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Project LEAP— Phase 1

Spatial And Numerical Dimensions of Young Minority Children Exposed to Low-Level Environmental Sources of Lead

SPATIAL AND NUMERICAL DIMENSIONS OF YOUNG MINORITY CHILDREN EXPOSED TO LOW-LEVEL ENVIRONMENTAL SOURCES OF LEAD

BY

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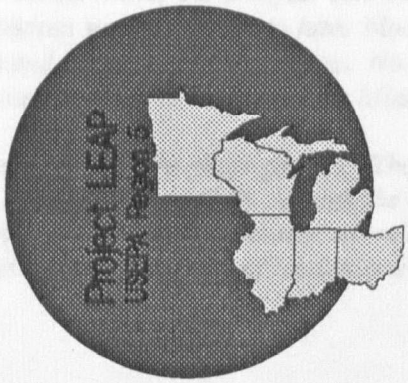

United States Environmental Protection Agency
Region 5, Chicago
Environmental Sciences Division
Project LEAP (Lead Education and Abatement Program)
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ABSTRACT

SPATIAL AND NUMERICAL DIMENSIONS OF
YOUNG MINORITY CHILDREN EXPOSED TO LOW-LEVEL
ENVIRONMENTAL SOURCES OF LEAD

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A population comparative risk algorithm was developed as a pilot study for the Agency's Lead Strategy, as a Region 5 Comparative Risk initiative, and as an environmental equity project. All known environmental sources of lead in 83 cities in the Midwest were assessed to develop a population comparative risk analysis for childhood exposure to lead. A secondary objective was to discern the association of proximity of transportation corridors, to elevations in blood-lead levels. The selected at risk population were African-American and Hispanic children under seven years of age. Measured and postulated values were derived to approximate lead concentrations in air, drinking water, soil, and dust. Sources included in the analysis were major point sources of lead and lead compounds (from the Toxic Release Inventory national data base), ambient air concentrations, reported drinking water concentrations, municipal waste combusters, abandoned hazardous waste sites, and operating hazardous waste facilities. Using concentrations specific to census tracts within each city, the EPA Uptake Biokinetic Model was used to estimate the probability distribution of blood-lead levels for each area, and to estimate the percent of children expected to exceed a criterion value of 10 μg/dL blood-lead.

Although considered to be conservative, the analysis concluded that in 1988 a total childhood population of 154,000 Midwest children were expected to have blood-lead levels exceeding 10 μg/dL, including 55,000 African-American and 12,000 Hispanic children. No association was found for proximity of transportation corridors to elevated lead-blood levels, for the Minneapolis/St. Paul, Minnesota, study area.

This report constitutes Phase 1 of a three phase project. The purpose of this phase is to screen a large number of cities for future lead reduction efforts through the use of a comparative risk analysis. Phase 2 will include testing in a small number of communities, as well as public education and outreach. Phase 3 will be remediation of environmental sources of lead in one or more communities.

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This study relies heavily upon previously gathered data, by design, and I want to thank those too numerous to mention that provided assistance and data for the analysis. Three individuals and organizations are, however, particularly noteworthy: Mr. Douglas M. Benson, Coordinator, Lead Program, Division of Environmental Health, Minnesota Department of Health, for graciously providing the blood-lead database for the Minneapolis/St. Paul Blood-lead Survey; the Minnesota Pollution Control Agency for providing the counterpart soil-lead data for the Twin Cities; and the U.S. Department of Housing and Urban Development, for providing the raw database used for the National Housing Survey.

Finally, my deepest appreciation to a husband and wife team in the U.S. Environmental Protection Agency, Environmental Sciences Division, Mr. Larry Lehrman and Mrs. Loretta Lehrman. The computer support, both hardware and software, and technical/programming assistance proved invaluable for efficacy in conducting the study. But, as important, their personal support and encouragement proved indispensable to the spirit, as I struggled through to completion.

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EXECUTIVE SUMMARY

This research considers the known environmental sources of lead in 83 cities in the Midwest, estimates the probability distribution of lead in African-American and Hispanic children (as well as the total childhood population) under seven years of age in each of the cities, and compares the numbers of children at risk. The approach thus developed is a population comparative risk screening methodology for ranking geographic areas as to potential lead toxicity. This data analysis report is the first phase of a three phase effort. Phase 2 will be to conduct sampling in a small number of communities, as well as to begin public outreach and education on the dangers of environmental exposure to lead. Phase 3 will be to conduct remediation of environmental sources of lead (e.g., soil and dust) in one or two communities.

The objective of Phase 1 is to estimate relative blood-lead levels in childhood populations and to compare geographic areas to ascertain the severity. For each metropolitan statistical central city area, environmental data were obtained for the major sources of exposure. This included stationary source air facilities, municipal waste combusters, ambient air quality measurements, drinking water supplies, and operating as well as abandoned hazardous waste sites. Where available, actual concentrations were used. Default values were established for each environmental medium where actual measurements had not been taken. Major air emission sources were modeled to calculate associated air concentrations. The results were used in a qualitative assessment of environmental exposure.

Demographic data were obtained from a geographic information systems application (provided by the Geographic Information Systems Management Office, Region 5, U.S. EPA). That office provided data at the census tract and community area (aggregation of census tracts) levels for each city. In general, a census tract has a population of about 4,000 people. Environmental data (air, drinking water, soil and dust

concentrations) associated with each tract were obtained in order to calculate blood-lead level distributions in affected populations.

Based upon these environmental concentrations for each census tract/community area, the Uptake Biokinetic Model (described in Section 4.7) was run to calculate an expected percent exceedance for each area. The percentage, applied against the population data for the tract, provided an estimate of the number of children under seven years of age at risk of lead exposure. Further aggregations allowed for a city total, as well as a numerical ranking of cities.

Data from a single geographical area, Minneapolis/St. Paul,Minnesota, was selected to test the methodology. That area had available measured blood-lead levels, along with pertinent demographic information. Two statistical procedures were performed. A simple correlation analysis was conducted to ascertain whether modeled blood-lead levels, based primarily upon the environmental data for the area, were associated with actual measured blood-lead levels. An association would indicate the viability of the approach in comparing cities. The correlation analysis indicates a correlation coefficient of 0.3. It is only statistically significant, however, at the 0.10 level.

The second statistical procedure was conducted to further analyze the contribution of environmental pathways of exposure to elevations in blood-lead levels and, in particular, to ascertain whether mobile sources (i.e., proximity to a major transportation corridor) could account for a portion of the elevation in blood-lead levels. No association was found.

An analysis of environmental data indicates that a tremendous quantity of lead is still being released into the environment, and that quite typically a small (relative) number of sources contribute most of the contaminant. For the six Midwest states, industry released nearly 450,000 pounds of lead and lead compounds into the air in 1988. Seventeen sources out of nearly 350 reporting facilities accounted for almost one-half of the total emissions. Nevertheless, air quality, based upon measurements of the ambient air, was excellent, with few exceedances of the primary air quality standard for lead. Point sources of

emissions, although many in number, generally do not cause concerns (a measurable increase in the ambient air-lead concentration). The notable exceptions are a few high emitting industries. For those industries, the increased ambient air-lead concentration, as modeled, is expected to occur near the source. Although there is a large amount of lead emitted into the air, only a few sources emit lead and lead compounds in sufficient amount to exceed the ambient air quality standard for air (refer to Section 5.5.2). Only two of 17 modeled stationary sources of air-lead emissions had calculated maximum point downwind air-lead concentration values projected to exceed the air quality standard of 1.5 $\mu g/m^3$. Drinking water supplies are also typically safe, although exposure does continue in some communities. Violations of the drinking water standard are rare.

Exposure to lead through soil and dust, associated with operating and abandoned hazardous waste sites, may occur in a few cities. The majority of sites, however, are located beyond the boundaries of the central cities assessed and, consequently, do not generally pose a threat.

The research placed special emphasis on the risk posed by low-level environmental sources of lead to African-American and Hispanic children. These populations are thought to be at particular risk (refer to Section 2.8). For children residing in central cities of one million population or more, and annual family income less than $6,000, 68 percent of African-American children are projected to have blood-lead levels exceeding 15 μg/dL. For white children in the same socioeconomic strata, the percent projected to exceed that value is much smaller, at 36 percent.

Seven cities in Region 5 are in the top 10 of the 83 cities assessed in the Midwest by virtue of having both the highest percentages of children as well as the greatest numbers of children that may exceed 10 μg/dL blood-lead concentration. Those cities are Milwaukee, Wisconsin; Detroit, Michigan; Minneapolis and St. Paul, Minnesota; and Cincinnati, Akron, and Cleveland, Ohio.

The analysis indicates that the States of Illinois and Michigan had the largest numbers of African-American and Hispanic children under seven years of age calculated to exceed 10 μg/dL blood-lead level.

This includes 28,000 and 16,000 minority children, in the respective states, due to environmental sources of lead. Every Region 5 state has community areas where elevated blood-lead levels are of concern.

For the six Region 5 states, all cities combined, the total childhood population under seven years of age was 1,359,000 in 1988. The findings indicate that 154,000 children, or 11 percent of the total, would have blood-lead levels exceeding 10 μg/dL. The predominant environmental sources are lead contaminated soil and dust. This includes 55,000 African-American and 12,000 Hispanic children.

The cities with the highest potential for sizable numbers of African-American and Hispanic children with blood-lead levels calculated as above 10 μg/dL are Chicago, Illinois, 27,000; Detroit, Michigan, 13,000; Milwaukee, Wisconsin, 5,000; Cleveland, Ohio, 4,000; Cincinnati, Ohio, 2,000; and Indianapolis, Indiana, 2,000.

It is important to note that this methodology is for population screening purposes. It expands upon the use of an Uptake Biokinetic Model for derivation of blood-lead levels. Such use of the model has not been attempted before. The Uptake Biokinetic Model was developed specifically for application at abandoned hazardous waste sites for which measured environmental lead concentrations are known. The Uptake Biokinetic Model has only been validated at that spatial scale. This methodology applies it at a much larger spatial scale. It includes both estimated and measured environmental concentrations, and uses the model as part of a population risk screening approach. Consequently, the results may have no practical value as a prediction of the actual number of children expected to have elevated blood-lead levels. Nor was that the intent of the methodology. The value of the approach is in the comparison between cities. It is specifically to locate areas within a city that may be expected to have higher rates of lead exposed children than other areas. The intent of the population screening methodology is to use the relative number to set priorities for intervention efforts within a city or region. The reader is particularly cautioned that the numbers of children cited in this research are as derived by the computerized methodology. The methodology is a screening tool. It is not a methodology to predict

actual number of children at risk.

1. INTRODUCTION

The insidious effects that lead causes on the health of children have received increased attention in recent years. Due to mouthing behavior, increased uptake of lead compared to adults, nutrition and other factors, children under seven years of age present a subpopulation at increased risk to the adverse effects of lead exposure. Within this population subgroup, it has been well demonstrated that African-Americans, particularly in lower socio-economic situations, are a subpopulation group at even greater risk. Hispanic children may also be at higher risk. The reasons for a dissimilarity between white and African-American children are unclear. It is clear, however, that the difference is seen at all socioeconomic levels. Measurements and projections of blood-lead levels for African-American children consistently reflect elevated blood-lead levels.

Reports from the second National Health and Nutrition Examination Survey, based upon data from 1976 to 1980, illustrates the substantial difference in blood-lead prevalence levels based upon ethnicity. Among African-American children six months to five years of age, only 2.5 percent of African-American children, compared to 14.5 percent of white children, had blood-lead levels less than 10 μg/dL (Lin-Fu, 1992). For families with an annual family income < $6,000, 18.5 percent of African-American children, contrasted to only 5.9 percent of white children, exceeded 30 μg/dL (children aged six months to five years). The percentage was 10.9 percent exceeding 30 μg/dL for all races. For that same age group, the geometric mean blood-lead level was 19.6 μg/dL for African-American children, 14 μg/dL for white children, and 14.9 μg/dL for all races. Although complete data are not available for children of Hispanic origin, the Agency for Toxic Substances Disease Registry (ATSDR, 1988) postulates that it is reasonable to assume that the association between high blood-lead levels and lower socioeconomic income status would hold true for this population as well. Hispanic children, accordingly, may also be at elevated risk.

As research continues, the level of blood-lead concentration of concern continues to be lowered.

More and more studies add to the weight of evidence for health effects in children at levels previously thought to be safe. The fact that lead is a transplacental contaminant is even more alarming because internal exposure can begin in the fetus. The exposure can continue to contribute to body tissue burden of the young child if the child is subsequently brought into a lead-contaminated environment. A significant evolving concern is that many of the effects of low-level lead exposure are not readily observable in the individual child, unlike physical manifestations caused by acute lead poisoning. Acute (observable) effects are usually associated with lead-based paint. Health effects are generally ascertained not through clinical diagnosis of the individual patient, but rather through epidemiologic study of large groups of children already suffering from the chronic effects of lead exposure. These chronic effects are generally not observed in the individual child. Effects may include lower intelligence and other neuropsychologic deficits, hearing impairment, stunted growth, reduction in attention span, and other reported health impacts. Some studies suggest the lack of a threshold. This is extremely problematic. Even though acute poisoning and exposure have been recognized, generally associated with lead-based paint contamination, chronic exposure and effects caused by low-level lead exposure in the environment are difficult to recognize.

This nation has experienced a tremendous reduction in lead emitted into the environment by the phase down of lead in gasoline. The reduction has been paralleled by a significant concomitant reduction of the average blood-lead levels in this country. Lead, however, remains pervasive in our environment. It is in the homes of tens of millions of families and serves as a continuous source of contamination and exposure via lead paint. Lead remains in some sources of drinking water in the home. It remains in soil and dust, caused potentially by both exterior and interior lead-based paint, as well as historical or ongoing deposition from mobile sources of nearby industry. Even the nation's food supply still contains some lead, albeit in small quantity. The aggregate effect from multiple sources, in a specific geographic area, may be sufficient to cause concern.

The major objective of this research is to examine environmental sources of lead that may be linked to chronic health effects in young children. In particular, such effects may be exacerbated by an aggregation of low-level environmental exposures to lead and lead compounds that result from multiple pathways of exposure. The research effort does not account for the direct effects of lead-based paint consumption. The methodology does take into account the indirect contribution to exposure from lead-based paint via lead-contaminated soil and dust. It is recognized, nevertheless, that lead-based paint provides the largest contribution to elevated blood-lead levels. This is particularly the case for acute lead-poisoning events. This effort, however, is to assess the extent to which low-level environmental sources of lead may also contribute to elevated blood-lead levels. It constitutes the first phase of Project LEAP: analysis of existing environmental data pursuant to a comparative risk analysis of childhood exposure to lead for the study cities. The goal is to discern a logical direction for future lead reduction efforts. Phases 2 and 3 will follow, to address lead testing and remediation, respectively.

This report documents the development of a management tool to identify and prioritize geographic areas having children with elevated environmental exposures to lead which may constitute a health risk to young children. The methodology explores the application of an Uptake Biokinetic Model, developed by the U.S. Environmental Protection Agency for site specific application, on a much larger scale than its original design and intent.

2. LITERATURE REVIEW

2.1. Toxicological Profile

"The EPA (1986a) and ATSDR (1988) are concerned that the emerging evidence of a consternation of effects, including inhibition of ALA-D activity and pyrimidine-5'-nucleotidase activity and reductions in serum 1,25-dihydrooxyvitamin D levels, is indicative that low-level lead exposure has a far reaching impact on fundamental enzymatic, energy transfer, and calcium homeostatic mechanisms in the body, which are expressed through subtle effects on neurobehavioral indices, growth, and blood pressure"(ATSDR,1990).

The evidence that low level lead exposure is a health concern, particularly for young children, has emerged from a host of studies concomitant with the recognition that today, such exposure has become pervasive in the United States. This is especially alarming as we gain a fuller understanding of the aggregate effects of the multitude of (external) environmental exposures that contribute to internal exposures. That internal exposure is typically assessed via ascertainment of the blood-lead (Pb-B) level, a measure historically associated not with low level, chronic exposure, but rather with the acutes effect caused by lead-in-paint poisoning.

2.1.1. Internal Exposure

For decades, scientists have recognized that high exposure to lead results in encephalopathy, colic, anemia, nephropathy, and electrocardiographic abnormalities. High exposure can cause spontaneous abortions in females, and decreased fertility in men (ATSDR, 1990). McMichael et al. (1986) reported on miscarriage and still births among pregnant women. ATSDR (1990) notes that the primary source of lead (Pb) in children is via the gastrointestinal tract. It is distributed in blood, soft tissue, and bone. In human blood, 99 percent of the lead in blood in attached to erythrocytes (with over 50 percent of this pool bound to hemoglobin) (ATSDR, 1990). The balance is deposited in blood plasma, and can be transported to soft tissues. Lead in bones is found in two components, an inert pool with a half life of decades, and

a labile pool having the ability to exchange readily between bone and blood or soft tissue (ATSDR, 1990). According to a model proposed by Rabinowitz et al. (1976), the blood component half life is 36 days, soft tissue is 40 days, and bone is 10^4 days (about 27 years). A number of age related differences exists between lead distribution and body burden of children, in comparison to adults. In a controlled experiment, Griffin et al. (1975) found that blood levels returned to near-normal after about two months subsequent to termination of exposure to airborne lead. In contrast, the biological half life in two year old children has been measured to be about 10 months, (Succop et al., 1987). Further, in adults, about 95 percent of the total body burden is in bones, while in children, the percentage is approximately 73 percent (ATSDR, 1990). It is noted that lead accumulation in most soft tissues (the kidney, brain, and liver) is of much smaller proportion than lead which accumulates in bone. Blood-lead which is not retained in one of these compartments is excreted by the kidney, or is excreted through biliary clearance into the gastrointestinal tract (ATSDR, 1990). It is also noted that the physiological stress of pregnancy can mobilize lead from maternal bone. This creates additional exposure for the developing fetus, resulting, consequently, in greater danger to the fetus. The transplacental transfer of lead has been cited in a number of studies over the years. In a Glasgow, Scotland, study of 236 mothers and infants, the geometric mean blood-lead levels were found to be 14 μg/dL for mothers, and 12 μg/dL for infants (Moore et al., 1982). According to the Public Health Service (ATSDR, 1988), there is no metabolic barrier to fetus uptake of lead; consequently, exposure of women during pregnancy results in lead uptake by the fetus (i.e., physiological stress results in increased exposure of the fetus). Differential internal exposure risk appears to continue after birth. Infants from birth to two years have been shown to retain 32 percent of the total amount of lead absorbed, according to a study by Ziegler et al. (1978); whereas a study by Rabinowitz et al. (1977) discerned a one percent retention rate in adults of the absorbed amount of inspired lead, derived by ATSDR from the Rabinowitz et al. study data. The Rabinowitz et al. study itself found that the average respired lead intake of 14 μg/day, inhaling air containing 2 $\mu g/m^3$ lead,

resulted in a calculated increase of 0.06 μg/gm in the blood-lead level.

The interaction of lead with other chemicals in the body has also been extensively studied, and is a matter of concern. "In humans, the interactive behavior of lead and various nutritional factors is appropriately viewed as particularly significant for children, since this age group is not only particularly sensitive to the effects of lead, but also experiences the greatest changes in relative nutrient status" (ATSDR, 1990). Data supporting this conclusion is available from a number of sources. Studies have found that calcium intake is inversely correlated with increasing blood-lead levels (ATSDR, 1990). Watson et al. (1980) reported that iron deficient adults absorbed lead two to three times greater than lead-replete adults (thus 20 to 30 percent of dietary input, versus 10 percent). Studies have found increased lead absorption with low dietary calcium, increased lead absorption and toxicity with iron deficiency, and that low zinc in the diet increases lead absorption (ATSDR, 1990). Mahaffey (1990) found that lead absorption and toxicity increased for subjects with diets low in calcium. He also found that long term iron deficiency, as well, increased the absorption and retention of lead. Mahaffey concluded that longitudinal, prospective studies are needed to evaluate the effectiveness of nutrition as a preventive strategy for lead intoxication.

Lower-level exposures affect the synthesis of heme, and decreases the circulating levels of the active form of vitamin D, 1,25-dihydroxyvitamin D, in children. "This form of vitamin D is largely responsible for the maintenance of calcium homeostasis in the body" (ATSDR, 1990). In a study by Rosen et al. (1980), the researchers found that lead-exposed children with Pb-B levels of 33 to 120 μg/dL had notable reductions in serum levels for both 1,25-dihydroxyvitamin D and Pb-B over the entire range of blood-lead levels measured in the study. EPA (1986a) concludes that lead's interference with heme synthesis may be the basis for the effects on vitamin D metabolism.

Low-level lead exposures causes inhibition of erythrocyte ALA-D, down to the lowest observed blood-lead levels of approximately three to five μg/dL (ATSDR, 1990). This has been confirmed

particularly for child studies by Secchi et al. (1974) (minimum subject Pb-B value of 16 μg/dL), Wada et al. (1973), Hernberg and Nikkanen (1970), Chisolm et al. (1985a), and Roels et al. (1976) (mimimum subject Pb-B value of 4.7 μg/dL). The lowest observed adverse effects level (LOAEL) for ALA-D and heme synthesis is thought to occur below 10 μg/dL (ATSDR, 1990).

Based upon a review of studies by EPA (1986a), ATSDR (1988), and Grant and Davis (1989) the threshold for accumulation of erythrocyte protoporphyrin (EP) or zinc protoporphyrin is approximately 15 μg/dL, the presumed Lowest Observed Adverse Effects Level (LOAEL) for children. EPA (1986a) concluded that inhibition of the enzyme erythrocyte pyrimidine-5'-nucleotidase may occur in workers at Pb-B levels at or exceeding 44 μg/dL, and in children that inhibition of the enzyme is seen down through the lowest blood-lead levels of approximately seven μg/dL, based upon data of Angle et al. (1978) and Angle et al. (1982). The LOAEL for children consequently appears to occur at less than 10 μg/dL under intermediate and chronic exposure scenarios. A study by Rosen et al. (1980) found strong indication of an inverse correlation between Pb-B and serum 1,25-dihydroxyvitamin D, that was observed in children over the blood-lead levels measured in the study, from 33 to 120 μg/dL.

2.1.2.Encephalopathy/Lethality

For the oral route of exposure, the range of blood-lead levels associated with encephalopathy in children was about 90 to 700 or 800 μg/dL, with a mean of approximately 330 μg/dL (ATSDR, 1990). The range associated with death is approximately 125 to 750 μg/dL, with a mean of 327 μg/dL. For the inhalation route of exposure in adults, lead encephalopathy is the most severe neurobehavioral effect (ATSDR, 1990). Early symptoms include dullness, irritability, poor attention span, headache, muscular tremor, loss of memory, and hallucinations. The condition can worsen to delirium, convulsions, paralysis, coma, and, ultimately, death. This is generally not observed in adults until levels exceed 120 μg/dL. Such studies of signs and symptoms indicate that the lowest observed-effect levels for overt signs and symptoms of neurotoxicity is in the range of 40 to 60 μg/dL (ATSDR, 1990). In children, acute lead poisoning other

than signs of encephalopathy have been observed at levels of approximately 60 to 450 μg/dL. Acute lead poisoning in children causes death from Pb-B levels equal to or exceeding 125 μg/dL, as reported by NAS (1972), based upon studies by Chisolm (1962) and Chisolm and Harrison (1956). Although the Chisolm studies did not address lethality directly, the latter study noted four deaths, and estimated the total lead in the soft tissues of the individuals to be 20 to 100 mg. Grant and Davis (1989) suggest that Pb-B levels that can produce death are basically the same as those associated with acute encephalopathy. Such effects are usually observed in children from approximately 100 μg/dL.

2.1.3. Neurological Impairment

ATSDR (1990) paraphased an EPA (1986a) report, that concluded "that the consistent pattern of lower IQ values and other neuropsychologic deficits among the higher lead exposure children in these studies indicate that cognitive deficits occur in apparently asymptomatic children with markedly elevated blood-lead levels (starting at 40 to 60 μg/dL and ranging up to ≥ 70 to 80 μg/dL)." EPA concluded that approximately five IQ decrement points is a reasonable estimate of the extent of IQ decrements associated with markedly elevated blood-lead levels (mean approximately 50 to 70 μg/dL) in children that do not exhibit signs and symptoms of lead poisoning (EPA, 1986a). IQ deficits of approximately four points are associated with blood-lead levels of 30 to 50 μg/dL (ATSDR, 1990). In studies reported in 1986 and 1987, Hawk et al. (1986) replicated the study with a cohort of 75 African-American children, aged three to seven years old. All were of low socioeconomic status. Using a backward stepwise multivariate regression analysis statistical technique, they found a "highly significant linear relationship between the Stanford-Benet IQ scores and contemporary blood-lead levels, over the entire range of 6 to 47 μg/dL". Hawk et al. (1986) and Fulton et al. (1987) reported a significantly inverse linear association between cognitive ability and blood-lead levels. There was no evident threshold down to the lowest Pb-B of approximately 6 μg/dL. The LOAEL for IQ effects is, consequently, thought to be less than 10 μg/dL (ATSDR, 1990).

A study by Robison et al. (1985) of 75 African-American children aged three to seven years old. The study determined that hearing decreased for the study group, and that the severity of hearing loss increased linearly with historical blood-lead levels, in the range of 6.2 to 56.0 μg/dL. Schwartz and Otto's (1987) logistic regression analysis of NHANES II (the second National Health and Nutrition Examination Survey) data suggests the probability of elevated hearing thresholds with significant increases across the entire range of blood-lead levels of < four μg/dL to > 50 μg/dL. The study involved 4,519 children aged four to 19 years, and was controlled for several confounding variables available from the data set. A study of the effects on peripheral nerve function suggests an increased susceptibility to lead neuropathy, among children with sickle cell disease (Erenberg et al., 1974).

2.1.4. Developmental Toxicity

Developmental toxicity of lead has been assessed in several studies. A study in Boston by Needleman et al. (1984) found an association between lead exposure and congenital abnormalities, including undescended testicles (Hydrocele). Several epidemiological studies have also been conducted. A Port Pirie, South Australia study of 595 children was reported by Vimpani et al. (1989) as well as Baghurst et al. (1987). These studies determined that the geometric mean values of Pb-B increased from approximately 14 μg/dL at six months of age, to approximately 21 μg/dL at 15 and 24 months of age. Depressed Mental Development Index (MDI) scores were found to be significantly associated with higher post-natal blood-lead levels, as well as with six month blood-lead levels, although such an association was not found with pre-natal delivery or with cord Pb-B level. The study found a two point deficit in MDI at age 24 months, for every 10 μg/dL increase in Pb-B at age six months. In a Bellinger et al. (1985a,b, 1986a,b,1987) prospective study of 249 middle-to-upper-middle-class children in Boston, Massachusetts, the researchers determined that the high-lead group (having a mean cord blood level of 14.6 μg/dL) demonstrated an average deficit of 4.8 points on a "covariate-adjusted" MDI score, when compared to the low-lead group (having a mean of 1.8 μg/dL cord blood level). The difference was 5.8 points at six

months, and 7.3 points at 12 months.This inverse relationship held for ages six, 12, 18, and 24 months of age. The Bellinger study (1985a) considered several variables, including demographics (race, parental age, education, marital status, occupation), medical/reproductive history, index pregnancy, labor and delivery, neonatal status (such as birth weight and infections), post natal status (such as hospitalizations and temperature), postnatal environment (including HOME, maternal IQ, family stress, and feeding method), and cord-blood level as an ordinal categorical value (low, medium, or high). The HOME (Home Observation for Measurement of the Environment) assesses the quality of the rearing environment. The study found a statistically significant association of blood cord levels and MDI scores, when the MDI scores were adjusted for length of gestation and total HOME score.

