared with those whose last test was prior to or at the time of delivery (median BLL = 10 and 18 µg/dL; median Δ = 16 and 10 µg/dL, respectively).

Blood lead test results on the newborns ($n = 25$) are summarized in Table 3. Among the 22 unchelated newborns, the median initial BLL was 16 µg/dL (range 6–51 µg/dL). Almost half had BLLs under 10 µg/dL. There were 10 newborns (45% of those tested) who received a second blood lead test; the average decline from the first test was 6 µg/dL, a 28% reduction ($P < .01$). The average decline between the first and last test was 11 µg/dL or 53% ($P < .01$).

Possible Sources of Exposure

Interviews and/or home inspections were completed for 32 cases (97%). Exposure sources are summarized in Table 4. A confirmed or possible current lead exposure was identified in 25 cases; another 4 women reported a history, but no current, lead exposure. All of the current sources were nonoccupational. Four women reported more than one possible source of exposure.

Pica There were 13 women (39%) who reported consuming nonfood items (pica) during their current pregnancies, including the 3 women with the highest BLLs. In

TABLE 3. Distribution of blood lead levels (BLLs) among newborns born to pregnant women with blood lead levels ≥ 20 µg/dL, New York City, 1996–1999

Blood lead levels, µg/dL	Blood Lead Test			
	All newborns ($n = 25$),*	Newborns with second lead test and not chelated†		
		(n = 10)		
	Test 1	Test 1	Test 2	Last test‡
<20	18	6	7	8
20–39	4	3	2	2
≥ 40	3	1	1	0
Median	12	16	9	8
Mean	20	21	15	10
Range	3–51	6–51	1–43	1–23
Median number since last test	—	—	143	173
Range	—	—	1–343	24–542
			(First–second)	(First–last)
Mean change since first BLL	—	—	6 µg/dL	11 µg/dL
			$t = 2.8$	$t = 3.1$
			$P < .01$	$P < .01$

*Birth outcome and/or newborns' BLL were not reported for 6 of 33 cases; 2 cases were known to have resulted in miscarriage.

†Excludes 3 newborns who underwent chelation therapy and 12 whose initial BLLs were <20 µg/dL.

‡Includes all newborns who had two or more blood lead tests. The last test represents the second test for five newborns, the third test for four newborns, and the fourth or more test for one newborn.

TABLE 4. Possible and confirmed sources of lead exposure among pregnant women with blood lead levels ≥ 20 $\mu\text{g}/\text{dL}$

Source	Reported number*	Suspect material tested	Method†	Test results
Pica	13	6		
Soil, dirt, clay	10	5	AA	Range 96–1000 ppm
Pottery	2	1	AA	223,000 ppm
Soap	1	0		
Imported pottery use	7	6	Lead check	All +; 1 leachate test = 7 $\mu\text{g}/\text{g}$
Imported spices, tea, food	8	8	AA	Range 1.48–1084 ppm
Vitamins, supplements	3	3	AA	Range 0.44–35 ppm
History of exposure‡	4	—		
Unknown	4	—		

*Includes three women who reported more than one possible source of lead exposure.

†AA, atomic absorption/mass spectrophotometry; lead check = colorimetric lead swab test.

‡Includes history of working in a ceramics factory, soldering, use of ceramic ware, and pica.

addition, 3 women reported a history of pica, but denied current pica behavior. Among the 13 women reporting current pica behavior, 8 claimed to have eaten dirt; 2 reported pulverizing and eating pottery; 2 ate both dirt and pulverized pottery. None reported eating paint chips in their homes. In addition, 1 claimed to eat “soap.” (It is not clear whether this could have been her source of lead exposure since the item was not available for testing.) Soil samples were collected and analyzed from 5 persons reporting consumption of dirt. Results ranged from 96 to 1,000 parts per million (ppm). (In the remaining 3 cases reporting dirt consumption, soil samples could not be collected because either the location was not identified or could not be accessed.) The woman with the highest BLL (130 $\mu\text{g}/\text{dL}$, FEP 25 $\mu\text{g}/\text{dL}$) claimed to have eaten half of a glazed Mexican bowl the day before she was tested for lead. The bowl was pulverized, and the lead content was analyzed and found to contain 223,000 ppm of lead.

Imported Pottery Use Seven women (21%) reported using imported pottery for cooking. Six pieces of ceramic ware were available, and all tested positive for lead using a colorimetric swab test. In addition, one woman reported that she had been a ceramic factory worker in Mexico when she was between the ages of 10 and 19 years old.

