

The Decline in Blood Lead Levels in the United States

The National Health and Nutrition Examination Surveys (NHANES)

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Objective.—To describe trends in blood lead levels for the US population and selected population subgroups during the time period between 1976 and 1991.

Design.—Two nationally representative cross-sectional surveys and one cross-sectional survey representing Mexican Americans in the southwestern United States.

Setting/Participants.—Participants in two national surveys that included blood lead measurements: the second National Health and Nutrition Examination Survey, 1976 to 1980 (n=9832), and phase 1 of the third National Health and Nutrition Examination Survey, 1988 to 1991 (n=12 119). Also, Mexican Americans participating in the Hispanic Health and Nutrition Examination Survey, 1982 to 1984 (n=5682).

Results.—The mean blood lead level of persons aged 1 to 74 years dropped 78%, from 0.62 to 0.14 $\mu\text{mol/L}$ (12.8 to 2.8 $\mu\text{g/dL}$). Mean blood lead levels of children aged 1 to 5 years declined 77% (0.66 to 0.15 $\mu\text{mol/L}$ [13.7 to 3.2 $\mu\text{g/dL}$]) for non-Hispanic white children and 72% (0.97 to 0.27 $\mu\text{mol/L}$ [20.2 to 5.6 $\mu\text{g/dL}$]) for non-Hispanic black children. The prevalence of blood lead levels 0.48 $\mu\text{mol/L}$ (10 $\mu\text{g/dL}$) or greater for children aged 1 to 5 years declined from 85.0% to 5.5% for non-Hispanic white children and from 97.7% to 20.6% for non-Hispanic black children. Similar declines were found in population subgroups defined by age, sex, race/ethnicity, income level, and urban status. Mexican Americans also showed similar declines in blood lead levels of a slightly smaller magnitude over a shorter time.

Conclusions.—The results demonstrate a substantial decline in blood lead levels of the entire US population and within selected subgroups of the population. The major cause of the observed decline in blood lead levels is most likely the removal of 99.8% of lead from gasoline and the removal of lead from soldered cans. Although these data indicate major progress in reducing lead exposure, they also show that the same sociodemographic factors continue to be associated with higher blood lead levels, including younger age, male sex, non-Hispanic black race/ethnicity, and low income level. Future efforts to remove other lead sources (eg, paint, dust, and soil) are needed but will be more difficult than removing lead from gasoline and soldered cans.

(JAMA. 1994;272:284-291)

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LEAD has been dispersed in the environment in substantial quantities over a long period of time. Compelling evidence from the scientific community on a wide range of adverse health outcomes has placed lead in the forefront of environmental health concerns. In the 1970s, federal regulatory and legislative efforts were undertaken to reduce lead hazards, including actions to limit the use of lead in paint and gasoline.¹ The second National Health and Nutrition Examination Survey (NHANES II, 1976 to 1980) established baseline lead measurements for the US population and demonstrated the pervasiveness of lead

See also pp 277 and 315.

exposure across race, urban and rural residence, and income levels.² Data from NHANES II showed a decline in blood lead levels from the beginning to the end of the survey period that was closely correlated to declines in the use of leaded gasoline during these years.³

Since 1980, intensive federal, state, and local actions directed at primary prevention have been taken to further reduce lead exposure from gasoline, paint, solder, and other sources. Secondary prevention activities, such as screening for early detection and lead education programs, have also been implemented. New data from phase 1 of the third National Health and Nutrition Examination Survey (NHANES III phase 1, 1988 to 1991) permit examination of changes in blood lead levels since 1980 in the US population and evaluation of the impact of these regulatory

actions. The Hispanic Health and Nutrition Examination Survey (HHANES, 1982 to 1984) provides data on Mexican Americans at an intermediate time point. These analyses of trends in blood lead levels serve both to evaluate the effectiveness of prevention programs and to develop new strategies to further reduce lead exposure in the United States.

METHODS

Design and Data Collection

The National Health and Nutrition Examination Surveys (NHANES) are designed to measure and monitor the health and nutritional status of the US population. The general design of the NHANES is a stratified multistage probability cluster sample of households whose target population is civilian non-institutionalized persons residing in the United States. Blood lead levels were determined in NHANES II (1976 to 1980), HHANES (1982 to 1984), and NHANES III (1988 to 1994). The estimates from NHANES II and NHANES III are based on a national sample, whereas HHANES sampled three Hispanic subgroups.^{4,6}

National trends of blood lead levels presented in this article were based primarily on comparisons of data from NHANES II and NHANES III phase 1 (1988 to 1991). Trends for Mexican Americans were based on a comparison between the estimates from HHANES and NHANES III phase 1. The HHANES also provides an intermediate point in time between NHANES II and NHANES III. The HHANES sample of Mexican Americans included only those residing in the southwestern United States whereas the NHANES III phase 1 sample represented Mexican Americans residing in the entire United States.

Venous blood lead measurements were obtained for persons aged 6 months to 74 years in NHANES II; persons aged 4 to 74 years in HHANES; and persons aged 1 year and older in NHANES III phase 1. Analysis was limited to persons aged 1 to 74 years for national trends and aged 4 to 74 years for trends in the Mexican-American population. The final samples used for analyses included 9832 and 12119 for the national trends from NHANES II and NHANES III phase 1, respectively, and 5682 and 4067 Mexican Americans from HHANES and NHANES III phase 1, respectively. Data from all of the surveys were collected using a household interview followed by a detailed medical examination in a mobile examination center.

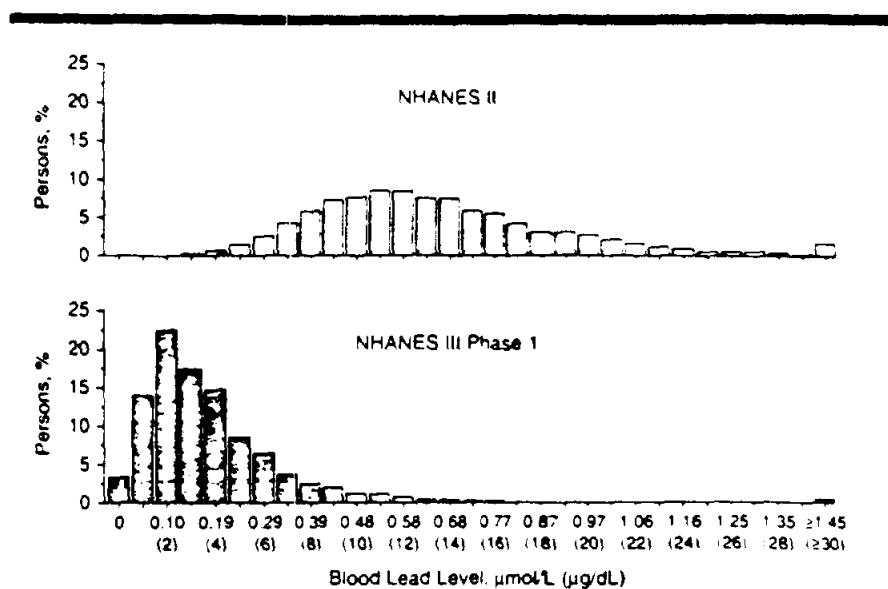


Fig 1.—Blood lead levels for persons aged 1 to 74 years: United States, second National Health and Nutrition Examination Survey (1976 to 1980, top) and phase 1 of the third National Health and Nutrition Examination Survey (1988 to 1991, bottom).

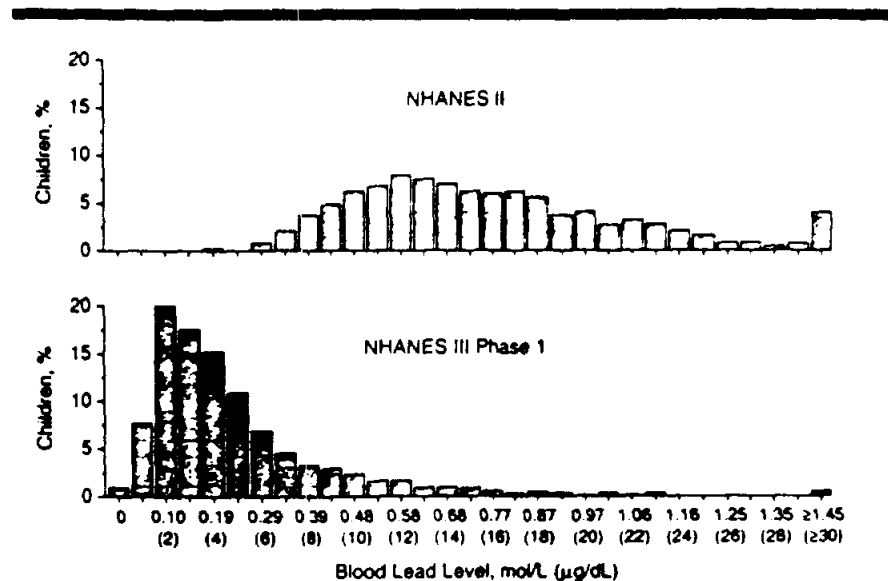


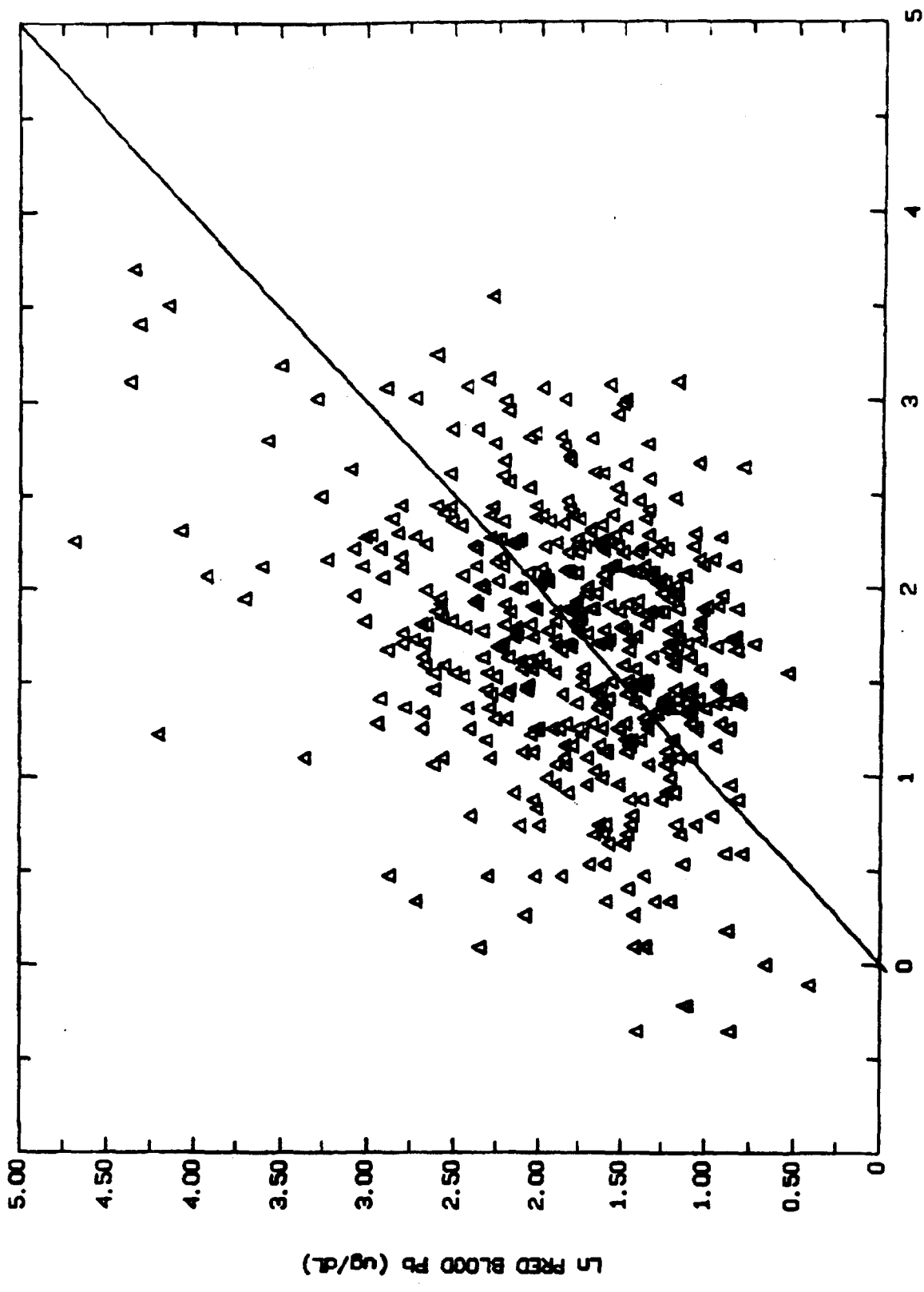
Fig 2.—Blood lead levels for children aged 1 to 5 years: United States, second National Health and Nutrition Examination Survey (1976 to 1980, top) and phase 1 of the third National Health and Nutrition Examination Survey (1988 to 1991, bottom).