A further analysis by Bellinger et al. (1990) found that children with high (10 to 25 μg/dL) umbilical cord-blood levels achieved significantly lower MDI scores through two years of age, than infants with low (< three μg/dL) or medium (six to seven μg/dL) cord blood levels. The cord blood level, however, was found not to be significantly related to performance (using the McCarthy Scales of Childrens' Abilities) at age 57 months. The study found that delta Z, a derived index for a child's "developmental trajectory" between 24 and 57 months of age, to be significantly related to higher HOME scores, higher social class, and more intelligent, older mothers. It was not, however, significantly related to gender or ethnicity. According to the report, "The associations between performance trajectory between ages 24 and 57 months and several of these characteristics, including high social class, high HOME score, and high maternal IQ, are consistent with the hypothesis that environmental enrichment facilitates the rate and extent of recovery on compensation" [from lead associated cognitive deficit].

EPA (1986a), Davis and Svendsgaard (1987), Grant and Davis (1989), and ATSDR (1988) concluded from several studies of neurobehavioral effects of pre-natal lead exposure (Ernhart et al., 1985; Wolf et al., 1985; Davis and Svendsgaard, 1987; Winnede et al.,1985a,b), that neurobehavioral effects, indeed, are associated with prenatal internal exposure levels. Maternal or cord blood-lead concentrations

of 10 to 15 μg/dL, and possibly lower were found to be associated with such effects (ATSDR, 1990). (The Ernhart 1985 study, however, for which cord Pb-B levels ranged from 2.6 μg/dL to 14.7 μg/dL with a mean of 5.8 μg/dL, concluded that "... the results do not provide a reasonable level of support for the hypothesis of adverse effects due to intrauterine low-level Pb exposure".) ATSDR (1990) notes the criticism of the flaws of the studies reviewed, that showed both positive and no effects at low blood-lead levels. The ATSDR further notes that a 2 to 8 point deficit for an individual child may not be clinically significant, but that a 4 point reduction in a normal distribution of MDI scores for a given population of children, would result in an increase of 50 percent of the children scoring below 80, which the report called "a grave consequence" (ATSDR, 1990).

The Cincinnati Lead Program Project continues to follow study subjects into their early school years to discern whether early deficits (i.e., decrements in Bayley mental index scores) persist into later life (that is, do the observed effects of low level lead exposure continued at the same magnitude over time). Dietrich et al. (1990) reports on the relationship between prenatal and postnatal lead exposure and development status of two-year-old infants. Families for the study were recruited from "lead-hazardous areas" of Cincinnati, Ohio, based upon pediatric case histories of lead poisoning. A total of 297 infants, with a mean blood-lead level of 17.45 μg/dL, participated. The sample was 86.2 percent African-American. The mothers were predominantly from lower social classes, unmarried, and on some form of public assistance.

Developmental assessments were conducted at ages three, six, 12, and 24 months. The three part Bayley Scales of Infant Development were used: Mental Development Index (MDI), Psychomotor Development Index (PDI), and Infant Behavior Record (IBR). The researchers collected social as well as medical background data to test as potential confounders, including race, maternal age and tobacco use. The study employed multiple regression analysis with backward elimination of nonsignificant covariates and confounders (in the reduced model, while all variables were included in the multiple regression

analysis). The lead variables were analyzed both in terms of μg/dL and of a transformation to their natural logarithms.

The prenatal and neonatal blood-lead levels were found to be low, with a few subjects exceeding 25 μg/dL. Most reached the highest blood-lead level during the second year. About 25 percent had at least one serial blood-lead of 25 μg/dL during the second year. Prenatal blood-lead was found to be significantly related to six month MDI after statistical adjustment for 10 potential covariates and confounders, at six months, but only for males. It was insignificant for females at this age. The study did not provide reasons for the gender difference. The study also found, for Hollingshead socioeconomic status scores below the sample median of 17, a covariate adjusted reduction of 0.757 MDI points for each μg/dL increase of neonatal blood-lead ($p = 0.0316$). This was statistically insignificant, however, for a status score above 17. A two year follow-up determined that there was no statistically significant relationships between prenatal or postnatal blood-lead level variables and Bayley MDI. The relationship with Bayley IBR factor scores also had statistically insignificant results. Dietrich et al. conclude that the lack of inverse relationships suggests that those infants of mothers with higher prenatal blood-lead levels may have overcome their early developmental deficits. The authors note that these results are inconsistent with previous studies by Bellinger and the Port Pirie study of 1988, and cite as caveats the limitation of Bayley scales of measurement. The authors also noted that the two other studies did find continuing harmful effects at two years of age.

The documented toxic effects of lead on the human fetus include a lowering of the gestational age, reduction in birth weight, and reduced mental development, all of which may occur at relatively low Pb-B levels (ATSDR, 1990). McMichael et al. (1986) found that the risk of pre-term delivery increases about four times as cord or maternal Pb-B increases from ≤ eight to >14 μg/dL. Dietrich et al. (1986; 1987a) reported a significant inverse association between prenatal Pb-B levels in the mother, and birth weight, with the effect observed down to 12 to 13 μg/dL.

A Bellinger et al. (1987a) study reported significant deficits of 4.8 points in the Bayley MDI at ages six to 24 months of age, in children whose Pb-B at birth ranged from 10 to 25 μg/dL, contrasted to children whose Pb-B level at birth was less than three μg/dL. Dietrich et al. (1987a) also reported an inverse correlation between prenatal or neonatal blood-lead levels and MDI, in the range of one to 25 μg/dL.

2.1.5. Aggregated Studies Analysis

Needleman et al. (1990a) performed what was termed a meta-analysis of 12 of 24 studies that used multiple regression analysis to study the effect of childhood exposure to lead on IQ. They found overall evidence of a strong link between low-dose lead exposure and intellectual deficit in children. The analysis concluded that even though the studies had significant variation in their individual power to find an effect, 11 of 12 of the studies reviewed reported an association between adverse health effects and lead exposure.

2.1.6. Growth

The effects of lead exposure upon growth in the young child have been recognized as far back as 1929, when Nye (1929) reported on runting (stunted growth) and chronic nephritis in overtly lead-poisoned children in Australia. (Nye, in turn, cites a report by A. Jefferis Turner of lead poisoned children in Brisbane, in the year 1892.) Schwartz et al. (1986), based upon data for 2,695 children under seven years of age from the second National Health and Nutrition Evaluation Survey (NHANES II) study, provides even stronger evidence of this effect. Through the use of a stepwise multiple regression analysis technique, the Schwartz group concluded that blood-lead levels for the range of five to 35 μg/dL, were a "statistically significant predictor of children's height ($p<.0001$), weight ($p<.001$), and chest circumference ($p<.026$), after controlling for age in months ... race, sex, and nutritional covariates." The strongest relationship found was between Pb-B and height, with regression models indicating no threshold down to the lowest observed Pb-B of five μg/dL. There was no indication of a threshold within the study range.

2.1.7. Toxicological Summary

These studies indicate that there are several effects of major concern regarding low-level exposure, including neurobehavioral effects Mental Development Index (MDI) and Intelligence Quotient (IQ) deficits, as well as elevated hearing thresholds, and growth retardation (for young children with pre-natal exposure as well as for children suffering from post-natal exposure) (ATSDR, 1990). There appears to

be no indication of a threshold down to the lowest level of internal exposure (Pb-B < 10 μg/dL) (ATSDR, 1990). Health impact are summarized in Table I.

TABLE I
Health Effects Summary

Study	Health Effect	Pb-B Value (μg/dL)
Rosen et al., 1980	Interference with heme synthesis, decreased level of 1,25-dehydroxyvitamin D	33 to 120
Secchi et al.,1974; Wada et al., 1973; Hernberg and Nikkenen, 1970; Chisolm et at., 1985a; and Roels et al., 1976	Inhibition of erythrocyte ALA-D	< 3 to 5
ATSDR, 1990	Lowest observed adverse effect level (LOAEL) for ALA-D and heme synthesis	< 10
EPA, 1986a; Grant and Davis, 1989	Accumulation of erythrocyte pyrinmidine (LOAEL)	15
Angle et al., 1978; Angle et al., 1982	Inhibition of enzyme erythrocyte pyrinmidine-5-nucleotidase	≥ 44
ATSDR, 1990	Encephalopathy in children	90 to 700
NAS, 1972; Chisolm, 1962; Chisolm and Harrison, 1956	Death	125 to 750
Hawk et al., 1986; Fulton et al., 1987	Decreased IQ	6 to 47
ATSDR, 1990; EPA 1986a	Decreased IQ of 4 points; of 5 points	30 to 50; 50 to 70
Robison et al., 1985; Schwartz and Otto, 1987	Decreased hearing acuity	6.2 to 56.0; <4 to >50
Needleman et al., 1984	Hydrocele (undescended testicle)	Not specified
Ernharrt et al., 1985; Wolf et al., 1985; Davis and Svendsgaard, 1987; Winnede et al., 1985a,b	Neurobehavioral effects	< 10 to 15
McMichael et al., 1986	Pre-term delivery risk	≤ 8 to 14
Dietrich et al., 1986, 1987a	Decreased birth weight associated with mother's Pb-B	12 to 13
Dietrich et al., 1987a	Mental Development Index deficit	1 to 25
Nye, 1929	Stunted growth	Not specified
Schwartz et al., 1986	Reduction in height and weight	< 5 to 35

In a speech given on October 7, 1991, Health and Human Services Secretary Louis Sullivan cited

an announcement by the Centers for Disease Control, for a lower "threshold of concern" for blood-lead levels in children (Sullivan, 1991). The new threshold is 10 μg/dL, coupled with recommendations for "...levels of action for intervention." Dr. Sullivan called lead poisoning "...the number one environmental threat to the health of children in the United States."

2.2. Adequacy of Studies

The United States Congress, in Section 110(3) of the Superfund Amendments and Reauthorization Act (SARA) of 1986, tasked the ATSDR with preparing a toxicological profile for each of 100 most significant hazardous substances found at the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) National Priority List (NPL) sites, and for each profile to provide "An examination, summary, and interpretation of available toxicological information and epidemiologic evaluations on a hazardous substance in order to ascertain the levels of significant human exposure for the substance and the associated acute, subacute, and chronic health effects" (ATSDR, 1990). The result of that mandate has been the preparation of a very intensive and extensive review of the literature on the human health effects of lead. This was presented in pertinent part in the preceding section. Congress also required ATSDR to determine whether sufficient information existed to ascertain the levels of exposure for a given chemical that posed endangerment to human health.

ATSDR categorized the sufficiency of data from human studies for specific health endpoints as sufficient, some, or no information available to make a definitive determination for each endpoint, for both cancer and noncancer (ATSDR, 1990). ATSDR judged, for combined oral and inhalation studies, that some information exists for lethality, acute systemic toxicity, reproductive toxicity, and carcinogenicity. Sufficient information is deemed to be available, based upon the extensive literature review, for intermediate systemic toxicity, chronic systemic toxicity, and developmental toxicity. No information (derived from human health studies) is available for the dermal route of exposure for any of the seven health endpoints reviewed.

Information is available, however, for animal data. There appears to be sufficient information available on reproductive toxicity (oral), along with some evidence of carcinogenicity via the dermal exposure route (ATSDR, 1990). It is noted that the Interagency Regulatory Assessment Group has rated lead as a Group B-2 possible human carcinogen (1987), and that EPA (1991) has determined lead to be a Class B-2 probable human carcinogen.

Noting the difficulties in ascertaining the length of exposures and that distinctions are somewhat arbitrary, ATSDR recommends the joint consideration of intermediate and chronic systemic toxicity data together (ATSDR, 1990). ATSDR has determined that the data do not clearly indicate NOAELs for humans, recognizing that the associations between blood-lead levels and neurobehavioral indices, blood pressure, growth, and heme synthesis, occur over a wide range of Pb-B concentrations. Further, there are no indications of threshold values through the lowest Pb-B levels. More than 100 ongoing federally sponsored projects involving lead toxicity have been identified, including several prospective studies on the effects of lead toxicity on neurobehavioral changes in childhood populations (ATSDR, 1990). The existence of a large data base relating Pb-B levels to measured lead concentrations in air, diet, drinking water, dust and soil, has also been noted (ATSDR, 1990). ATSDR, nonetheless, considers such measures to be an imperfect measure of body tissue burden. The better measure is the level of lead in teeth and bones, together with Pb-B, to better measure both past exposures and current body burden. ATSDR specifically cites the EPA lead uptake biokinetic model (EPA, 1991a) for estimating blood-lead levels. The model is based upon exposures, and has been validated by an investigation of young children living near industrial lead sources that contribute to lead concentrations in ambient air, soil, and dust.

2.3. Biological Monitoring Techniques

As noted, however, blood-lead measurement is the most common method of assessing exposure to lead. The half life of lead in human blood is 28 to 36 days (ATSDR, 1990). It is noted that the detection limit at most clinical laboratories is three to five μg/dL (ATSDR, 1990).

The use of erythrocyte protoporphyrin (EP) measurement for screening asymptomatic children for lead toxicity was recommended (CDC, 1985; American Academy of Pediatrics, 1987), recognizing that elevated EP was one of the earliest and most reliable indications of impairment of heme biosynthesis. Further, EP is used because of the contamination problems in measuring blood-lead levels (ATSDR, 1990). In humans, it is noteworthy that Pb-B values are distributed in a log-normal distribution. Accordingly, researchers should use the geometric mean and the geometric standard deviation when analyzing the distribution data (ASTDR, 1988).

2.4. Typically Encountered Environmental Levels

EPA (1986a) found that the relationship of blood-lead levels to lead concentrations in air, food, and water is curvilinear, such that the increase in Pb-B is less at high levels than at low exposure levels. The clear implication is that concern is warranted when children, in particular, are subject to even low levels of environmental exposure. The range of normal air concentrations is 0.1 to 2.0 $\mu g/m^3$ (ATSDR, 1990). The median blood-lead-level/inhalation-concentration for children is approximately 1.92 $\mu g/dL$ blood per $\mu g/m^3$ air, based upon three major studies (ATSDR, 1990). Aggregate values, including indirect blood-lead contribution from dust and soil, range from three to five $\mu g/dL$ per $\mu g/m^3$ air (Brunekreff, 1984). Angle et al. (1984) defines the value at four to five $\mu g/dL$ for indirect exposure, additive to the direct inhalation contribution.

The Centers For Disease Control (1985) has determined that concentrations of lead in soil of 500 to 1,000 $\mu g/g$ result in Pb-B in children exceeding background levels of Pb-B. EPA's (1985) estimate of the contributions of blood-lead levels from various media have been provided based upon background levels, and the resultant levels from the addition of incremental concentrations of lead in air (EPA 1986, 1986a). Mean background contributions for non-air sources are 2.37 $\mu g/dL$ from food, water, and beverages; 0.30 $\mu g/dL$ from dust; and 1.65 $\mu g/dL$ from air. The total background contribution is 4.32 $\mu g/dL$. The background Pb-B range is 4.32 $\mu g/dL$ to 16.72 $\mu g/dL$ (including 9.40 $\mu g/dL$ from ingested

dust, and 3.00 μg/dL from ingested air, at the upper value). EPA found the estimates to be higher than predicted when compared to observations in children living in areas with measured lead in air concentrations. By comparison, Piomelli et al. (1980), during an expedition ascending the Marsyandi River in the Manang district of Nepal in the foothills of Annaparna and Dhaulagiri, sampled the blood of local inhabitants. The geometric mean Pb-B concentration was 3.4 μg/dL. Only 10 of 103 individuals tested exceeded 10 μg/dL.

In 1986, the U.S. production of refined lead from primary sources totaled 808 million pounds, and from secondary sources totaled another 1,356 million pounds (ATSDR, 1990). It is noted that recycling of old scrap metal supplies 45 percent of U.S. demand. In that year, consumption in the nation was 2,480 million pounds, including 319 million pounds of lead imported to this country (ATSDR, 1990).

Anthropogenic emissions constitute the primary source of lead in the environment, and as of 1984, gasoline combustion, in particular, was responsible for approximately 90 percent of all anthropogenic emissions (ATSDR, 1990). This percentage has been reduced dramatically, however, due to the phase down of lead in gasoline. Atmospheric deposition is the largest source of lead found in both soils and surface waters. The lead particles are removed from the atmosphere principally via wet and dry deposition. ATSDR (1990) states that soil and sediments appear to be important sinks. The average residence time in the atmosphere is seven to 30 days, during which period lead can be transported up to thousands of kilometers. According to EPA (1986a), natural emissions of lead from volcanoes and windblown dust are thought to be of minor significance. EPA (1986a) has also estimated the anthropogenic lead emissions into the atmosphere for the year 1984. The 1984 estimate for gasoline production was 34,881 tons/year, or 89.4 percent of the total emissions of 39,016 tons/year. Based upon a current gasoline standard of 0.1 g Pb/gallon-gasoline, the estimated 1988 lead emission would be 1,100 tons/year.

In the atmosphere, lead exists mostly in particulate form (ATSDR, 1990). Large size particles,

particularly those with aerodynamic diameters exceeding 2 μm, settle out of the atmosphere fairly rapidly and are deposited relatively proximate to the emission source. Smaller particles may travel thousands of kilometers. In waters, at a pH > 5.4, the total solubility of lead is about 30 μg/l in hard water, and 500 μg/l in soft water (ATSDR, 1990). The ratio of lead in suspended solids to lead in dissolved form has been found to vary from four to one in rural streams, to 27 to one in urban streams (EPA, 1986a). It is noted that lead does not appear to be biomagnified in the food chain, but may accumulate in flora and fauna. In aquatic organisms, the lead concentrations are typically highest in benthic organisms , such as algae, and are lowest in the upper-trophic predators (e.g., carnivorous fish) (ATSDR, 1990).

Most lead is retained strongly in soil, and very little is transported into surface or groundwater, according to reports by EPA (1986a) and the Zimdahl and Hassett (1977). In soils with a pH ≥ 5 having more than five percent organic content, atmospheric lead is retained in the upper two to five cm of soil, if it is left undisturbed. EPA (1986a) estimates that four to five million metric tons of lead from gasoline combustion remain in the dust, soils, and sediments of the U.S. Although lead does have a high degree of immobility in soil (Zimdahl and Hassett, 1977), wind action may induce mobilization to the atmosphere and thus downwind transport. This soil entrainment may be of significance to the atmospheric burden downwind from stationary sources, particularly smelters and superfund sites (ATSDR, 1990).

As noted previously, preschool age children, pregnant women, and their fetuses constitute the population at highest risk. ATSDR also notes the increase risk to white males aged 40 to 59 years old (ATSDR, 1990). The health endpoint of concern for this latter risk group is hypertension.

ATSDR (1990) estimates the baseline intake for a two year old child to be 46.6 μg/day, and for an adult female to be 37.5 μg/day. Additional exposure results from residing in an urban environment, proximity to stationary lead sources, residences and other building structures containing lead-based paint, pica (eating disorder of some younger children), both primary and secondary occupational exposure, smoking (from tobacco products containing lead), and wine consumption (for wines containing lead)

(ATSDR, 1990). In addition, proximity to a superfund site is thought to increase risk of exposure. At the time of the ATSDR (1990) report, lead and lead compounds had been discovered at 635 of 1,177 National Priority List (NPL) sites.

The levels of lead in the ambient air range from 0.000076 $\mu g/m^3$ in remote areas, to over 10 $\mu g/m^3$ near stationary lead sources (ATSDR, 1990). Confined places (e.g., parking garages, tunnels, and toll booths) may have unusually high concentrations of lead in air. In surface waters in the United States, EPA (1986a) has found typical levels of five to 30 µg/l. Sediments contain a considerably elevated concentration of approximately 20 µg/kg (ATSDR, 1990). In ground water, the typical range is one to 100 µg/l (EPA, 1986a).

EPA (1988b) estimates that 99 percent of 219 million people in the United States that utilize public drinking water supplies, are exposed to water with levels of lead < 0.005 mg/l, and that about two million consumers are exposed to drinking water exceeding this value. The range is, on average, 10 to 30 µg/l in households, schools, and office building drinking water supplies, although corrosive water, lead pipes, and lead solder joints can, singly or in combination, produce much higher concentrations (EPA, 1989b).

Soils adjacent to roads traveled since 1930 may have as much as 10,000 µg/g lead (EPA, 1986a), while soils near homes with exterior lead-based paint may have even higher soil-lead concentrations. ATSDR (1990) draws upon studies conducted in Baltimore, Maryland, and Minnesota, to conclude that the highest soil-lead levels generally occur in inner city areas, especially in areas where there has been an historically high amount of traffic.

Lead is also found in dairy products, meat, fish, poultry, fruits, sugar, and beverages (EPA, 1986a). Canning processes, in particular, can increase the concentration of pre-canned foods from eight to 10-fold. According to the Food and Drug Administration (Gunderson, 1988), the baseline intake via food consumption for the years 1982 to 1984 was 23.0 µg/day for a two year old child, 29.6 µg/day for

an adult female, and 40.9 μg.dL for an adult male. Elias (1985), based upon an analysis of food residues and using the 1984 U.S. Food and Drug Administration's Marketbasket Survey, postulates total food consumption to be 25.8 μg/Pb/day (including 2.8 μg/day from water) for a two year old child. For a male aged 25 to 30 years, Elias estimates a lead consumption of 54.7 μg/day. It is also noted (ATSDR, 1990) that additional exposure through dietary exposure, from atmospheric dust, is experienced by those living in an urban environment, at 91 μg/day for children, and 28 μg/day for adults.

Lead content of dusts can be a significant source of exposure, particularly for young children (ATSDR, 1990). It is estimated that children ingest five times more dust particles than adults do (EPA 1986a). Lead-based paint confounds the problem for young children. EPA (1986a) has found concentrations of 1,000 to 5,000 $\mu g/cm^2$ for lead-based paint chips. Chisolm (1986) estimates that between 40 to 50 percent of the currently occupied housing in the United States may contain lead-based paint on exposed surfaces.

Cigarette smoke is yet another source of lead exposure, with each cigarette containing approximately 2.5 to 12.2 μg lead (ATSDR, 1990). From two to six percent of the lead may be inhaled in the smoke. Consequently, given the greater propensity for lead uptake by children, secondary smoke poses yet another threat to children under seven years of age, as well as to the developing fetus. Additional exposure to children in the home, as well as to others, is also plausible via secondary occupational exposure from workers in lead processing industries. Workers may bring home lead dusts on their clothing.

Other sources of lead exposure, such as housing renovation activity, are also now being more fully recognized. Marino et al. (1990) reported on an outbreak of severe lead-based paint poisoning in a family that was exposed to lead dust and fumes generated during the removal of lead-based paint in the family's rural farm house. Multiple coats of lead-based paint were being removed over a 10 week period. The removal methods were sanding, torching, and the use of heat guns. These methods produced wood

particles, fine dusts, and fumes that could be ingested or inhaled. Symptoms were first noticed in the family dog, found by the veterinarian upon examination to be weak, dehydrated, and depressed. The animal was determined to be lead-poisoned, and subsequently died. The mother of the family began feeling weak and tired. The daughter complained of stomach aches in the mornings. The father suffered severe nausea during weekends of renovation work. All were found to have elevated blood-lead levels.

EPA (1986a) concluded, appropriately, that "... lead is a pervasive environmental contaminant that causes a wide variety of adverse health effects in humans. In short, lead is potentially toxic wherever it is found, and it is found everywhere".

2.5. At Risk Population

Section 118(f) of SARA requires the ATSDR to prepare a comprehensive study on lead poisoning in children.

ATSDR (1988) noted that much of the data needed to prepare the report was not available in peer-reviewed literature, and, consequently, was developed specifically for the report to Congress.

In a given year, ATSDR estimates that an estimated 400,000 fetuses are exposed to maternal Pb-B > 10 μg/dL, within Standard Metropolitan Statistical Areas (SMSAs). For other exposures, the estimation problem is more problematic. ATSDR (1988) found that "The actual number of children exposed to lead in dust and soil at concentrations adequate to elevate Pb-B levels cannot be estimated with the data now available." The opinion expressed is that the regulatory actions of the 1970's to address existing lead-based paint in housing "have been a clear failure" (ASTDR, 1988). The current average Pb-B levels in the United States today, in some segments of the population, are 15 to 30 times higher than the theoretical mean value of 0.5 μg/dL, calculated for pre-industrial humans (ASTDR, 1988).

ATSDR (1988) selected 1984 as the base year for estimating the number of children at or above selected Pb-B levels, because all of the required enumerations were available for that year. The findings from NHANES II were utilized to derive prevalences for demographic and socioeconomic strata within

the childhood population, in order to judge the numbers of exposed children. The method was to allocate the total number in each Standard Metropolitan Statistical Area (SMSA) for selected strata of age, race, income, and (where possible) urbanization categories. The various strata were then added to obtain national totals for each strata. Each stratum population number was then multiplied by the prevalence for the three selected Pb-B levels (using national prevalence rates), adjusting prevalence from 1978 to 1984 levels, to account for the reduction of lead in gasoline. It is noted that NHANES II did not report Pb-B levels for specific geographic areas, but rather reported for socioeconomic, demographic, and ethnic strata for the nation as a whole. Consequently, due to the lack of geographic specificity of data, the ATSDR report considers SMSAs collectively, and not individually. The data (ATSDR, 1988) is further limited to young white and African-American children, because NHANES II did not include sufficient numbers of Hispanic and other-race children, in order to enumerate prevalences in those ethnic sub-populations. The strata analyzed were African-American and white; 0.5- to two-year-old children, three- to five-year-old children (although these age bands were subsequently merged to derive a 0.5- to five-year-old child age band); urban status (central city, outside of central city); and family income. The size of SMSA (< or > one million population) was also provided. It is noted with specificity that just as the ASTDR study adjusted the 1978 prevalence rates to account for reductions in lead-in-gasoline and lead-in-food from 1978 to 1984, so too are these prevalence rate estimates now overly conservative, due to the further reductions of lead-in-gasoline and food for today (recall in particular the significant reduction in leaded-gasoline emissions to 1,100 metric tons by the year 1990).

2.5.1. Spatial/Numerical Estimate of At Risk Population

ASTDR (1988) Pb-B criteria values of ≥15, ≥20, and ≥ 25 μg/dL were as calculated from NHANES data by EPA's Office of Policy, Planning, and Evaluation (ASTDR, 1988) , using logistic regression analysis techniques to update prevalences to 1984. Tables are provided, separately for central cities and outside central cities, on the projected percentages of children 0.5 to five years old that are

estimated to exceed selected Pb-B levels by family income, race, and urban status within SMSAs. The range is from 36 percent > 15 μg/dL for white children in populations centers ≥ one million and income < $6,000, to 0.5 percent > 25 μg/dL for white children, with family income >$15,000. For African-American children, in the same categories, the range is from 67.8 percent > 15 μg/dL, to 2.2 percent >25 μg/dL. Similar patterns are presented for children residing outside central cities.