Imported Spices, Tea, and Food Eight women (24%) reported consuming imported spices, tea, and/or food. Samples of available material were collected from the homes of all eight women and analyzed for lead content. Spice samples from the homes of two women were found to have maximum levels of 189 and 1,084 ppm, respectively. In the six remaining cases, only lead levels ranged from 1.6 to 14 ppm. The woman who used spices with a maximum lead content of 1,084 ppm had emigrated to the United States from Bangladesh 2 months earlier and had daily use of the spices she brought with her in cooking. (Her three other children, ages 9, 6, and 3 years old, were all found to have lead poisoning, with BLLs of 46 $\mu\text{g}/\text{dL}$

dL, 50 µg/dL, and 43 µg/dL, respectively.) A home inspection revealed lead-based paint hazards, and the family was brought to a lead-safe house.

Demographic and Exposure-Related Factors

Analysis of demographic and exposure-related factors according to BLL found that, on average, women with higher incident BLLs were slightly younger and more advanced in their pregnancies and reported living in the United States for a shorter period of time, although none of the differences were statistically significant. Women in the group with the higher BLL were more than four times as likely to report pica during their current pregnancies than women with lower BLLs ($P \leq .01$).

Reporting Facilities

Cases were reported from 14 different PNC facilities. Of these, 10 were also birthing facilities. Through telephone conversations, we were able to confirm that 7 of the 14 facilities conducted routine blood lead screening of all their pregnant patients, generally as part of the first PNC visit. Prenatal/birthing facilities employing universal screening policies accounted for over three fourths of the cases (77%), yet accounted for only about 11% of the total births occurring in New York City during the years covered by the study period. Less than one quarter of the cases (23%) were reported by facilities employing targeted screening policies; these facilities also accounted for about 11% of total births.²⁰

Estimated Incidence Rates

To estimate the incidence rate of lead poisoning, the total number of women tested at facilities employing a policy of universal testing of pregnant women was requested from NYSDOH. Data were available from two facilities (two of the three with the largest number of reported cases: 6 cases each). The total number of women between the ages of 18 and 45 years who were tested at these facilities between January 1, 1997, and December 31, 1998, was ascertained. The number of incident cases reported during the same period was divided by the total number of women tested to obtain an incidence rate. At one facility, the incidence rate was 6 of 2,645 or 2.3 per 1,000 pregnant women tested; at the second facility, the incidence was 6 of 5,102 or 1.2 per 1,000 pregnant women tested.

To estimate citywide rates of reported lead poisoning among pregnant women during these years, the number of reported cases was divided by the number of live births and reported spontaneous abortions. (Induced terminations of pregnancy were excluded from these calculations because cases tended to be diagnosed during the late second or early third trimester of pregnancy, beyond the period when induced abortions occur.) The incidence rates of lead poisoning among pregnant women living in New York City for these years was 20 of 227,855 or 8.8 per 100,000—more than two orders of magnitude lower than the incidence rates derived from the facilities in which universal screening was performed. (Altogether, 89% of pregnancies among New York City women resulted in live births, and the remaining 11% resulted in spontaneous abortions, which is similar to the percentage in this cohort.²¹)

DISCUSSION

This report documents that lead poisoning among pregnant women in urban, multi-ethnic settings is a significant problem. Among the cases reported here, the most important risk factors were pica, use of imported pottery, and immigration. Over

one third of the cases reported ingestion of nonfood items—predominantly geophagia (compulsive eating of dirt or clay). Measurable quantities of lead in soil, pottery, spices, and other items, if available, were documented. Pica during pregnancy has been previously documented in both rural and urban parts of the United States, with one study of Mexican American women living in California reporting a 31% prevalence.^{22,23} The specific risk factors identified here are consistent with a recent study of predominantly Latino pregnant women in South Central Los Angeles, which found that pica and recent immigration to the United States (as well as low calcium intake) were the strongest predictors of BLLs.²⁴

There was also some tendency for higher BLLs to be associated with younger maternal age, more advanced EGA, and more recent immigration to the United States. It is possible that these trends would have reached statistical significance if extended to a larger population. Interestingly, none of our cases reported current occupational lead exposure, which accounts for 90%–95% of adult lead poisoning in the United States.²⁵ The women involved in the cases were almost twice as likely to be foreign born compared with all women who gave birth in New York City in 1997 and 1998 (90% vs. 48%).²⁰

It can be hypothesized that “immigrant status” is a risk factor for lead poisoning among pregnant women in at least three ways: First, immigrant pregnant women from countries where lead exposure is endemic may carry high cumulative body lead burdens. Among the cases described here (as in the United States as a whole),²⁶ the country with the single largest number of immigrants was Mexico, where high endemic levels of lead exposure in the general population and among pregnant women have been reported,²⁷ especially in comparison with their American counterparts.^{28,29}

Several women reported a history of lead exposure in their country of origin and were without current exposures. Lead can accumulate in the bone. Significant skeletal mobilization of lead can occur during pregnancy and lactation, and endogenous lead released from bone can be a major contributor to BLLs,³⁰ especially during the last half of pregnancy.³¹ The implications of historical lead exposure for the cases reported here remains purely speculative, however, since the degree to which such exposures (e.g., from bone and soft tissue) were reflected in the BLLs could not be assessed.