The response rates for blood lead collection in the three surveys ranged from 61% to 69%. Previous nonresponse bias analyses conducted for NHANES II, HHANES, and NHANES III phase 1 indicated that there was no apparent bias due to nonresponse.^{7,8}

Laboratory Methods

All venous blood specimens were collected in the mobile examination centers, frozen, and shipped on dry ice to the NHANES laboratory, Division of Environmental Health Laboratory Sci-

ences, National Centers for Environmental Health, Centers for Disease Control and Prevention, Atlanta, Ga, for analysis. The methods for determining lead in blood, including descriptions of quality control and assurance procedures, have been described for each survey.⁹⁻¹¹ Comparability has been established for the method used in NHANES II and HHANES (modified Delves cup) and that used in NHANES III phase 1 (graphite furnace atomic absorption spectrophotometry), as described by Miller et al.¹² In each of the three sur-

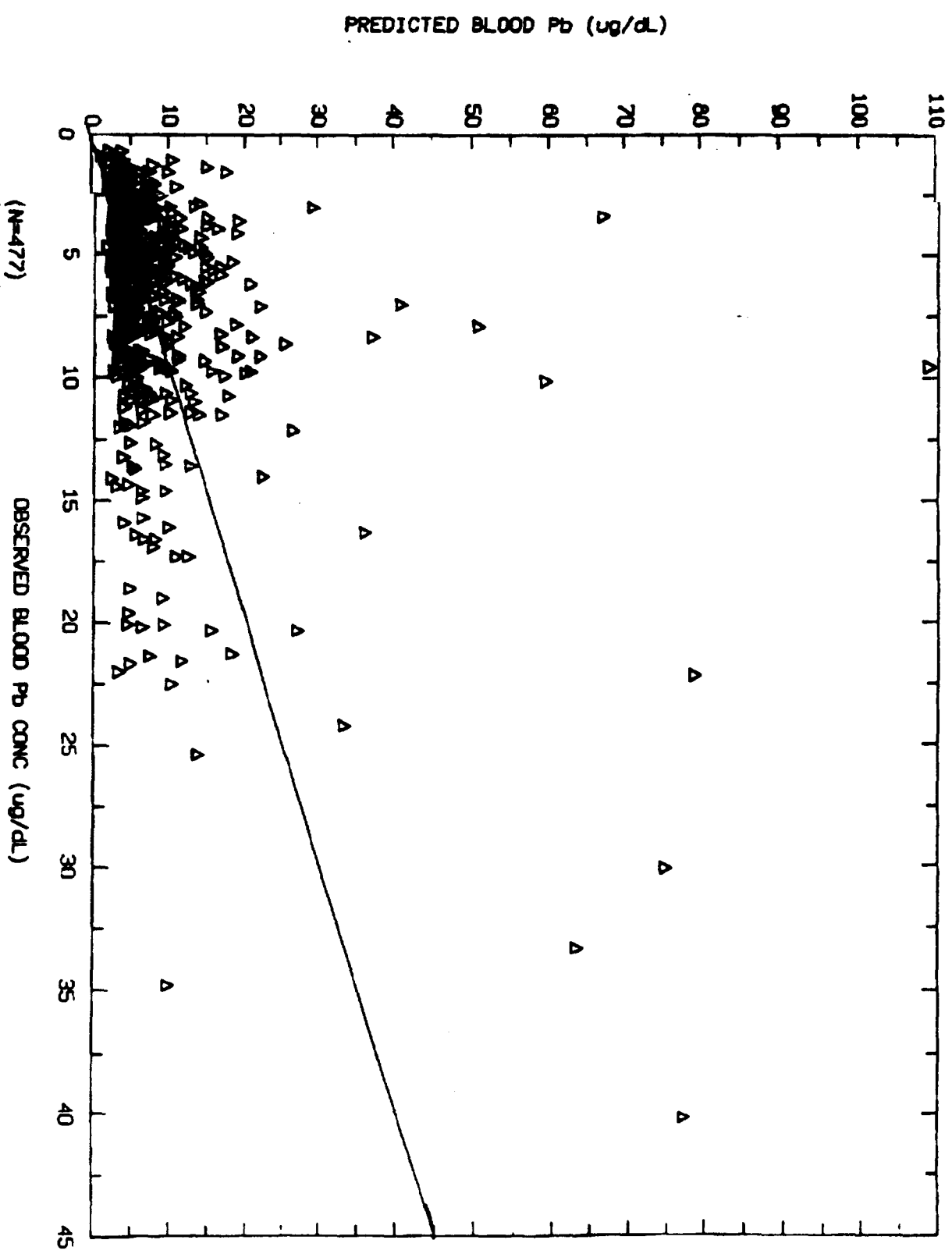


(N=477)
(r=0.34)
(13 Missing Value(s))

Ln OBSV BLOOD Pb CONC (ug/dL)

GCVALID01.ASC

FIGURE 19



GCVAL101.ASC

FIGURE 20

HOURS OF PLAY ON FLOOR VS. DISTANCE FROM NL SITE

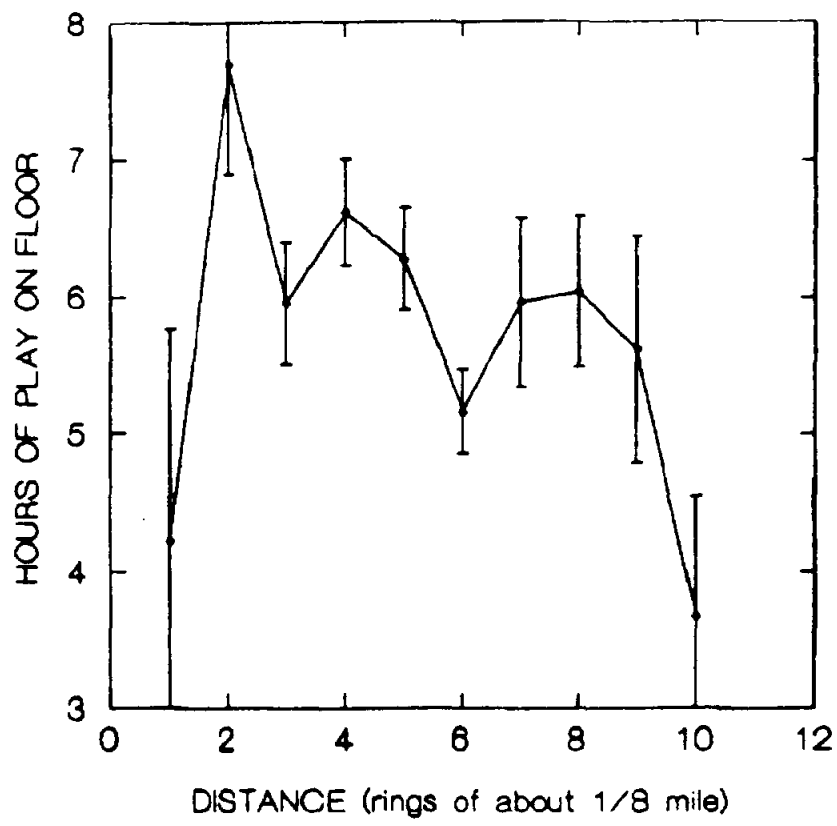
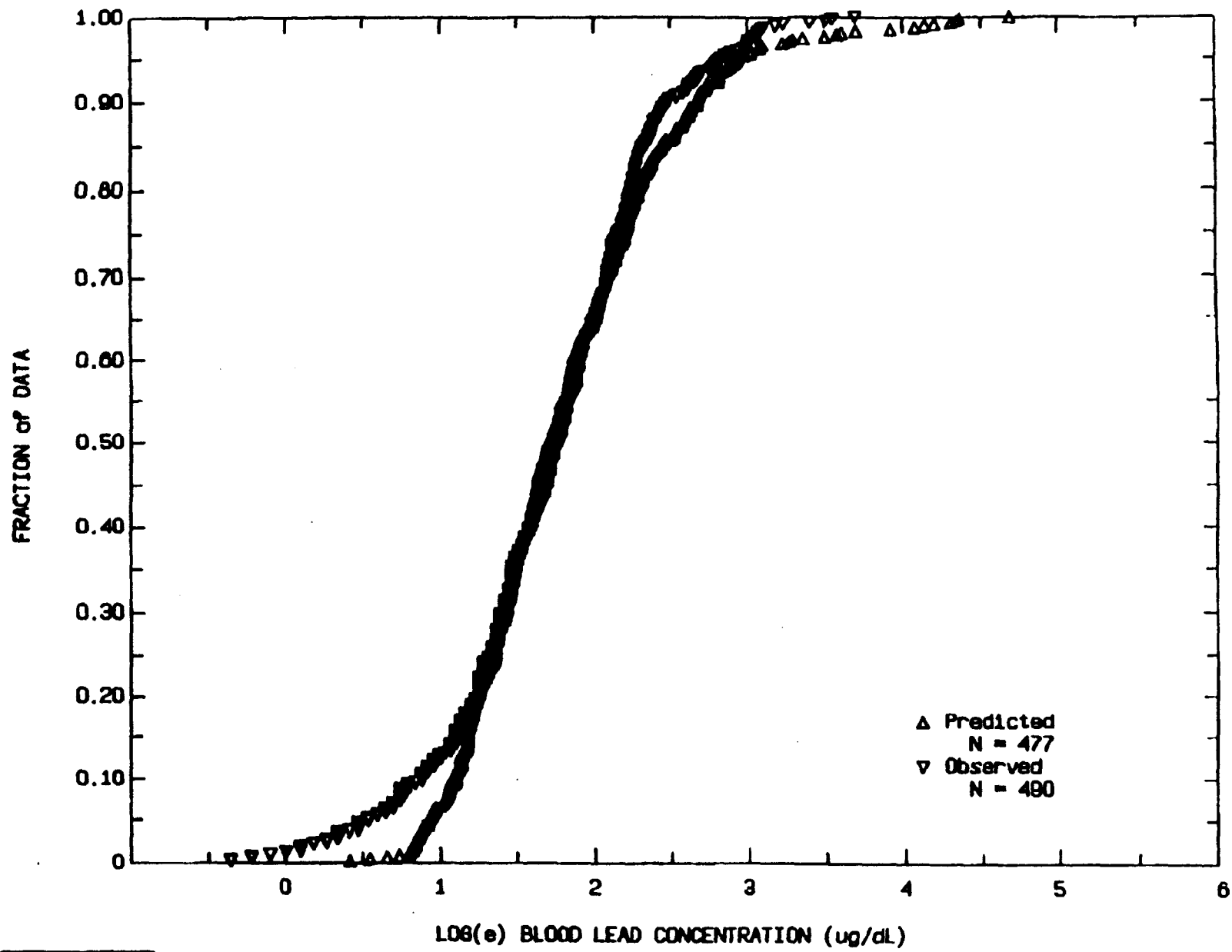


