Demographic Risk Factors Associated with Elevated Lead Levels in Texas Children covered by Medicaid

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Despite its preventability, lead poisoning remains a common pediatric problem (1–3). Lead primarily affects three target organs in children: brain, kidney, and blood-forming organs (4). Lead is especially toxic for young children (4–8). Immature organs are most susceptible to damage at their time of most rapid growth, accounting for the observation that the brain is most vulnerable in the first 2 years of life (8). The blood–brain barrier takes 3 years to complete development; therefore, lead ingested by toddlers enters the central nervous system more readily (3–7). Furthermore, absorption and bioavailability of ingested lead is four times greater in children than in adults—40% versus 10%. Risk of lead ingestion is increased in young children by pica and normal hand-to-mouth exploratory behavior (5–9). In 1990, the EPA estimated that 3 million of the nation’s children under 6 years of age had blood lead levels >10 μg/dl, the level statistically associated with subsequent lower intellectual performance and other adverse health effects (10–14). Not only can lead-intoxicated children have impaired intelligence but they also are frequently overactive, aggressive, more distractible, disorganized, and less able to follow directions (15–17). Longitudinal studies of young children with high lead levels have shown lower class standing in the final year of high school, with increased absenteeism, lower vocabulary scores, and impaired motor function (8,18). The sources and pathways of lead exposure are well known so that serious preventive efforts intended to eradicate childhood lead toxicity are now under way (4–6,9,14,18).

Lead screening has been conducted throughout Texas since 1973 as part of the Early Periodic Screening, Diagnosis and Treatment (EPSDT) program. The national standard for acceptable blood lead concentrations was originally set at <40 μg/dl in 1973, and this standard was used to test Texas Medicaid recipients (19). In 1976, the Texas Department of Health laboratory changed its lead screening methodology to erythrocyte protoporphyrin (EP) testing coupled with blood lead analysis for detecting potential lead problems (20). Only children under 6 years of age were required to be tested; the level considered normal in 1976 was an EP level below 50 μg/dl, with a corresponding blood lead level below 30 μg/dl. In 1986, the threshold for a normal blood lead level was further lowered to 25 μg/dl, with a corresponding EP level of 35 μg/dl (21). In October 1991, the Centers for Disease Control and Prevention (CDC) set the current level for acceptable blood lead concentration at <10 μg/dl, a level believed not to be harmful to children (5). In October 1992, the Texas Department of Health Laboratories began using a graphite furnace atomic absorption spectrophotometer (GFAAS) analytic method, which allowed more accurate determination of blood lead concentrations at these lower levels (22).

The aim of this study was to determine demographic risk factors for high lead levels in Texas children tested during routine Medicaid screening, and it focused on gender, ethnicity, and age.

Design

The study population consisted of all the Texas children covered by Medicaid screened for blood lead for 6 months (1 January–30 June 1993). The specimens were collected by well-child clinics in local health departments and by office-based private physicians. Samples were either venous or capillary specimens, at the physician’s discretion. Program guidelines request that all follow-up specimens be venous in order to reduce the possibility of skin lead contamination, which may occur with poorly collected capillary specimens. Venous specimens for verification of initial high results were requested at 3-month intervals following therapeutic intervention. All specimens were analyzed by the Texas Department of Health (TDH) laboratory in Austin, Texas, using GFAAS methodology (22). Submitters received laboratory reports 7–10 days after specimen collection.

Age of subjects was imputed as the difference between date of birth and 30 June 1993, the last date of the 6-month collection period. The dates of specimen collection were not included in the magnetic data file made available for this study so exact ages of subjects were not available for this study.

Analysis was performed using SPSS (Statistical Package for Social Sciences; SPSS Inc., Chicago, IL). Specific hypothesis testing or parameter estimation was considered inappropriate for this exploratory study. Only descriptive statistics were used: frequency distributions, cross-tabulations, and percentiles/quantiles.

Results

During the first 6 months of 1993, TDH received 92,900 blood specimens for lead testing from Texas children covered by Medicaid who were at least 6 months of age. This total includes multiple blood specimens for children receiving repeat tests during this period. Capillary and venous specimens could not be examined separately because there was no coding of specimen type in the

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records reviewed. Of the 92,900 specimens collected, 98.9% were adequate for valid lead testing. Incomplete demographic data information affected 7,624 (8.2%) of the specimens with valid lead determinations.