Estimates of the numbers of children in the age band who are projected to exceed the three criteria levels of Pb-B are provided by family income and race. For central cities, with SMSAs < one million population, the projections are 301,100 children >15 μg/dL, 93,800 > 20 μg/dL, and 27,500 > 25 μg/dL. For central cities with SMSAs > one million population, the numbers are even greater, 901,800 children > 15 μg/dL, 301,700 >20 μg/dL, and 86,200 > 25 μg/dL. Overall, for the 1984 United States childhood population of 13,840,000, 2,381,000 are expected to have Pb-B values >25 μg/dL (ASTDR, 1988). Specific concerns with the limitations on accuracy of these projections are noted in the ASTDR report. In particular, the Hispanic child population is not included, and that population segment is experiencing high birth and growth rates. Some of this population is associated with lower economic and central city strata and, consequently, are expected to have higher predicted prevalence rates across the criteria Pb-B levels. "The most important finding, however, is that no strata of these children are totally exempt from risk of Pb-B levels high enough to represent a potentially adverse health impact" (ASTDR, 1988).

2.5.2. Lead Screening Programs

Lead has a long history of use by man as well as harm to man, extending hundreds of years back in time. In this country, not much public concern was evident in the early part of this century. In the early 1930's, however, the Baltimore Health Department became interested in lead poisoning (Lin-Fu, 1982). Not much attention was shown by health officials in other cities until the early 1950's. At that time, New York, Chicago, and Philadelphia began case finding as well as public education efforts. During this period through the mid-1960's, health officials found hundreds of cases of lead poisoning in several

large older cities (Lin-Fu, 1980). These included Baltimore, New York, Philadelphia, and Chicago. Between 1959 and 1963, Cook County Hospital in Chicago treated 182 children for lead encephalopathy (Lin-Fu, 1982). Of the cases, 51 died.

A mass of data on childhood lead poisoning was published in the 1950's to early 1060's (Lin-Fu, 1982). Most of the public was unaware of the problem. Many in the public health profession failed to react. According to Lin-Fu (1982), the turmoil and awakening of social conscience of the mid-1960's brought a sudden realization of the magnitude of childhood lead poisoning in this country. It was during this period that it was discovered that lead poisoning was epidemic in the inner city slums (Lin-Fu, 1980). In 1966, Chicago began the first mass blood-lead screening program in the nation. New York and other cities did the same. It was also during this decade that health officials unexpectedly discovered asymptomatic children with elevated blood-lead levels. This discovery sounded an alarm that health care workers needed to recognize lead absorption in preventing lead poisoning disease, and that subclinical toxic effects of lead were a concern (Lin-Fu, 1980). The pervasive source of lead exposure in children, from lead in dust and soil, also became apparent (Lin-Fu, 1992). Childhood screening programs discovered a high prevalence of elevated blood-lead levels in children that could not be fully explained by ingestion of lead contaminated paint chips.

The U.S. Surgeon General issued a statement in 1970 which effectively shifted the emphasis of health care workers from case finding to lead poisoning prevention. He advocated mass screening to find cases of elevated blood-lead levels (Lin-Fu, 1982). Shortly thereafter, the 1971 Lead-Based Paint Poisoning Prevention Act became law. The Act provided funds for mass screenings. Mass screening funded by the Act began in mid-1971. From January 1972 through December 1978, 2,485,320 children were screened by federally funded projects (Lin-Fu, 1980). Of these, 170,738 children were found to have elevated Pb-B or EP levels.

In fiscal year 1982, these lead screening programs, along with other public health protection

programs, were incorporated into the Maternal and Child Health (MCH) Block Grant Program (ASTDR, 1988). The screening programs are targeted primarily at case finding for children with Pb-B levels serious enough to warrant medical intervention. The classification schemes have changed over the years. Analysis for elevated EP has been the first step in screening, although it is recognized that some children having elevated Pb-B will pass the EP test; accordingly, the EP screening test does produce false negatives. Table II, based upon data presented in the ATSDR (1988) report, presents the results of screening programs in 16 cities in the Midwest region of the country, for fiscal year 1981, using the 1978 Centers for Disease Control classification of lead toxicity of 30 μg/dL Pb-B and 50 μg/dL EP.

In 1988 Congress enacted the Lead Contamination Control Act. Among other provisions, the Act authorized the Centers For Disease Control to Provide grants to States and local health agencies to fund childhood lead poisoning prevention programs (DHHS, 1991c). The grants are to screen children for lead poisoning; to ensure environmental as well as medical follow up for lead-poisoned children; and to provide education about lead poisoning.

TABLE II

Blood-Lead Screening Program Results for Children in 16 Midwest Cities in 1981[1]

Program Location	Number Screened	Number with Elevated Pb-B
Chicago, IL	32,861	2,070
Kankakee, IL	2,464	56
Madison County, IL	2,288	105
Rockford, IL	2,341	30
Waukegan-Lake Col, IL	3,570	35
Illinois (other programs)	5,184	145
FT. Wayne, IN	532	19
Detroit, MI	19,281	926
Grand Rapids, MI	688	19
Wayne Co., MI	1,818	75
St. Paul, MN	2,107	15
Akron, OH	4,637	149
Cincinnati, OH	9,085	191
Cleveland, OH	14,151	921
Beloit, WI	779	15
Milwaukee, WI	6,640	316

[1] Elevated Blood-lead (Pb-B) is based upon the Centers for Disease Control lead toxicity classification of ≥ 30 μg/dL Pb-B and ≥ 50 μg/dL erythrocyte protoporphyrin.

ATSDR has also compiled the number of lead poisonings determined by screening programs for fiscal year 1983 (ASTDR, 1988). The numbers of children screened and cases of confirmed lead toxicity, by state are Illinois: 25,340 and 136; Indiana: 1,265 and 1; Michigan: 14,700 and 434; Minnesota: 1,816 and 18; Ohio: 19,543 and 416 (the number evaluated for lead toxicity did not necessarily include all those screened who may have been lead poisoned); and Wisconsin: 4,322 and 187 (some of the 187 cases are estimates by respondents, not necessarily the result of Pb-B testing) (ASTDR, 1988). The cases ranged from 0.1 to 4.3 percent of the children screened. It appears that the rate of chronic lead poisoning in young children is decreasing somewhat (ATSDR, 1988). The numbers of children with elevated blood-lead levels, as well as the percentages of screened children that have lead toxicity, indicates that lead poisoning is a continuing problem. That conclusion is supported by an analysis of lead-screening statistics for the Chicago Department of Health from 1981 to 1985 (ASTDR, 1988). That analysis suggests that there has been minimal change over these years in the percent of children screening positive for lead poisoning. The prevalence of Pb-B levels above 30 μg/dL in young children sampled by NHANES II was higher than that which would be predicted from the state and local screening data (ASTDR, 1988). It would appear, consequently, that screening programs may not be addressing the totality of the at-risk population.

2.6. At Risk Population Estimates By Sources/Routes Of Exposure

2.6.1. Lead-Based Paint

Clearly the greatest amount of attention and data in recent years has been on the contamination and health problems caused by lead-based paint. A prospective study of inner-city children conducted by Clark et al. (1985), that found that children who have the highest Pb-B levels lived in the worst housing. The housing-quality accounted for more than 50 percent of the Pb-B variability in 18 month-old children. The study also found that children in public (versus private) housing, near a heavily used interstate

highway, had the lowest Pb-B levels. This result indicates that, for the study, air-lead from highways had only a very limited impact on blood-lead in children. Further, the study found that although rehabilitated housing contained lower lead paint levels than public housing, children in rehabilitated housing had higher blood-lead levels than those in public housing, suggesting to the authors that lead sources in the immediate neighborhood of the rehabilitated housing may be a factor. A more plausible explanation, however, is the probability of the inadequacy of rehabilitation. Performed incorrectly, such units pose substantial risks of reexposure of children returned to the housing units.

Chisolm et al. (1985b) found, in a prospective study of children in Baltimore, that children returned to homes subsequent to lead paint abatement/removal actions, experienced significantly higher Pb-B levels than children returned to public housing that was free of leaded paint. ATSDR (1988) noted a "great decline" in the number of very severe cases of lead poisoning in the U.S., but notes that "... the basic epidemiological picture characterizing paint-lead associated toxicity has not materially changed for chronic interaction."

To derive the number of children at risk via this route of exposure, ATSDR's (1988) method was to use estimates of the ratio of children under seven years of age per 1,000 housing units, together with estimates of categories and numbers of lead-painted houses with problems such as peeling paint, broken or cracked plaster, or holes in walls. Problem dwellings were as defined by the American Housing Survey of the U.S. Bureau of the Census (ASTDR, 1988). The fraction of total housing units to that of such defined "problem" units was used to derive estimates of the total number of children in lead-painted homes, and the number of children in lead-painted homes categorized as problem dwellings. The ATSDR study used estimates and calculations by Pope (1986), whose efforts addressed four major areas of the country, including the Midwest.

ATSDR (1988) also notes a comprehensive unit-by-unit study that was conducted in the city of Chicago in 1978, that assessed the Pb-B levels and the presence of leaded paint in 80,000 individual

housing units. In general, however, ATSDR found "a general dearth of nationwide studies that estimate the number of children living in paint-containing homes who have elevated Pb-B levels..." The study approach was to take the number of children living that the U.S. Census Bureau estimates to be living in deteriorated housing having 100 percent lead paint, and then to approximate "the most logical" prevalence for the stratum (as discussed earlier), that would be applicable to children in such housing (ASTDR, 1988). The stratum chosen was inner city, dense population, and lowest income, with the further assumption that many of the children in such areas would be African-American.

Paint with a lead concentration ≥ 0.7 mg/cm^2 was chosen as the criterion value for distribution, with an estimation that 99 percent of pre-1940 housing stock, 70 percent of houses built from 1940-59, and 20 percent of the housing stock built during 1959-74, would exceed this value. Thus, for the U.S. housing inventory of 80, 390,000 (1983 Survey, U.S. Bureau of the Census), ATSDR estimates that 52 percent (41,964,000) of the units exceed the criterion value (ASTDR, 1988). It is further noted that the 0.7 mg/cm^2 criterion value is based upon a CDC (1985) statement. Pope's method (Pope, 1986) was used to classify housing for age groups by unsound housing (i.e., deteriorating paint).

The study derived, via these considerations, a best national estimate of 1,772,000 children, and a national upper bound estimate of 1,996,000 children under seven years of age living in unsound lead-painted housing. For the Midwest region, the derived numbers are (derived from ATSDR, 1988):

TABLE III

Children Under 7 years of age in the Midwest Residing in Unsound Lead-Painted Housing[2]

Age of Housing	Housing with Peeling Paint	Number of Children
Pre-1940	264,000	74,000
1940-1959	159,000	47,000
1960-1974	47,000	14,000
Pre-1980	470,000	139,000

For comparison purposes, the numbers for all four regions of the nation and all housing, are 1,840,000 housing units, and 520,000 children. It is noted that these estimates (based on Pope's work) include non-SMSA housing stock, and exclude potential exposures that may result from renovation of older urban housing (the so-called urban gentrification phenomenon, as discussed previously, for example, in the study by Marino et al. of a rural farm house renovation) (ASTDR, 1988), due to an inability to quantify such units.

A recently released study, the Comprehensive and Workable Plan for the Abatement of Lead-Based Paint in Privately Owned Housing (HUD, 1990), determined there to be no correlation between lead-based paint and household income, and that more units have lead paint on the exterior walls than on interior walls. The national survey estimated that 38 percent of all homes occupied by families with young children have priority hazards, and notes that blood-lead screening programs reach only five percent of the young children in the nation.

[2] Source: The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress, ATSDR, 1988.

The objectives of the national housing survey were to determine the incidence of lead-in-dust in dwelling units, as well as lead-in-soil in and around residences; and to define the characteristics of housing with varying levels of potential lead hazard in order to determine priorities for abatement. The study population was the United States population residing in pre-1980 housing stock. A statistically based survey was derived for the nation. A sample size of 284 housing units was chosen to represent 77 million housing units. The survey assessed interior and exterior paint (concentration and condition) by year built, the type of housing, the threshold level of lead concentration, and the census region. The stratification was on type (privately owned single-family and privately owned multifamily) and construction date (before 1940, 1940 to 1959, and 1960 to 1979). The sample units were geographically clustered in 30 counties (of 3,000 in the nation). The researchers employed X-ray fluorescence (XRF) to test for lead paint concentration, and also collected and analyzed dust and soil samples. It is noted with particularity that XRF does not distinguish between paint lead on the surface and lead beneath the surface (e.g., old paint under a fresh cover, or lead pipes).

The national survey found that 57.4 million homes, representing 74 percent of the study population (residing in 77 million homes), contained lead-based paint (LBP[3]). This included 9.9 million homes whose families have young children. The percent of LBP housing units by strata was determined to be 90 percent for pre-1940 housing, 80 percent for 1940-59 housing, and 62 percent for 1960-79 housing. The Midwest census region had 76 percent LBP housing, compared to the 74 percent national value. Distribution for the housing unit was determined to be 14 percent LBP interior only, 23 percent LBP exterior only, and 37 percent LBP on both interior and exterior walls (for a total of 74 percent). Thus lead-based paint was determined to be more common on the exterior of homes.

Nonintact paint was estimated to exist in 13.8 million (of 57.3 million) units containing LBP. Of

[3] LBP is defined as greater than of equal to 1.0 mg/cm^2, measured by XRF, in accordance with the Federal Standard for LBP established in Section 566, Housing and Community Development Act of 1987 (HUD, 1990).

the 13.8 million, 5 percent are interior only, 11 percent are exterior only, and 2 percent are estimated to be both interior and exterior surfaces. Thus, 18 percent of the total housing stock is estimated to contain nonintact LBP, defined as exceeding five ft^2 of LBP in a dwelling being defective (HUD, 1990). Further, the paint is estimated to be damaged in 21 percent of units with exterior LBP, and in 13 percent of the units with interior LBP. Citing the interim guidelines developed by the Department of Housing and Urban Development on clearance levels for dust, post abatement[4], the authors note that fully 17 percent of the occupied homes that contain LBP exceed the guidelines. Only four percent of the homes free of LBP, in contrast, were determined to have excessive dust-lead. The chance of having excessive dust-lead if lead-based paint exists versus no lead-based paint was thus calculated to be 17:4.

Surprisingly, the study found that the incidence of dust-lead is almost as low for homes with interior LBP only, as for homes with no LBP, while the incidence is approximately the same for units with interior or exterior LBP. The study concludes, consequently, that interior dust-lead-contamination is more likely generated by exterior LBP than by interior LBP. The incidence was found to be highest for units containing both interior and exterior LBP. Most dust was found to be located around windows.

2.6.2. Leaded Gasoline

Recent consumption of leaded gasoline in the United States alone shows that in the 10-year period from 1975 to 1984, inclusive, this country consumed 654.6 X 10^9 gallons of gasoline, resulting in the dispersal of 1,087.8 X 10^3 metric tons of lead in the U.S. (ASTDR, 1988). "Gasoline lead makes a sizable contribution (about 90 to 95 %) to the total atmospheric lead burden in developed countries such as the United States" (ASTDR, 1988). From 1975 to 1984, however, U.S. gasoline lead consumption decreased by 73 percent. ASTDR states that studies and data indicate that past gasoline lead consumption resulted in airborne lead that "added significantly to atmospheric and soil/dust/food burdens, and that via both direct and indirect routes, such input contributes 20 to 25% to Pb-B levels." The pathway can be very

[4] 200 $\mu g/ft^2$ for floors, 500 $\mu g/ft^2$ for window sills, and 800 $\mu g/ft^2$ for window wells (DHUD, 1990).

significant as a route of exposure for children, with elevated blood-lead to airborne lead concentration ratios of five to six μg/dL Pb-B rise for each $\mu g/m^3$ increase in air-lead concentration. The NHANES II data supports the high correlation between reduction in leaded-gasoline and the decrease in Pb-B levels in the general population (ATSDR, 1988).

The methodology utilized for estimating numbers exposed was to restrict the enumeration to the 100 largest cities, where the highest exposures, due to mobile sources (including soil/dust routes of exposure resulting from past deposition), were expected to occur. For the estimated 1984 population of 50,597,300 residents of these areas, 11 percent (5,565,700) are estimated to be children under seven years of age (ASTDR, 1988). ATSDR, estimating the numbers of children falling below criteria Pb-B levels, and then projecting to the year 1990, found that gasoline lead phase down alone, will not be sufficient to reduce all Pb-B levels down to levels considered to be acceptable. The agency determined that, in the year 1990, the numbers of children estimated to be below criteria Pb-B levels, as a result of lead in gasoline phaseout, are 25 μg/dL-119,000; 20 μg/dL-400,000; and 15 μg/dL-1,252,000 (ASTDR, 1988).

2.6.3. Stationary Sources

This nation has 11 lead mines, five primary smelters and refineries, 60 secondary smelters, and 132 plants, the latter for manufacture of lead-acid batteries (ASTDR, 1988). Soil and dust levels near these sources range from 500 to 5,000 ppm, with exponential decreases with distance from the source. A 1977 investigation by Yankel et al. (1977) of one- to nine-year-old children living near a smelter in Silver Valley, Idaho, clearly demonstrates an association of airborne lead concentration as well as (elevated) Pb-B levels with distances from the source. That study modeled the natural log of blood-lead, house dust, soil, age, occupational factors, and air concentration. The researchers determined that 99 percent of the children adjacent (within 1.6 km) to a smelter had Pb-B levels exceeding 40 μg/dL. Air concentration alone accounted for 55 percent of the variance in Pb-B levels. A CDC study in two smelter communities in Montana (CDC, 1986a) and Idaho (CDC, 1986b) found that the only significant

environmental source causing elevated blood-lead levels in children was lead in the soil and house dust, resulting from smelter operations. Thus previous fallout remains a main contributor to elevated Pb-B levels. It is also noted that blood-lead remains elevated even when airborne levels have been reduced to low levels. Consequently, ATSDR (1988) recommends that closed facilities be included in studies, to account for the impact of previous lead emissions and deposition.

Based upon previous studies, ATSDR estimates that between 1 and 26 percent of children living near primary lead smelters would exceed the CDC criteria for lead toxicity of 25 μg/dL Pb-B and 35 μg/dL EP. Four percent of children residing near secondary smelters would also exceed the criteria values (ASTDR, 1988). The report quotes an estimate by the EPA Office of Air Quality Planning and Standards, of 21,000 children exposed via primary lead smelters (within five km of the source), and 187,000 exposed via secondary lead smelters (within two km of the source). Some 25,000 children are estimated to be exposed from lead-acid battery plants (within a one km radius), for a total childhood exposure count of 233,000.

2.6.4. Dust and Soils

Brunekruf et al. (1983), in a study conducted in the Netherlands, determined that household dust-lead concentration increases by 400 to 700 ppm for each $\mu g/m^3$ rise in airborne lead. This was as reported by ATSDR, apparently based upon data presented in the Brunekruf study. The Bruenekruf study found a Pb-B to air-lead concentration of one to two μg/dL per $\mu g/m^3$. The outdoor measured air values ranged from 0.10 to 0.27 $\mu g/m^3$. Soil-lead generally was found to be less than 500 ppm. Dust-lead ranged from geometric mean values of 58 to 81 $\mu g/m^2$ for two inner city areas studied, with a range of values from 22 to 740 ppm. EPA (1986a) also reviewed reports on the relationship of lead in soil and dust to Pb-B levels. Generally, the review found that lead in soil and dust of 500 to 1,000 ppm begins to affect Pb-B levels in children (Baker et al., 1977; Mielke et al., 1984). The Mielke Twin Cities, Minnesota, study of inner city areas found that 50 percent of the individuals with lead poisoning lived in housing containing

soil-lead levels of 500 to 999 μg/g, and that 40 percent lived in homes with values of >1,000 μg/g. The authors cite both house paint and leaded gasoline as contributors. The study also found, from right-of-way soil samples, that lead levels low to high correspond to light to heavy traffic (however, the report did not indicate a p-value or other indication of statistical significance for this finding). Over half the Minneapolis homes in the study had soil-lead levels exceeding 50 μg/g. The variability of measured soil-lead concentrations, sometimes a 100-fold order of magnitude difference in concentrations between the front and back entrance of the home, was noted as a precaution in interpretation of the soils data. Clark et al. (1987) determined an increase of Pb-B by 6.2 μg/dL for each 1,000 ppm increase in soil-lead concentration. Studies (EPA, 1986a) show a range of values, from 0.6 to 6.8 μg/dL rise in Pb-B level for 1,000 ppm incremental increases in soil-lead concentration.

Recognizing that soil/dust information was not available at the time of the report (beyond a limited number of site specific studies), the (ATSDR, 1988) report authors recommend the use of multiple linear regression analysis to account for different contributions to a child's Pb-B level, preceded by a representative sampling of dusts and soils from the urban and rural areas of each of the nation's four major regions. Because such a statistically based representative sampling program was not available at that time, the report used an admitted overestimate of exposure by combining the major routes: paint lead in pre-1940 housing with the highest lead content - 5.9 million children; gasoline lead in the 100 largest cities - 5.6 million; and stationary sources - 0.2 million, for a total of 11.7 million exposed children (ASTDR, 1988). A reliable method to apportion Pb-B values to primary contributors was called for by ATSDR.

That call was answered in part by the Comprehensive and Workable Plan (DHUD, 1990) that derived soil/paint correlations, and speculated about the contributions to elevated blood-lead levels. From multiple regression and pathway analyses, the HUD report determined that excessive dust-lead levels occur more often in houses with LBP (intact or not) than in housing without LBP. Elevated blood-lead levels were associated more often with housing with nonintact LBP on exterior walls, than with intact exterior

LBP. HUD concludes that young children in homes having nonintact LBP, or excessive dust-lead, are at highest risk. Of 57 million occupied homes having LBP, less than 10 million are occupied by families with children under the age of seven. Of these homes, 3.8 million units have high dust-lead levels or nonintact paint.

According to the survey report, soil-lead is within the guidelines[5] of 500 ppm, 79 percent of the time that LBP is present. The analysis estimated the numbers of occupied dwellings with soil-lead associated with the presence and condition of exterior LBP. The percentages of homes exceeding the guidelines by strata were estimated to be 6 percent for homes with no LBP, 21 percent for homes with intact LBP, 48 percent for nonintact LBP, and 27 percent with homes containing any exterior LBP. Overall, 18 percent of 63 million occupied housing units are estimated to have soil-lead exceeding 500 ppm. A strong statistical association was thus found between the presence of lead-based paint and lead-contaminated soil. The probability of excessive soil-lead was derived as 4:1 for LBP exterior compared to LBP-free exterior.

The report analyzed hypothesized pathways from paint to dust, by determining the correlation coefficients between the natural logarithms of the pairs of survey measurements of lead associated with a pathway. The correlation coefficients determined were paint-on-wall:dust-on-window-sill—0.25; dust-on-window-sill:dust-in-window-well—0.46; dust-in-window-well:soil-at-drip-line—0.42-0.45; and soil-at-drip-line:soil-at-remote-location—0.68. All of the correlations were found to be statistically significant at or below 0.05, with some at the 0.001 level. Thus, if high lead concentrations were found at one location, values tended to be high everywhere.

The regression of dust variables on paint variables support the conclusion that paint is one of the sources of lead in dust. Derived R^2 values to discern the fraction of the dependent variable explained by

[5] The DHUD report refers to an EPA interim guideline having a range of values for soil lead concentration of 500 to 1000 ppm.

the independent variables yielded values ranging from 0.11 to 0.30. From the regression analysis, the authors conclude that lead from exterior paint is brought inside the house, and that lead from interior paint contaminates the soil outside the house. When the age of housing is added to the regression analysis, that variable helped explain lead levels in most of the regressions. The older the dwelling, the higher the estimated lead levels. The regression values ranged from 0.13 to 0.43. HUD postulates that age of the home "merely proxies for lead-based paint", and that age may measure other sources, such as auto emissions. HUD notes, however, the difficulty in estimating the percent of lead in dust and soil that can be attributed to LBP. From the regression analysis, approximately 20 to 25 percent of the variation in dust and soil-lead is explained by paint variables (HUD notes that this could be low). Consequently, the source of most of the lead in soil is not explained by the model.

Thorton et al. (1990) studied lead in garden soils and household dusts in England, Scotland, and Wales. They found that 10 percent of the floor dusts exceeded 2,000 μg/g. The two-year-olds and their home environs were sampled for inside dust, soil, road soil, wipes, food and water, and venous blood. The intent of the study was to assess lead intake from dusts in relation to other sources. The study reported a geometric mean for lead in the surface (zero to five cm) garden soils to be 266 μg/g and for house dust to be 561 μg/g. In London, the mean values were 654 μg/g for soils, and 1010 μg/g for dust. A highly significant correlation between household dust and garden soil was determined ($r= 0.531$, $p= 0.001$, $n=4512$). Overall, the geometric means were determined to be 11.7 μg/dL blood-lead; playroom air 0.27 $\mu g/m^3$; bedroom air 0.26 $\mu g/m^3$; external air 0.43 $\mu g/m^3$; dust soil 424 μg/g; soil 313 μg/g; dust loading 60 $\mu g/m^2$; handwipes 5.7 μg; food and beverage 161 μg/week; and water 19 μg/l. The study found that the correlation of Pb-B levels with indoor air concentration to be virtually zero. The correlation of blood-lead levels with dust-lead was determined to be $r= 0.34$, with water to be $r= 0.39$, and with soil-lead to be $r= 0.18$. The association with dietary variables was found not to be statistically significant.

The researchers used multiple linear regression to assess the relative importance of various sources,

with the model log Pb-B (μg/100 dl) = 0.55 + 0.10 log xi + 0.14 log PbW (μg/l) + 0.07 S, where

Pb-B = blood-lead concentration

PbW = water lead concentration

xi = dust loading x rate of hands touching all objects, and

S = 0,1 depending upon whether parents smoked cigarettes or not.

The analysis determined that adding air-lead concentrations, soil-lead concentrations, or dietary-lead intake gave nonsignificant regression coefficients, and only marginal improvements to the R^2 value. The study concluded that the Birmingham study for the first time demonstrated a relationship between levels of environmental lead within the home and blood-lead in a two-year-old child.

In this country, a comprehensive study was concluded in 1987 by the Minnesota Pollution Control Agency (MPCA) and the Minnesota Department of Health (MPCA, 1987). The report "provides the results of soil testing throughout Minnesota and blood-lead screening of children residing near sites in Minneapolis and St. Paul identified by the MPCA as having at least 1,000 parts per million (ppm) of lead in soil." The study sampled soils in five major cities and 27 counties in Minnesota, including census tracts in Minneapolis, St. Paul, Duluth, Rochester, and St. Cloud. A total of 2,485 soil samples were taken. Overall, 85.8 percent of the samples were found to be <500 ppm[6]. Only 7 percent of the samples were found to exceed 1,000 ppm. For areas designated as play areas, only five of 564 samples exceeded 500 ppm, and none exceeded 1,000 ppm. For foundation samples (defined as being within five ft of a structure), however, 53 percent (of 413 samples) exceeded 500 ppm, and 31 percent exceeded 1,000 ppm. Surprisingly, only 4 percent (22 samples) of the street side samples (generally, the parkway areas adjacent to the street) exceeded 500 ppm, and only one of 593 samples exceeded 1,000 ppm. As expected, the study found that samples taken from sites occupied by industrial point sources had very high lead

[6] The study notes that soil lead levels can vary by > 50 percent, depending upon the (laboratory) analytical method used, and also that soil samples from the same yard may vary by a factor of 100. Consequently, the mean soil concentration values are deemed to be of questionable value.

concentrations.