FIGURE 17a



BCVALID1.ASC

FIGURE 18

PERCENT WITH AIR CONDITIONING VS. DISTANCE FROM NL S

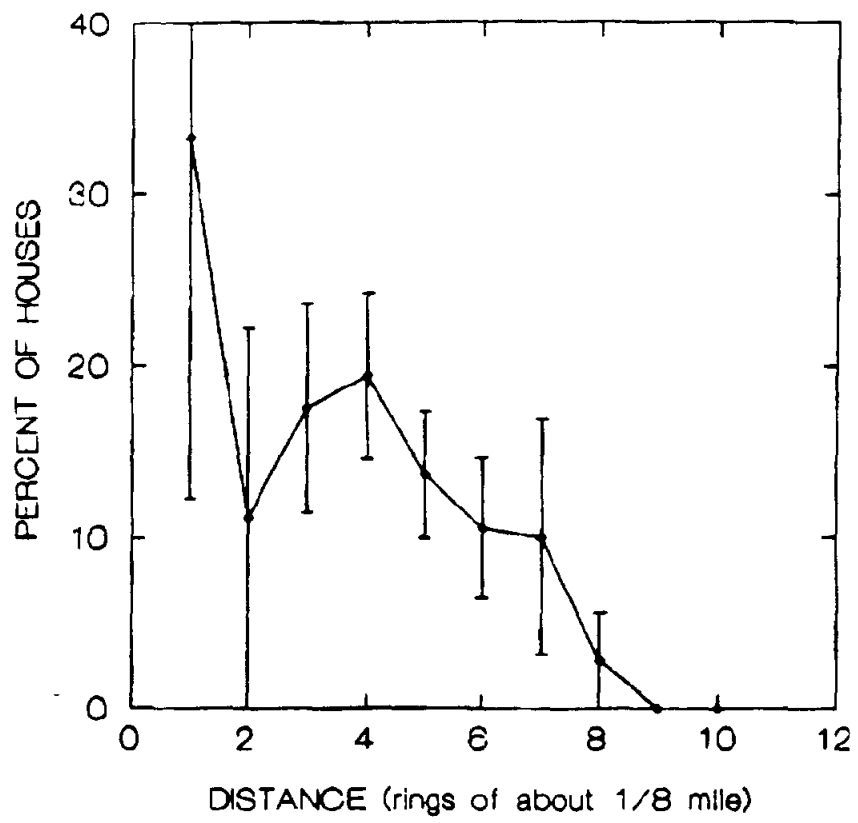


FIGURE 16

HOURS OF OUTDOOR PLAY VS. DISTANCE FROM NL SITE

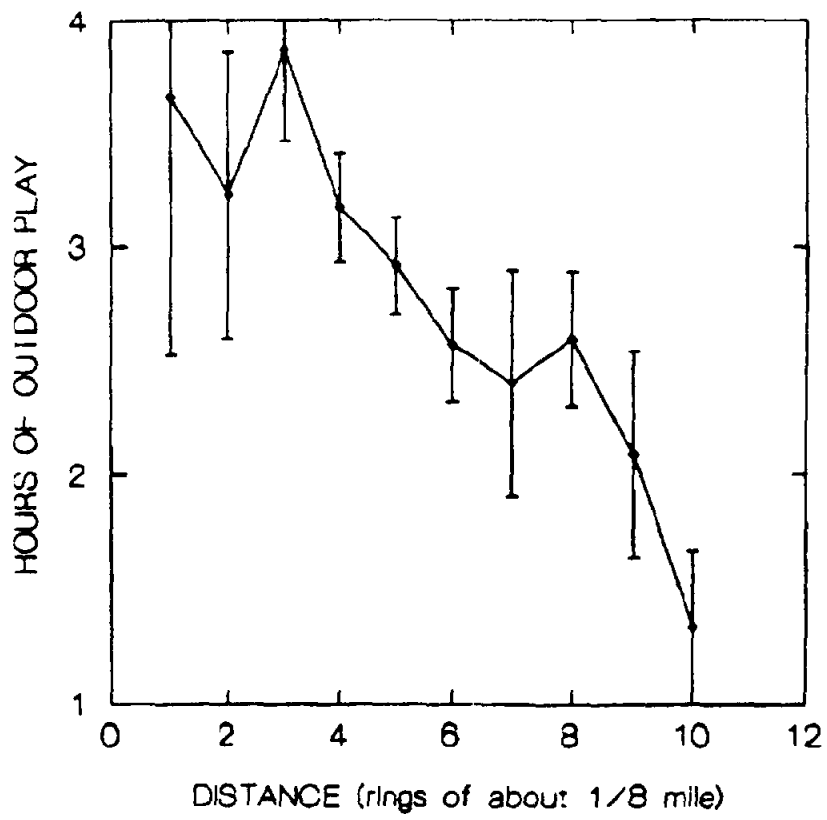


FIGURE 17

NUMBER OF CHILDREN IN HOUSE VS. DISTANCE FROM NL S

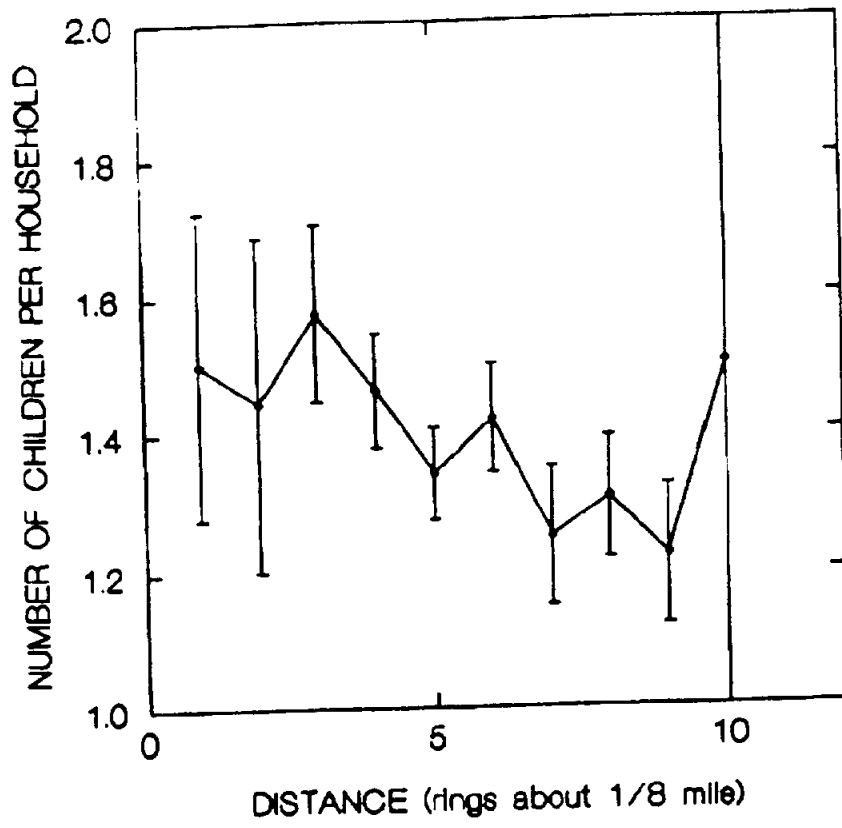


Figure 14

MEAN BUILDING CONDITION VS. DISTANCE FROM NL SITE

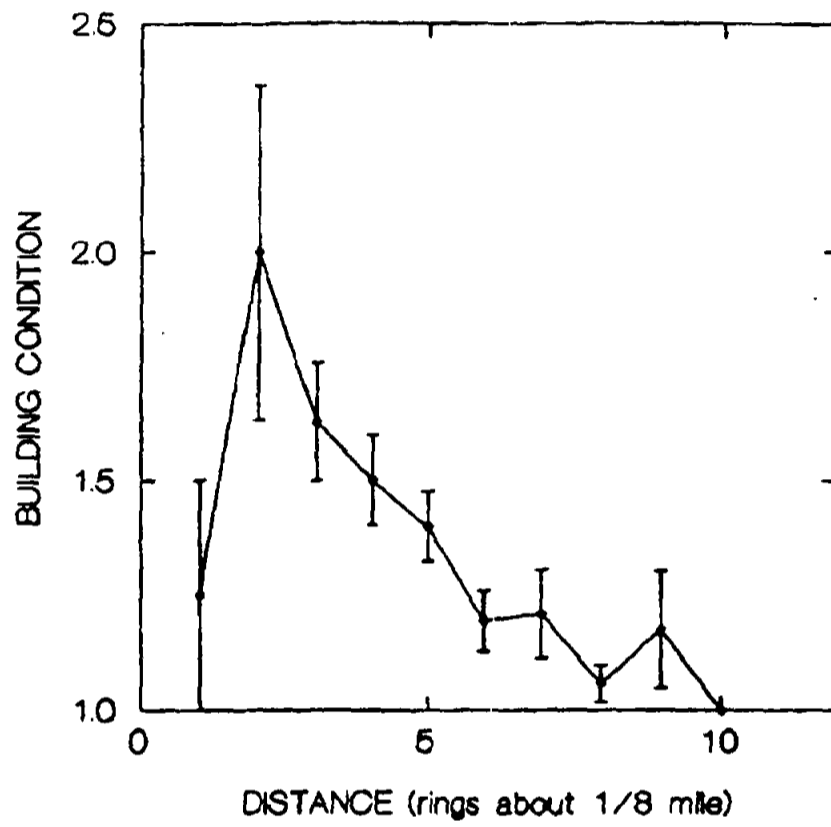


Figure 15

MEAN YEARS OF EDUCATION VS. DISTANCE FROM NL SITE

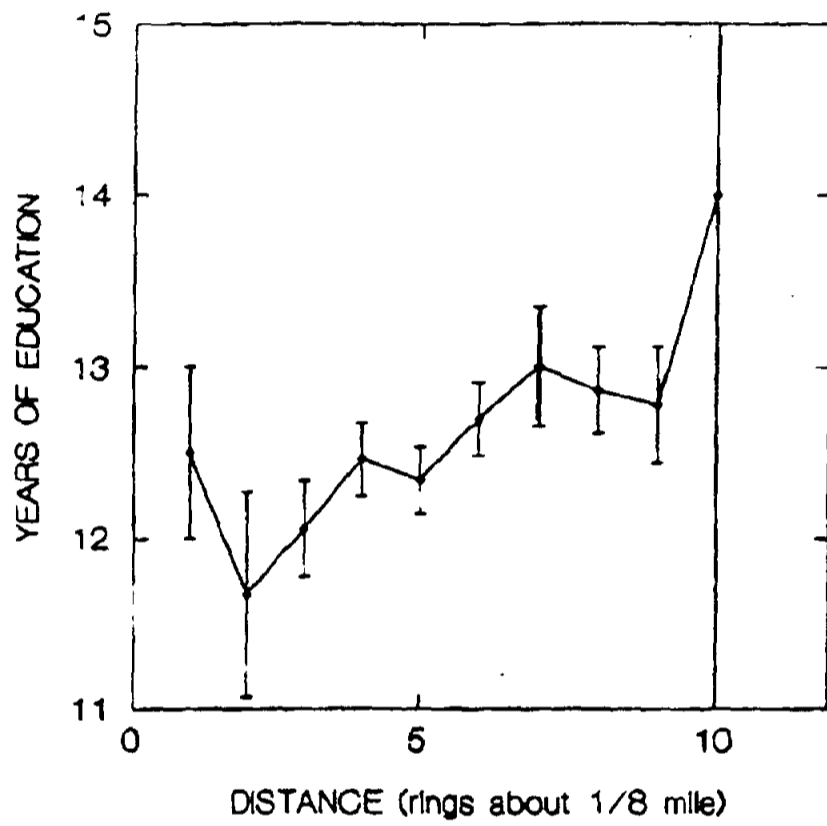


Figure 12

MEAN INCOME VS. DISTANCE FROM NL SITE

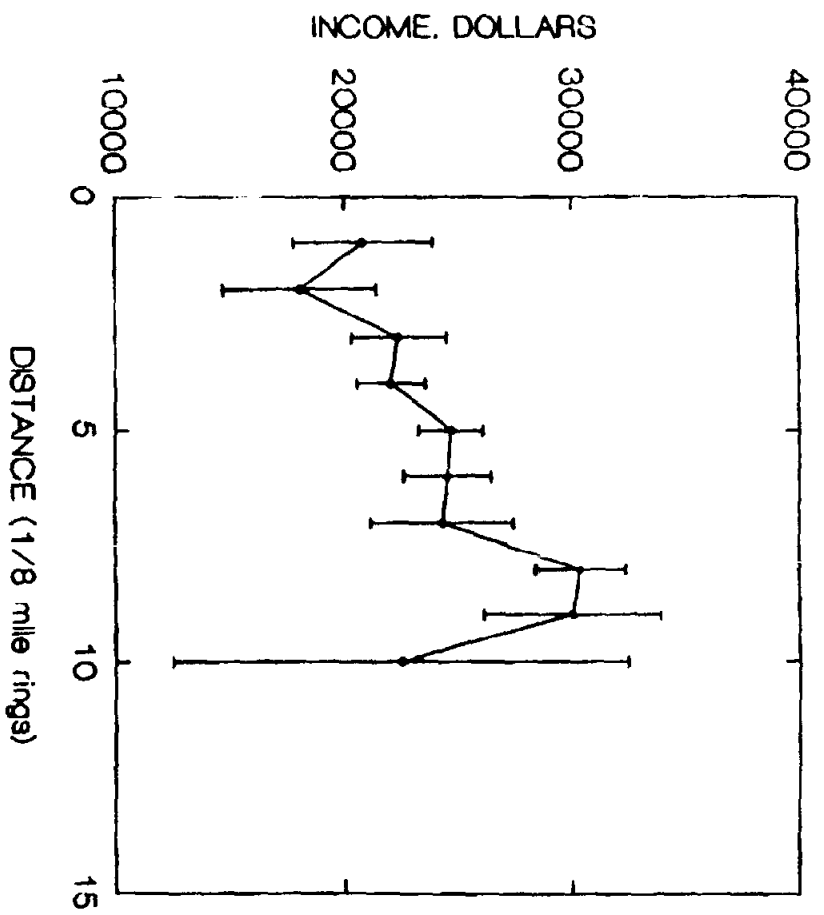


Figure 13

TOTAL DUST LOADING VS. DISTANCE FROM NL SITE

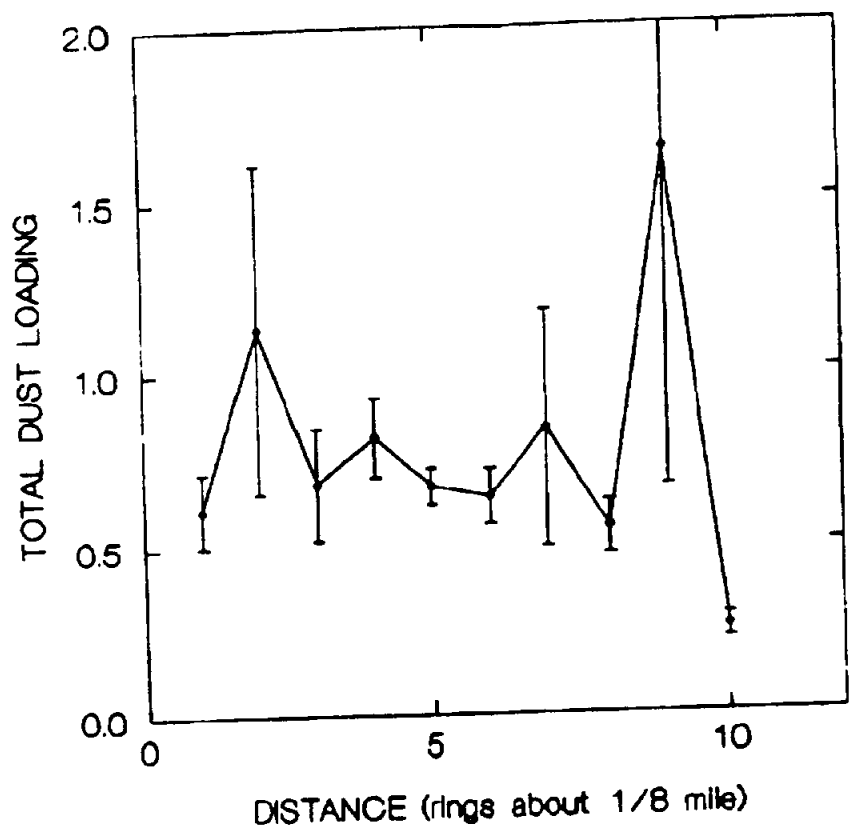


Figure 10

LOG OF DUST LEAD LOADING VS. DISTANCE FROM NL SITE

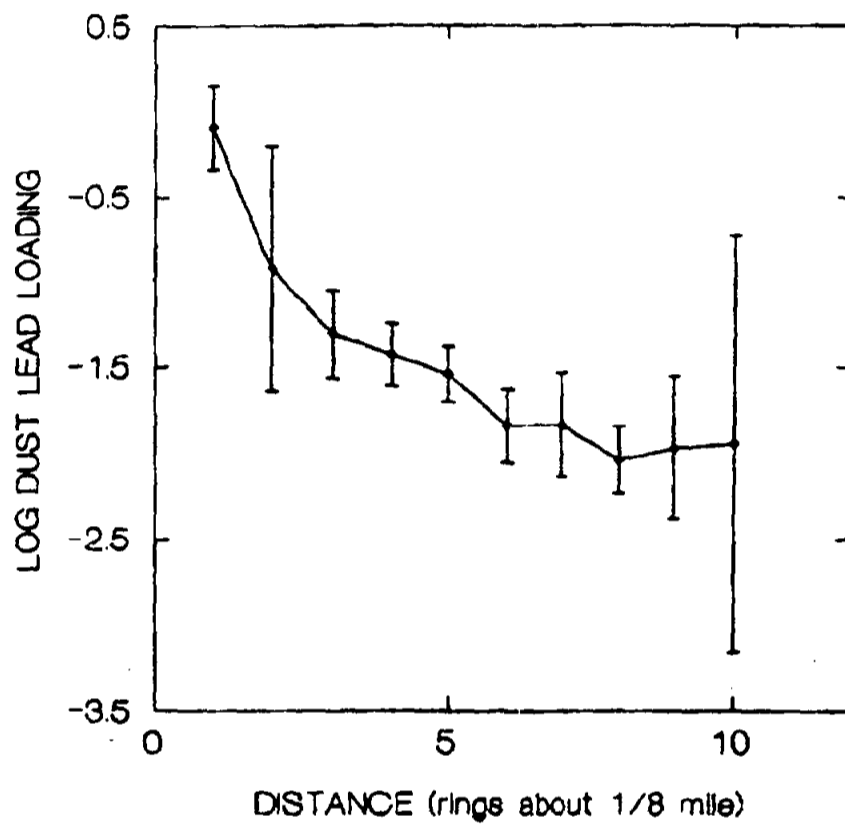


Figure 11

LOG EXTERIOR LEAD PAINT VS. DISTANCE FROM NL SITE

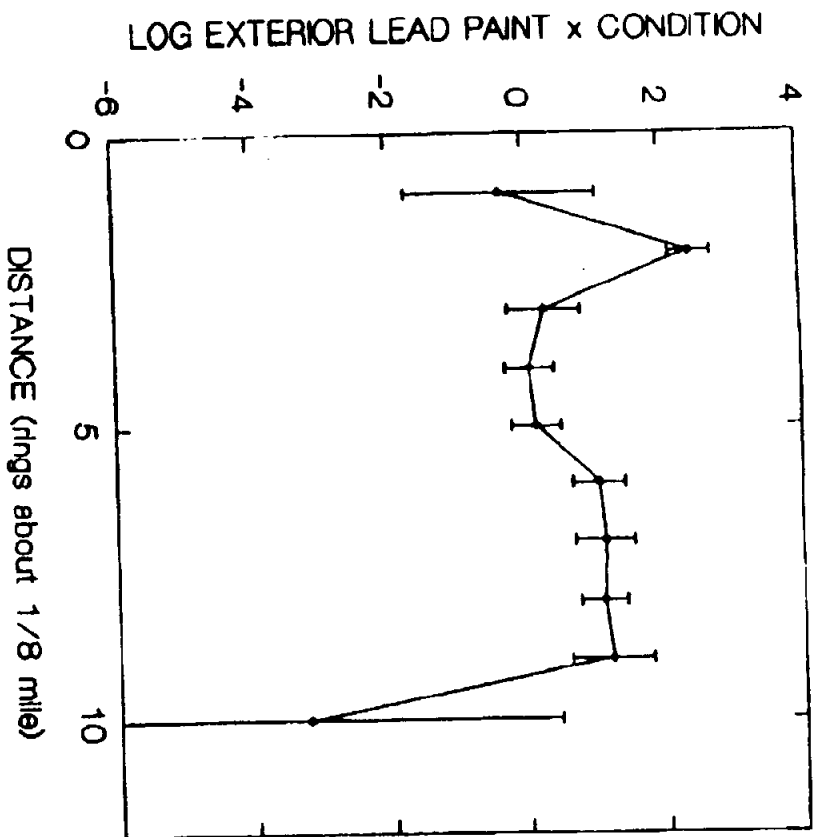


Figure 8

LOG OF WATER LEAD CONC. VS. DISTANCE FROM NL SITE

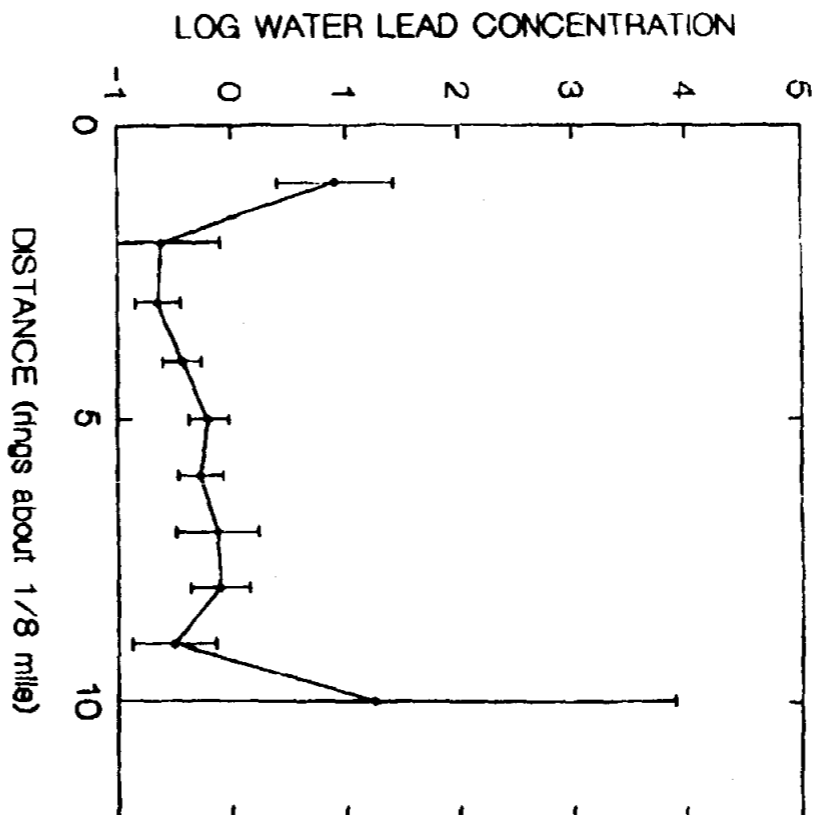


Figure 9

LOG OF DUST LEAD CONC. VS. DISTANCE FROM NL SITE

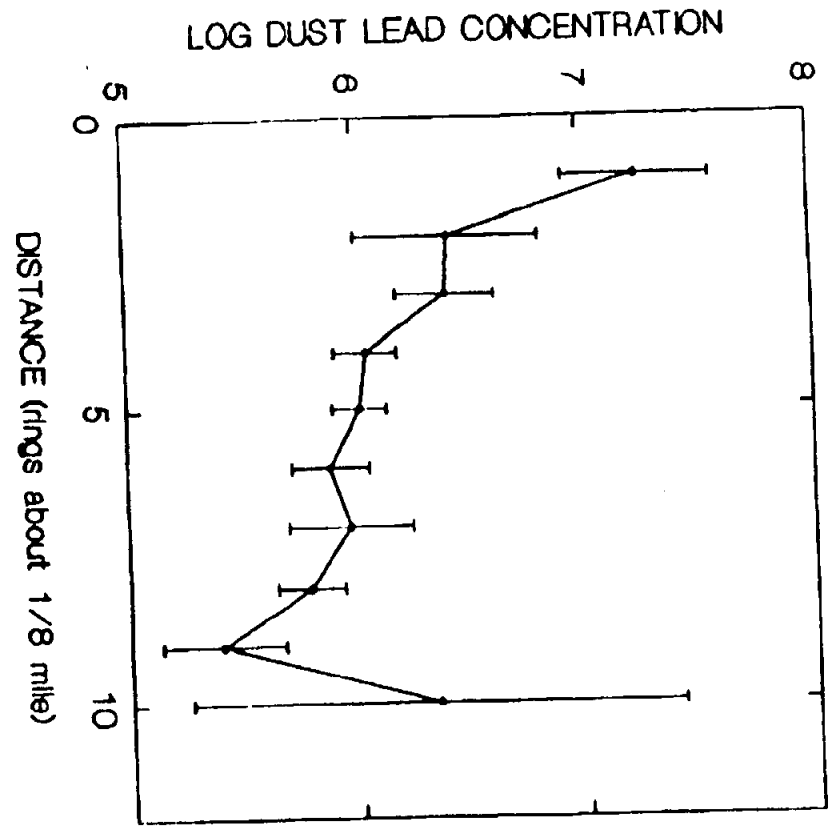


Figure 6

LOG INTERIOR LEAD PAINT VS. DISTANCE FROM NL SITE

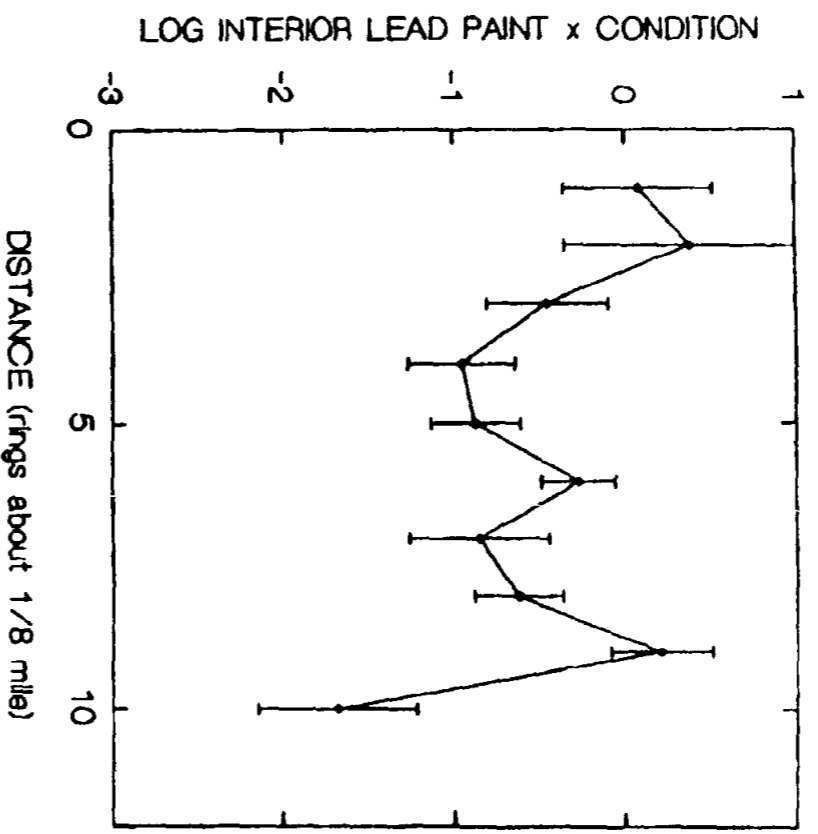


Figure 7

MEAN LOG BLOOD LEAD VS. DISTANCE FROM NL SITE

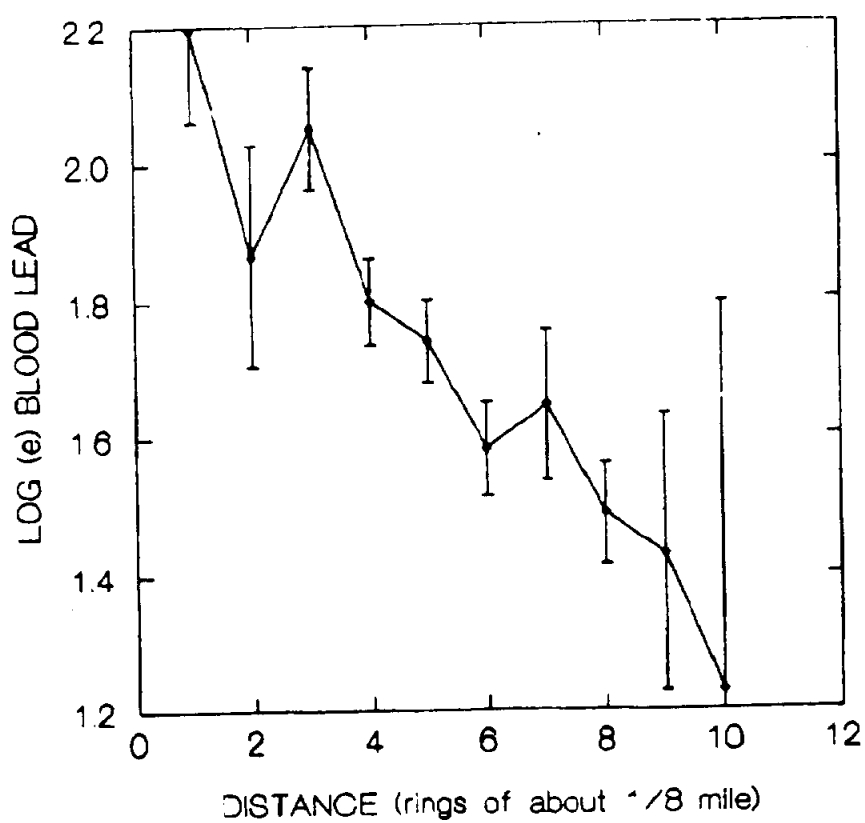


Figure 4a

LOG OF SOIL LEAD CONC. VS. DISTANCE FROM NL SITE

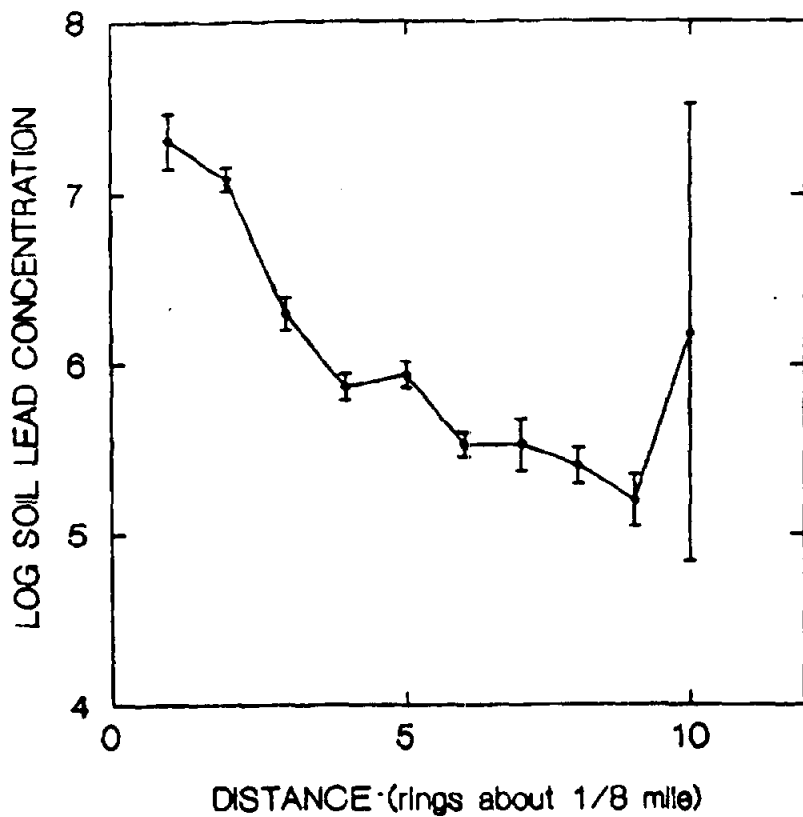


Figure 5

PERCENT WITH BLOOD LEAD 20+ VS. DISTANCE FROM NL S

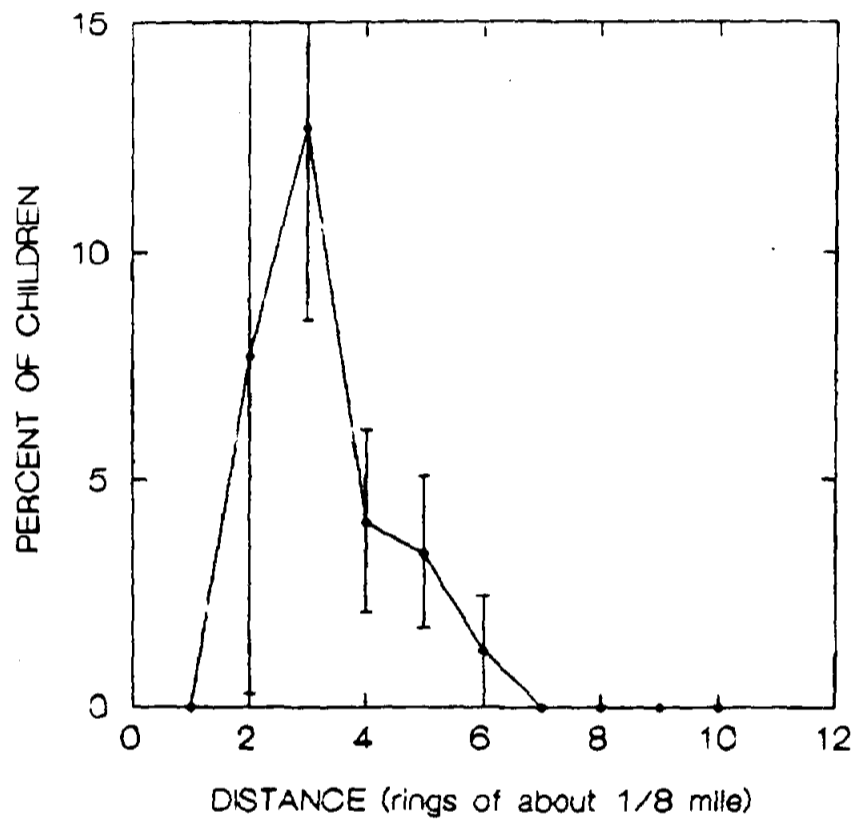


FIGURE 3

MEAN BLOOD LEAD VS. DISTANCE FROM NL SITE

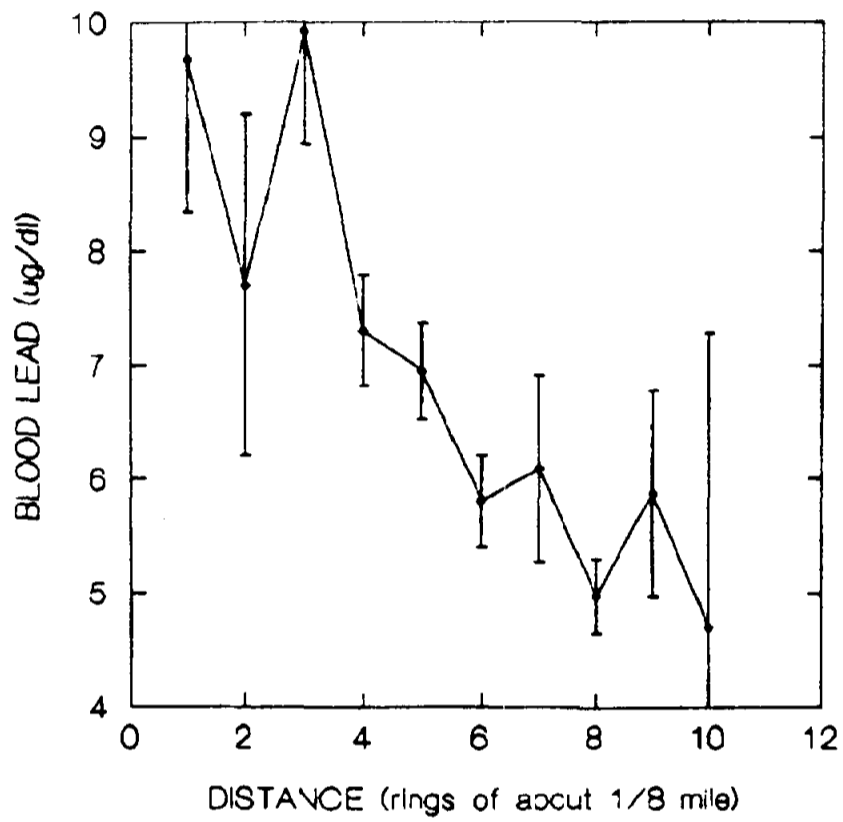


Figure 4

PERCENT WITH BLOOD LEAD 10+ VS. DISTANCE FROM NL S

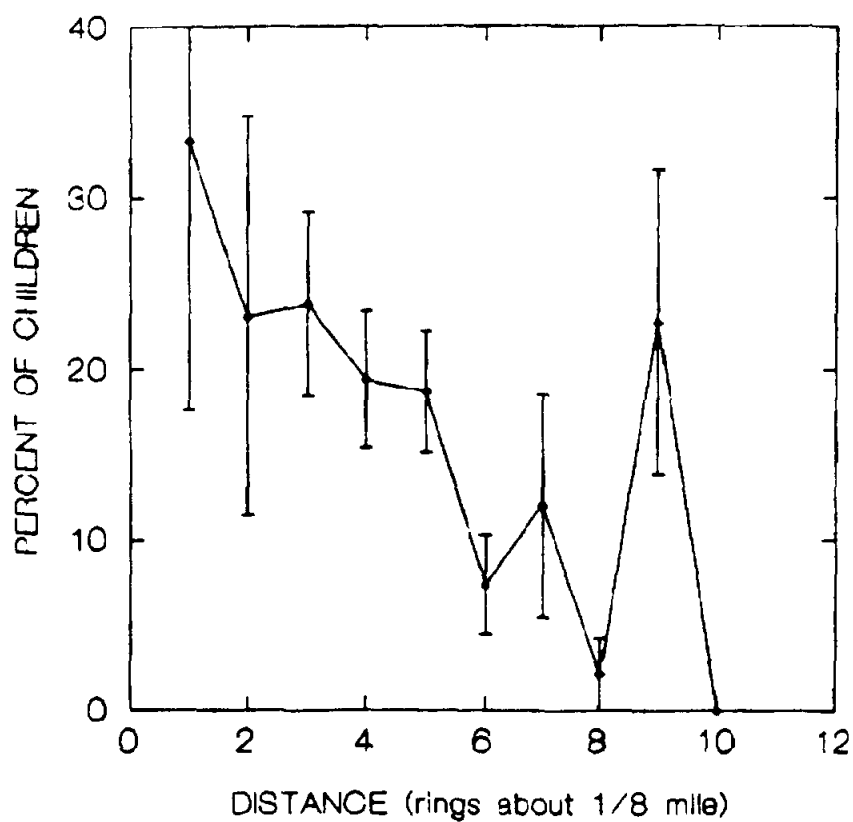


FIGURE 1

PERCENT WITH BLOOD LEAD 15+ VS. DISTANCE FROM NL S

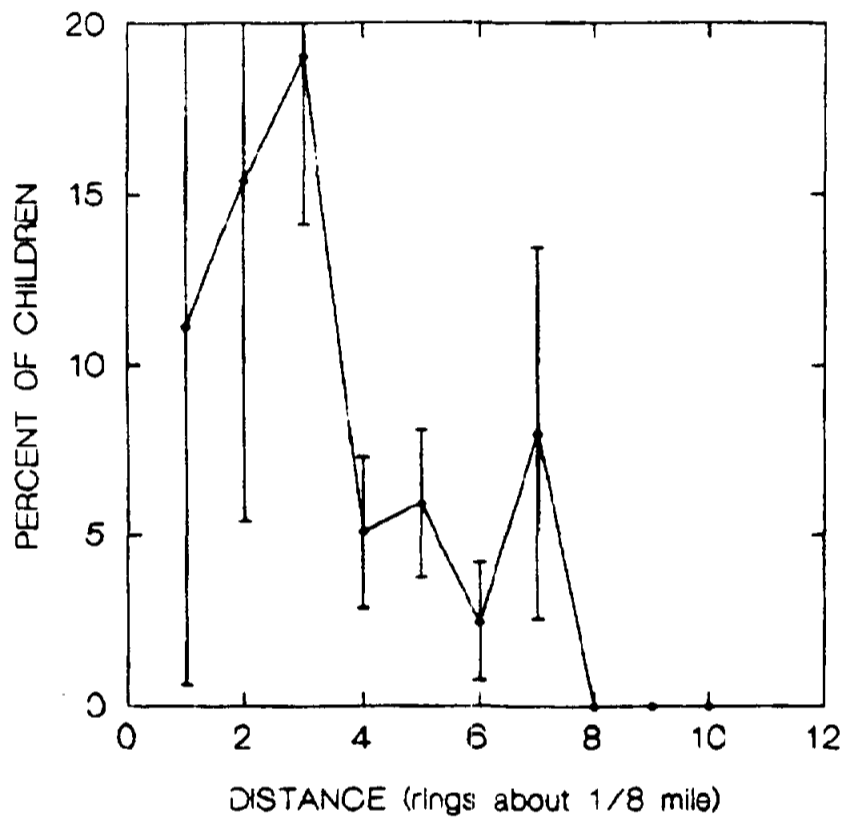


FIGURE 2

and predicted blood lead. The over-all goodness of fit was comparable or better to that for the calibration community, the lead smelter community of Midvale, Utah. (Figures 18, 19, 20).

3. Sensitivity analyses were based on a range of values for the contribution of lead-contaminated soil to household dust. The default assumption, that the concentration of soil-derived lead in house dust is 0.70 of the soil lead concentration, was judged to be appropriate, and also provided a very good fit to the child blood lead data from the Madison County study. Alternative values in the sensitivity analyses were based on statistical analyses from study data: 0.29 (distances up to 1/4 mile), 0.46 (all data), and 0.55 (distances to 3/8 mile). The curvilinearity in the blood lead vs. environmental lead relationship was characterized by a passive-to-total gut lead absorption fraction of 0.20, as found from in-vitro studies. The higher dust/soil coefficients of 0.70 and 0.55 are more appropriate for risk assessment, more realistic for properties of the site, and provide a good fit to the data.