Of the 91,783 valid lead determinations reviewed, 90.3% were not elevated, 6.1% were in the 10–14 μg/dl range, 1.4% were in the 15–19 μg/dl range, and only 0.9% were ≥20 μg/dl (see Fig. 1). Figures 1 and 2 show that elevated lead blood occurred mostly in children of 1–5 years of age (13–72 months). Children 25–36 months old had the highest rate of elevated levels (14.3%), followed by those 19–24 months old (13.0%) and 37–48 months old (12.0%).

Of the specimens with ethnicity recorded, 53.3% were Hispanic, 20.8% were white, and 20.0% were African American. The remainder (5.9%) comprised diverse ethnicities, each of which was too small a subgroup for meaningful statistical interpretation. African Americans had the highest prevalence rates in each age group (see Table 1). The highest prevalence rate was among African American children in the 2–4 year-old age group (17.3%); this was 3.5 times higher than that of the lowest prevalence group (whites over 4 years of age (4.9%).

Males constituted 48.9% of the children tested, and 48.1% were females. Gender was not given in 3% of the specimens submitted; these were excluded in the comparisons involving gender. This estimated sex ratio of 1.02 suggests no overall effect of gender on the likelihood of receiving lead screening. Males had a higher prevalence rate of elevated lead at all ages. The age-specific rate ratios resemble the sex ratios for elevated lead, with the overall sex ratio for all ages being 1.15. This indicates a 15% greater likelihood of an elevated lead level in males. In the youngest age group, 0–6 months, the sample size was too small, and perhaps not representative enough, to enable reliable interpretations. The remaining 12 older age groups suggest consistently increased risk in boys. The prevalence rate ratios ranged from 1.02 to 1.29 for boys in age groups under 4 years and 1.14 to 2.13 for boys in the older age groups. The average combined prevalence rate ratio for all young males under 48 months was 1.1 and for all males over 48 months was 1.4.

The highest blood lead concentration was 70 μg/dl, which occurred in an African American female in the 25–36 months age group. The characteristics of this case are those expected in terms of age and ethnicity, but this level was not typical in a female.

**Discussion**

This study was limited by the inability to identify: 1) duplicate specimens for individual children, 2) false positives or false negatives, and 3) remote past exposure or a lifetime peak exposure (this cross-sectional study of single blood specimens reflects predominantly recent exposure to lead). Nonetheless, this study suggests three risk factors for excessive lead blood in children: ethnicity (African American), gender (male), and age (13–72 months of age). Results did not show variation in prevalence rates of high blood lead by geographic location (not shown); the distribution of lead values appeared similar in residents of urban and rural counties of Texas. Because

![Figure 1](image1.png)

**Figure 1.** Percent of abnormal blood specimens from Texas children covered by Medicaid by age and lead concentration, January through June 1993. *Denotes the number of children in that category.*

| Table 1. Number of blood lead specimens and percent with elevated lead concentration (>10 μg/dl) by age and ethnicity* |
|---|---|---|---|---|---|---|
| Age (year) | White | | | | | |
| | No. Elevated, % | No. Elevated, % | No. Elevated, % | No. Elevated, % | | |
| <2 | 9,501 | 6.03 | 22,300 | 7.41 | 7,440 | 8.01 |
| 2–4 | 3,832 | 10.46 | 11,229 | 12.72 | 4,316 | 17.26 |
| >4 | 5,545 | 4.87 | 15,031 | 7.08 | 6,471 | 9.32 |
| Total | 18,878 | 6.59 | 48,560 | 8.53 | 18,227 | 10.67 |

*Excluded were 6,233 specimens with missing data for age and/or ethnicity; 1,002 specimens were excluded as being coded to ethnic groups other than white, African American, or Hispanic.

![Figure 2](image2.png)

**Figure 2.** Percent of blood lead specimens from Texas children covered by Medicaid by age group and ethnicity, January through June 1993.
the study population consisted of Texas Medicaid recipients who are necessarily near or below the federally defined poverty level, these findings cannot be generalized directly to the remainder of the childhood population in Texas. The data do not provide information on the prevalence of lead in children of affluent families.