The researchers performed a regression analysis and found a general tendency for streetside soil-lead concentrations to increase with increasing traffic, but judged the relationship to be weak, with 29 percent of variation in streetside soil-lead concentration attributable to average daily traffic count. The study also noted that the degree of contamination in the streetside samples, ostensively from vehicular traffic, is far less than soil-lead concentration along foundations. Maximum soil-lead concentration accounted for little variation ($R^2 = 0.0541$, $p = 0.0060$) of measured blood-lead. The average daily traffic count accounted for some of the variation in street side lead concentration ($R^2 = 0.2888$, $p = 0.0001$). The report concludes that the relationship between soil-lead and blood-lead appears to be weak.

The Minnesota Department of Health (MDH) also conducted a lead screening program for youths aged 6 months to 6 years living near the sites with soil-lead concentrations exceeding 1,000 ppm. Of 743 children screened (742 EP test and 656 blood-lead tests), 13 were determined to have lead toxicity in accordance with the CDC criteria of > 25 μg/dL blood-lead and 35 μg/dL EP. Another 24 had elevated blood-lead (>25 μg/dL Pb-B but < 35 μg/dL EP), and 65 were determined to have iron deficiency. Twenty percent (134) of the inner city children tested had Pb-B equal to or exceeding 15 μg/dL. The Minnesota Department of Health noted that the children tested "live in older, poor, inner city neighborhoods dominated by lead painted housing, high traffic density, and the highest residential soil-lead concentrations found in the study." The average blood-lead level of screened children was 10 μg/dL, which the study compared to the NHANES 1980 national average Pb-B of 16 μg/dL for children under five years old.

2.6.5. Drinking Water

Most contamination via this source results from domestic plumbing and plumbing in public buildings, including lead pipe connections, lead-based solder in copper plumbing, and corrosive water in plumbing (ASTDR, 1988). Water fountains and drinking water coolers in schools and other public

buildings are potentially important sources, as well. EPA (1986a; ATSDR 1988) and ATSDR (1988) note that lead is absorbed in the human body at 35 to 50 percent from water, compared to 10 to 15 percent from food; consequently, lead in water poses a three to five multiple risk compared to food having the same lead concentration. Lead absorption rates are even higher for children, resulting in even higher increased risks of exposure. EPA (1986b) estimates that 42 million people in the U.S. may be exposed to lead in drinking water exceeding 20 µg/l at the tap. This is based upon 772 samples from a random grab sampling program conducted in 580 cities in 47 states. Data from this survey indicate that 16 percent of water from U.S. kitchen taps exceed 20 µg/l, noting also a problem of lead leaching from new water connections, that was not considered in the survey. In addition, the survey did not consider drinking water from water coolers in schools, another documented potential source of lead contamination.

ATSDR (1988) assessed exposure by age of housing stock, considering the use of lead pipes for pre-1920 homes, iron pipes for homes built between 1920 and 1949, the use of lead solder during the period 1950-1984, and that fresh solder may have been used during the two most recent years preceding the ATSDR report, 1985-6. Using this approach, the estimated population at risk is set at 1.8 million children in new housing, and 4.89 million in older housing (assuming 1\3 of the housing built before 1939, or 10 percent of the housing stock) contained lead pipes. The effects of corrosivity are also noted. From the 42 million people estimated to be exposed above 20 µg/dL (thought to result in an increase in Pb-B levels), 3,780,000 children (9 percent of 42 million) are estimated to be exposed. Levels in drinking water can be high (up to 1,000 µg/l) due to leaching of lead from lead pipe and leaded solder joints (EPA, 1991a). The concentration varies with the amount of lead in the plumbing and with the corrosiveness of the water. Soft or acidic waters tend to be more corrosive, and consequently tend to contain higher concentrations of dissolved lead. An analysis performed for the Environmental Protection Agency, which included public water supply systems' data for the States of Indiana, Michigan, and Minnesota, indicates that these states have only 1.6 percent of the public water suppliers delivering highly corrosive water (EPA

1988). In general, water with a pH of eight or above and high alkalinity is less corrosive than water with a pH < eight and low alkalinity (highly corrosive) (EPA, 1991b). EPA estimates the water from lead service lines to be 10 μg/l for water systems with highly corrosive water, and five μg/l for systems with moderately corrosive water (Memorandum, Cohan, 1991). In an EPA (1986b) analysis of the benefits of reducing the lead in drinking water standard, EPA estimated that 241,000 children had blood-lead levels exceeding 15 μg/dL due to lead in drinking water (as a result of the action of corrosive water on aged piping), including 11,000 having Pb-B levels exceeding 30 μg/dL.

2.6.6. Lead in Food

Lead enters food processing mainly through lead-soldered cans, which practice was to be phased out beginning in the late 1970s (ASTDR, 1988). Studies have found varying levels of lead intake in children, based upon foods consumed. Recognizing the centralized food distribution in this country, that all children (indeed the entire population) are exposed via this route, ATSDR estimates that 9 percent of the 1985 population, or 21 million children, are exposed by food intake. By making a series of assumptions and relying on the results of previous surveys, the report estimates that a maximum of 5 percent of children five months to six years of age are "at or approaching a dietary lead exposure that pushes their body burden close to that associated with early toxicity if they are also exposed to other typical lead sources" (ASTDR, 1988). The continual decline of lead in food, however, is noted, along with a myriad of uncertainties associated with the five percent estimate. By excluding children zero to five months of age, the population estimate of 21,405,000 (citing the World Almanac 1987) is reduced to 19,474,000. A 5 percent exposure rate would then result in 973,000 children at risk, based upon an Pb-B increase of 10 μg/dL.

2.7. Special Concern For Exposure Of The Fetus

To estimate exposure of the yet-to-be-born, ATSDR considered women in SMSAs of childbearing age for the year 1984, with four strata: white and African-American women, and age ranges 15-19 and

20-44. Using estimated prevalence and logistic regression to extend NHANES II Pb-B levels in the general population to the year 1984, the authors then applied the prevalence to the four strata for 1984. The resultant geometric mean Pb-B levels were 3.4 μg/dL for white females 15-19 years of age; 5.2 μg/dL for white females 20-44 years old; 5.1 μg/dL for African-American females 15-19 years old; and 7.3 μg/dL for African-American females 20-44 years of age. An estimated 41,300,000 females are thought to be in the four strata. ATSDR estimates that 3,595,000 could be pregnant (in a given year), with 403,200 (at risk annually, for fetuses of white and African-American women living in SMSAs) having Pb-B levels ≥ 10 μg/dL; 69,400 ≥ 15μg/dL; 14,500 ≥ 20 μg/dL; and 3,800 ≥ 25 μg/dL. The report acknowledges both overestimation and underestimation errors due to the limitations of methodology, data availability, and assumptions. Estimations were not calculated for individual SMSAs.

2.8. Special Emphasis: Ethnicity

A crucial finding of the ATSDR (1988) study is the substantial difference in estimated prevalence of blood-lead levels based upon ethnicity. The Agency provided projected percentages of children 6 months to 5 years old expected to exceed 15, 20, and 25 μg/dL Pb-B, who live inside central cities of Standard Metropolitan Statistical Areas with populations greater than one million. The starkest difference is at the lower socioeconomic level with annual family incomes of less than $6,000. For African-American children, an astounding 68 percent are projected to exceed 15 μg/dL Pb-B, compared to 36 percent for white children. A difference is indicated across all socioeconomic strata. For annual family income exceeding $15,000, 26.6 percent of African-American children are projected to exceed 15 μg/dL, contrasted to 7.1 percent for white children. This is compelling evidence of an increased exposure risk for African-American children. Because the projections rely upon NHANES II data, a similar comparison was not provided for Hispanic children. Data were not available from NHANES for such analyses. It is plausible, however, given similar socioeconomic circumstances of the African-American and Hispanic population, that Hispanic children could also be at increased risk of the harmful effects of low-level lead

exposure. There is, moreover, city specific analyses, based upon blood-lead screening programs, to support this contention.

Evidence that suggests that elevated blood-lead values are a significant concern in the Hispanic community as well as the African-American community is based upon screening programs, rather than upon epidemiological studies. Fernadez et al. (1990) studied the demographic patterns of 485 lead-poisoned children in the City of Chicago. Ninety-four of the cases studied were minority. Their analysis indicated that African-American and Hispanic children are disproportionately affected by lead poisoning. The study found that 69 percent of the cases were African-American children, and that 25 percent of the children were Hispanic. In contrast, Chicago's African-American population is 41 percent, and the Hispanic population is 17 percent. The analysis found that even in community areas with a "fairly even racial/ethnic composition", African-American and Hispanic children suffered disproportionately from lead poisoning. The authors noted limitations in the analysis. The sample data were not representative of the entire population of the city. True incidence could not be calculated. Further, the data was sometimes incomplete. The relationship to socioeconomic characteristics of the neighborhoods studied was also noted.

Data from the Minnesota Department of Health 1986-87 Blood Lead Survey supports this conclusion (Memoranda, Benson, 1991). The survey was conducted for 451 children in Minneapolis and 584 children in St. Paul. Data for St. Paul indicated an average blood-lead value of 9 μg/dL for African-American children, and 7 μg/dL for both Hispanic and white children. The population size for the latter, however, was quite small at 7 children. For Minneapolis, the average values were 9 μg/dL for African-American children also, but 8 μg/dL for white children, and 12 μg/dL for Hispanic children. Overall, for five Minnesota cities including Minneapolis and St. Paul, the analysis determined the percent of those screened exceeding 10 μg/dL blood-lead. The percentages were 33.3 percent for African-American children, 25.7 percent for white children, and 43.9 percent for all others (including Hispanic, but excluding

American Indian children). The author notes also that the screened children are not necessarily representative of the entire population of the cities.

The Public Health Service (PHS), in Healthy People 2000 (Health and Human Services, 1991a), has set an objective to "Reduce the prevalence of blood-lead levels exceeding 15 μg/dL and 25 μg/dL among children aged six months through five years to no more than 500,000 and zero, respectively." The baseline for the objective is an estimated three million children with Pb-B levels exceeding 15 μg/dL, and 234,000 children with Pb-B levels exceeding 25 μg/dL, in the year 1984. The 1984 baseline of inner-city low-income African-American children (having an annual family income <$6,000 in 1984 dollars) was 234,900 exceeding 15 μg/dL (with a year 2000 target objective of reduction to 75,000 children), and 36,000 children exceeding 25 μg/dL (with the corresponding year 2000 target objective of a reduction to no children). The Public Health Service refers to this as a special population target. Such a special emphasis is supported by the findings of Danford et al. (1982a). Danford and her colleagues found, in a study population consisting of mentally retarded individuals, that 30 percent of African-American children aged one to six years have abnormal ingestion behavior, compared to 10-18 percent in the same age strata for white children. Danford (1982b) notes, however, that interpretation of survey results on the incidence of pica is complicated by several factors, including limitations on statistical methods used, inconsistent definitions of pica, and (statistically) small numbers of subjects. Danford also cites a cultural hypothesis for pica. In some African cultures, the consumption of soil during pregnancy is thought to suppress nausea. She asserts that, "given the deeply ingrained geophagy of the African cultures that supplied the bulk of slaves to the New World, it is not surprising that the practice persists in the black subculture of the United States." Consumption of lead-contaminated soil, as a consequence of such practices, would cause elevated blood-lead levels.

PHS further, in its Strategic Plan for the Elimination of Childhood Lead Poisoning (HHS, 1991), asserts that "Poor, minority children in the inner cities, who are already disadvantaged by inadequate

nutrition and other factors, are particularly vulnerable to this [lead poisoning] disease." The Strategic Plan focuses heavily upon lead poisoning because of its importance to public health protection.

Needleman (1990) speculates about the social cost of exposure, based upon his ongoing study of a cohort of children followed into the 19th year of life. Needleman and David Bellinger had found, when the cohort was in grade five, that the incidence of grade retention at that time was significantly higher in the high (blood) lead group. Further, the attention span of the high lead group was disturbed. Needleman, in retesting 132 of the children, found the relative risk for not graduating from high school, associated with lead, to be 4.8. He asserts that the high lead group in adult years are clumsier, have poorer reading scores, more depression, and higher rates of hard drug use (no statistical presentation, however, was provided in the paper). Further study is to be done. Bellinger et al. (1990) add that "Children already stressed by sociodemographic disadvantages may be less able to weather the additional stress of high prenatal lead exposure."

The Needleman analysis also indicates that lead is associated with increased risk for attention deficit disorder (ADD) (attributable risk of 0.51), and that attention deficit disorder in turn is a risk factor for antisocial behavior. Needleman determined the attributable risk for antisocial behavior, given ADD, to be 0.58. Using these findings, he postulates a joint probability of delinquency, given lead exposure, to be that 20 percent of (juvenile) delinquency is lead-associated. He is currently examining this relationship.

2.9. Research Needs

The Public Health Service (HHS, 1991b) calls for research studies to determine the relative contributions of various pathways of lead to children's blood-lead levels, particularly from paint, dust, soil, air, food, water, parental occupations, and hobbies. HHS notes particularly that the dietary contribution of lead in calcium supplements, especially when consumed by pregnant women, should be assessed.

The ATSDR (1988) report aptly describes the current situation on lead exposure: *"At the same*

time that progress is being made to reduce some sources of lead toxicity, scientific determinations of what constitute 'safe' levels of lead exposure are concurrently declining even further. Thus, increasing percentages of young children and pregnant women fall into the 'at-risk' category as permissible exposure limits are revised downward. Accompanying these increases is the growing dilemma of how to deal effectively with such a widespread public health problem. Since hospitalization and medical treatment of individuals with Pb-B levels below approximately 25 μg/dL is neither appropriate nor even feasible, the only available option is to eliminate or reduce the lead in the environment" (emphasis added). In concluding its report to Congress, ATSDR (1988) cites the need for comprehensive studies, at the regional level, of the impact and geographic distribution of lead sources upon exposed populations.

The need to eliminate low-level environmental sources of lead is clear. Far too many children are still exposed to concentrations of lead in dust and soil that cause unacceptable blood-lead levels. A lessor number are exposed to excessive air-lead and lead in drinking water. The relatively higher risk that confronts African-American and Hispanic children, compared to the general population, is also apparent. It is uncertain, however, where these children are located, and in what numbers, due to such environmental exposures. Gathering actual data for all environmental pathways of exposure for the entire population is neither practical nor feasible. Even creating such a data base for the much smaller minority childhood population would be a daunting task. Consequently, as an alternative, a population screening methodology to guide public health officials to geographic areas where children are at high risk, is needed.

3. STUDY OBJECTIVES

Children under seven years of age having low blood-lead levels, resulting from environmental exposures to lead from multiple pathways of exposure, experience a significant health threat. Further, the danger posed to specific communities within the Midwest region of the nation, is oftentimes not detected via either environmental monitoring of exposures to lead and lead compounds, or via biological measurements such as ascertainment of blood-lead levels. Consequently, large numbers of children at risk to low level exposure to lead are undetected and thus, unprotected.

OBJECTIVE 1: Develop a population comparative risk approach for estimating the number and location of African-American and Hispanic children under seven years of age, at risk of exposure to lead with blood-lead levels exceeding 10 μg/dL. Include a "hot spot" selection scheme that accounts for all known routes of environmental exposure to lead.

OBJECTIVE 2: Conduct an analysis to ascertain the predictive ability of the approach for selecting "hot spot" areas, by comparing modeled blood-lead levels to measured blood-lead levels.

OBJECTIVE 3: For a selected city, examine the association of elevated blood-lead levels with proximity of children to transportation corridors (lead exposure due to historical deposition of lead in gasoline and/or current emissions from mobile sources).

4. METHODOLOGY

4.1. Study Scope and Methodology Overview

In 1987 the USEPA published a document entitled *"Unfinished Business"*, which provided a best professional judgment review of agency programs and environmental problems from the perspective of comparative risk. Since that time, each individual medium program office at USEPA headquarters, as well as each of the 10 regional offices, were tasked with development of a comparative risk analysis pertinent to the program or geographic region of concern. The intent of the approach was to discern and prioritize environmental problems affecting human health and the environment, to determine whether Agency programs were adequately addressing the existing and emerging environmental concerns, and to assess whether resource shifts (generally at the margin) could impact priority environmental problems that otherwise would not be addressed. The Region 5 office's comparative risk study was completed in the summer of 1990. Several cross-cutting concerns were identified. Lead was identified by several program areas as one of the multi-program pollutants of concern. The region selected lead as a priority area, and tasked the program managers, and a project director, with development of a comprehensive strategy and implementation plan to address and remediate lead contamination in the six state region.

The group recognized that lead poisoning in children is now considered to be a national epidemic by many in the public health community. Lead exposures from exterior and interior residential paint, in particular, as well as exposures from contaminated soils and dust in and around structures present in most urban areas, drinking water, air emissions, food, occupational settings, and hobby activities, result in multiple pathways of exposure. These exposures are responsible for a number of adverse health effects in humans, especially in children. Because children are at elevated risk, a targeted population has been chosen to be children under seven years of age. Within this population group, African-American and

Hispanic children are particularly targeted in recognition of increased body burden susceptibility and thus vulnerability to the uptake and effects of lead exposure.

Project LEAP is a multi-media and multi-program approach having four basic components: data analysis and targeting; pollution prevention; education and intervention activities; and abatement activities. The project is being implemented in three phases. It is a component of the Agency Lead Strategy. Project LEAP Phase 1 focuses on data analysis, air modeling of major sources, prioritization of sources and areas for targeting purposes, and selection of geographic areas for attention during the subsequent phases of the Project. Phase 2 will focus upon specific geographic areas of concern with an emphasis upon on-site measurement, e.g., of soil and dust concentrations. Phase 2 will also include continuation of pollution prevention efforts, and initiation of public education and outreach efforts in coordination with other agencies. Phase 3 is envisioned to be actual abatement activities for a selected communicity.

Lead exposures from exterior and interior residential paint, in particular, as well as from contaminated soils and dust in and around structures present in most urban areas, drinking water, air emissions, food, occupational settings, and hobby activities, result in multiple pathways of exposure. These exposures are responsible for a number of adverse health effects in humans, especially in children. Because children are at elevated risk, a targeted population has been chosen to be children under seven years of age, as well as the fetus. Within this population group, African-American and Hispanic children are particularly targeted in recognition of the vulnerability of this population to the uptake and effects of lead exposure.

The approach of this effort was to estimate the probability distribution of blood-lead in childhood populations. Determination of severity for each city would then allow for comparisons of geographic areas. For each metropolitan statistical area central city, environmental data were obtained for the major sources/routes of exposure (i.e., point sources of air emissions, municipal waste combusters as a special case categorical source of air emissions, ambient air quality measurements, drinking water supplies, and

operating as well as abandoned hazardous waste sites). Where available, actual concentrations were used. Default values were established for each environmental medium where actual measurements had not been taken. Sensitivity analyses were conducted to assess the impact of assumed (default) values on the blood-lead uptake estimate.

Demographic information was obtained from a geographic information systems application (derived and provided by the Geographic Information Systems Management Office, Region 5, EPA). Information was provided at the census tract or community area (aggregation of census tracts) levels for each city. Environmental data (i.e., media concentrations) associated with each tract were provided in order to calculate blood-lead level distributions in affected populations.

A single geographical area, Minneapolis/St. Paul, Minnesota, was selected to test the viability of the approach. That area had measured blood-lead levels available, along with pertinent demographic information. A simple correlation analysis was conducted to ascertain whether modeled blood-lead levels were associated with actual measured blood-lead levels. An association would indicate the viability of the approach in comparing cities.

Based upon environmental concentrations for each census tract/community area, the Uptake Biokinetic Model (described in Section 5.5) was run to calculate an expected percent lead exceedance for the pertinent area. The percentage, applied against the population data for the tract, provided an estimate of the number of children under seven years of age at risk. Further aggregations of geographic areas provided city totals.

4.2. Study Area

The study area includes 83 cities located in 60 metropolitan statistical areas in the Midwest. These cities represent the central cities in all of the metropolitan statistical areas in the States of Illinois, Indiana, Minnesota, Wisconsin, Michigan, and Ohio. Each city is shown, along with selected demographic information, in TABLE IV.

TABLE IV
Metropolitan Statistical Area Central City Demographics[7]

City	Population	% White	% African-American	% Hispanic	Total Births 1984	Birth rate per 1,000 Pop.
Rock Island	43,720	82.48	15.18	3.36	695	15.3
Moline	44,500	95.64	1.17	5.38	667	14.5
Chicago	3,005,072	49.59	39.83	14.05	53912	18.0
Kankakee	27,220	70.53	28.19	1.08	543	19.1
Peoria	110,290	81.49	16.69	1.39	1931	16.5
Bloomington	46,250	92.80	5.70	1.38	860	18.5
Normal	36,790	91.99	6.07	.79	368	9.8
Champaign	59,180	84.52	12.74	1.23	800	13.3
Urbana	35,770	84.08	9.99	1.76	560	16.4
Rantoul	N/A[8]					
Springfield	100,290	88.04	10.79	.66	1817	17.9
E. St. Louis	49,470	4.16	95.56	.94	1474	28.7
Granite City	35,150	98.76	.20	1.61	560	15.7
Rockford	135,760	84.27	13.19	2.89	2294	16.8
Total State of Illinois	11,548,000	80.81	14.66	5.56	179274	15.6
Gary	136,790	25.16	70.84	7.10	2574	18.0
Hammond	86,380	89.48	6.40	8.30	1224	13.7
E. Chicago	36,950	47.85	29.66	42.27	617	16.6
South Bend	107,190	79.50	18.29	2.36	1862	17.4
Mishawaka	41,400	97.93	1.08	.71	602	14.6
Elkhart	44,180	86.02	12.56	1.28	866	20.1
Goshen	N/A					

[7] Source: County and City Data Book 1988, U.S. Department of Commerce, Bureau of the Census.

[8] N/A not available. Data was not attainable for these cities.

City	Population	% White	% African-American	% Hispanic	Total Births 1984	Birth rate per 1,000 Pop.
Ft. Wayne	172,900	83.24	14.55	2.20	3166	19.1
LaFayette	44,240	97.14	1.63	1.14	848	19.2
Kokomo	45,610	90.57	8.11	1.41	843	18.6
Anderson	61,020	85.65	13.70	.63	828	13.4
Muncie	72,600	89.47	9.54	.80	969	13.1
Indianapolis	719,820	77.10	21.78	.88	12812	18.0
Terre Haute	57,920	90.11	8.49	.77	891	15.2
Bloomington	52,500	91.10	4.31	1.59	688	13.2
Evansville	129,480	90.36	8.83	.49	1954	15.0
New Albany	37,260	94.32	5.19	.61	566	14.9
Total State of Indiana	5,504,000	91.15	7.55	1.59	80084	14.6
Saginaw	72,470	57.37	35.55	9.01	1557	21.1
Bay City	39,700	94.69	1.79	4.68	701	17.6
Midland	35,890	96.26	1.39	1.42	537	14.2
Muskegon	39,810	76.04	21.42	2.98	867	21.9
Grand Rapids	186,530	80.93	15.73	3.16	3937	21.5
Lansing	128,980	80.42	13.94	6.32	2566	20.1
East Lansing	48,120	90.31	5.22	1.80	404	8.6
Flint	145,590	56.17	41.43	2.49	3129	21.0
Detroit	1,086,220	34.38	63.07	2.41	18523	17.0
Ann Arbor	107,810	85.10	9.33	2.08	1414	13.1
Battle Creek	54,080	75.00	22.79	1.90	948	17.4
Jackson	36,970	82.45	15.43	2.03	705	18.7
Kalamazoo	77,230	81.40	15.60	1.87	1416	18.3
Benton Harbor	N/A					
Total State of Michigan	9,155,000	84.99	12.95	1.75	136076	15.0

City	Population	% White	% African-American	% Hispanic	Total Births 1984	Birth rate per 1,000 Pop.
Moorhead	28,360	97.80	.46	1.02	426	14.6
Duluth	82,380	96.98	.83	.45	1298	15.2
St. Cloud	42,850	97.70	.53	.44	718	17.1
Minneapolis	356,840	87.30	7.66	1.26	6301	17.6
St. Paul	263,680	90.01	4.92	2.91	5040	19.0
Rochester	58,130	97.37	.65	.71	1297	22.3
Total State of Minnesota	4,075,970	96.56	1.31	.79	66716	16.0
Toledo	340,680	80.06	17.41	3.01	5594	16.3
Cleveland	535,822	53.55	43.80	3.10	10162	18.6
Akron	222,060	76.78	22.23	.65	3451	15.2
Lorain	72,210	79.44	11.89	14.36	1114	15.3
Canton	87,110	83.06	15.99	1.31	1508	16.9
Steubenville	23,580	84.72	14.25	.72	319	13.1
Wheeling	N/A					
Marietta	N/A					
Youngstown	104,690	64.43	33.34	3.32	1641	15.2
Warren	52,900	81.12	18.13	.66	934	17.3
Mansfield	51,340	83.08	16.05	1.11	955	18.4
Lima	45,990	78.72	20.41	1.10	881	19.1
Dayton	178,920	62.05	36.89	.86	3535	19.5
Springfield	69,500	81.87	17.24	.73	1158	16.5
Columbus	566,030	76.24	22.11	.82	10406	18.4
Hamilton	65,050	91.75	7.16	.69	1208	18.9
Middletown	46,090	88.00	11.57	.46	818	18.7
Cincinnati	369,750	65.15	33.85	.78	7312	19.7
Total State of Ohio	10,752,000	88.68	9.97	1.11	158519	14.7

City	Population	% White	% African-American	% Hispanic	Total Births 1984	Birth rate per 1,000 Pop.
Eau Claire	54,580	98.66	.25	.38	793	14.7
Wausaw	32,240	98.80	.07	.30	519	16.3
Green Bay	93,470	97.25	.25	.68	1542	17.1
Oshkosh	51,190	98.35	.59	.52	741	14.8
Neenah	N/A					
Milwaukee	605,090	73.34	23.10	4.10	11800	19.0
Racine	82,440	81.91	14.74	.34	1642	19.7
Kenosha	74,960	93.89	3.62	4.00	1232	16.3
Madison	175,830	94.33	2.70	1.31	2580	15.1
Janesville	51,790	98.95	.22	.71	901	17.5
Beloit	33,760	86.99	11.30	1.00	583	17.1
LaCrosse	47,650	98.75	.29	.48	710	14.9
Sheboygan	47,410	98.28	.12	1.60	812	17.0
Appleton	64,190	98.27	.08	.55	1052	16.9
Total State of Wisconsin	4,785,000	94.42	3.88	1.34	73187	15.4

4.3. Contribution to Childhood Lead Levels From Air Emissions

4.3.1. Industrial Source Complex Long Term Model

Air-lead concentrations resulting from significant point sources were estimated using the Industrial Source Complex Long Term (ISCLT) Model, Personal Computer Version. The model is an advanced Gaussian plume model that uses the steady-state Gaussian plume equation for a continuous source to calculate concentrations for point sources. The model uses statistical wind summaries to calculate seasonal or annual concentration values, and a wind-profile exponent law to adjust the observed mean wind speed from the measurement height to the emission height for plume rise and other parameters. Plume rise is calculated due to momentum and buoyancy as a function of downwind distance for stack emissions. Pasquill's method is used to account for buoyancy induced dispersion.