Second, immigrants may “transport” lead-containing products and cultural and behavioral practices with them when they move to the United States from their countries of origin. Among the cases analyzed here, almost half the women reported current use of imported items containing lead, such as pottery, spices, food, and vitamin supplements. The use of imported glazed ceramic ware from Mexico, in particular, has been well documented as a source of significant lead exposure.^{32,33} These data also suggest that other imported items—such as spices, foods, and vitamin supplements—may contain significant amounts of lead.

Third, recent immigrants may face cultural, linguistic, economic, and legal barriers to early prenatal care and, as a consequence, delayed identification and management of lead poisoning. The ACOG/NYSDOH guidelines are aimed at primary prevention of childhood lead poisoning: By identifying elevated BLLs among mothers-to-be early in their pregnancies and reducing sources of exposure as quickly as possible, maternal-fetal transmission of lead and its adverse sequelae can be minimized. Yet, among the group of lead-exposed women reported here, it is clear that screening failed to achieve its primary prevention aim. The median EGA at diagnosis was 25 weeks, and almost half the women were diagnosed during their third

trimester of pregnancy; well past the time when lead can cross the placenta. For many women, this was their first PNC visit. Although significant reductions in maternal BLLs occurred once cases were identified and newborn BLLs were well below incident maternal levels, significant maternal-fetal lead transmission may still have occurred in early pregnancy and may have continued to occur throughout pregnancy and lactation—periods of significant bone lead mobilization and transport.

Another striking pattern from these data is related to the apparent relationship between case identification and blood lead screening practices. Facilities employing universal blood lead testing policies reported disproportionate numbers of cases (77%) relative to total births (11%). Also, for the 2 years for which complete data were available, the incidence rate of lead poisoning among pregnant women tested at such facilities was about two to three orders of magnitude higher than in the general New York City population. This finding suggests that if a policy of universal testing had not been in place at those facilities, many cases might have been missed. It also raises the possibility that if additional facilities employed a policy of universal testing, the number of reported cases could have been substantially higher. Informal conversations with staff at several facilities revealed that, despite recommendations for universal risk assessment and targeted screening of women found to be at risk for current high-dose lead exposure, high case loads and time pressure, coupled with linguistic and cultural diversity, created significant barriers to administering and interpreting standard risk assessment questionnaires.

In spite of the important findings on current risk factors and sources of lead exposure among pregnant women in a multiethnic urban setting, the data contain several limitations and raise many questions that will require further research, surveillance, policy, and/or programmatic activities to address fully.

The incidence of elevated BLLs among pregnant women in New York City reported here underestimates the true population values. It is well documented^{4,5} that lead can cross the placental barrier and produce toxic effects at BLL levels below 20 $\mu\text{g}/\text{dL}$ —the case definition used here. It is expected that the incidence rate of pregnant women with would have been higher than reported if a lower case definition (say, 10 $\mu\text{g}/\text{dL}$ rather than 20 $\mu\text{g}/\text{dL}$) had been employed. Indeed, whenever the case definitions of “elevated BLLs” have been lowered, an increase in the number of reported cases has been observed among women¹⁷ and children.³⁴

While there is some evidence to suggest that, among a population of high-risk immigrant women, universal screening at the time of the first PNC was associated with increased case identification, firm conclusions about the most effective screening policy—universal versus targeted—cannot be drawn from these data. Information on screening practices was only collected from those facilities from which one or more cases were reported. Such facilities only accounted for about 20% of all birthing centers and 22% of all births.²⁰ To more conclusively determine the most effective approach to early identification of lead poisoning cases among pregnant women, especially among recent immigrants from countries where lead exposure is endemic, more comprehensive surveillance systems and epidemiologic studies are needed. In particular, information on screening practices, demographic characteristics of all women giving birth (cases and noncases), and screening rates and results at all PNC facilities (not just those from which cases were reported) must be collected and analyzed. Such data were not readily available at the time of this analysis. This would also help to assess incidence and prevalence rates more accurately.