4. Remediation goals for soil abatement were calculated from the IEUBK Model so as to generate not more than 5 percent of children of ages 6 to 84 months with blood lead 10 ug/dl or greater. The calculated soil lead concentrations depended on the assumptions one made about soil to dust transport, but otherwise assumed only default parameters. The soil remediation levels ranged from 340 ppm (soil-to-dust coefficient = 0.70) to 480 ppm (soil-to-dust coefficient of 0.29). This suggests a range of soil lead cleanup values of 400 to 500 ppm. The results are shown in Table 1.

REFERENCES

1. Illinois Department of Public Health, 1994. Madison County Lead Exposure Study. Granite City, Illinois. Draft for public comment. Springfield, Illinois, Feb. 1994.
2. Marcus A.H., Hogan K., White P., Van Leeuwen P. 1994. Comments on Madison County Lead Exposure Study. Granite City, Illinois. In-house memo, U.S. Environmental Protection Agency, May 1994; corrected draft, Sept. 18, 1994. Research Triangle Park, NC.
3. Agency for Toxic Substances and Disease Registry, 1994. Comments on Multisite Lead and Cadmium Exposure study with Biological Markers Incorporated. Review draft. Atlanta, Georgia.
4. Wilkinson, L. 1992. SYSTAT: The System for Statistics. Systat Inc., Evanston IL.

TABLE 1
SENSITIVITY ANALYSES FOR SOIL LEAD CLEANUP LEVELS

SOIL CONTRIBUTION TO HOUSE DUST	SOIL LEAD CLEAN-UP CONCENTRATION (ppm)
0.70 (default)	340
0.55	370
0.46	420
0.29	480

factor in childhood blood lead in Madison County, and is much less important than soil lead and dust lead. (Figures 4, 5, 6, 7, 8).

4. Tap water lead concentrations are highest, on average, in the area closest to the NL smelter, but show little relation to distance from the site farther away. This suggests that tap water lead may be a contributing factor, but is not the primary environmental factor in childhood blood lead in the Madison County study. (Figure 9).

5. Total dust loading shows almost no relationship to distance from the NL site, on average, so that increased dustiness of homes cannot explain the higher household dust lead loadings found near the NL site; it is the higher concentration of lead in household dust that accounts for higher dust lead loadings near the NL site. Further studies are needed to determine whether this is an artifact of the method of house dust sampling, or whether this is a generalizable conclusion. (Figures 10, 11).

6. Lead in household dust is the primary exposure pathway for young children. Lead in soil and lead in deteriorating lead-based paint are primary sources for lead in house dust, with substantial variability from one household to another.

7. Many socio-demographic factors are related to individual childhood blood lead concentrations, and also show a systematic relationship to distance from the NL site. These include increasing parental education, increasing income, and decreasing numbers of pre-school children per household with increasing distance from the smelter. Households in the study with the most children and the fewest resources to cope with lead poisoning are located closest to the NL site. (Figures 12, 13, 14, 15).

8. Individual child-specific behaviors may affect blood lead concentration, with substantial differences among children. These include hours of outdoor play, frequency of mouthing non-food objects, and hours of indoor play on the floor. There are some systematic relationships, such as a highly significant tendency for children in the study who live closer to the site to have more hours of outdoor play, on average, than children who live farther away (Figures 16, 17) and 17a.