The ISCLT requires input data arrays of the joint frequency of occurrence of wind speed and direction for each Pasquill stability category and season (when the season option is selected); an array of the mean ambient air temperatures as a function of stability category and season; and an array of the median mixing layer heights as a function of wind speed, stability category, and season. Source specific information needed includes emission release rate, stack height and diameter, gas exit velocity, and gas exit temperature. The "regulatory default" option of the model was selected for the analysis. The regulatory default option includes final plume rise at all receptor locations, stack-tip downwash, buoyancy induced dispersion, default wind profile coefficients, default vertical potential temperature gradients, and revised wake effect procedures.

The particle size distribution was added to the model, in accordance with Agency recommendations (Rothblatt Memorandum, "Refined Metals Lead Modeling Analysis, December 8, 1989), as shown in Table V. This particle size distribution provides a better estimate of the actual particle sizes expected, in comparison to the default particle size distribution in the ISCLT model.

TABLE V
Particle Size Distribution Input to
Industrial Source Complex Model

Mean Mass Diameter (μm)	Settling Velocity (m/sec)	Settling Fraction	Reflection Coefficient
.079	.000129	.237	1.0
4.08	.00363	.157	1.0
11.1	.0262	.015	.68
20.4	.0877	.20	.52
30.27	.194	.16	.26
40.19	.342	.12	0
50.15	.532	.08	0
60.11	.764	.04	0

Meteorological input arrays were obtained by the following process. Surface meteorological data files were obtained from the National Climatologic Data Center weather monitoring stations closest to the source to be modeled, along with upper air data files. A "STAR" (Stability Array) program was run on each set of meteorological data in order to convert the data into the format used by the ISCLT model. The STAR program converted the raw meteorological data into the proper format required by the ISCLT model for the joint frequency of occurrence of wind speed and direction for each Pasquill stability category A through F. A file of hourly temperatures was also created. The temperature and upper air data files were further processed using a statistical analysis program to derive the mean air temperatures and mixing layer heights, and to calculate the median mixing heights by stability and wind speed categories. The statistical analysis program was used as a convenient method for calculating mean and median values, and for sorting data for use by the ISCLT program. The three data arrays were incorporated into a single file specific to each source.

Emission rates are those reported in the Toxics Release Inventory data base for 1988 (raw data was retrieved from the U.S. EPA national computer center). Steady-state emissions were assumed for the source to calculate a gram per second emission rate from the annual loading.

The Aeorometric Information and Retrieval Facility Subsystem (AIRS-FS) was utilized as the data source for facility specific information on physical properties of emission releases, that were required to model emissions at 17 selected facilities. As an approximation, multiple stacks were combined into a single stack by weight-averaging emissions. For modeling purposes, the derived stack height, stack diameter, temperature of gas at release, and gas exit velocity were used, together with the Toxic Release Inventory (TRI) reported emission rate for the facility. The AIRS-FS also contained emission data for 532 sources of lead emissions. That information, however, was deemed inappropriate for use by the project. Much of the data had been estimated. The estimation factors are currently being updated. The use of the quantity of lead data in AIRS-FS, based upon the existing emission factors, would provide questionable results. Consequently, quantity emission from the TRI data base was used. Stack height, stack diameter, and stack gas exit temperature were obtained from the AIRS-FS data base for eight of the 17 sources: LaClede Steel Co., Alton, Illinois; Chemetco, Inc., Hartford, Illinois; Refined Metals Corp., Beech Grove, Illinois; Quemetco, Inc., Indianapolis, Indiana; Inland Steel Co., East Chicago, Indiana; Kohler Co., Kohler, Wisconsin; Gopher Smelting and Refining Co., Eagan, Minnesota; and North Star Steel, St. Paul, Minnesota. The ISCLT model was run for these sources, and for the additional seven sources using default values.

4.3.2. ISCLT Sensitivity Analysis

Recognizing that upper air data (used to discern mixing heights in the model) are available for a very small number of weather stations (Flint, Michigan; Dayton, Ohio; Green Bay, Wisconsin; and St. Cloud, Minnesota) for 1988, the ISCLT model was run using each, and the resulting lead concentrations compared at the four locations. The locations included x and y coordinates (-2000,2000), (0,2000),

(200,0), and (2000,2000) in meters (Figure 1). The Flint, Green Bay, and St. Cloud upper air stations (resulting in three observations for each x,y coordinate location) provided extremely close air-lead concentrations, as shown in Table VI. Running the ISCLT model with data for the three upper air stations results in a mean value of 1.693 $\mu g/m^3$ air-lead. The standard deviation of 0.004 is quite small. Consequently, the choice of upper air station for inclusion in the modeling of air-lead concentrations is basically irrelevant, especially given the other assumption made in order to run the model. Green Bay, which provided values close to the mean values, was selected.

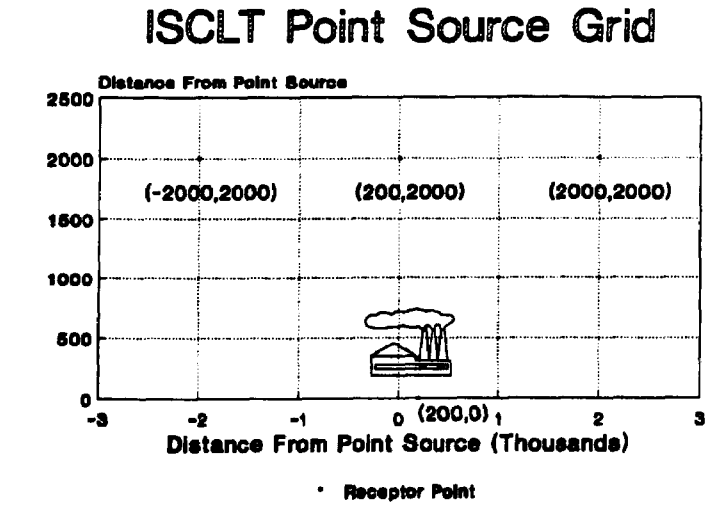

Figure
Industrial Source Complex Long Term Point Source Grid

TABLE VI
Upper Air Data Analysis

Analysis Variable: Lead Concentration

Location of x,y coordinate	No. of Observations	Minimum ($\mu g/m^3$)	Maximum ($\mu g/m^3$)	Mean ($\mu g/m^3$)	Standard Deviation
1	3	0.043570	0.050765	0.047196	0.003630
2	3	0.111866	0.130074	0.120823	0.009107
3	3	1.693079	1.700655	1.697299	0.003861
4	3	0.033252	0.041245	0.037243	0.003996

A similar series of model runs were conducted with varying source specific inputs of exit gas temperature, stack diameter, and stack height, recognizing that those parameters were not available for all sources. A model default of stack temperature of 432 degrees Kelvin, stack diameter of 2.4 meters, stack height of 35 meters, and stack gas exit velocity of 11.4 meters/second were compared to varying inputs for a source at the same emission rate. The modeled lead concentrations, for a selected point -200,0 meters west of the source, are shown in Table VII.

TABLE VII
Industrial Source Complex Long Term
Model Run Comparative Analysis

Run No.	Temperature (Degrees K)	Stack Height (meters)	Stack Diameter (meters)	Exit Velocity (m/sec)	Concentration of Air-Lead ($\mu g/m^3$)
Default	432	35	2.4	11.7	0.055
Run 1	426	34	1.7	11.7	0.085
Run 2	121	22	7.2	11.7	0.044
Run 3	432	35	2.4	35.1	0.023

Decreasing the stack diameter from 2.4 to 1.7 meters, along with very minor changes to the exit temperature and stack height, results in an increase above the "default" run from 0.055 $\mu g/m^3$ to 0.085 $\mu g/m^3$ at selected grid point (-200,0). Both values indicate that associated quarterly values would be well below the ambient air quality standard of 1.5 $\mu g/m^3$ quarterly average. A reduction in the stack gas temperature from 432 degrees K to 121 degrees K, along with a lower stack height (35 m to 22 m) and a larger stack diameter (2.4 m to 7.2 m) results in a slight decrease in the concentration from 0.055 $\mu g/m^3$ Pb to 0.044 $\mu g/m^3$. Tripling the gas exit velocity from 11.7 to 35.1 m/sec, while holding all other parameters constant, results in a decrease in the concentration to 0.023 $\mu g/m^3$. Consequently, choosing the default value for a source in place of a source specific exit velocity, where the actual exit velocity at the source is greater, would result in a conservative (i.e. higher) estimate of air-lead concentrations.

4.3.3. Ambient Air Data

Lead concentrations in the ambient air are reported as part of the National Ambient Monitoring System/State and Local Monitoring System (NAMS/SLAMS). The network of monitoring stations is administered by State and local agencies. Monitors are sited to ascertain compliance with criteria air pollutant standards, including lead. Although the monitors are not strategically placed to be statistically representative of a geographic area, the measured air quality at the stations do provide an indication of overall air quality in an area. Many are sited near point sources or in locations expected to experience maximum spatial concentrations.

By nature of the siting criteria, many of the Metropolitan Statistical Area cities had actual concentration data for lead. The data quality is excellent, because the EPA conducts a rigorous quality assurance program for the NAMS/SLAMS system. Flagged data indicates that the data is of questionable quality. The results of 1988 monitoring data were obtained for project analysis. Where monitored data were available, the annual average concentration was used to characterize air quality for a city. The

program default of 0.20 $\mu g/m^3$ was selected when actual monitoring data was not available. The ambient data was provided to the EPA Geographic Information Systems Management Office to create a spatial data coverage for comparison to estimated concentrations derived from point source modeling.

A summary of the ambient air concentrations is contained in Appendix A.

4.3.4. Air Emissions

The Toxic Release Inventory (TRI) data base was utilized as a source of information for point sources of lead emissions, particularly for air emissions. The national computer center was queried for a listing of all releases in the Midwest of lead and lead compounds, and that data was subjected to further analysis.

TRI was chosen because it is the most comprehensive data base available on toxic releases into the environment. The data is provided to the EPA and the states as required by the Emergency Planning and Community Right-to-Know Act of 1986. According to the 1990 report, "Toxics in the Community National and Local Perspectives" (EPA, 1990), an analysis of data quality and completeness found the data to be quite accurate in the aggregate. On-site visits by Agency personnel determined that the total volume of reported releases were just 2 percent lower than corrected figures. The audit found that almost 80 percent of all release estimates were without error. It is noted, however, that only two-thirds of the companies nationwide that were required to report, did so. Further, not all manufacturing facilities must report; consequently, the TRI data base does not account for all toxic emissions.

4.3.5. Municipal Waste Combusters

Municipal Waste Combusters (MWCs) were analyzed as a special category of potential air emissions of lead. The sources, which are not required to report by the Emergency Planning and Community Right-to-Know Act of 1986, but could have substantially large emissions of lead due to the incineration of lead in the municipal waste stream. The EPA Region 5 Municipal Waste Combuster Coordinator provided a listing of MWCs in the six states. Further information on the facilities was

obtained by direct written and verbal communication with State agency personnel and facility operators. The sources were to be modeled to discern air concentrations resulting from operations, if a facility were deemed to be an important source (i.e., operations would be expected to result in a measurable increase in the ambient lead concentrations).

4.4. Drinking Water Data

The Federal Data Reporting System (FRDS), operated at the EPA National Computer Center, was accessed for information on violations of the drinking water standard. FRDS tracks community water supplies' compliance with monitoring requirements, maximum contaminant level (MCL) exceedances, variances, enforcement actions, and population.

State agency records were also obtained. Community water supplies are required to participate in a quality assurance program, and to report the results of data analysis to the state agency. Data quality is consequently considered to be excellent. The test results for a city was used, when available. When a non-detect value is reported, one-half of that value was used, in accordance with EPA risk assessment guidelines (EPA, 1989). Otherwise, a UBK program default value of 4.0 μg/l was used. Corrosivity of drinking water supplies was taken into account to recognize the contribution from lead pipe leads, by assuming a higher value for drinking water in homes built prior to 1949. Housing age data was provided only as prior to 1949. This was done as a substitution for homes built before 1920, which are more likely to have lead-pipe leads).

4.5. Soil and Dust Contributions to Elevated Blood-lead Levels

4.5.1. RCRA and Operating Landfills

There is no central data base to query for Resource Conservation and Recovery Act (RCRA) facilities that currently treat, store, or dispose of lead and lead-based compounds. Consequently, information on facilities operating in the MSA cities was obtained by contacting RCRA program personnel at EPA and the state agencies. In many cases, facility operators were contacted to obtain more specific

information. The TRI data base was accessed to find facilities disposing of lead and lead compounds on site (designated operating landfills). Each site was then characterized as to the potential for human exposure.

4.5.2. Abandoned Hazardous Waste Sites Data

A November 1989 listing, Final and Proposed NPL (National Priority List) Sites With Lead, was used to identify NPL Sites in the six states that listed lead as a primary or major constituent of concern. Specific information on sites located in the MSA cities was obtained from summary sheets and from the more comprehensive reports on file for each facility.

4.5.3. Derivation of Soil and Dust Values

Data developed and utilized by the Department of Housing and Urban Development to prepare the Comprehensive And Workable Plan (DHUD, 1990) were obtained to derive values for soil and dust concentrations. Although data were coded by region, which would allow assessing data specific to the Midwest states, the national data base was selected to avoid weakening the representativeness of the statistically based sample. The data were generated under a rigorous quality control regimen, and is of good quality, except for dust concentrations reported in ppm. Problems with use of that information was flagged by DHUD due to problems in the laboratory. The weight of the filter, upon which dust was collected, could not be accurately measured. Consequently, the values in the data base may not be accurate. The calculated values, however, although flawed, are ordinate indications of dust concentrations, in that lead-dust concentration increases with age of dwelling. The data were therefore judged to be adequate for use when categorized by housing age bands. The validity of this judgement is assessed in the UBK sensitivity section 4.7.1.

In order to calculate mean soil and dust concentrations from data in the DHUD data base, several soil and dust sampling locations were selected for analysis. The locations were standard points of reference from which DHUD obtained samples at each home. For example, soil concentrations were

determined at the front entrance and rear yard locations for each home. Selected locations included dust mass concentrations in ppm at six locations, and dust sample results at six locations in $\mu g/ft^2$. A statistical program was run on these to obtain minimum, maximum, mean, and standard deviations of lead, by housing age category. The calculated values from two locations for each home, one for dust and one for soil, were used to characterize soil and dust concentration for each census tract. These values were used for inclusion in the Uptake Biokinetic (UBK) Model.

Results of a statistical analysis of selected variables from the National Housing Survey, including derived variables used to calculate soil arithmetic means and geometric means for dust concentration samples, are shown in Appendix B. Dust concentration in ppm at the common-entrance location (of all the houses surveyed), and soil concentration at the dwelling-entrance location, were selected for use in the study. Values are shown in Table VIII. These values, rounded and prorated to reflect actual housing counts in each area, were used in the UBK model as the soil and dust concentrations associated with the age of housing for each census area (see page 84).

Table VIII
Soil and Dust Concentrations For Pb Based Upon
DHUD National Housing Survey Data

Year House Built	No. of Observ.	Dust Value (ppm)				Soil Value (ppm)			
		Min.	Mean	Max.	Stand. Dev.	Min.	Mean	Max.	Stand. Dev.
1960-1979	120	0	20	1520	145	1	90	2500	285
1940-1959	87	0	55	1410	225	1	175	4040	465
Pre-1940	77	0	565	33130	3780	1	555	6260	1060

4.6. Lead Uptake Biokinetic Model

The Uptake Biokinetic Model was developed by the U.S. EPA and was validated using blood-lead and associated environmental concentrations for individual children (e.g., soil and dust values for the child's home). The model has not been validated at the large scale as applied by the study methodology. ***It is specifically emphasized that this research explores the application of the model on a scale much different from the original design and intent of the model. The effort is to determine whether the UBK model can be used as an effective risk management tool to suggest which areas might have comparatively more children at risk to environmental sources of lead. Thus, previously the UBK model has been used to predict site-specific distributions of blood-lead levels in childhood populations in the vicinity of lead point sources.*** This alternative use is to compare modeled levels between cities and areas within cities.

The model uses assumptions regarding behavioral and physiologic parameters that determine intake and absorption of lead from air, soil, dust, drinking water, and lead point sources. Behavioral and physiologic assumptions vary by age of child, and include time spent indoors and outdoors; time spent sleeping; breathing volume; deposition efficiency in the respiratory tract; diet (based upon a national food basket survey, and not specific to the Midwest or to individual cities); and absorption efficiency in the gastrointestinal and respiratory tracts.

The Uptake Biokinetic Model PC Version 0.5 (EPA, 1991d) is thus a mathematical simplification of lead exposure-effect relationships. The model uses estimates of exposures to predict the distribution of blood-lead concentrations in populations, for user selectable age ranges of children. This analysis uses the full age range of the model, 0 to 84 months of age, and 10 μg/dL as the cutoff point for exceedances.

Marcus' study suggests that the default value Geometric Standard Deviation (GSD) of 1.42 used by the UBK model, based upon the nationwide NHANES II study, may be too small (Marcus, 1991). In U.S. communities having much lower blood-lead values, and where there are a diversity of lead sources,

for some children in smelter and mining towns indicated a range of unadjusted GSD values from 1.67 to 1.79, in three very disparate types of communities (Marcus, 1991). The communities assessed were Kellogg, Idaho; East Helena, Montana; Leadville, Colorado; Telluride, Colorado; and Midvale, Utah. Marcus analysis calculated both raw and adjusted GSDs for these communities. He determined, for purposes of his analysis, that a GSD value of "... 1.66 fits neatly between the maximal raw GSD and the minimal adjusted GSD in all cases ...". Although the default value for the geometric standard deviation (GSD) of blood-lead values is 1.42, appropriate for point sources of lead, this analysis uses a standard deviation of 1.7. The wider value is more appropriate for area sources of lead. In addition, the wider GSD better reflects the uncertainty of the spread in blood-lead data values for a population.

Table IX provides the UBK model default values for indoor air concentration, diet (based upon Food and Drug Administration National Food Basket Survey for 1988), soil and dust, and paint. The UBK model Calculated Blood Pb and Pb Uptakes shown in Table X are those derived from the default values shown. The associated Figure 2 displays the probability density function for the selected age range of children, along with the percentage of the population expected to exceed the cutoff value. The probability distribution function is a mathematical representation of how blood-lead levels would be distributed in a given population. The impact of various assumptions regarding lead concentration of environmental sources (air, water, soil, and dust), as well as selection of a GSD value, is assessed in the following section.

TABLE IX[9]
Uptake Biokinetic Model Default Values

ABSORPTION METHODOLOGY: Linear Absorption

AIR CONCENTRATION: 0.200 µg Pb/m³ Default
Indoor Air Pb Concentration: 30.0 % of outdoor.
Other Air Parameters:

Age	Time Outdoors (hr)	Ventilation Rate (m^3/day)	Lung Absorption (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: Default
DRINKING WATER Concentration: 4.00 µg Pb/L Default
SOIL & Dust:
Soil: Constant Concentration
Dust: Constant Concentration

Age	Soil (µg Pb/g)	House Dust (µg Pb/g)
0-1	200.0	200.0
1-2	200.0	200.0
2-3	200.0	200.0
3-4	200.0	200.0
4-5	200.0	200.0
5-6	200.0	200.0
6-7	200.0	200.0

PAINT Intake: 0.00 µg Pb/day Default
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Concentration: 7.50 µg Pb/dL

[9] Based upon program default values in the Uptake Biokinetic Model

TABLE X
Model Default Blood-lead and Lead Uptake:

Age in Years	Blood Level (µg/dL)	Total Uptake (µg/day)	Soil+Dust Uptake (µg/day)
0.5-1	3.30	9.38	6.00
1-2	3.01	10.03	6.00
2-3	2.98	10.56	6.00
3-4	3.04	10.48	6.00
4-5	3.12	10.41	6.00
5-6	3.15	10.72	6.00
6-7	3.18	11.11	6.00

Uptake Year	Diet Uptake (µg/day)	Water Uptake (µg/day)	Paint Uptake (µg/day)	Air (µg/day)
0.5-1	2.94	0.40	0.00	0.04
1-2	2.96	1.00	0.00	0.07
2-3	3.40	1.04	0.00	0.12
3-4	3.29	1.06	0.00	0.13
4-5	3.18	1.10	0.00	0.13
5-6	3.38	1.16	0.00	0.19
6-7	3.74	1.18	0.00	0.19

Based upon information pertinent to each medium pathway/source contribution, as discussed previously, the model was run for subareas within each of 83 cities, at the community area level (an aggregation of census tracts). The population age range for the model runs was zero to 84 months of age. The "percent above" percentages were then used to calculate numbers of children expected to exceed 10 µg/dL Pb-B.

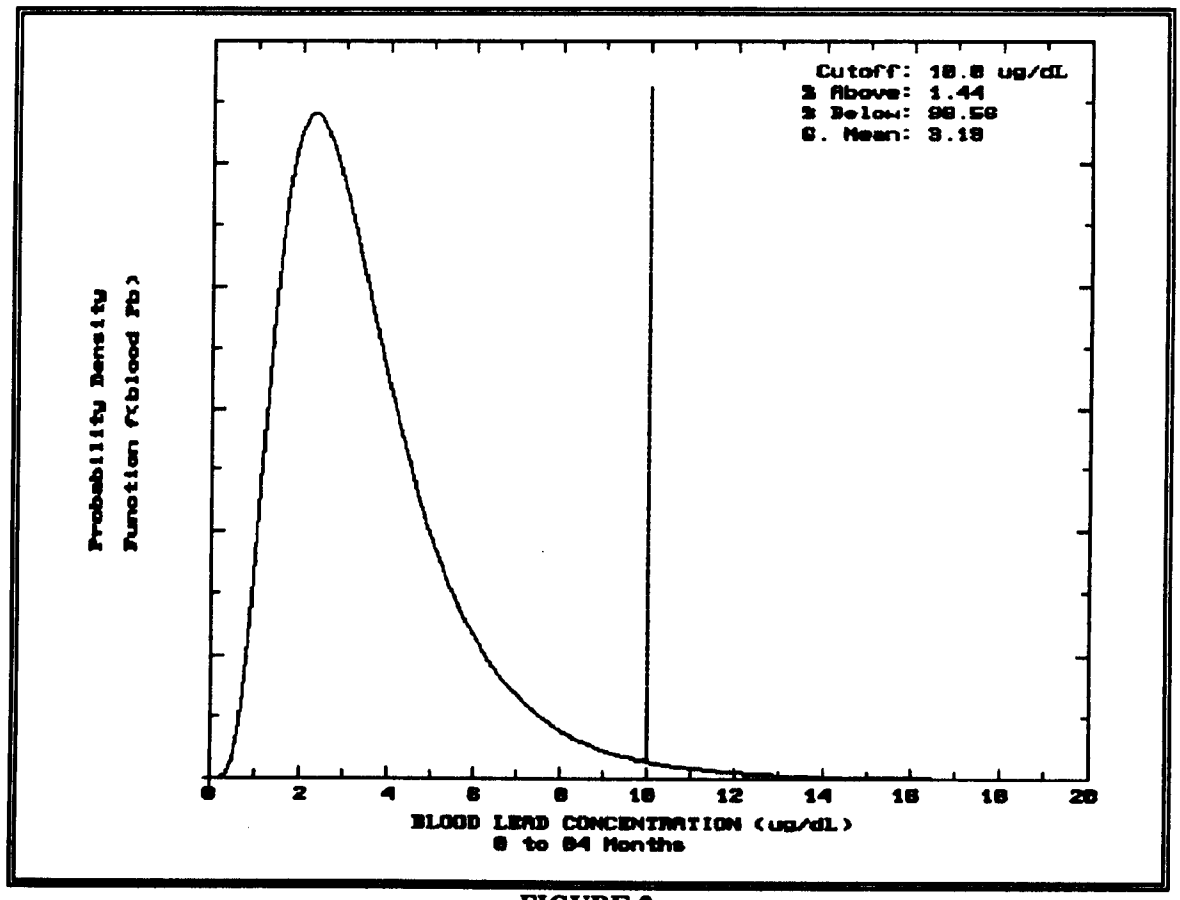

FIGURE 2
Uptake Biokinetic Model Default Concentration Curve

4.6.1. UBK Sensitivity Analysis

A sensitivity analysis was conducted in order to ascertain the impact of various assumptions made in conducting the study, and to provide a sense of how a range of environmental media concentrations affects the blood-lead level outputs from the model. The analysis considered various concentration levels for drinking water, soil and dust, and outdoor air, as well as different geometric standard deviation values. For each model run, except for the parameter of concern, all other values were held constant at the model default values.

Table X displays how varying environmental media concentrations affect mean blood-lead concentrations. The model runs use a Geometric Standard Deviation (GSD) of 1.7. Associated Figures 3, 4, and 5 show the probability density functions for each medium. Table XI summarizes information from the figures. The blood-lead levels vary only slightly when drinking water concentration is increased from 0 μg/l to 4.0 μg/l. The latter is the model default value. Consequently, drinking water concentrations in these ranges would be expected to contribute only a small amount, as an environmental pathway, to the numbers of children expected to exceed 10 μg/dL.

Soil and dust, however, can contribute significantly. Particularly for housing built prior to 1949, the concentrations of lead in soil and dust, due to historical deposition as well as lead-based-paint, can result in high numbers of potentially exposed people. To assess the impact of using a value for dust-lead concentration that may not be accurate, the model was run using the same soil concentration as dust concentration. This was in order to compare an assumption of soils concentration equals dust concentration, to the use of the calculated dust concentrations in combination with the calculated soils concentration. Using the same soil as dust value versus the calculated dust value of 565 ppm does not change the percent expected to exceed 10 μg/dL Pb-B. Therefore, for the oldest housing age category, there is no impact of using 565 ppm versus 555 ppm soil-dust, each together with a soil lead concentration of 555 ppm. For housing age category 1940 to 1959, using a 175 ppm dust value in place of the

calculated dust-lead concentration of 55 ppm, results in an elevation in the percent expected to exceed 10 μg/dL of less than 1 percent. For the newest housing age category, substituting a dust-lead concentration of 90 ppm for the calculated dust-lead value of 20 ppm, results in virtually no change in the percent estimate of exceedance. Thus the model is not sensitive to using the calculated dust-lead values.

Running the model for dust alone (with model default values for air, drinking water, and diet) indicates that, except for the highest dust-lead concentration, there is minimal contribution to the percent expected to exceed the criterion Pb-B value. For housing age category prior to 1940, the modeled percent exceedance of 4.55 percent indicates an increase of slightly more than 3 percentage points associated with increasing dust concentration from 200 ppm to 565 ppm. Compared to the model derived percent exceedances when soil and dust values are both held at 0, essentially the full 4.55 percent is associated with the dust concentration of 565 ppm.

Outdoor air, at low levels (generally expected, except where a significant point source is in the vicinity of a population), is not expected to contribute greatly to an increase in Pb-B levels. When the air quality standard is greatly exceeded, however, as may be caused by a point source or lead-contaminated dust, the Pb-B levels of nearby residents (within one to two km) are expected to rise significantly. As indicated in the table, an air concentration ten times the standard would result in a percent exceedance of almost 13 percent.