Although the data show that BLLs in pregnant women and their offspring de-

clined markedly over time, it is impossible to calculate precisely the rate of decline or to measure the contributory causes. Because data were collected through passive surveillance of multiple institutions, the number and timing of blood lead testing varied widely between cases, thus making it difficult to measure precisely the rate of decline. Measures such as erythrocyte protoporphyrin and free erythrocyte protoporphyrin were not uniformly available, limiting our ability to assess lead toxicity and exposure duration.

Once cases were identified, the majority received environmental investigation/risk reduction education (97%) and follow-up care (85% returned to their providers for follow-up blood lead testing and care). But, the relative effectiveness of the various interventions, such as case management, lead hazard reduction and education, and continued pre- and postnatal care, cannot be measured here. Also, additional unmeasured factors may have contributed to the observed changes—such as calcium intake, release of bone lead stores during the postpartum and lactation periods, and regression to the mean.

In many instances, the sources and quantities of lead exposure could not be verified through environmental sampling—either because the reported sources were not available for testing or because the materials tested did not indicate high lead concentrations. Even when environmental samples were collected and analyzed, it was not possible to estimate total exposure. Moreover, established standards may not be directly applicable to the exposure sources and pathways identified here. For example, the recently established EPA soil lead standard of 400 ppm³⁵ applies to children's play areas—where toddlers might inadvertently get soil on their hands and ingest it through normal hand-to-mouth activity—not to intentional ingestion by adults.

In spite of the limitations noted here, there are several public health implications of these data. The promulgation of rules and guidelines for lead poisoning prevention and control among pregnant women have been useful in identifying cases that may not have otherwise come to light. At the same time, the data suggest that the recommended approach—anticipatory guidance and risk assessment—may not be as effective as universal screening among high-risk immigrant populations, although more comprehensive surveillance and epidemiologic studies are needed to evaluate and identify the most effective approach. Finally, there is a need for additional public health interventions for pregnant women in urban multicultural centers, including (1) developing outreach strategies to increase access to and utilization of early prenatal care; (2) developing culturally sensitive, linguistically appropriate education of patients and PNC providers around specific lead risk factors, most notably pica; (3) expansion of lead risk assessments to include imported items such as spices, food, and vitamin supplements.

REFERENCES

1. US Centers for Disease Control and Prevention. Update: blood lead levels—United States, 1991–1994. *MMWR Morb Mortal Wkly Rep.* 1997;46(7):141–146.
2. US Centers for Disease Control and Prevention. Blood lead levels in young children—United States and selected states, 1996–1999. *MMWR Morb Mortal Wkly Rep.* 2000; 49(50):1133–1137.
3. US Centers for Disease Control. *Preventing Lead Poisoning in Young Children.* Atlanta, GA: Agency for Toxic Substances and Disease Registry, US Dept of Health and Human Services, Public Health Service; 1991.