9. In view of correlations that were found between distance from the NL site, blood lead, environmental lead, household socio-demographic characteristics, and typical child behavior, there are some concerns that the sample of children may not be representative of the community. This study used volunteer subjects. The lowest response rate was in the zone farthest from the NL site, 39 percent. Response rates were similar in the other three zones closer to the NL site, respectively 51 percent in the closest zone, 60 percent in the next closest, and 53 percent in the next closest. One must assume that there were no systematic biases in recruitment related to important factors that affect child blood lead, such as socio-economic status or behavior.

10. Percentage of explained variability in the logarithm of blood lead is not a

useful criterion in model assessment, since it depends on the range and the distribution of predictor variables within the data set. In comparison with all other studies of child blood lead data that EPA has performed, including urban and rural sites, active and inactive lead smelter or lead mining sites, the child blood lead data from the Madison County study has a higher percentage of explained variation (40 percent) than most other studies, and environmental lead explains a comparable percentage of variance (18 percent in our analyses) to other inactive smelter sites. Both the magnitude and strength of the relationships between blood lead and environmental lead are comparable to those we have seen at other sites. The linear regression relationships for blood lead vs. lead in soil, dust, and drinking water are statistically significant in all appropriate model specifications.

3. CALCULATION OF SOIL LEAD CLEANUP CONCENTRATIONS.

3.1. SUMMARY AND CONCLUSIONS: SITE-SPECIFIC PROPERTIES FOR RISK ASSESSMENT

1. The NL/Taracorp site appears to have properties that are characteristic of other recently inactive lead smelter sites. The areas closest to the site have soil and dust lead concentrations that are appropriate to airborne particulates from smelter emissions. These particles are generally easily transported from exterior soil into household dust, and are likely to be small, soluble, and highly bioavailable.

2. These analyses, plus observation of Granite City neighborhoods closest to the NL site, show that:

- (i) there are many young children in the community,
- (ii) children often play outdoors for much of the day,
- (iii) residential yards often contain large bare areas without grass cover,
- (iv) adjacent yards are often not fenced and are readily accessible to young children,
- (v) the residential areas are surrounded by industrial areas and by transportation routes that contribute to the total environmental impact on these children.

3.2. SOIL LEAD CLEANUP LEVELS USING THE IEUBK MODEL

1. Site-specific parameters were based on our judgement and analyses that the NL site had many points of similarity to the calibration site, Midvale, and that it is appropriate to assume no mitigating factors that may reduce childhood exposure to dust and soil.