Since the late 1970s, ongoing contamination of the U.S. environment by lead has been substantially reduced as major uses of lead in house paint, gasoline, water distribution systems, and food cans have been reduced or eliminated (5). Between 1976 and 1980, the second National Health and Nutrition Examination Survey (NHANES II) collected blood lead data from selected populations and from convenience samples, confirming a continued decline in blood lead levels (23-25). For the last half of the 1970s, that survey found that African Americans had a 12.2% prevalence rate compared to 2.0% in whites for blood lead levels over 30 μg/dl among children 6 months-5 years of age in the income group $6,000-$15,000/year and living in large urban areas (23-25). There were significant but complex associations between income, ethnicity, and lead concentration. There was a stronger inverse relation between income and lead levels in African Americans than in whites. About one-sixth (18.5%) of African American children from low-income households (less than $6,000/year) had blood lead concentrations high enough to qualify for medical follow-up (23-25).

Phase I of the NHANES III survey took place from October 1988 through October 1991 and showed a 78% decline in the estimated geometric mean in blood lead levels in the U.S. population (26-28). This decrease was similar across all age groups, leaving the cross-sectional age pattern virtually unchanged (26-28). The highest geometric mean was for children 1-2 years old (4.1 μg/dl) and the lowest was for those 12-19 years old (1.6 μg/dl). The prevalence of blood lead levels ≥10 μg/dl among children aged 1-5 years decreased substantially from 88.2% during NHANES II to 8.9% during NHANES III, Phase I. Prevalence of elevated blood lead levels continued to vary by race/ethnicity, income, and residence (26-28). Despite the decline in childhood lead exposure, approximately 1.7 million children aged 1-5 years still have blood lead concentrations at or above 10 μg/dl (26-28).

Results of this study of Texas children covered by Medicaid are consistent with the NHANES II and Phase I of the NHANES III results in identifying the role of ethnicity and income. The demographic pattern of elevated blood lead levels in Texas children covered by Medicaid probably reflects the distribution of the two major remaining environmental reservoirs of lead contamination: deteriorated indoor lead paint in older housing and urban soil and dust contaminated by past emissions of leaded gasoline and by exterior paint on structures (1,26). While it is not possible to arrive at valid incidence rates from the data due to the limitations previously described, the existence of clinically significant levels of lead exposure in this study group is undeniable. As many as 8% of the children tested may have lead levels in excess of 10 μg/dl and thus may be at risk for developmental problems. These data indicate the need for continued vigilance in order to minimize exposure to environmental sources of lead, especially in toddlers and preschoolers, and to evaluate the lead risk in Texas children in greater detail, the data-reporting system will need to identify duplicate results and whether the specimen is capillary or venous. Steps have been taken to accommodate these needs through recent data collection changes in the Texas EPSPD program. This study has also confirmed the need for expanded consumer education regarding the risks of increased blood lead concentrations. The need for expanded screening of young children is supported by these data. A risk assessment study in children not covered by Medicaid would be desirable.