The model is very sensitive to the choice of blood-lead GSD for the population. The GSD model default value of 1.42, thought to be applicable to point sources of lead, as mentioned, results in a 0.05 percent exceedance of 10 μg/dL. A GSD of 1.7, selected as discussed earlier, results in a 1.44 percent exceedance, while a GSD of 1.8 results in an even greater exceedance of 2.47 percent (Figure 6). At higher input levels of environmental concentrations, the spread would be even more dramatic. When applied to a population, for example of 1,000 children, the number of children expected to exceed 10 μg/dL for the model default concentrations would vary from 0 to 14 to 25 children at GSDs of 1.42, 1.70,

and 1.80, respectively. Consequently, the choice of a GSD value for the model has a significant effect upon the estimated number of children at risk of elevated blood-lead.

TABLE XI
Uptake Biokinetic Model
Sensitivity Analysis[10]

Parameter Concentration Value	Geometric Mean (µg/l)	% exceeding 10.0 µg/dL Children aged 0-84 months	UBK Run No.	Remarks
Drinking Water (µg/l)				
0	2.92	0.98	1	
2.5	3.09	1.29	2	For level of detection 5.0 µg/l
3.5	3.16	1.44	3	For level of detection 7.0 µg/l
4.0	3.19	1.44	4	Default value.
15.0	3.94	3.81	5	New drinking water standard.
50.0	6.33	18.69	6	Old drinking water standard.
Soil & (Dust) (µg/g)				
0 (0)	1.47	0.01	1	
200 (200)	3.19	1.44	2	Default value.
500 (500)	5.78	14.56	3	Superfund lower range value.
1,000 (1,000)	10.10	49.00	4	Superfund upper range value.
555 (565)	6.3	17.56	5	Housing age prior to 1940
175 (55)	2.41	0.34	6	Housing age 1940-1959
90 (20)	1.91	0.00	7	Housing age 1960-1979
555 (555)	6.25	17.56		Assumes soil concentration = dust concentration
175 (175)	3.02	1.15		
90 (90)	2.29	0.25		
0 (565)	4.17	4.55		Dust concentration only

[10] Note that the level of detection for lead in drinking water varies by state, either 5.0 µg/l or 7.0 µg/l. The significance of the soil and dust values is discussed in Section 4.6.6.

Parameter Concentration Value	Geometric Mean (µg/l)	% exceeding 10.0 µg/dL Children aged 0-84 months	UBK Run No.	Remarks
0 (55)	1.79	0.55		
0 (20)	1.62	0.02		
Outdoor Air ($\mu g/m^3$)				
0	3.16	0	1	
0.20	3.19	1.44	2	Default value.
1.50	3.41	2.13	3	Quarterly average standard.
15.0	5.63	12.86	4	Ten times standard

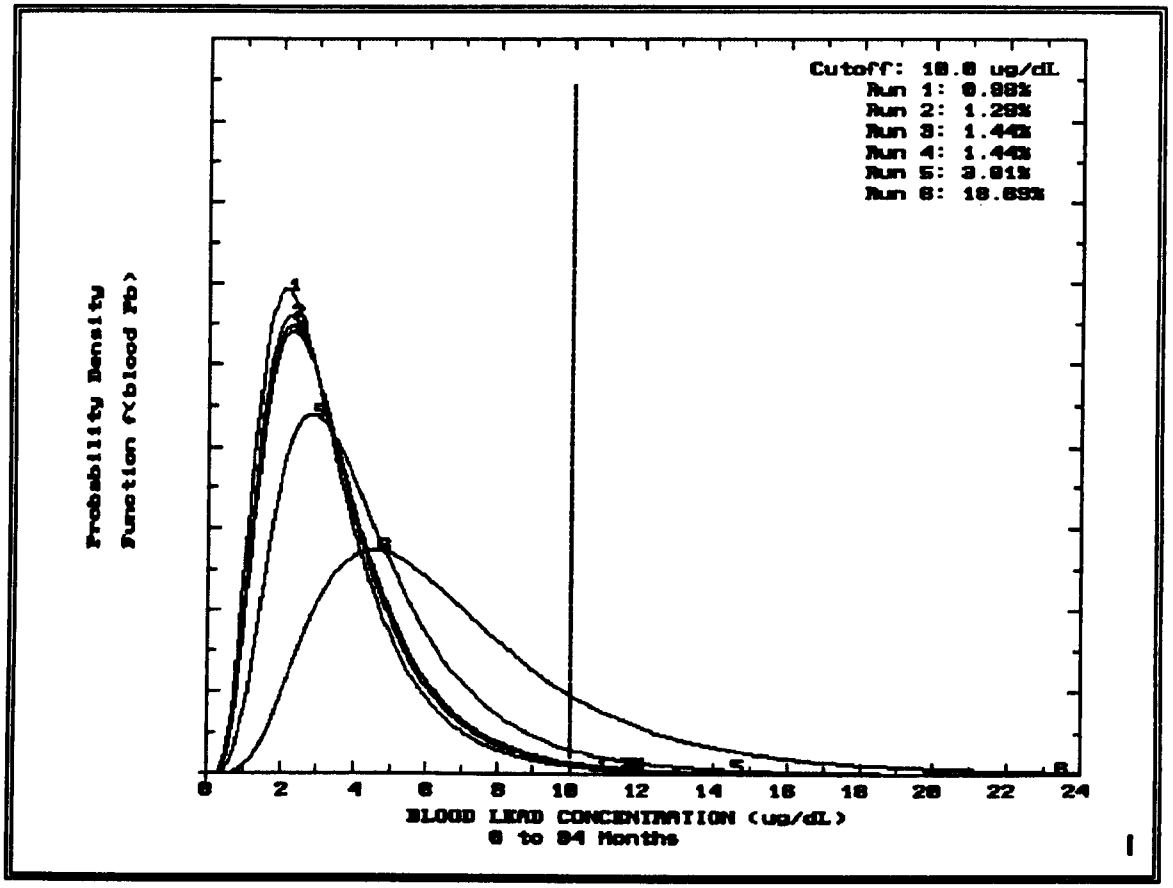

FIGURE 3
Select Drinking Water Concentrations

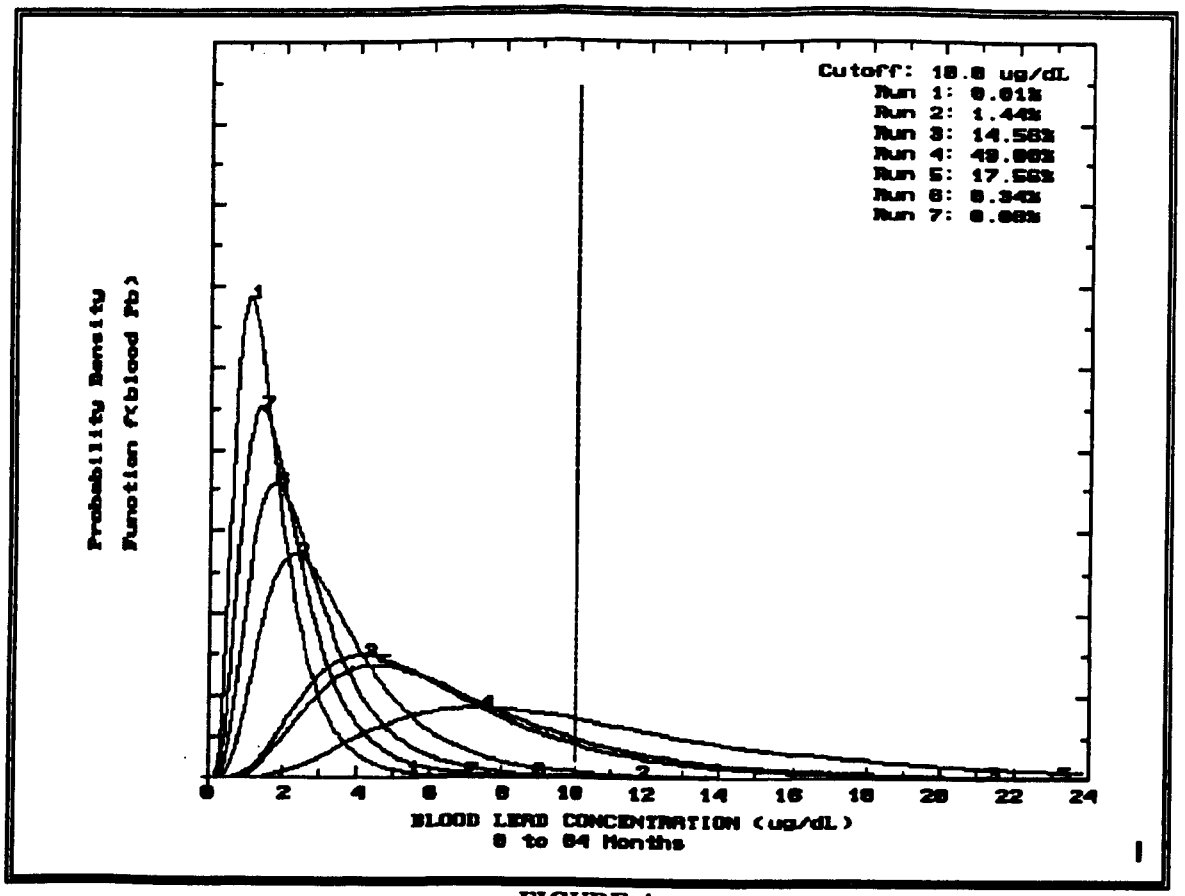

FIGURE 4
Select Soil and Dust Concentrations

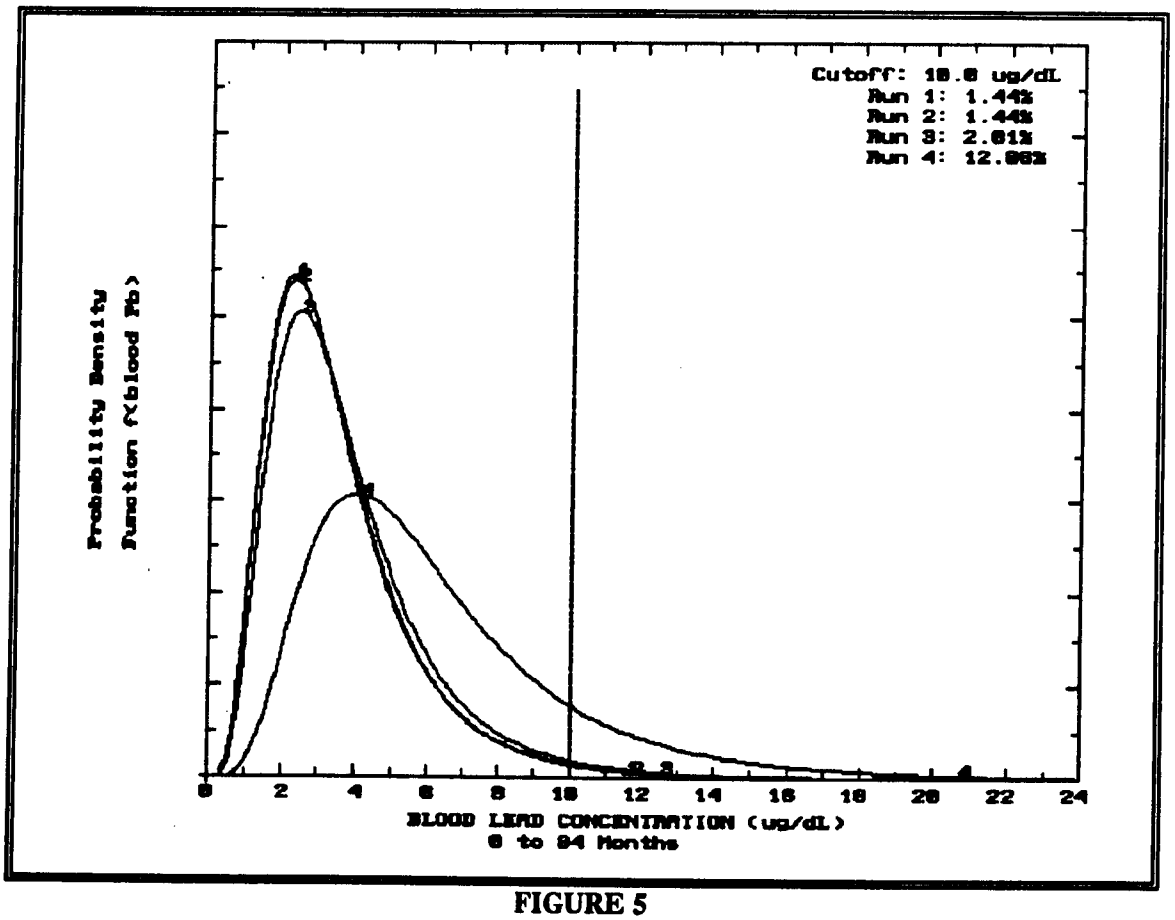

FIGURE 5
Select Ambient Air Concentrations

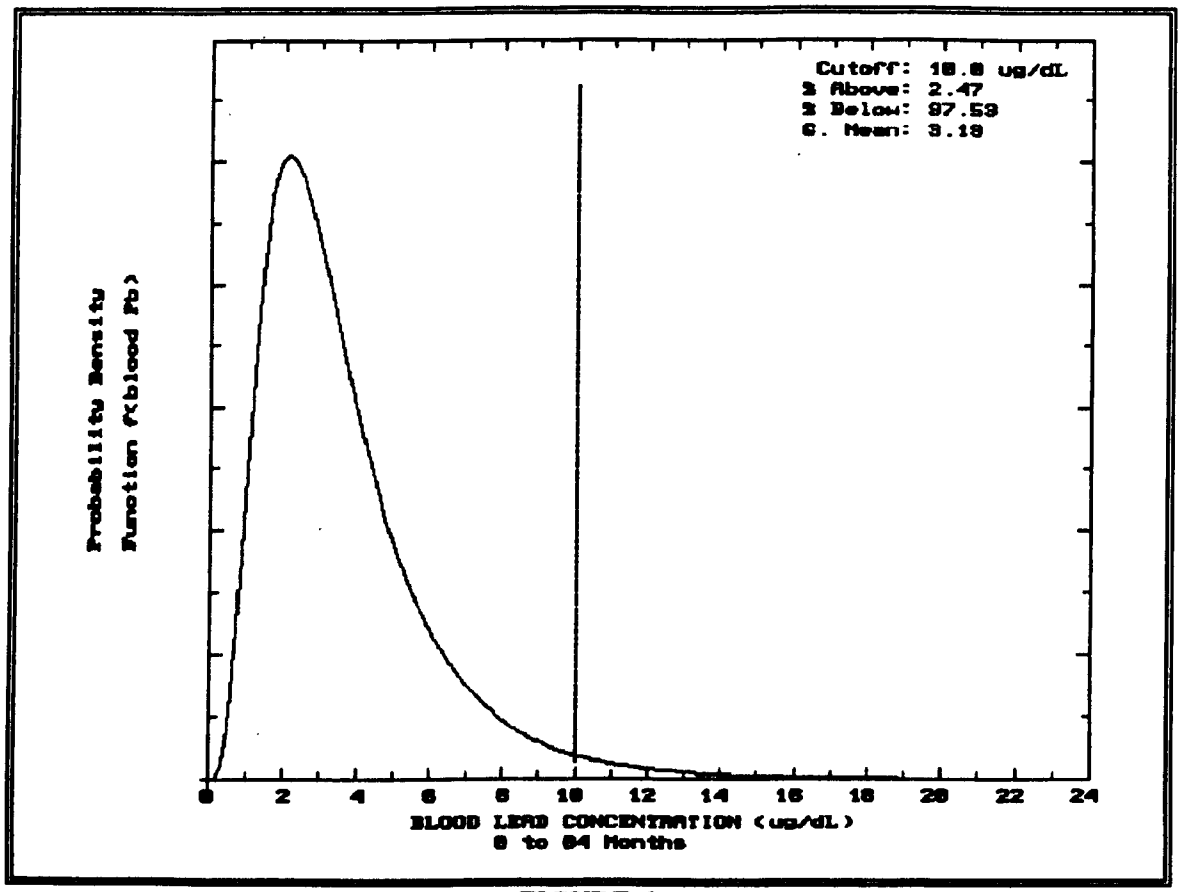

FIGURE 6

UBK Default Concentrations with Geometric Standard Deviation of 1.8

4.7. Selected Area for Verification of Lead Screening Approach: Minneapolis/St. Paul

4.7.1. Minneapolis/St. Paul Demographic, Biological, and Soils Data

Minneapolis/St. Paul MSA was selected for verification of the population screening approach (Objective 2) and for analyzing the association of blood-lead level to mobile sources (Objective 3) because its data were available in computerized format. The Minnesota Department of Health (MDH) conducted blood-lead testing in the area during the years 1986 and 1987. Reports of the results of the Minnesota Department of Health 1986-87 Blood-lead Survey were reported by memoranda from the Lead Program Coordinator, Division of Environmental Health, MDH, (Douglas Benson, Office Memorandum, October 11, 1991). A total of 1,410 children were surveyed, mostly in the Twin Cities, to ascertain blood-lead values and to find lead-poisoned children. The data collected in support of the survey were provided on computer disk. The data base contained 1,034 records for Minnesota and St. Paul. Data included blood-lead level, census tract of home, ethnicity, gender, birth date of child, years of education for the father and mother, and year blood-lead sample was taken. It is important to note that the blood-lead survey was conducted in geographic areas where soil-lead values had been previously determined to exceed 1,000 ppm.

Environmental and demographic data were added to each record. Blood-lead-modeled values were calculated from the UBK model for the age and census tract location for each child/record. Environmental concentrations ascertained for the census tract of residency for each child were included to account for relevant routes of exposure. Most particularly, soils data for each census tract were obtained from the Minnesota Department of Pollution Control. The data base was that used by the MDH to prepare the Soil Lead Report to the Minnesota State Legislature (MPCA, 1987). Geometric mean soil-lead concentrations were calculated from the raw data, for each census tract. The geometric mean was selected in order to compare values with modeled blood-lead values. The UBK model assumes a lognormal distribution. The

model calculates a geometric mean.

A Geographic Information Systems applications was use to determine the distance from a transportation corridor for each census tract. That distance was then included in the data set for each child. The distance between the center of each census tract and the closest heavy duty transportation corridor[11] was determined for each census tract.

4.7.2 Minneapolis/St. Paul Statistical Analyses

Two statistical procedures were conducted. To gauge the predictive ability of the comparative risk approach, the UBK model was run for each child's data set, using the child's age and the environmental data (concentrations) pertinent to the census tract of residency. The geometric mean estimated blood-lead levels were then compared to the measured blood-lead levels, using a simple correlation procedure. The correlation analysis was then repeated, grouping the children by census tract. The mean Pb-B values for each group was then compared to the UBK modeled Pb-B value for the census tract.

A multivariable regression analysis procedure was employed to discern the contributions of various pathways of exposure. The procedure was limited by the lack of variation of some of the data. Neither drinking water nor air concentrations varied (sufficiently) across geographic areas and, consequently, the variables were not included in the regression analysis. The predominant housing age for each census tract was assigned to each record. Housing age was included in the model to account for historic deposition from mobile sources, both exterior and interior lead-based-paint, and deposition from point sources. Insufficient information is available, in general as well as for this analysis, to distinguish between and partition the contributions from these sources. For the remaining data/variables (full model on the following page), a regression analysis using stepwise comparison/replacement of independent variables was conducted. The decision point p-value for selecting a variable in the model was $p = 0.05$.

[11] Interstate Routes 35, 94, 494, and 694.

The full model is of the form:

$$\text{Log Pb-B}_{act} = \beta_0 + \beta_1 \text{Log Pb-B}_{mod} + \beta_2 \text{HAC} + \beta_3 \text{Dist} + \beta_4 \text{AGE} + \beta_5 E_1 + \beta_6 E_2 + \beta_7 E_3 + \beta_8 \text{GEN} + \beta_9 \text{FAT} + \beta_{10} \text{MOT} + \beta_{11} \text{Soil} + \beta_{12} \text{INC}$$

Where

Pb-B_{act}	= measured lead blood from survey (μg/dL)
Pb-B_{mod}	= model estimate of lead blood level (μg/dL)
HAC	= 1 if house built before 1949 2 if house built 1950-59 3 if house built 1960-69 4 if house built 1970-79
DIST	= distance from centroid of census tract to nearest heavy duty highway (meters)
AGE	= Age of child (years)
E1	= 1 if ethnicity is white, 0 otherwise
E2	= 1 if ethnicity is African-American, 0 otherwise
E3	= 1 if ethnicity is American Indian, 0 otherwise
GEN	= 1 if gender is female, 0 otherwise
FAT	= number of years of father's education
MOT	= number of years of mother's education
SOIL	= soil lead concentration measured for census tract (ppm)
INC	= family income for census tract (dollars)

4.8. Derivation of City Exceedance Estimates

To derive an estimate of exceedance of the 10 μg/dL blood-lead value for each group of census tracts, the UBK model was run with data pertinent to the area. Ambient air concentrations for the city (refer to Section 4.4.3.) and drinking water concentrations for the city (refer to Section 4.5) were used as input. No data from the air quality modeling efforts were used in the city computations. The results of the air quality modeling for the 17 sources were used in the qualitative analysis only.

Soil and dust concentrations were calculated as a weighted average of the actual number of houses in each housing age category, for each census tract group, based upon the soil and dust concentration values derived from HUD data (refer to Section 4.6.3.). To illustrate, consider a census tract with 200 homes built before 1940, 300 homes built between 1940 and 1959, and 100 homes built from 1960 to 1979. Calculation of soil and dust values for the census tract, for input into the model, would be as follows:

Housing Age	Number of Homes	Calculated Soil Value (ppm)	Calculated Dust Value (ppm)
1960-1979	100	(100/600)090= 015.0	(100/600)020= 003.3
1940-1959	300	(300/600)175= 087.5	(300/600)055= 027.5
pre- 1940	200	(200/600)555= 185.0	(200/600)565= 188.3
Total/Ave.	600	287.5	219.1

Thus, the soil and dust concentrations for the census tract, for input into the UBK model, would be 287.5 ppm and 219.1 ppm, respectively.

The percentage of population expected to exceed 10 μg/dL, derived from the UBK model, was then multiplied by the total, African-American, and Hispanic childhood counts to derive the number of children expected to exceed the criterion Pb-B value for the census group area. The numbers for all census tract groups were then totaled to derive an exceedance number for each city.

The number of new borne by ethnic category was calculated by applying the city specific birth rate to the total, African-American, and Hispanic populations. The UBK derived percentages were multiplied by those numbers to derive an estimate of fetuses that would exceed 10 μg/dL Pb-B. Census tract groups were similarly aggregated to derive city totals.

It is important to note that this methodology is for population screening purposes. The results may have no practical value as a prediction of the actual number of children expected to have elevated blood-lead values. Nor was that the intent of the methodology. The value of the approach is in the comparison between cities, and specifically to areas within a city that may be expected to have higher rates of lead exposed children than other areas. The intent of the population screening methodology is to use that indication to set priorities for intervention efforts within a city or region. The reader is particularly cautioned that the numbers are as derived by the computerized methodology and therefore appear to be precise. They are not.

5. RESULTS

5.1. Overview/Introduction to Results

Results are presented for each environmental category, along with the modeling results for 17 air emission sources, soil and dust derivations, and a qualitative summary of the results for each city. An environmental profile and the results of statistical analyses pertinent to Minneapolis and St. Paul, Minnesota, are provided. Finally, the results of Pb-B modeling for each city, are presented.

5.2. Environmental Data Categorical Assessments

5.2.1. Ambient Air

During 1988, there were few exceedances of the air quality standard for lead (three month average of 1.5 $\mu g.m^3$). Most monitors reported in the tenths or hundreds of a $\mu g/m^3$. The notable exception is Eagan, Minnesota, with a single fourth-quarter exceedance of 1.8 $\mu g/m^3$.

The average lead concentrations utilized as the air concentration values in the UBK model are listed in Appendix C. Cities for which a program default values were used are also listed.

5.2.2. Air Emissions

Tables showing total emissions in the six states and total emissions in the 83 cities are provided as Appendices D and E, respectively, based upon the Toxic Release Inventory data base. Figure 7 shows total air emissions of lead by state.

The inventory contains 497 facility emission reports. Total air releases from 342 sources reporting release to air equals 449,304 pounds for calendar year 1988. Twenty-one sources, or 6.1 percent of the total number of sources, release 261,051 pounds annually, or 58.1 percent of the total air emissions. In the MSA areas, 226 sources account for 314,904 pounds annually. Seventeen sources with annual releases to the air greater than 4,000 pounds release 216,459 pounds annually. This accounts for 48.2 percent of total air emissions in the six states. These 17 sources are listed in Table XII. They constitute a mere 5 percent of the number of sources in the six states. Appendix F provides locational information.

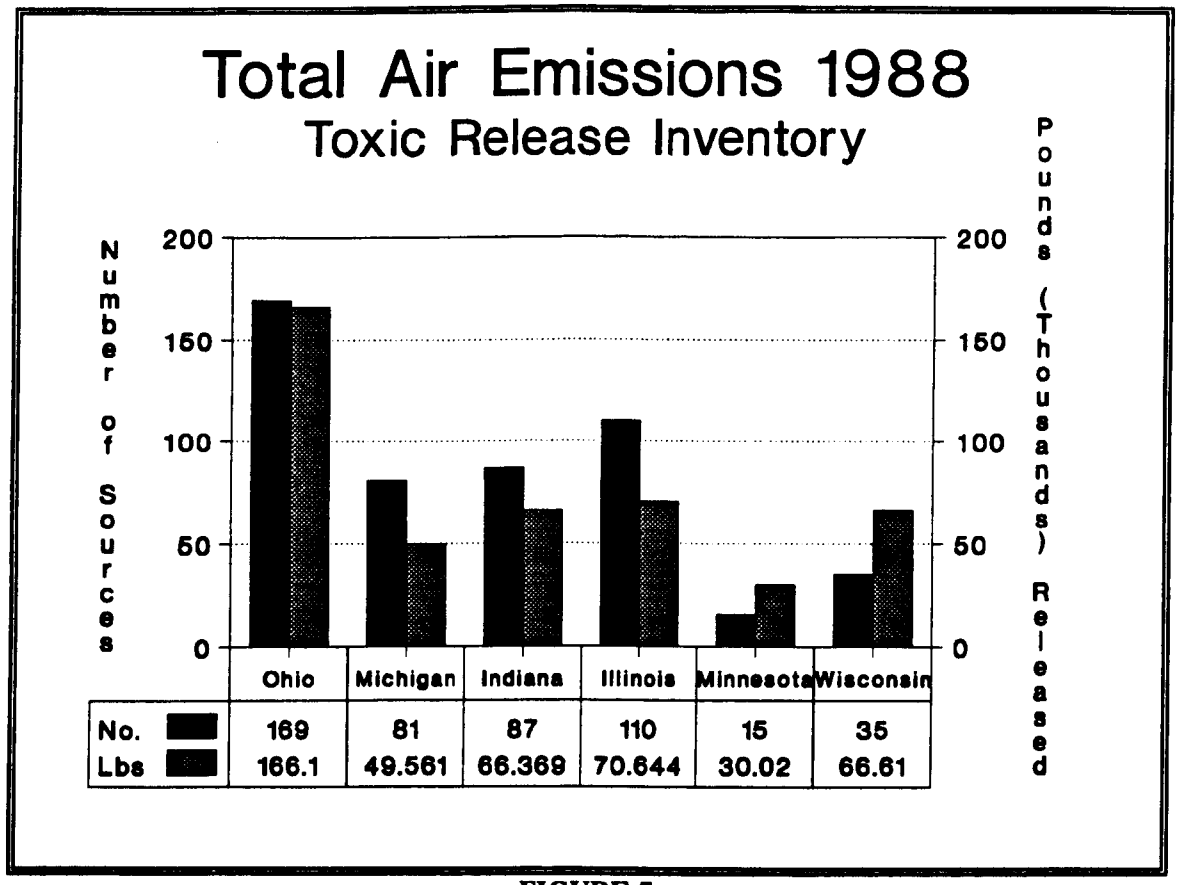

FIGURE 7
Total Air-lead Emissions 1988

TABLE XII
Sources with TRI Reported Total Air Emissions
Exceeding 4,000 Pounds/Year in 1988

Facility Name	Location	Air Emissions (Pounds)
OI-Neg TV Products, Inc.	Columbus, OH	61,300
DuPont Toledo Plant	Toledo, OH	6,711
Oatey Co.	Cleveland, OH	4,200
Copperweld Steel Co.	Warren, OH	5,843
Republic Engineered Steels, Canton Works	Canton, OH	4,600
Empire Detroit Division	Mansfield, OH	7,231
Acustar Dayton Thermal Products Division	Dayton, OH	4,250
Refined Metals Corp.	Beech Grove, IN	9,870
Quemetco, Inc.	Indianapolis, IN	5,485
National Steel Great Lakes Division	Ecorse, MI	11,590
Federal-Mogul	Saint Johns, MI	5,740
Gopher Smelting & Refining Co.	Eagan, MN	13,812
Chemetco, Inc.	Hartford, IL	11,570
Inland Steel Co.	East Chicago, IN	17,900
Kohler Co.	Kohler, WI	29,200
North Star Steel Minnesota	Saint Paul, MN	12,480
LaClede Steel Co.	Alton, IL	4,677
Total for sources		216,459

Figure 8 shows the spatial distribution of the sources of lead and associated annual release amounts.