2. The default parameters with an assumed soil-to-dust coefficient of 70 percent provided a very good fit to the blood lead data, in terms of geometric mean blood lead, percentiles of the blood lead distribution, and reasonable correlation between observed

PRELIMINARY ASSESSMENT OF DATA FROM THE MADISON COUNTY LEAD STUDY AND IMPLICATIONS FOR REMEDIATION OF LEAD-CONTAMINATED SOIL

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1. INTRODUCTION

The data on blood lead, environmental lead, and family interviews for 490 children in Madison County, Illinois, were provided to us by the Institute for Evaluating Health Risks (IEHR) through the U.S. Department of Justice. The study was carried out in 1991 by U.S. EPA, Illinois EPA, the Illinois Dept. of Public Health (IDPH) and the Agency for Toxic Substances and Disease Registry (ATSDR). We requested access to the data from IDPH, which was provided by their contractor, IEHR. Evaluating Health Risks (IEHR). The data were sent to us on diskette in ASCII format. We converted these data into a SYSTAT (Wilkinson 1992) data file, from which all subsequent analyses reported here were performed. Additional analyses will require creation of SAS data sets. Analyses of the data were reported by IDPH (February 1994) and in a more compact form by ATSDR (May 1994). U.S. EPA provided a critical review of these analyses (Marcus et al., May 1994).

The purposes for our reanalyses of the data are:

1. To assess the results described in (IDPH 1994) for use in evaluating childhood lead exposure in Madison County;
2. To provide site-specific information about relevant parameters in the EPA Integrated Exposure, Uptake, and Biokinetic Model for Lead (IEUBK Model);
3. To evaluate the proposed soil remediation level of 500 ppm using this recent information.

This is only a very preliminary report of results. A more detailed report will be prepared, describing the methods used in the analyses, a complete set of results, and the basis for our conclusions. This report is divided into the following sections. Section 2 reports the results of the preliminary analyses. Complete technical details will follow in a subsequent report. Section 3 presents the basis for a soil lead cleanup level. Section 3.1 describes the empirical basis for the findings, which combines personal observation of the site, results of data analyses, and professional judgements about the site. Section 3.2 describes the soil lead results when input values for the IEUBK Model from Section 3.1 are used, and describes the reevaluation of the soil lead cleanup level for the site.

2. SUMMARY AND CONCLUSIONS FROM DATA ANALYSES

A large number of graphs using these data are attached. The following results are based on these figures. Note that with few exceptions, the logarithms of the environmental lead concentrations and blood lead concentrations were used since the distribution of each variable is skewed to the right. All are "natural" base e logarithms, not common base 10 logarithms. The error bars on the graphs show the standard error of the (geometric) mean. The exact distance of the household from the NL site is not in the data set, only location within (approximate) 1/8 mile rings around the smelter. No information is provided about the quadrant or direction from the smelter site, and no information is provided about the city or township where each child lives.

We reanalyzed data on 490 pre-school age children in 351 households in Madison County, Illinois. These data contain no information on location within the study area, apart from approximate distance of the child's household from the NL/Taracorp site ("NL site"), and in particular do not identify the child's community of residence, so the results cannot be ascribed to any locale such as Granite City, Madison, or Venice. Our analyses of these data showed that:

1. Sixteen percent of the children had blood concentrations of 10 ug/dl or greater in the Madison County study area as a whole, but the percentage of lead-burdened children increased with decreasing distance from the NL smelter site. The increase was from 7 percent of children living at a distance greater than 0.75 miles from the NL site to about 26 percent of children in the study who lived in the area closest to the site. The areas closest to the site would be considered as appropriate for remediation, based on remedial investigations, by current EPA criteria. (Figure 1)

2. The percentage of children in the study with blood lead concentrations of 15 ug/dl or greater increased from about 2 percent of children living at a distance greater than 0.75 miles from the NL site to about 9 percent of children within 0.4 miles of the site. None of the children living at a distance of a mile or more from the NL site had a blood lead of 20 ug/dl or greater, whereas about 5 percent of the pre-school children who lived within half a mile from the NL site had a blood lead of at least 20 ug/dl. A blood lead concentration of 20 ug/dl is well above EPA's level of concern of 10 ug/dl and is associated with a substantially increased risk of permanent and irreversible neurobehavioral damage. Current CDC guidelines recommend individual environmental or medical intervention with children whose blood lead concentrations are at least 20 ug/dl. (Figures 2 and 3).

3. Blood lead concentration, soil lead concentration, and house dust lead concentration show very similar patterns of decreasing concentration with increasing distance from the NL site, on average. Loadings of deteriorating lead paint inside and outside the house show little or no relationship to distance from the NL site. This suggests that deteriorating lead-based paint is not the most important environmental

Table 3—Distribution of Blood Lead Levels for Mexican Americans Aged 4 to 74 Years by Age Category, Sex, and Income Level: 1982 to 1984 (Hispanic Health and Nutrition Examination Survey [HHANES]) and 1988 to 1991 Phase 1 of the Third National Health and Nutrition Examination Survey (NHANES III phase 1)

	No.	Geometric Mean, µmol/L (µg/dL)*	95% Confidence Interval, µmol/L (µg/dL)	Percentiles, µmol/L (µg/dL)						
				5th	10th	25th	50th	75th	90th	95th
All persons										
1982-1984	5682	0.41 (8.5)	0.40-0.42 (8.3-8.7)	0.19 (4.0)	0.24 (5.0)	0.29 (6.0)	0.43 (9.0)	0.58 (12.0)	0.77 (16.0)	0.87 (18.0)
1988-1991	3611	0.14 (3.0)	0.12-0.17 (2.5-3.5)	<0.05 (<1.0)	0.05 (1.1)	0.09 (1.9)	0.16 (3.3)	0.26 (5.4)	0.40 (8.3)	0.51 (10.6)
Ages 4-5 y										
1982-1984	269	0.53 (10.9)	0.50-0.56 (10.3-11.5)	0.24 (5.0)	0.29 (6.0)	0.39 (8.0)	0.53 (11.0)	0.68 (14.0)	0.92 (19.0)	1.11 (23.0)
1988-1991	349	0.17 (3.5)	0.14-0.21 (2.8-4.3)	<0.05 (<1.0)	0.07 (1.4)	0.12 (2.5)	0.18 (3.8)	0.26 (5.9)	0.40 (8.3)	0.48 (9.9)
Ages 6-19 y										
1982-1984	2331	0.39 (8.0)	0.38-0.40 (7.8-8.2)	0.19 (4.0)	0.24 (5.0)	0.29 (6.0)	0.39 (8.0)	0.53 (11.0)	0.68 (14.0)	0.82 (17.0)
1988-1991	1188	0.12 (2.5)	0.10-0.15 (2.0-3.2)	<0.05 (<1.0)	<0.05 (<1.0)	0.06 (1.8)	0.14 (2.8)	0.23 (4.7)	0.36 (7.4)	0.47 (9.8)
Ages 20-74 y										
1982-1984	3082	0.42 (8.7)	0.40-0.43 (8.3-9.0)	0.19 (4.0)	0.24 (5.0)	0.29 (6.0)	0.43 (9.0)	0.58 (12.0)	0.77 (16.0)	0.92 (19.0)
1988-1991	2074	0.15 (3.2)	0.13-0.18 (2.7-3.7)	<0.05 (<1.0)	0.05 (1.1)	0.10 (2.0)	0.16 (3.4)	0.28 (5.7)	0.42 (8.6)	0.54 (11.1)
Males										
1982-1984	2638	0.50 (10.4)	0.49-0.51 (10.2-10.5)	0.24 (5.0)	0.29 (6.0)	0.39 (8.0)	0.48 (10.0)	0.68 (14.0)	0.87 (18.0)	1.01 (21.0)
1988-1991	1797	0.19 (4.0)	0.16-0.23 (3.3-4.8)	0.06 (1.2)	0.08 (1.8)	0.12 (2.5)	0.20 (4.2)	0.31 (6.5)	0.45 (9.4)	0.57 (11.8)
Females										
1982-1984	3044	0.34 (7.0)	0.32-0.35 (6.7-7.2)	0.14 (3.0)	0.19 (4.0)	0.24 (5.0)	0.34 (7.0)	0.43 (9.0)	0.58 (12.0)	0.68 (14.0)
1988-1991	1814	0.11 (2.2)	0.09-0.13 (1.8-2.7)	<0.05 (<1.0)	<0.05 (<1.0)	0.07 (1.4)	0.12 (2.4)	0.19 (3.9)	0.31 (6.4)	0.41 (8.4)
Income level, low†										
1982-1984	2460	0.42 (8.8)	0.42-0.44 (8.6-9.1)	0.19 (4.0)	0.24 (5.0)	0.29 (6.0)	0.43 (9.0)	0.58 (12.0)	0.77 (16.0)	0.92 (19.0)
1988-1991	1664	0.16 (3.3)	0.13-0.19 (2.7-4.0)	<0.05 (<1.0)	0.06 (1.2)	0.10 (2.0)	0.17 (3.6)	0.26 (5.6)	0.43 (9.0)	0.54 (11.1)
Income level, mid†										
1982-1984	2032	0.40 (8.3)	0.38-0.42 (7.9-8.7)	0.19 (4.0)	0.24 (5.0)	0.29 (6.0)	0.43 (9.0)	0.58 (12.0)	0.77 (16.0)	0.87 (18.0)
1988-1991	1024	0.13 (2.6)	0.11-0.15 (2.2-3.1)	<0.05 (<1.0)	<0.05 (<1.0)	0.06 (1.6)	0.14 (2.9)	0.23 (4.8)	0.36 (7.5)	0.44 (9.2)
Income level, high†										
1982-1984	674	0.39 (8.1)	0.37-0.39 (7.6-8.6)	0.19 (4.0)	0.24 (5.0)	0.29 (6.0)	0.39 (8.0)	0.53 (11.0)	0.72 (15.0)	0.82 (17.0)
1988-1991	393	0.11 (2.3)	0.09-0.14 (1.8-2.9)	<0.05 (<1.0)	<0.05 (<1.0)	0.06 (1.6)	0.12 (2.5)	0.19 (4.0)	0.28 (5.7)	0.35 (7.3)

*For each grouping, the geometric means from HHANES and NHANES III phase 1 are statistically different ($P<.01$).

†Income level was defined by poverty-income ratio (PIR) categorized as low ($0<PIR<1.30$), mid ($1.30\leq PIR<3.00$), and high ($PIR\geq 3.00$).

housing. The distribution of blood lead levels in the NHANES reflects exposure in the general population, whereas studies focusing on high-risk populations, such as persons living in older, deteriorating housing, may find a different blood lead distribution. Data from national housing surveys indicate that in 1980 about 24.2 million (30.3%) occupied houses in the United States were built before 1940 when lead-based paint was in common use. By 1989, this number had decreased by 3.4 million to 20.8 million (22.2%), suggesting that population exposure to lead-based paint may have decreased slightly.^{21,22} On the other hand, the continuing deterioration of lead-based paint in existing houses could increase the likelihood of exposure for per-

sons in the 20.8 million households who remained in these older houses. On a population scale, it is not clear whether the net effect is an increase or decrease in exposure to lead-based paint.

The consistent decline in blood lead levels across broad population categories of age, sex, race/ethnicity, urban status, and income level most probably reflect changes in exposure to major population-wide lead sources. In addition, selected population groups within the United States are likely to have benefited from other changes in exposure, such as reductions in lead in community water supplies and reduction of lead emissions from local industry.

The public health impact of the observed decline in blood lead levels of the

US population is dramatic, especially for children. The change in the proportion of children aged 1 to 5 years with blood lead levels 0.48 µmol/L (10 µg/dL) or greater was at least 70% for non-Hispanic whites, non-Hispanic blacks and Mexican Americans. Although the decline in blood lead levels is encouraging, the number of children with lead levels 0.48 µmol/L (10 µg/dL) or greater remains substantial and disproportionately higher for non-Hispanic black children (one in five children), as discussed in the accompanying article in this issue.⁹

At least 99.8% of lead in gasoline has already been removed, and domestically produced cans are no longer lead soldered. Therefore, to achieve additional

Table 4.—Percentage of Mexican Americans Aged 4 to 74 Years at or Above Selected Blood Lead Level Cutoffs by Age, Sex, and Income Level: 1982 to 1984 (Hispanic Health and Nutrition Examination Survey) and 1988 to 1991 (Phase 1 of the Third National Health and Nutrition Examination Survey)

	No.	Blood Lead Levels of Population Group, %					
		≥1.45 μmol/L (≥30 μg/dL)	≥1.21 μmol/L (≥25 μg/dL)	≥0.97 μmol/L (≥20 μg/dL)	≥0.72 μmol/L (≥15 μg/dL)	≥0.48 μmol/L (≥10 μg/dL)	≥0.24 μmol/L (≥5 μg/dL)
All persons							
1982-1984	5682	0.4	1.3	3.6	12.8	41.2	91.2
1988-1991	3611	0.0	0.2	0.4	1.5	5.9	28.9
Ages 4-5 y							
1982-1984	269	2.4	4.9	8.8	24.7	61.5	96.4
1988-1991	349	0.0	0.0	0.0	0.1	4.9	32.7
Ages 6-19 y							
1982-1984	2331	0.3	0.5	2.0	9.0	35.8	90.1
1988-1991	1188	0.0	0.1	0.4	0.9	4.5	23.8
Ages 20-74 y							
1982-1984	3082	0.3	1.5	4.2	14.1	42.9	91.5
1988-1991	2074	0.0	0.2	0.4	1.9	6.6	31.0
Males							
1982-1984	2838	0.6	2.1	6.2	21.1	58.4	96.8
1988-1991	1797	0.0	0.2	0.5	2.2	8.7	40.5
Females							
1982-1984	3044	0.1	0.4	1.1	4.5	23.8	85.6
1988-1991	1814	0.0	0.1	0.3	0.7	2.8	16.4
Income level, low*							
1982-1984	2460	0.5	1.4	4.1	14.6	45.2	92.2
1988-1991	1684	0.0	0.1	0.4	1.7	7.3	33.5
Income level, mid*							
1982-1984	2032	0.4	1.3	3.4	11.4	38.0	90.1
1988-1991	1024	0.0	0.0	0.3	1.1	4.2	23.6
Income level, high*							
1982-1984	674	0.2	0.8	2.7	10.0	35.8	91.2
1988-1991	383	0.0	0.0	0.0	0.0	1.6	14.7

*Income level was defined by poverty-income ratio (PIR) categorized as low (0<PIR≤1.30), mid (1.30<PIR<3.00), and high (PIR≥3.00).

reductions in blood lead levels in the US population, sources other than lead in gasoline and lead in solder need to be

addressed further. The major remaining sources are lead in paint and lead that has already accumulated in dust

and soil. Without efforts to reduce these exposures, population blood lead levels are unlikely to continue to decline.