REFERENCES

1. Bellingr D, Stiles K, Needleman H. Low-level lead exposure, intelligence and academic achievement: a long-term follow-up study. Pediatrics 90:855-860 (1992).
2. Landrigan P. Commentary: environmental disease—a preventable epidemic. Am J Public Health 82:941-943 (1992).
3. Mushak P. Perspective: defining lead as the premier environmental health issue for children in America: criteria and their quantitative application. Environ Res 59:281-309 (1992).
4. Committee on Measuring Lead in Critical Populations, National Academy of Sciences. Measuring lead exposure in infants, children, and other sensitive populations. Washington, DC:National Academy Press, 1993.
5. CDC. Preventing lead poisoning in young children: a statement by the centers for disease control. Atlanta, GA:Centers for Disease Control, 1975.
6. CDC. Preventing lead poisoning in young children: a statement by the centers for disease control. CDC publication no. 00-2629. Atlanta, GA:Centers for Disease Control, 1978.
7. CDC. Preventing lead poisoning in young children: a statement by the centers for disease control. CDC report no. 99-2230. Atlanta, GA:Centers for Disease Control, 1985.
8. Parsons PJ, Slavin W. A rapid Zeeman graphite atomic absorption spectrophotometric method for the determination of lead in blood. Spectrochim Acta 48B:925-939 (1993).
9. Mahaffey KR, Annest JL, Roberts J, Murphy RS. National estimates of blood lead levels: United States, 1976-1980. N Engl J Med 320:573-579 (1989).
10. Mahoney M. Four million children at risk: lead paint poisoning victims and the law. Stanford Environ Law J 9:46-83 (1990).
11. ATSDB. The nature and extent of lead poisoning in children: a report to Congress. Atlanta, GA:Agency for Toxic Substances and Disease Registry, 1988.
12. Centers for Disease Control. Lead levels—United States, 1988-1991. MMWR 41(30):545-548 (1994).
13. Brody DJ, Pirkle JL, Kramer RA, Flegal KM, Matte TD, Gunter EW, Paschal DC. Blood lead levels in the U.S. population. JAMA 272:277-283 (1994).
14. Pirkle JL, Brody DJ, Gunter EW, Kramer RA, Paschal DC, Finlay KM, Matte TD. The decline in blood lead levels in the United States. JAMA 272:284-291 (1994).
15. Lin-Fu J. Vulnerability of children to lead exposure and toxicity. N Engl J Med 289:1229-1233 (1973).
16. Bender S. Childhood lead poisoning. JAMA 269:1679-1681 (1993).
17. Centers for Disease Control. State activities for prevention of lead poisoning among children—United States, 1992. MMWR 42(19):165-172 (1993).
18. Berney B. Round and round it goes: the epidemiology of childhood lead poisoning, 1950-1990. Milbank Quarterly 71:3-39 (1993).
19. Needleman HL, Gunnoe C, Leviton A, Reed R, Pereisie H, Maher C, Barre HP. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. N Engl J Med 313:689-695 (1991).
20. Needleman HL, Garsonis C. Low-level lead exposure and the IQ of children: a meta-analysis of modern studies. JAMA 262:673-678 (1990).
21. Needleman HL, Schell A. The long-term effects of exposure to low doses of lead in childhood: an 11-year follow-up report. N Engl J Med 327:884-888 (1993).
22. Needleman HL. The current status of childhood lead toxicity. Advances Ped 40:125-139 (1993).
23. Needleman HL. Correction: lead and cognitive performance in children [letter]. N Engl J Med 326:673-678 (1990).
24. Mahaffey K. Exposure to lead in childhood. N Engl J Med 327:1308-1309 (1990).
25. CDC. Increase lead absorption and lead poisoning in young children: a statement from the center for disease control. Atlanta, GA:Centers for Disease Control, 1975.
26. CDC. Preventing lead poisoning in young children: a statement by the center for disease control. CDC publication no. 00-2629. Atlanta, GA:Centers for Disease Control, 1978.
27. CDC. Preventing lead poisoning in young children: a statement by the centers for disease control. CDC report no. 99-2230. Atlanta, GA:Centers for Disease Control, 1985.
28. Parsons PJ, Slavin W. A rapid Zeeman graphite atomic absorption spectrophotometric method for the determination of lead in blood. Spectrochim Acta 48B:925-939 (1993).
29. Mahaffey KR, Annest JL, Roberts J, Murphy RS. National estimates of blood lead levels: United States, 1976-1980. N Engl J Med 320:573-579 (1989).
30. Mahoney M. Four million children at risk: lead paint poisoning victims and the law. Stanford Environ Law J 9:46-83 (1990).
31. ATSDB. The nature and extent of lead poisoning in children: a report to Congress. Atlanta, GA:Agency for Toxic Substances and Disease Registry, 1988.
32. Centers for Disease Control. Lead levels—United States, 1988-1991. MMWR 41(30):545-548 (1994).
33. Brody DJ, Pirkle JL, Kramer RA, Flegal KM, Matte TD, Gunter EW, Paschal DC. Blood lead levels in the U.S. population. JAMA 272:277-283 (1994).
34. Pirkle JL, Brody DJ, Gunter EW, Kramer RA, Paschal DC, Finlay KM, Matte TD. The decline in blood lead levels in the United States. JAMA 272:284-291 (1994).