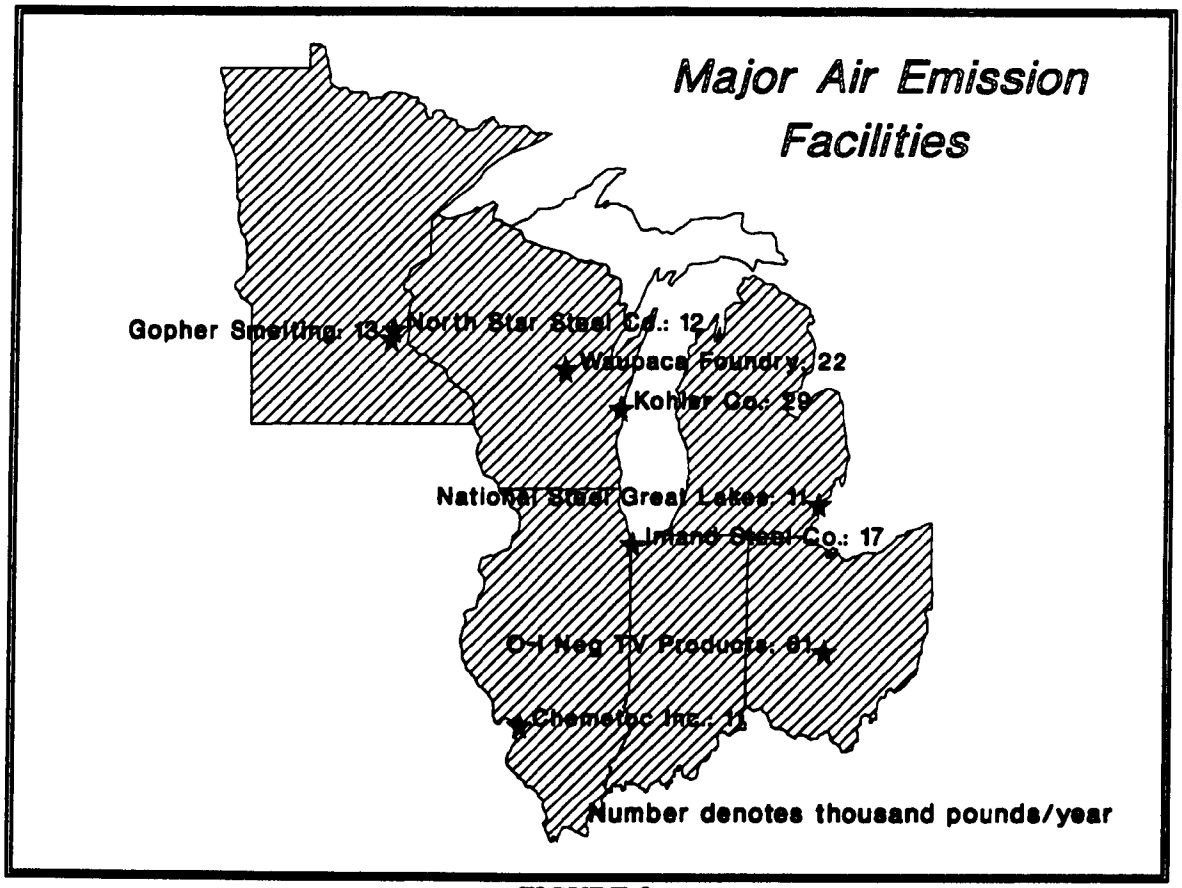

FIGURE 8
Major Air-lead Emission Facilities

5.2.2.1. ISCLT Modeling Results

Model run results for the seventeen major air emission facilities, included in Appendix G, were provided to the GISMO for spatial representation and to relate to census tracts. The resultant concentrations of air-lead were cross checked with ambient air data, where the latter was available.

Table XIII summarizes the concentrations of lead determined for each of the 17 sources at a comparable grid point (200, 200) meters. This is generally the point of maximum concentration. Beyond 200 meters from the source, concentrations begin to decrease rapidly, generally to tens and hundredths of a $\mu g/m^3$. At the extremities of the model grid, 2,000 meters from the source, concentrations were in the thousandths of a $\mu g/m^3$, for all but the largest sources. All sources were less than hundredths of a $\mu g/m^3$ at the extreme points.

The maximum concentrations for the 17 sources varied from a low of 0.118 $\mu g/m^3$ downwind from Acustar Dayton Thermal Products Division in Dayton, Ohio, to the two highest maximum concentrations values calculated at 1.792 $\mu g/m^3$ for Kohler Co., Kohler, Wisconsin, and 1.693 $\mu g/m^3$ for OI-Neg TV Products, Inc., Columbus, Ohio. Except for these two sources, all sources had annual concentrations of less than unity.

TABLE XIII
Maximum Concentrations of Lead
for Modeled Sources

Facility Name	Location	Estimated Concentration ($\mu g/m^3$)
OI-Neg TV Products, Inc.	Columbus, OH	1.693
DuPont Toledo Plant	Toledo, OH	0.138
Oatey Co.	Cleveland, OH	0.132
Copperweld Steel Co.	Warren, OH	0.151
Republic Engineered Steels, Canton Works	Canton, OH	0.135
Empire Detroit Division	Mansfield, OH	0.229
Acustar Dayton Thermal Products Division	Dayton, OH	0.118
Refined Metals Corp.	Beech Grove, IN	0.584
Quemetco, Inc.	Indianapolis, IN	0.244
National Steel Great Lakes Division	Ecorse, MI	0.306
Federal-Mogul	Saint Johns, MI	0.136
Gopher Smelting & Refining Co.	Eagan, MN	0.179
Chemetco, Inc.	Hartford, IL	0.912
Inland Steel Co.	East Chicago, IN	0.527
Kohler Co.	Kohler, WI	1.792
North Star Steel Minnesota	Saint Paul, MN	0.302
LaClede Steel Co.	Alton, IL	0.146

5.2.3. Municipal Waste Combusters

There are 32 municipal waste combusters in the Agency's inventory, although three facilities are not currently operating. The three are all outside the study area. A significant finding is that estimated emissions exceed actual emissions gathered by stack test emissions, generally by an order of magnitude or more. Appendix H provides information on each of the facilities, including design capacity, estimated emissions, stack test emissions, and comments pertinent to each facility.

The 32 facilities, when all were operating, had annual emissions of 62,288 pounds of lead, based upon estimated emission factors and stack test emissions. Stack test emissions, available for 17 of the 32 sources, were utilized when available. Of the 32 sources, 15 are located in the project MSA cities. The facilities are listed in Table IV. Figure 9 shows the location of the facilities.

An analysis of the 17 sources with emissions data indicates the problem of using estimated emission factors. For those sources, estimated emissions total 349.13 pounds/day, while emissions based upon stack test information total only 46.78 pounds/day. Notably large differences for estimated and stack test emissions, respectively, include the Indianapolis facility, 48.60 and 0.06 pounds/day; Detroit, 92.40 and 1.82 pounds/day; NSP-Red Wing (Minnesota), 27.00 and 0.34 pounds/day; and Columbus, Ohio, 56.00 and 7.60 pounds/day. Analysis of the seven facilities with stack data, located in the MSA cities, shows a similar spread of 251.50 pounds/day estimated emission estimate, and 30.89 pounds/day stack test emissions, for an aggregate annual (stack test) emission of 11,275 pounds for the seven sources. Several sources, including the Chicago facility with estimated emissions of 35.20 pounds/day, appear to be significant sources and to warrant modeling. No municipal waste combuster has been modeled, however, due to the uncertainty of the estimated values, and as well as the significant differences between estimated emissions and stack test emission results. Consequently, the lead emissions from this categorical source was not incorporated into the study modeling and subsequent estimates of children exposed to lead. Large

sources, nonetheless, may prove to be a concern, when actual stack test data is derived. The Chicago, South Montgomery County (Ohio), and North Montgomery County (Ohio) facilities are planning or have recently obtained stack test data. Stack test data for those facilities may indicate the need for additional consideration.

TABLE XIV
Municipal Waste Combuster Inventory
Metropolitan Statistical Area Cities in Region 5

Facility Name	Location	Waste Design Capacity (Tons/Day)	Estimated Air-lead Emissions (Pounds/Day)	Lead Stack Test Emissions (Pounds/Day)
Chicago NW	Chicago, IL	1600	35.20	N/A
East Chicago	East Chicago, IN	450	9.90	N/A
Indianapolis	Indianapolis, IN	2200	48.40	0.06
Jackson	Jackson, MI	200	4.40	N/A
Detroit	Detroit, MI	3300	92.40	1.82
Kent Co.	Grand Rapids, MI	625	13.75	N/A
WLSSD-Duluth	Duluth, MN	110	3.10	0.04
Olmsted Co.	Rochester, MN	200	4.40	0.37
Hennepin Energy Res.	Minneapolis, MN	1,000	22.00	0.06
N. Montgomery Co.	Dayton, OH	900	19.80	N/A
S. Montgomery Co.	Dayton, OH	900	19.80	N/A
Akron	Akron, OH	900	25.20	20.94
Columbus	Columbus, OH	2000	56.00	7.60
Sheboygan	Sheboygan, WI	96	2.10	N/A
Madison-G&E	Madison, WI	75	2.10	N/A
Total		14556	358.55	

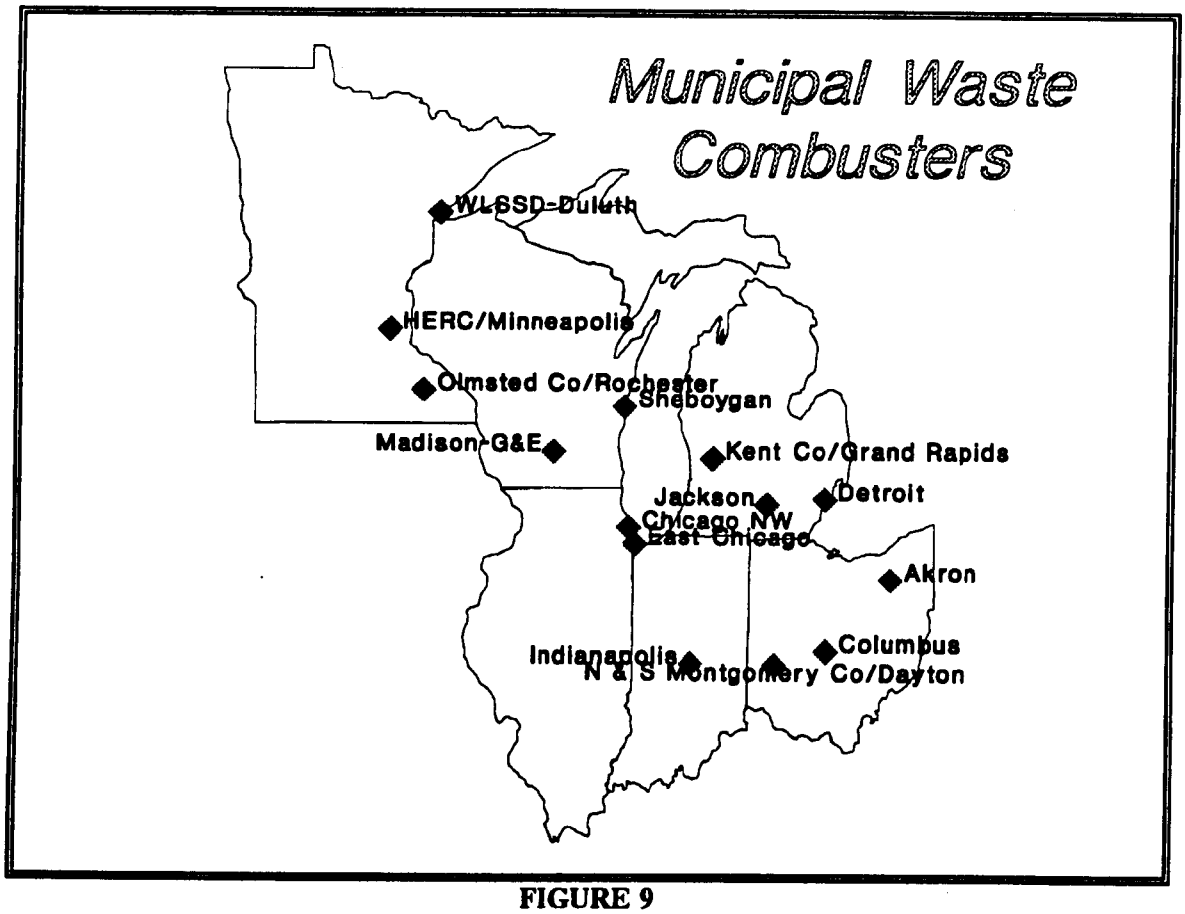

FIGURE 9
Municipal Waste Combusters in U.S. EPA Region 5

5.2.4. Drinking Water

The Federal Data Reporting System(FDRS) data base for 1988 indicated that there were no violations of the (50 µg/l) Maximum Contamination Level (MCL) for Pb for any of the study area cities. Only exceedances of the MCL, however, are reported to the system. Consequently, the FRDS does not contain information on actual measured concentrations less than the MCL. State agency records indicate actual values from sampling results. These are summarized in Appendix I, with test results, number of

samples in each test, and the drinking water concentration value for modeling, for each city. Of the 83 cities, only 10 had test results above the level of detection, while 27 cities showed non-detect levels. The drinking water suppliers for 46 cities did not report sample results in 1988. For the latter, 4.0 μg/l was assumed. The largest values reported were for Wausau, Wisconsin at 1500 μg/l (reported value is suspect and was not used in the study) and 7 μg/l ; Milwaukee, Wisconsin at 25.0 μg/l; Youngstown, Ohio at 12.0 μg/l; and Madison, Wisconsin at 10.2 μg/l. Thus of all the cities sampled, only two would exceed a standard of 15.0 μg/l.

5.2.5. RCRA and Operating Landfills

RCRA facilities as a category do not appear to present a significant risk of lead exposure. That assessment is qualified, however, due to the difficulty in obtaining information about a particular parameter, lead, at a given facility. Generally, the facilities may treat or otherwise process a limited number to a wide variety of pollutants, depending upon the facility's operating permit and type of operation. A total of 27 RCRA facilities were assessed to determine whether lead was processed at the facility, and a determination was made on potential exposure.

Appendix J lists the 27 RCRA facilities with comments on each and a T/F (true or false) notation as to potential for exposure. No information was obtained for seven facilities. For the 20 facilities assessed, only four appeared to have a potential for off-site exposure that could result in human exposure, generally via the air pathway from lead-contaminated piles and wind blown dust. These are Saint Louis Lead Recyclers, McLean Steel, Kemeto, and Olin, all located in Granite City, Illinois. Response action is ongoing in Granite City, Illinois, at the NL/Taracorp Site, that will result in capping a 240,000 ton lead-bearing waste pile situated adjacent to the former lead smelter, along with residential soil cleanup in a 55 square block area. Soils with lead concentration exceeding 500 ppm will be excavated and replaced with clean soil. EPA (Superfund program) is currently preparing the remedial design for the project.

Granite City is also one of three study areas that is part of a tri-state lead study being conducted

jointly by EPA and the Agency for Toxic Substances Disease Registry. In addition to determination of soil and dust contamination, the Illinois Department of Public Health has conducted extensive blood-lead testing for the project. This ongoing area-wide study in the city, should elucidate the potential for human exposure to lead from these facilities.

Operating facilities that dispose of lead on-site were obtained from the TRI data base. Sixteen facilities in the MSA cities reported on-site disposal, ranging from a diminutive seven pounds annually to 566,000 pounds annually, for a total of 2,138,048 pounds/year disposed of on-site in 1988. Figure 10 provides a spatial representation of the largest facilities, with annual amounts indicated for the facilities shown.

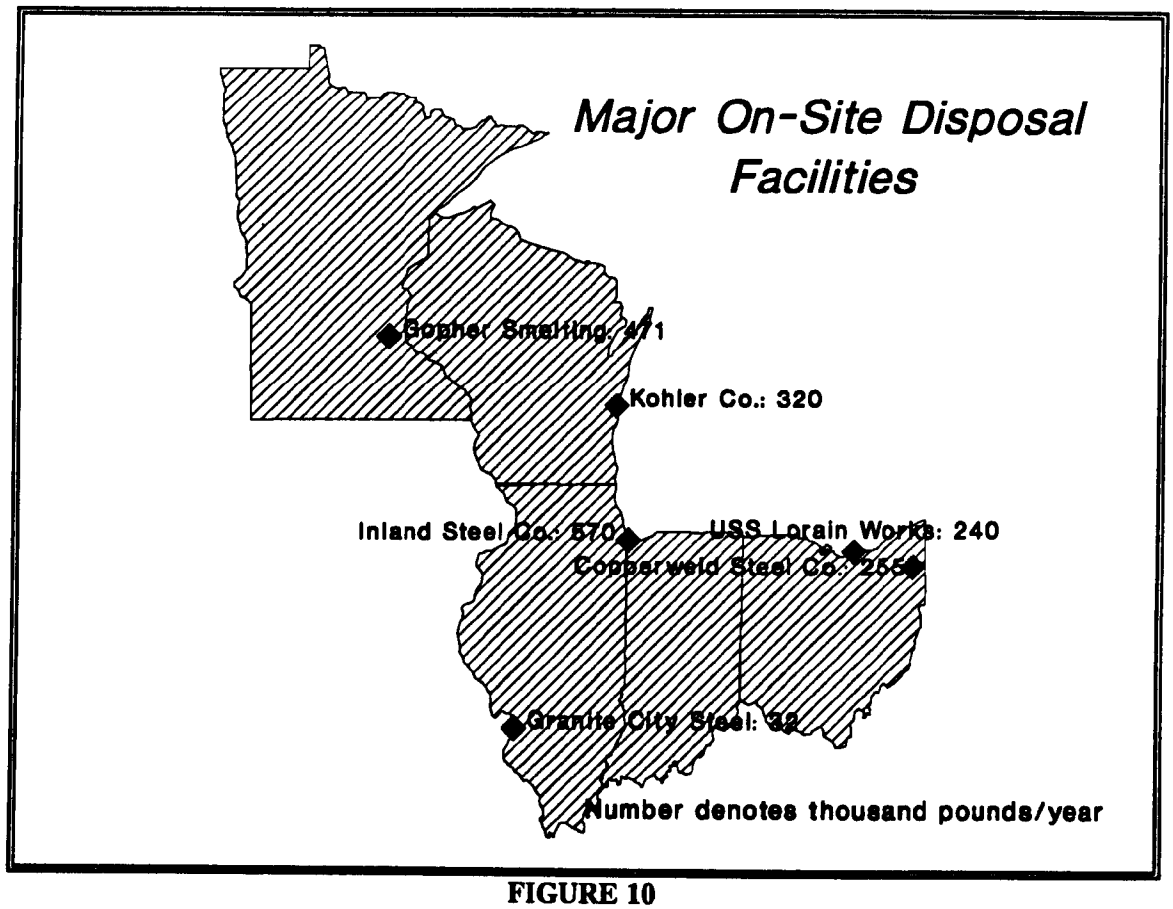

FIGURE 10
Major On-Site Lead Disposal Facilities

Appendix K contains information on facilities having on-site lead disposal, along with a categorical judgement on the potential for off-site contamination. Of the 16 facilities, five appear to have the potential for off-site lead-contamination, as described in Table XV. As is the case for RCRA facilities, minimal data is available to characterize the concentration and spatial extent of lead-contamination that may result from landfilling/on-site disposal at the facilities.

TABLE XV
Toxic Release Inventory Reported On-Site
Disposal in 1988 for MSA Cities

Facility Name	Location	On-site Disposal (Pounds)	Remarks
Keystone Steel & Wire Co.	Peoria, IL	41,000	Ground water around the facility is contaminated with lead. Facility is seeking closure. Arc furnace dust pile addressed previously. Near residential area and Peoria State Hospital. Potential dust source. High level exposure to population.
Granite City Steel	Granite City, IL	45,000	Facility sends 15-20 different types of waste streams to landfill, including blast furnace flue dust, settling pond sludge, etc. Some potential for off-site contamination of residents proximate to main street side of facility.
Inland Steel	East Chicago, IN	560,000	No information on-site disposal operations.
USX Gary	Gary, IN	7,400	Good potential for electric arc furnace dust to get off site. Residential area.
Cooperweld Steel Co.	Warren, OH	2,001	Main waste is arc furnace dust. State is handling the closure of waste piles.

5.2.6. Abandoned Hazardous Waste Sites (Superfund)

The National Priority List (NPL) listing contained 95 sites in the six states that listed lead as a major contaminant. Of these, 17 facilities are located in the MSA cities. These are indicated on the map, Figure 11. Appendix L lists all sites in the six state area, with a designation of final or proposed pertaining to designation status as an NPL site. Appendix M provides definitive information on the sites located in MSA cities.

The data base consists of sites that were both proposed for listing and final, at that time, so that the extent of information about the sites vary greatly. In particular, the proposed sites tended to have much less information on the extent of lead contamination. Of course, lead is just one of the pollutants that could be on any given site, consequently, there is no requirement or particular reason for a file to contain more extensive data on lead. The more extensive site investigation step, development of a remedial investigation/feasibility study (to better characterize the extent of contamination and to develop alternatives for abatement) had not been initiated at many of the sites. Extensive sampling results, therefore, were not available. It is important to note, further, that the investigations are not undertaken solely to determine lead concentrations. Lead is only one of a host of contaminants of concern, and is most often not the prime pollutant being investigated.

Table XVI lists the 17 sites located in the MSA cities. Most of the sites are abandoned landfills, many municipally owned and operated, and have been assessed primarily for potential groundwater contamination both on and off site. Soil contamination investigations, at this stage of investigation of site conditions, has almost exclusively focused upon on-site concentrations. Soil-lead contamination has been documented on-site for most of the facilities, although the primary route of exposure appears to be through contaminated groundwater. Information is, at best, sparse, particularly for those sites that have been

proposed for the National Priority List, as contrasted to final NPL sites. For the former, only preliminary information, and few actual physical measurements of concentrations of groundwater, soil, or on site materials (e.g., in barrels or sludge lagoons) had been taken. There are two sites that are notable exceptions. The Barrels, Inc., site, in Lansing, Michigan, appears to have potential for off-site contamination. The NL Industries/Taracorp Lead Smelter site in Granite City, Illinois, has well documented and significant contamination in the residential area surrounding the site and is, consequently, currently being addressed by the EPA Superfund program.

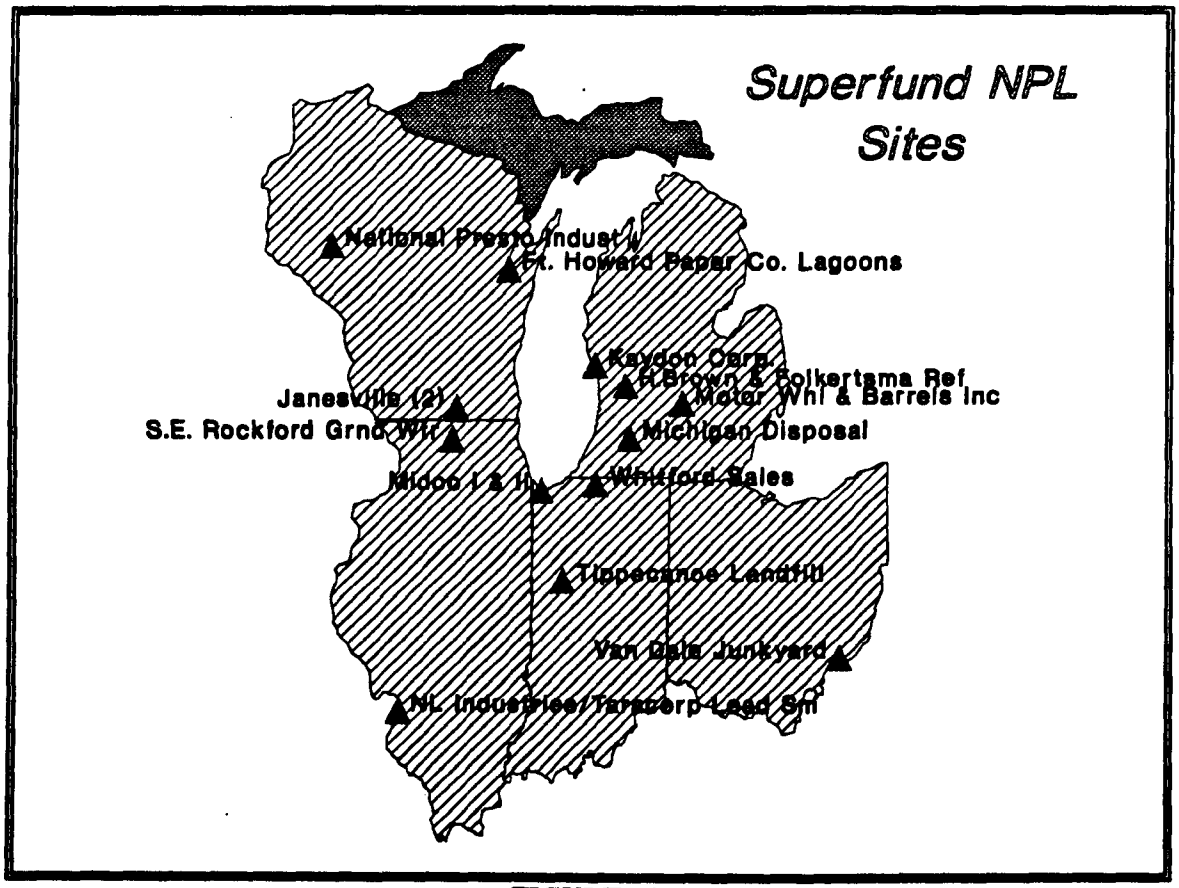

FIGURE 11
Superfund National Priority List Sites with Lead Contamination

TABLE XVI
National Priority List Facilities in Metropolitan Statistical Area Cities
with Lead as of November 1989

Facility Name	Location	Potential (Y/N)[12]
NL Industries/Taracorp Lead Smelter	Granite City, Il	Y
Southeast Rockford Groundwater Contamination	Rockford, Il	N
MIDCO I	Gary, In	N
MIDCO II	Gary, In	N
Tippecanoe Sanitary Landfill, Inc.	Lafayette, In	N
Whitford Sales & Service	South Bend, In	N
Michigan Disposal (Cork Street Landfill)	Kalamazoo, Mi	N
Motor Wheel, Inc.	Lansing, Mi	N
H. Brown Co., Inc.	Grand Rapids, Mi	N
Folkertsma Refuse	Grand Rapids, Mi	N
Barrels, Inc.	Lansing, Mi	Y
Kaydon Corp.	Muskegon, Mi	N
Van Dale Junkyard	Marietta, Oh	N
Janesville Ash Beds	Janesville, Wi	N
Janesville Old Landfill	Janesville, Wi	N
National Presto Industries, Inc.	Eau Claire, Wi	N
Fort Howard Paper Co. Lagoons	Green Bay, Wi	N

[12] Potential for off-site lead contamination. Y indicates that potential exists. N indicates that no potential exists for off-site lead contamination.