4. Lanphear BP, Dietrich K, Auinger P, Cox C. Cognitive deficits associated with blood lead concentrations <10 µg/dL in US children and adolescents. *Public Health Rep.* 2000; 115(6):521–529.
5. US Centers for Disease Control. Agency for Toxic Substances and Disease Registry. *Toxicological Profile for Lead (Update)*. Atlanta, GA: Agency for Toxic Substances and Disease Registry, US Dept of Health and Human Services, Public Health Service; July 1999.
6. US Department of Labor, Occupational Health, and Safety Administration. *Final Standard for Occupational Exposure to Lead*. Washington, DC: US Dept of Labor, Occupational Health, and Safety Administration; 1978. 29 CFR 1910.1025.
7. US Department of Labor, Occupational Safety, and Health Administration. *Lead in Construction, Interim Final Rule*. Washington, DC: US Dept of Labor, Occupational Health, and Safety Administration; 1993. 29 CFR 1926.62.
8. *Title X, Residential Lead Poisoning Control Act of 1992*. 42 USC §63.
9. Lancranjan I, Popescu HI, Gavanescu O, et al. Reproductive ability of workmen occupationally exposed to lead. *Arch Environ Health.* 1975;30(8):396–401.
10. Min YI, Correa-Villasenor A, Stewart PA. Parental occupational lead exposure and low birth weight. *Am J Ind Med.* 1996;30(5):569–578.
11. Nordstrom S, Beckman L, Nordensen I. Occupational and environmental risks in and around a smelter in northern Sweden: V. Spontaneous abortion among female employees and decreased birth weight in their offspring. *Hereditas.* 1979;90(2):291–296.
12. Baghurst PA, Robertson EF, Michael AJ, et al. The Port Pirie cohort study: lead effects on pregnancy outcome and early childhood development. *Neurotoxicology.* 1987;8(3): 395–401.
13. Hu H, Pepper L, Goldman R. Effect of repeated occupational exposure to lead, cessation of exposure and chelation on levels of lead in bone. *Am J Ind Med.* 1991;20(6):723–735.
14. McMichael AJ, Baghurst PA, Wigg NR, Vimpani GV, Robertson EF, Roberts RJ. Port Pirie cohort study: environmental exposure to lead and children's abilities at the age of 4 years. *N Engl J Med.* 1988;319(8):468–476.
15. Crocetti AF, Mushak P, Schwartz J. Determination of numbers of lead-exposed women of childbearing age and pregnant women: an integrated summary of a report to the US Congress on childhood lead poisoning. *Environ Health Perspect.* 1990;89:121–124.
16. Flanigan GD, Mayfield R, Blumenthal HT. Studies on lead exposure in patients of a neighborhood health center: Part II. A comparison of women of childbearing age and children. *J Natl Med Assoc.* 1992;84(1):23–27.
17. Fletcher AM, Gelberg KH, Marshall EG. Reasons for testing and exposure sources among women of childbearing age with moderate blood lead levels. *J Community Health.* 1999;24(3):215–227.
18. State of New York. *Lead Poisoning Prevention and Control*. Pub Hlth L 10 NYCCR §206(1)(n) Part 67. Available at: <http://www.health.state.ny.us/nysdoh/phforum/nycrr10.htm>. Accessed on January 10, 2001.
19. New York State Department of Health and American College of Obstetricians and Gynecologists District II. *Lead Poisoning Prevention Guidelines for Prenatal Care Providers*. Albany, NY: State of New York, Dept of Health; December 1995.
20. New York City Department of Health. Birth File 1996–1999 [computer file]. New York, NY: New York City Dept of Health, Office of Vital Statistics; 2001.
21. New York City Department of Health. *Summary of Vital Statistics 1997 and 1998* [database online]. 2000. Available at: <http://www.nyc.gov/html/doh/html/pub/pub.html>. Accessed on January 20, 2001.
22. Edwards CH, Johnson AA, Knight EM, et al. Pica in an urban environment. *J Nutr.* 1994;124(6 suppl):954S–962S.
23. Simpson E, Mull DJ, Longley E. Pica during pregnancy in low-income women born in Mexico. *West J Med.* 2000;173:20–24.

24. Rothenberg SJ, Manalo M, Jiang J, et al. Maternal blood lead level during pregnancy in South Central Los Angeles. *Arch Environ Health*. 1999;54(3):151–157.
25. US Centers for Disease Control and Prevention. Adult blood lead epidemiology and surveillance—United States, second and third quarters, 1998, and annual 1994–1997. *MMWR Morb Mortal Wkly Rep*. 1999;48(10):213–216, 223.
26. US Census Bureau. *Current Population Survey*. March 1997. Available at: <http://www.census.gov>. Accessed on January 13, 2001.
27. Rothenberg SJ, Darchmer S, Schnaas L, Perroni E, Zea F, Fernández Alba J. Changes in serial blood lead levels during pregnancy. *Environ Health Perspect*. 1994;102(10):151–60.
28. Hernandez-Avila M, Gonzalez-Cossio T, Palazuelos E, et al. Dietary and environmental determinants of blood and bone lead levels in lactating postpartum women living in Mexico City. *Environ Health Perspect*. 1996;104(10):1076–1082.
29. Hu H, Hashimoto D, Besser M. Levels of lead in blood and bone in women giving birth in a Boston hospital. *Arch Environ Health*. 1996;51:52–58.
30. Gulson BL, Jameson CW, Mahaffey KR, Mizon KJ, Korsch MJ, Vimpani G. Pregnancy increases mobilization of lead from maternal skeleton. *J Lab Clin Med*. 1997;130:51–62.
31. Hertz-Picciotto I, Schramm M, Watt-Morse M, Chantala K, Anderson J, Osterloh J. Patterns and determinants of blood lead during pregnancy. *Am J Epidemiol*. 2000;152(9):829–837.
32. Hernandez-Avila M, Romieu I, Rios C, Riverro A, Palazuelos E. Lead-glazed ceramics as a major determinant of blood lead levels in Mexican women. *Environ Health Perspect*. 1991;94:117–120.
33. Matte TD, Proops D, Palazuelos E, Graef J, Hernandez-Avila M. Acute high-dose lead exposure from beverage contaminated by traditional Mexican pottery. *Lancet*. 1994;344:1064–1065.
34. Decreasing childhood lead poisoning in New York City, 1970–1998. *J Urban Health*. 1999;76(4):545.
35. 40 CFR Part 745, Residential Lead Hazard Standards.