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Table 2.—Percentage of Persons Aged 1 to 74 Years at or Above Selected Blood Lead Level Cutoffs by Age, Sex, Race/Ethnicity, Urban Status, and Income Level: United States, 1976 to 1980 (Second National Health and Nutrition Examination Survey) and 1988 to 1991 (Phase 1 of the Third National Health and Nutrition Examination Survey)

	No.	Blood Lead Levels of Population Group, %					
		≥1.45 $\mu\text{mol/L}$ (≥30 $\mu\text{g/dL}$)	≥1.21 $\mu\text{mol/L}$ (≥25 $\mu\text{g/dL}$)	≥0.97 $\mu\text{mol/L}$ (≥20 $\mu\text{g/dL}$)	≥0.72 $\mu\text{mol/L}$ (≥15 $\mu\text{g/dL}$)	≥0.48 $\mu\text{mol/L}$ (≥10 $\mu\text{g/dL}$)	≥0.24 $\mu\text{mol/L}$ (≥5 $\mu\text{g/dL}$)
All persons							
1976-1980	9832	1.9	5.2	14.9	37.7	77.8	99.2
1988-1991	12119	0.2	0.4	0.8	1.1	4.3	23.3
Ages 1-5 y							
1976-1980	2271	4.1	9.3	24.7	52.6	88.2	99.8
1988-1991	2234	0.4	0.5	1.1	2.7	8.9	33.2
Ages 6-19 y							
1976-1980	2024	0.6	2.4	6.2	27.7	71.7	99.1
1988-1991	2963	0.0	0.2	0.4	0.8	2.6	12.2
Ages 20-74 y							
1976-1980	5537	2.3	5.9	16.7	40.3	79.4	99.2
1988-1991	6922	0.3	0.4	0.7	1.1	4.4	25.5
Males							
1976-1980	4895	3.3	9.0	24.1	53.1	89.6	99.8
1988-1991	6051	0.4	0.7	1.1	1.9	6.8	33.5
Females							
1976-1980	4937	0.6	1.6	6.2	23.0	66.7	98.7
1988-1991	6068	0.1	0.1	0.2	0.4	1.8	13.2
Non-Hispanic whites							
1976-1980	6816	1.7	4.8	14.0	36.0	76.9	99.2
1988-1991	4337	0.2	0.4	0.8	0.9	3.6	21.1
Non-Hispanic blacks							
1976-1980	1259	2.8	8.4	22.9	50.9	86.4	99.7
1988-1991	3274	0.2	0.4	1.2	2.6	8.5	33.7
Non-central city							
1976-1980	7112	1.9	4.9	13.9	35.3	75.7	99.0
1988-1991	7495	0.1	0.3	0.6	0.9	3.5	21.7
Central city, <1 million							
1976-1980	1612	1.9	6.1	17.1	43.1	82.1	99.8
1988-1991	2909	0.3	0.3	0.6	1.8	5.9	26.9
Central city, ≥1 million							
1976-1980	1106	1.8	6.0	18.4	44.4	84.8	99.9
1988-1991	1379	1.1	1.4	1.9	2.9	9.8	36.0
Income level, low*							
1976-1980	2546	2.9	6.8	18.0	39.6	78.4	99.2
1988-1991	4106	0.5	0.9	1.6	2.6	8.8	32.6
Income level, mid*							
1976-1980	4176	1.7	4.6	13.8	36.3	76.1	99.3
1988-1991	4050	0.2	0.3	0.5	0.9	3.4	22.9
Income level, high*							
1976-1980	2784	1.5	5.1	14.5	36.3	79.8	99.4
1988-1991	2781	0.1	0.3	0.4	0.6	2.7	18.4

*Income level was defined by poverty-income ratio (PIR) categorized as low ($0 < \text{PIR} < 1.30$), mid ($1.30 \leq \text{PIR} < 3.00$), and high ($\text{PIR} \geq 3.00$).

dL) or greater between NHANES II and NHANES III phase 1 was consistent across the entire age range (Fig 4).

The percentage of the population with blood lead levels at or above selected values is presented in Table 2. These levels were chosen in part because of their prior or potential use in public health policy. For those aged 1 to 74 years, the prevalence of blood lead levels $0.48 \mu\text{mol/L}$ ($10 \mu\text{g/dL}$) or greater decreased from 77.8% in NHANES II to 4.3% in NHANES III phase 1. For children aged 1 to 5 years during the same time frame, the prevalence of blood lead levels $0.48 \mu\text{mol/L}$ ($10 \mu\text{g/dL}$) or greater decreased from 88.2% to 8.9%.

The change in percentage of children at or above selected lead levels from NHANES II to NHANES III phase 1 is presented in Fig 5.

Separate analysis by race/ethnicity revealed that geometric mean blood lead levels declined by 77%, from 0.66 to $0.15 \mu\text{mol/L}$ (13.7 to $3.2 \mu\text{g/dL}$), for non-Hispanic white children and by 72%, from 0.97 to $0.27 \mu\text{mol/L}$ (20.2 to $5.6 \mu\text{g/dL}$), for non-Hispanic black children. The prevalence of blood lead levels $0.48 \mu\text{mol/L}$ ($10 \mu\text{g/dL}$) or greater for children in this same age group declined from 85.0% to 5.5% for non-Hispanic white children and from 97.7% to 20.6% for non-Hispanic black children.

Mean blood lead levels decreased from

0.73 to $0.20 \mu\text{mol/L}$ (15.2 to $4.1 \mu\text{g/dL}$) for children aged 1 to 2 years and from 0.71 to $0.16 \mu\text{mol/L}$ (14.8 to $3.4 \mu\text{g/dL}$) for children aged 3 to 5 years. During the same time period, the prevalence of blood lead levels $0.48 \mu\text{mol/L}$ ($10 \mu\text{g/dL}$) or greater also decreased from 88.3% to 11.5% for children aged 1 to 2 years and from 88.1% to 7.3% for children aged 3 to 5 years.

Mean blood lead levels decreased by 60% (1.16 to $0.47 \mu\text{mol/L}$ [24.0 to $9.7 \mu\text{g/dL}$]) for non-Hispanic black children from low-income families living in the central cities with populations of 1 million or more. This compares with an overall decrease in blood lead levels of 75% (0.72 to $0.18 \mu\text{mol/L}$ [14.9

to 3.6 $\mu\text{g/dL}$) for all children aged 1 to 5 years.

From HHANES (1982 to 1984) to NHANES III Phase 1 (1988 to 1991)

The HHANES was conducted from 1982 to 1984, between the second and third NHANES. Geometric mean blood lead levels were also found to be intermediate between the estimates of the two national surveys. The blood lead levels of Mexican Americans from HHANES were lower than overall levels observed in NHANES II, but not as low as levels of Mexican Americans sampled in NHANES III phase 1.

Geometric means, 95% confidence intervals, and percentiles of the blood lead distribution of Mexican Americans between HHANES and NHANES III phase 1 are presented in Table 3. Mexican Americans showed an overall decrease in geometric mean of 65%, from 0.41 to 0.14 $\mu\text{mol/L}$ (8.5 to 3.0 $\mu\text{g/dL}$). The geometric mean for children aged 4 to 5 years declined from 0.52 to 0.17 $\mu\text{mol/L}$ (10.9 to 3.5 $\mu\text{g/dL}$). As demonstrated in the comparison of NHANES II to NHANES III phase 1 estimates, the size of the decrease in blood lead levels was similar in both sexes and across age groups and income levels.

The proportion of the Mexican-American population at or above selected blood lead levels is shown in Table 4. Overall, prevalence of blood lead levels 0.48 $\mu\text{mol/L}$ (10 $\mu\text{g/dL}$) or greater among Mexican Americans decreased from 41.2% to 5.9%. The percentage of children aged 4 to 5 years with blood lead levels 0.48 $\mu\text{mol/L}$ (10 $\mu\text{g/dL}$) or greater decreased from 61.5% to 4.9%. These results demonstrate that one in 20 Mexican Americans aged 4 to 5 years continue to have blood lead levels of health concern.

COMMENT

The data from two national surveys of the US population, conducted more than a decade apart, demonstrate a substantial decline in blood lead levels. As the consequence of a shift in the overall distribution of lead levels, fewer persons have blood lead levels in the upper ranges. The decrease in mean blood lead levels was observed for the total population and within all race/ethnicity, sex, urban status, and income level subgroups examined in this article. The prevalence of blood lead levels 0.48 $\mu\text{mol/L}$ (10 $\mu\text{g/dL}$) or greater also decreased sharply from 77.8% to 4.3%.

As discussed herein, exposure to lead from major population-wide lead sources declined between 1976 and 1991. Consistent with this decline, the blood lead levels observed in HHANES (1982 to

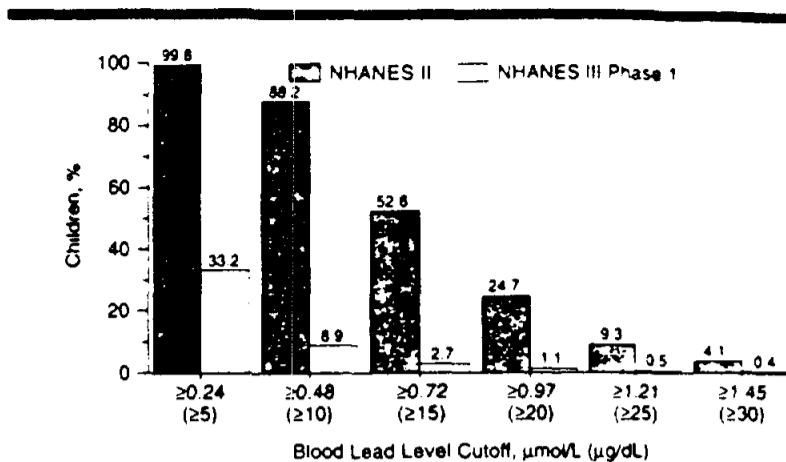


Fig 5.—Percentage of children aged 1 to 5 years at or above selected blood lead levels: United States, 1976 to 1980 (second National Health and Nutrition Examination Survey [NHANES II]) and 1988 to 1991 (phase 1 of the third National Health and Nutrition Examination Survey [NHANES III phase 1]).

1984) were intermediate between levels found in NHANES II (1976 to 1980) and NHANES III phase 1 (1988 to 1991). Consequently, the magnitude of decrease from HHANES to NHANES III phase 1 (65%) was less than from NHANES II to NHANES III phase 1 (78%). The percentage of Mexican Americans with blood lead levels 0.48 $\mu\text{mol/L}$ (10 $\mu\text{g/dL}$) or greater declined from 41.2% to 5.9%. In NHANES III phase 1, both mean blood lead levels and the prevalence of blood lead levels 0.48 $\mu\text{mol/L}$ (10 $\mu\text{g/dL}$) or greater of Mexican Americans were closer to those of non-Hispanic whites than to those of non-Hispanic blacks.

The decline in blood lead levels seen in these national surveys is consistent with the results of other studies of environmental lead levels,¹ which indicate that a continued reduction in exposure to lead sources began in the late 1970s and continued throughout the 1980s. Between 1976 and 1991, the three major sources of lead exposure common to the general population were lead in gasoline, soldered cans, and paint. In 1976, a total of 186.47 million kg (205 810 tons) of lead was used in gasoline in the United States.¹⁴ In 1983, this amount had dropped to 51.59 million kg (56 940 tons), and in 1990, lead used in gasoline had been reduced to 0.47 million kg (520 tons).¹⁵ From 1976 to 1990, the amount of lead used in gasoline decreased 99.8%. The reduction of lead in gasoline is most likely the greatest contributor to the observed decline in blood lead levels during the period of the national surveys.^{1,2,14,17}

Lead from gasoline and soldered cans contribute to lead in food. Since gasoline lead enters food through multiple pathways,^{1,14,16} it is difficult to make a quan-

titative estimate of the reduction in food lead that resulted from decreasing lead in gasoline. The amount of lead used in soldered cans decreased markedly throughout the 1980s. In 1980, 47% of food and soft drink cans were lead soldered. By 1985, this figure had dropped to 14%, and by 1990, only 0.85% of food and soft drink cans were lead soldered.¹⁸ As of November 1991, lead-soldered food or soft drink cans were no longer manufactured in the United States.¹⁸

The Food and Drug Administration uses "market-basket" surveys to estimate the average daily intake of lead from food for various population groups in the United States.¹⁹ For 2-year-old children, these surveys estimate the typical daily intake of lead to have dropped from 30 $\mu\text{g/d}$ in 1982 to 1.9 $\mu\text{g/d}$ in 1991.^{19,20} The Environmental Protection Agency estimated in 1986 that about 42% of lead in food came from lead-soldered cans.¹ Thus, reducing the amount of lead used in soldered cans has likely been a major factor in reducing food lead levels. Although it is difficult to quantitatively determine the decrease in blood lead levels attributable to reduced amounts of lead in soldered cans, the decline in the amount of lead used in this source probably contributed substantially to the observed decline in blood lead levels.

The manufacture of lead-based paint was limited to less than 0.06% by weight in 1978 by the Consumer Product Safety Commission.¹ Individuals who have left housing with lead-based paint or who reside in lead-abated homes have reduced their lead exposure. Still, lead-based paint remains a problem, predominantly in older, deteriorating housing.^{1,14,16} The NHANES do not specifically target persons who live in such

Table 1.—Distribution of Blood Lead Levels for Persons Aged 1 to 74 Years by Age Category, Sex, Race/Ethnicity, Urban Status, and Income Level: United States, 1976 to 1980 (Second National Health and Nutrition Examination Survey [NHANES II]) and 1988 to 1991 (Phase 1 of the Third National Health and Nutrition Examination Survey [NHANES III Phase 1])

	No.	Geometric Mean, µmol/L (µg/dL)*	95% Confidence Interval, µmol/L (µg/dL)	Percentiles, µmol/L (µg/dL)						
				5th	10th	25th	50th	75th	90th	95th
All persons										
1976-1980	9832	0.62 (12.8)	0.60-0.65 (12.4-13.3)	0.34 (7.0)	0.39 (8.0)	0.48 (10.0)	0.63 (13.0)	0.82 (17.0)	1.01 (21.0)	1.21 (25.0)
1988-1991	12 119	0.14 (2.8)	0.13-0.15 (2.7-3.0)	<0.05 (<1.0)	0.05 (1.0)	0.09 (1.8)	0.14 (3.0)	0.23 (4.8)	0.35 (7.3)	0.45 (9.4)
Ages 1-5 y										
1976-1980	2271	0.71 (15.0)	0.67-0.75 (14.2-15.8)	0.39 (8.0)	0.43 (9.0)	0.58 (12.0)	0.72 (15.0)	0.92 (19.0)	1.16 (24.0)	1.35 (28.0)
1988-1991	2234	0.17 (3.6)	0.16-0.19 (3.3-4.0)	0.05 (1.1)	0.07 (1.5)	0.11 (2.2)	0.18 (3.7)	0.28 (5.9)	0.46 (9.6)	0.59 (12.2)
Ages 6-19 y										
1976-1980	2024	0.56 (11.7)	0.54-0.60 (11.2-12.4)	0.29 (6.0)	0.34 (7.0)	0.43 (9.0)	0.58 (12.0)	0.72 (15.0)	0.92 (19.0)	1.06 (22.0)
1988-1991	2963	0.09 (1.9)	0.08-0.11 (1.7-2.2)	<0.05 (<1.0)	<0.05 (<1.0)	0.06 (1.3)	0.10 (2.1)	0.17 (3.5)	0.26 (5.4)	0.36 (7.4)
Ages 20-74 y										
1976-1980	5537	0.63 (13.1)	0.61-0.66 (12.7-13.7)	0.34 (7.0)	0.39 (8.0)	0.48 (10.0)	0.63 (13.0)	0.82 (17.0)	1.06 (22.0)	1.25 (26.0)
1988-1991	6922	0.14 (3.0)	0.14-0.15 (2.8-3.2)	<0.05 (<1.0)	0.06 (1.2)	0.10 (2.0)	0.15 (3.2)	0.24 (5.0)	0.36 (7.4)	0.46 (9.5)
Males										
1976-1980	4895	0.72 (15.0)	0.70-0.75 (14.5-15.5)	0.39 (8.0)	0.43 (9.0)	0.58 (12.0)	0.72 (15.0)	0.92 (19.0)	1.16 (24.0)	1.30 (27.0)
1988-1991	6051	0.18 (3.7)	0.17-0.19 (3.5-3.9)	0.06 (1.2)	0.08 (1.8)	0.12 (2.4)	0.18 (3.8)	0.28 (5.8)	0.42 (8.7)	0.52 (10.9)
Females										
1976-1980	4937	0.54 (11.1)	0.51-0.55 (10.6-11.5)	0.29 (6.0)	0.34 (7.0)	0.43 (9.0)	0.53 (11.0)	0.68 (14.0)	0.87 (18.0)	0.97 (20.0)
1988-1991	6068	0.10 (2.1)	0.10-0.11 (2.0-2.2)	<0.05 (<1.0)	<0.05 (<1.0)	0.07 (1.4)	0.11 (2.3)	0.18 (3.8)	0.28 (5.7)	0.36 (7.4)
Non-Hispanic whites										
1976-1980	6816	0.61 (12.6)	0.58-0.63 (12.1-13.1)	0.29 (6.0)	0.39 (8.0)	0.48 (10.0)	0.63 (13.0)	0.77 (16.0)	1.01 (21.0)	1.16 (24.0)
1988-1991	4337	0.13 (2.7)	0.12-0.14 (2.2-2.8)	<0.05 (<1.0)	0.05 (1.0)	0.08 (1.7)	0.14 (2.9)	0.22 (4.5)	0.33 (6.8)	0.43 (8.9)
Non-Hispanic blacks										
1976-1980	1259	0.70 (14.5)	0.66-0.74 (13.7-15.5)	0.39 (8.0)	0.43 (9.0)	0.53 (11.0)	0.72 (15.0)	0.92 (19.0)	1.11 (23.0)	1.30 (27.0)
1988-1991	3274	0.17 (3.5)	0.16-0.19 (3.3-3.9)	<0.05 (<1.0)	0.06 (1.3)	0.11 (2.2)	0.18 (3.7)	0.28 (5.9)	0.45 (9.3)	0.58 (12.1)
Non-central city										
1976-1980	7112	0.60 (12.5)	0.58-0.64 (12.0-13.1)	0.29 (6.0)	0.34 (7.0)	0.48 (10.0)	0.63 (13.0)	0.77 (16.0)	1.01 (21.0)	1.16 (24.0)
1988-1991	7495	0.13 (2.7)	0.12-0.14 (2.5-2.8)	<0.05 (<1.0)	0.05 (1.0)	0.09 (1.8)	0.14 (3.0)	0.22 (4.6)	0.33 (6.9)	0.43 (8.9)
Central city, <1 million										
1976-1980	1612	0.66 (13.6)	0.61-0.70 (12.7-14.5)	0.34 (7.0)	0.39 (8.0)	0.53 (11.0)	0.68 (14.0)	0.87 (18.0)	1.08 (22.0)	1.25 (26.0)
1988-1991	2908	0.14 (2.9)	0.12-0.16 (2.5-3.4)	<0.05 (<1.0)	0.05 (1.0)	0.09 (1.8)	0.14 (3.0)	0.25 (5.2)	0.40 (8.3)	0.50 (10.4)
Central city, ≥1 million										
1976-1980	1108	0.67 (13.9)	0.61-0.73 (12.7-15.1)	0.34 (7.0)	0.43 (9.0)	0.53 (11.0)	0.68 (14.0)	0.87 (18.0)	1.06 (22.0)	1.21 (25.0)
1988-1991	1379	0.19 (3.9)	0.17-0.21 (3.6-4.3)	0.06 (1.3)	0.09 (1.8)	0.12 (2.5)	0.19 (4.0)	0.29 (6.1)	0.48 (9.9)	0.64 (13.2)
Income level, low†										
1976-1980	2548	0.63 (13.1)	0.60-0.67 (12.4-13.8)	0.29 (6.0)	0.34 (7.0)	0.48 (10.0)	0.63 (13.0)	0.82 (17.0)	1.11 (23.0)	1.25 (26.0)
1988-1991	4106	0.16 (3.4)	0.15-0.16 (3.1-3.8)	<0.05 (<1.0)	0.06 (1.3)	0.10 (2.1)	0.17 (3.6)	0.28 (5.8)	0.45 (9.4)	0.57 (11.8)
Income level, mid†										
1976-1980	4178	0.61 (12.6)	0.58-0.63 (12.1-13.1)	0.34 (7.0)	0.39 (8.0)	0.48 (10.0)	0.63 (13.0)	0.77 (16.0)	1.01 (21.0)	1.16 (24.0)
1988-1991	4050	0.13 (2.7)	0.13-0.14 (2.6-2.9)	<0.05 (<1.0)	0.05 (1.0)	0.08 (1.7)	0.14 (2.9)	0.23 (4.7)	0.34 (7.1)	0.44 (9.1)
Income level, high†										
1976-1980	2784	0.63 (13.0)	0.60-0.65 (12.5-13.5)	0.34 (7.0)	0.39 (8.0)	0.48 (10.0)	0.63 (13.0)	0.82 (17.0)	1.01 (21.0)	1.21 (25.0)
1988-1991	2781	0.12 (2.5)	0.12-0.13 (2.4-2.7)	<0.05 (<1.0)	<0.05 (<1.0)	0.08 (1.7)	0.14 (2.8)	0.21 (4.3)	0.30 (6.3)	0.39 (8.0)

*For each grouping, the geometric means from NHANES II and NHANES III phase 1 are statistically different ($P<0.01$).
†Income level was defined by poverty-income ratio (PIR) categorized as low ($0<PIR<1.30$), mid ($1.30<PIR<3.00$), and high ($PIR≥3.00$).

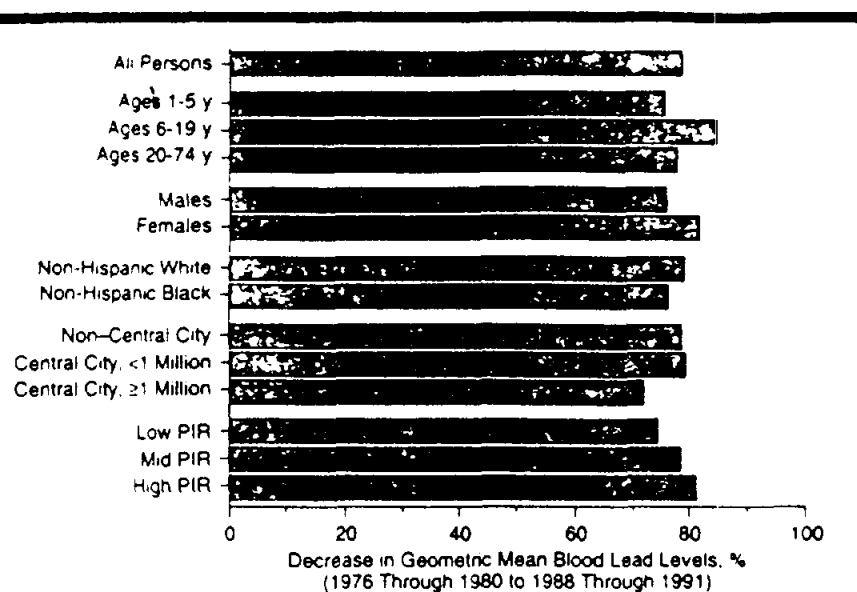


Fig 3.—Percentage decrease in geometric mean blood lead levels for persons aged 1 to 74 years by age category, sex, race, ethnicity, urban status, and income level: United States, 1976 to 1980 (second National Health and Nutrition Examination Survey) to 1988 to 1991 (phase 1 of the third National Health and Nutrition Examination Survey). Income level defined by poverty-income ratio (PIR) as low ($0 < \text{PIR} < 1.30$), mid ($1.30 \leq \text{PIR} < 3.00$), and high ($\text{PIR} \geq 3.00$).

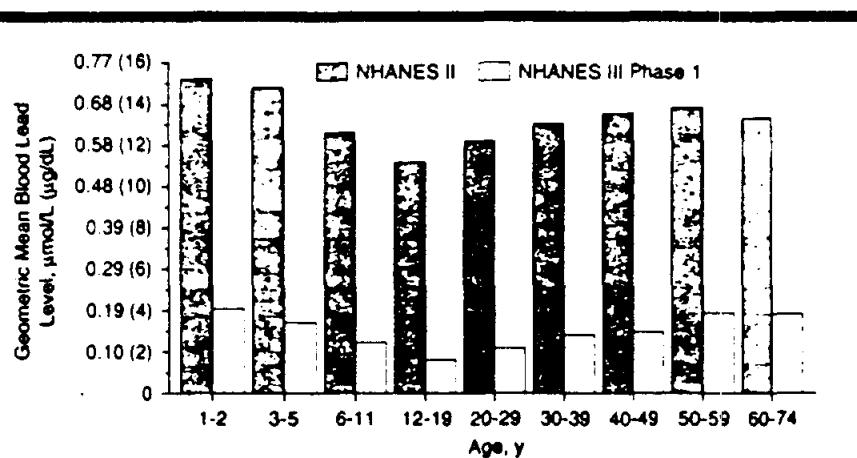


Fig 4.—Geometric mean blood lead levels for persons aged 1 to 74 years by age: United States, 1976 to 1980 (second National Health and Nutrition Examination Survey [NHANES II]) and 1988 to 1991 (phase 1 of the third National Health and Nutrition Examination Survey [NHANES III phase 1]).

veys, the blood lead measurements were calibrated using standards prepared from lead nitrate Standard Reference Material 928 obtained from the National Institute of Standards and Technology, Gaithersburg, Md. The consistent use of Standard Reference Material 928 for calibration assured a common accuracy base across surveys.

Demographic and Socioeconomic Covariates

The trends analysis included stratification by five sociodemographic variables: age, sex, race/ethnicity, urban status, and income level. Age was defined

in years and categorized as 1 to 5 years (4 to 5 years for Mexican Americans), 6 to 19 years, and 20 to 74 years for analysis. Race/ethnicity was categorized as non-Hispanic black, non-Hispanic white, and Mexican American. Because of small sample sizes, persons not defined by these three largest US race/ethnicity groups were included only in the overall estimates.

Definitions for income level and urban status were based on those previously determined by the US Bureau of the Census. Income level was defined by the poverty-income ratio (PIR): the total family income divided by a poverty

threshold. The PIR was divided into three categories: low ($0 < \text{PIR} < 1.30$), mid ($1.30 \leq \text{PIR} < 3.00$), and high ($\text{PIR} \geq 3.00$). Urban status was categorized as non-central city, central city with population less than 1 million, and central city with population 1 million or greater.

Statistical Analysis

Survey-specific sample weights were used in all statistical analyses. Geometric means and percentiles of blood lead were calculated using SAS.¹³ Log₁₀ transformed blood lead levels were used to normalize the distribution of blood lead levels. Geometric means were calculated by taking the antilog of the mean log₁₀ blood lead levels. SUDAAN,¹⁴ a statistical software package that incorporates the sample weights and adjusts for the complex sample design of the survey, was used to estimate the SEs.

RESULTS

The results of the trend analysis in blood lead levels are presented in two parts: first, the change in blood lead levels from NHANES II (1976 to 1980) to NHANES III phase 1 (1988 to 1991), and second, the change in blood lead levels for Mexican Americans from NHANES (1982 to 1984) to NHANES III phase 1 (1988 to 1991).

From NHANES II (1976 To 1980) to NHANES III Phase 1 (1988 To 1991)

The different distributions of blood lead levels for those aged 1 to 74 years from NHANES II and NHANES III phase 1 are presented in Fig 1. A decline of approximately 0.48 µmol/L (10 µg/dL) occurred in the geometric mean blood lead level as well as a clear change in the shape of the distribution. When the sample was limited to children aged 1 to 5 years, similar results were observed (Fig 2).

The geometric means, 95% confidence intervals, and percentiles of the blood lead distribution for the total population and stratified by the five sociodemographic factors are presented by survey in Table 1. For the total population, the geometric mean decreased by 0.48 µmol/L (10 µg/dL). Stratification of the data showed that the size of the decrease was fairly constant across sex, race/ethnicity, age groups, urban status, and income levels.

The decline represents an overall decrease in blood lead levels of 78% for persons aged 1 to 74 years and a decrease of 70% or more for selected subgroups (Fig 3). Children and youths aged 6 to 19 years showed the greatest decline in blood lead levels. However, a decline of 0.48 µmol/L (10 µg/dL)