5.2.7. Environmental Data Qualitative Summary

Table XVII is presented as a qualitative summary of potential routes of exposure to lead from environmental sources, for the 83 MSA cities. A positive indication for a categorical source for a particular city does not imply violations of an environmental standard or that there is necessarily an urgent public health concern caused by sources via the indicated medium. It does mean that, based upon current information, the potential for a problem exists. More definitive conclusions, in most instances, can only be drawn subsequent to on-site measurements.

The qualitative summary table is presented as a quick view method of understanding where environmental sources of lead may exists in the 83 cities. The existence of a point source, RCRA facility, landfill facility, or Superfund site does not necessarily indicate that there is an environmental problem. Similarly, the cities shown with ambient air concentrations exceeding 0.2 $\mu g/m^3$ and drinking water exceeding 4 $\mu g/l$, may be well below the standards for air and drinking water, respectively. The significance of the values in the table is merely to reflect the UBK default values. The table is presented to account for known sources of lead that may be above the norm for the 83 cities. A check mark indicates the existence of a facility, or measured air or water concentration above the concentrations shown in the table.

TABLE XVII
Qualitative Summary of Environmental Exposures[13]
to Lead for MSA Cities in 1988

City	Total No. of Area Concerns	No. of Air Point Sources	No. of RCRA Facilities	No. of Landfill Facilities	No. of Superfund Sites	Ambient Air > 0.2 μg/m³	Drinking Water > 4 μg/l
Rock Island							
Moline							
Chicago	1					✓	
Kankakee							
Peoria	1			✓			
Bloomington							
Normal							
Champaign							
Urbana							
Rantoul							
Springfield							
E. St. Louis	1	✓					
Granite City	7		✓✓✓✓	✓	✓	✓	
Rockford							
Total State of Illinois	10	1	4	2	1	2	
Gary	1			✓			
Hammond	1					✓	
E. Chicago	3	✓		✓		✓	
South Bend							
Mishawaka							
Elkhart							

[13] Air point source (in or proximate to city), RCRA facility, Landfill Facility, and Superfund facility numbers indicate facilities with potential to cause exposure to humans. Ambient air and drinking water exceedances pertain to the UBK model default values.

City	Total No. of Area Concerns	No. of Air Point Sources	No. of RCRA Facilities	No. of Landfill Facilities	No. of Superfund Sites	Ambient Air > 0.2 $\mu g/m^3$	Drinking Water > 4 µg/l
Goshen							
Ft. Wayne							
LaFayette							
Kokomo							
Anderson							
Muncie							
Indianapolis	3	✓✓				✓	
Terre Haute							
Bloomington							
Evansville							
New Albany							
Total State of Indiana	6	3		2		3	
Saginaw							
Bay City							
Midland							
Muskegon							
Grand Rapids							
Lansing	1				✓		
East Lansing							
Flint							
Detroit	1	✓					
Ann Arbor							
Battle Creek							
Jackson							
Kalamazoo							
Benton Harbor							
Total State of Michigan	2	1			1		

City	Total No. of Area Concerns	No. of Air Point Sources	No. of RCRA Facilities	No. of Landfill Facilities	No. of Superfund Sites	Ambient Air > 0.2 $\mu g/m^3$	Drinking Water > 4 µg/l
Moorhead							
Duluth							
St. Cloud							
Minneapolis	1	✓					
St. Paul	1	✓					
Rochester							
Total State of Minnesota	2	2					
Toledo							
Cleveland							
Akron	1						✓
Lorain							
Canton							
Steubenville							
Wheeling							
Marietta							
Youngstown	1						✓
Warren	1			✓			
Mansfield	2	✓					✓
Lima							
Dayton							
Springfield							
Columbus	1	✓					
Hamilton							
Middletown							
Cincinnati							
Total State of Ohio	6	2		1			3
Eau Claire							

City	Total No. of Area Concerns	No. of Air Point Sources	No. of RCRA Facilities	No. of Landfill Facilities	No. of Superfund Sites	Ambient Air > 0.2 $\mu g/m^3$	Drinking Water > 4 µg/l
Wausau	1						✓
Green Bay							
Oshkosh							
Neenah							
Milwaukee	1						✓
Racine							
Kenosha							
Madison	1						✓
Janesville	1						✓
Beloit							
LaCrosse							
Sheboygan	1	✓					
Appleton							
Total State of Wisconsin	5	1					4
Total Six States	33	10	4	5	2	5	7

5.4. Chosen Cities

5.4.1. Minneapolis/St. Paul Environmental Sources of Lead

Air quality monitoring data for NAMS stations located in Minneapolis and St. Paul indicate very low values of lead. Annual average air-lead concentrations, from the quarterly monitoring data, were 0.06 $\mu g/m^3$ and 0.05 $\mu g/m^3$ for Minneapolis and St. Paul, respectively.

Six sources in the Metropolitan Statistical Area reported air emissions to the Toxic Release Inventory. These were Gopher Smelting and Refining Co., Eagan, with 13,812 pounds/year total air emissions of lead and lead compounds; North Star Steel, St. Paul, at 12,480 pounds/year; American National Can Co., St. Paul, at 551 pounds/year; Honeywell New Hope Facility, Minneapolis, at 500 pounds/year; Bureau of Engraving, Inc., Minneapolis, at 500 pounds/year; and Whir-Air-Flow, Minneapolis, at 250 pounds/year. Both Gopher Smelting and Refining Co., and North Star Steel, were modeled to estimate air concentrations resulting from emissions. The maximum downwind concentrations were 0.18 $\mu g/m^3$ for Gopher Smelting, and 0.30 $\mu g/m^3$ for North Star Steel. Both values were derived in close proximity, 200 meters, to the emission source. The Eagan facility, consequently, would not contribute to increased lead-air concentrations in either Minneapolis or St. Paul, due to the distance. Generally, noting the relatively *de minimus* maximum concentration value, the exposures are rather limited.

The Hennepin Energy Res. Municipal Waste Combuster, located in Minneapolis, is the only other point source of lead emissions reported in the MSA. The amount of emission, 0.06 pounds per day, based upon stack test emissions, is quite small and, therefore, the source was not modeled to derive air concentrations.

Drinking water test results were not required and therefore were not conducted for the drinking water supplies for 1988 because supplies were sampled for lead every other year. There is no indication of a problem with the source drinking water. Consequently, the model default value of four $\mu g/l$ was

assumed.

On-site disposal was reported in TRI only for the Gopher Smelting facility. As noted above, the facility is far enough from the two central cities such that wind-blown lead contaminated soil and dust, if there were any, would not impact/contribute to soil-lead and dust-lead concentrations in Minneapolis or St. Paul.

Although the NPL Superfund sites with lead included in the data base, discussed earlier, did not list any sites in either city, a further review of NPL site summary documents and files found three sites: Union Scrap Iron and Metal Company located in Minneapolis, Twin Cities Air Force Reserve Base (Small Arms Range Landfill), also located in Minneapolis, and Pigs Eye Landfill, located in St. Paul. None of the sites appear to pose a threat to residents via wind-blown off-site lead contaminated dust.

The Pigs Eye Landfill is a 307 acre site that served as the City's municipal waste landfill and also accepted industrial waste. The soil on-site is contaminated with lead and other constituents. The area immediately surrounding the site is industrial. A residential area is located one-half mile east. Lead was detected in high concentrations in one well, and in low concentrations in soil. Indeed, soil samples taken near the facility indicate soil-lead concentrations of less than 150 ppm. The potential route of exposure is through contamination of 210 residential wells in the vicinity.

The Minneapolis sites are both small. Union Scrap Iron is an one-acre site used to crush lead battery fragments. Reportedly, 30,000 tons of lead-contaminated plaster and rubber fragments remain on-site, partially covered by tarp. A soil contamination study was to be conducted. The three-acre Twin Cities Air Force Base, Small Arms Range Landfill site, is located within and adjacent to the Minneapolis-St. Paul International Airport. Periodic flooding of the site has resulted in the release of lead and other contaminants into the Minnesota River. The primary potential for exposure is through contamination of drinking water wells. A hydrogeological investigation has been initiated.

Three additional sites in the two cities are National Priority List sites that do not cite lead as a

contaminant. The Whittaker Corp. property is a 10-acre site located in Minnesota. The General Mills/Henkel Corp. site, also in the City of Minneapolis, poses a threat to the groundwater aquifer from solvents disposed in a dry well. The 45-acre Koppers Coke site is located in St. Paul. The removal of lead-contaminated coal tar waste and contaminated soil from the site has begun.

The Minnesota Pollution Control Agency provided the raw data base for soil sampling conducted in Minneapolis and St. Paul, that provided a partial basis for the report to the Minnesota State Legislature (MPCA and MDH, 1987). Geometric mean soil concentrations are shown, by census tract, in Appendix N. Soil lead geometric mean values range from a low of 33 ppm to a high of 736 ppm. The geometric mean values are deceptive, however, in that the values do not truly represent soil concentrations in a tract. Indeed, a review of the individual samples taken for each census tract shows a wide range of values, with the highest concentrations generally from soil samples taken near house foundations. Foundation sample concentration values of 3,000 to 7,000 ppm are not uncommon, with the highest sample results in a single census tract in Minneapolis showing a value > 20,000 ppm. The two highest values of 38,850 ppm and 166,780 ppm were determined near an industrial facility in St. Paul. (It should be noted that the blood-lead counterpart data base contains no child blood-lead level measurements for that census tract). Recognizing the wide range of sample values within each census tract, for purposes of deriving modeled blood-lead values for each census tract, the foundation sample values were selected, where available, to represent soil concentration values.

To assess the contribution to elevated Pb-B levels from mobile sources, the distance from the centroid of each census tract was calculated using geographic information systems applications.

5.4.2. Blood-lead Data/Demographics

The Minneapolis and St. Paul blood-lead survey data contains the records of over a thousand children under the age of six for whom blood-lead levels were measured in 1986 and 1987. Table XVIII shows the number of children with elevated blood-lead levels by ethnicity. The dual heritage ethnic

category refers to those children listed as white/African-American, Hispanic/American Indian, or other races. Table XIX provides descriptive statistics for each ethnic group.

TABLE XVIII
Children Under Six Years of Age with
Blood-Lead Levels Exceeding 10 μg/dL based upon
1986 - 1987 Blood-lead Survey for Minneapolis and St. Paul

	Total	White	African-Amer.	Hispanic	Amer. Indian	Asian	Other[14]	Dual Herit.
Number > 10μg/dL	298	151	34	4	64	9	8	28
Total Number	1022	667	114	13	121	31	11	65
Percent Exceeding	29.1	22.6	29.8	30.7	52.8	29.0	72.7	43.1

For white, African-American, and American Indian children (those ethnic groups greater than 100 children), arithmetic blood-lead levels are 7.7 μg/dL, 9.2 μg.dl, and 13.2 μg/dL, respectively. Corresponding geometric mean blood-lead levels (Appendix P) are 5.8 μg/dL, 7.2 μg/dL, and 9.8 μg/dL, respectively. For the data set in total, blood-lead levels ranged from a minimum of 1.0 μg/dL to a maximum of 65 μg/dL, with a geometric mean of 6.6 μg/dL and a geometric standard deviation (GSD) of 2.2. Appendix Q provides comparable data by census tract. Geometric mean blood-lead levels ranged from 2.0 μg/dl (14 observations) to 65 μg/dL (one observation), for the census tracts.

[14] Other ethnic groups include children of dual heritage ethnicity and children for whom ethnicity was not recorded.

TABLE XIX
Blood-lead Values (µg/dL) by Ethnicity for
Minneapolis and St. Paul, Minnesota Blood-lead Survey in 1986 - 1987

Ethnicity	No. of Observations	Minimum (µg/dL)	Maximum (µg/dL)	Geometric Mean (µg/dL)	Standard Deviation
Not Specified	14	3.0000	19.0000	7.2142	4.9017
White	667	1.0000	65.0000	7.6971	6.1447
African-American	114	1.0000	37.0000	9.1666	6.6929
Hispanic	13	3.0000	28.0000	8.8461	6.6061
American Indian	121	1.0000	39.0000	13.2396	9.2032
Asian	31	3.0000	36.0000	10.0967	7.9177
Other[15]	11	1.0000	44.0000	15.4545	12.1767
White/African-American	30	2.0000	34.0000	12.6666	9.2263
White/Hispanic	8	3.0000	22.0000	10.2500	6.0886
White/ American Indian	8	1.0000	21.0000	11.3750	7.4630
White/Asian	3	1.0000	7.0000	4.0000	3.0000
White/Other	3	3.0000	5.0000	4.0000	1.0000
African-American/ Hispanic	8	3.0000	24.0000	15.1250	7.6613
African-American/ American Indian	1	8.0000	8.0000	8.0000	
Hispanic/ American Indian	4	3.0000	20.0000	8.5000	8.0208

Age of housing for each census tract in the Twin Cities was provided from census data via geographic information systems. As counted by the 1980 census, housing in the two cities is

[15] Children for whom ethnicity was not recorded.

overwhelmingly built before 1949, with 78.9 percent of then-existing housing stock built prior to that year, and 92.9 percent built prior to 1960. For the blood-lead data base, the housing stock reflects an even older pattern (as a result primarily of the selection criteria for the blood-lead survey); consequently, there is very little differentiation in housing age by census tract.

5.4.3. Minneapolis/St. Paul Correlation Analysis

A correlation analysis, using the Minneapolis/St. Paul blood-lead data and derived blood-lead levels from the UBK model, for each record, was performed to ascertain the validity of the methodology for finding geographic areas where environmental exposures to lead would result in increased Pb-B levels. Sources have been described earlier.

Selected zero-order correlations are shown in TABLE XX. The correlation of actual blood-lead levels to the corresponding modeled blood-lead levels is small, at 0.05, with a $p = 0.14$; consequently, the results indicate a failure to reject the null hypothesis of no correlation between the modeled and measured blood-lead values. The conclusion from this analysis would be that the modeled blood-lead levels do not predict the actual blood-lead levels at a statistically significant level. (Recognizing problems with the approach, however, a second analysis as employed. This provided better results, as discussed below.)

The analysis also determined very small correlation coefficients for actual Pb-B levels with housing age category, distance from an interstate highway, and soil-lead concentration.

The combined routes of exposure of air (due to ongoing emissions from mobile sources) and soils and dust contamination, due to past deposition from mobile sources, do not appear to contribute appreciably to Pb-B levels for the study population. Similarly, the expected finding of a strong correlation for soil with housing age category, was not determined. The analysis did, however, find a weak correlation of soil concentration with distance from a major highway ($r= 0.13$, $p= 0.0001$).

TABLE XX
Correlation Analysis of Minneapolis/St. Paul

Simple Statistics[16]						
Variable	Number	Log_{10} Mean	Standard Deviation	Sum	Minimum	Maximum
LPB-BA	1033	0.8198	0.3476	846.8810	0	1.8129
LPB-BM	901	0.9664	0.3013	870.7621	0.4941	3.8646
HAC	1033	0.9990	0.1841	1032	0	3.0000
DIST	1033	1135	937.76	1172375	0	5015
SOIL	1033	1863	2199	1924415	0	11162

Correlation Analysis Pearson Correlation Coefficients/ Probability > |R|: Rho = 0/ No. of Observations					
	LPB-BA	LPB-BM	HAC	DIST	SOIL
LPBBA	1.0000 0. 1033	0.0487 0.1436 901	0.0095 0.7593 1033	-0.0849 0.0063 1033	0.0795 0.0106 1033
LPBBM	0.0487 0.1436 901	1.0000 0. 901	-0.03855 0.2477 901	-0.0049 0.8825 901	0.7248 0.0001 901
HAC	0.0095 0.7593 1033	-0.0385 0.2477 901	1.0000 0. 1033	0.0093 0.7637 1033	0.0240 0.4408 1033
DIST	-0.0849 0.0063 1033	-0.0049 0.8825 901	0.0093 0.7637 1033	1.0000 0. 1033	0.1318 0.0001 1033
SOIL	0.0795 0.0106 1033	0.7248 0.0001 901	0.0240 0.4408 1033	0.1318 0.0001 1033	1.0000 0. 1033

[16] where LPBBA = measured blood-lead level (μg/dl); LPBBM = modeled blood-lead level (μg/dl); HAC = housing age category (range of years for each category), as defined in the regression model; DIST = distance from an interstate highway (meters); and SOIL = soil-lead concentration (ppm).

A significant problem with the correlation analysis was that different physical scales were being compared. The actual blood-lead values were associated with soil-lead values at or near the home of the individual child, and thus it was on a relatively small geographic scale. The modeled blood-lead values, in contrast, depended upon a soil-lead concentration for the census tract where the child resided. This much larger geographic area (scale) could result in a soil-lead concentration much different than the actual exposure concentration. Recognizing this, a second correlation analysis was employed to compare the geometric mean of the measured blood-lead levels to the geometric mean modeled blood-lead levels calculated for each census tract. Only census tracts having nine or more observations were selected, as displayed in Table XXI. The modeled values are higher particularly in that high soil lead values were used for census tract modeling, than is thought to be the actual exposure of children living in a census tract.

TABLE XXI
Selected Census Tract Data from Minneapolis/St. Paul

No. Obs	Census Tract	Pb-B Act.[17] (G.M.)	Pb-B Mod.[18] (G.M.)	No. Obs	Census Tract	Pb-B Act. (G.M.)	Pb-B Mod. (G.M.)
26	15	9.79	18.33	23	73	6.63	5.35
14	16	7.76	7.12	32	79	8.12	46.15
28	18	3.58	8.55	20	83	10.19	11.73
9	21	8.60	13.59	14	86	5.36	11.93
14	22	8.10	8.00	30	301	5.56	6.49
27	25	4.78	8.77	20	325	10.84	6.49
14	28	5.58	6.31	16	326	9.51	8.06
14	29	6.62	14.61	14	335	7.08	8.06
12	33	7.51	7.77	11	355	8.91	3.48
16	36	4.95	7.22	46	357	4.51	6.27
20	50	6.85	6.50	19	368	5.56	14.38
18	61	8.79	20.24	36	370	4.35	4.90
15	66	7.98	3.30	27	371	6.80	17.27
46	72	8.52	18.25				

Table XXII presents the results of a correlation analysis for modeled blood-lead and actual blood-lead variables listed in the table. The correlation improved from 0.13 to 0.3. The results were not statistically significant, with p> 0.10. Given the constraints inherent in the methodology, however, the approach works reasonably well. It is interesting to note that for the modeled Pb-B mean values, some census tracts were quite close to the measured Pb-B mean values. The larger estimated values from the model results in a small Pearson correlation coefficient.

[17] Geometric mean value of measured blood-lead level.

[18] Geometric mean value of modeled blood-lead level.

TABLE XXII
Correlation Analysis- Minneapolis/St. Paul Census Tract Level

Simple Statistics[19]						
Variable	Number	Mean	Standard Deviation	Sum	Minimum	Maximum
LPBBA	28	6.8867	2.3324	197.830	0	10.840
PBBM	28	10.6828	8.5733	299.120	0	46.150

CORRELATION ANALYSIS Pearson Correlation Coefficients/ Probability > |R| under Ho: Rho = 0/ N=28		
	LPb-BA	Pb-BB
LPBBA	1.000 0.0	0.3167 0.1005
PBBB	0.3167 0.1005	1.000 0.0

In particular, one outlier results in a skewing of the data which causes a smaller pearson correlation coefficient and a smaller p-value. Figure 12 is a scatter plot of the 27 data points in Table XXI, comparing the geometric mean modeled blood-lead levels with the actual blood-lead levels for children in each of the 27 census tracts. As shown in the scatter plot, there appears to be a definite linear association between the two variables, with modeled values generally increasing with increasing measured blood-lead levels.

[19] where LPBBA = measured blood-lead level, and PBBM = modeled blood-lead level.

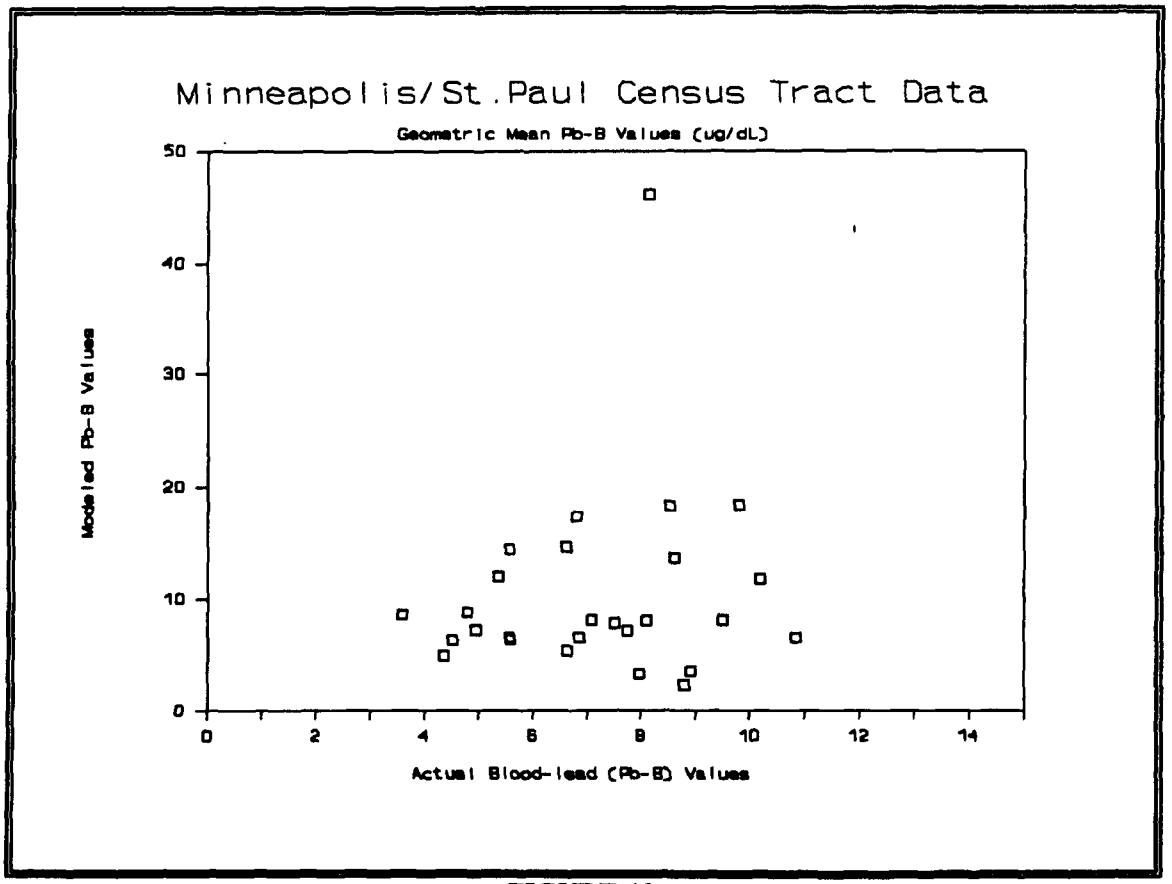

FIGURE 12
Scatter Plot of Modeled Blood-lead Values Vs. Actual Blood-lead Values

5.4.4. Minneapolis/St. Paul Regression Analysis

A second use of the blood-lead data was to partition the actual blood-lead levels found among environmental sources. For the data set, distance from a major thoroughfare was added to each record in order to ascertain whether lead from vehicle exhaust (thought to be primarily via lead-contaminated dust

and soil from historic deposition from vehicles) would explain a portion of the variation. In addition, housing age was added to each record (by category) to recognize the contribution of lead-based paint. This would also account for historic deposition from area and point sources.

Results of the regression analysis yielded the following for the full model and the final model. Regression analysis for the full model shows a very small R^2 value of only 0.08, although the result is statistically significant (p= 0.0001). This is an expected result, given the minimal Pearson correlation coefficients derived for selected variables. The results are tabulated as Appendix Q.

The final model was derived by using a stepwise regression procedure, with a criterion level of significance of p= 0.05 for inclusion of an independent variable in the formula. Non-significant regression coefficients were not carried forward from the full to the final model. The results are tabulated in full as Appendix R, and in summary form as TABLE XXIII. The final model is

$$\text{Log } PbB_{act} = 1.007 - 0.017\ AGE - 0.092\ E1 + 0.099\ E3 + 0.001\ FAT - 0.004\ INC$$

Where

PbB_{act}	= Measured blood-lead level from survey (μg/dL)
AGE	= Age of child (years)
E1	= 1 if ethnicity is white, 0 otherwise
E3	= 1 if ethnicity is American Indian, 0 otherwise
FAT	= number of years of father's education
INC	= family income for census tract (thousands of dollars)

TABLE XXIII

Summary of Stepwise Procedure for Dependent Variable
Actual Blood-lead Concentration for Minneapolis/St. Paul

Step 5 Variable FAT Entered R-square = 0.0763566 C(p) = 11.19800486					
	Deg. of Freedom	Sum of Squares	Mean Square	F	Prob>F
Regression	5	8.49494	1.69898	14.88	0.0001
Error	895	102.20900	0.11420		
Total	900	110.70394			

Variable[20]	Parameter Estimate	Standard Error	Type II Sum of Squares	F	Prob >F
INTERCEP	1.00708	0.05457	38.88745	340.52	0.0001
AGE	-0.01748	0.00657	0.80810	7.08	0.0800
E1	-0.09168	0.02840	1.18992	10.42	0.0013
E3	0.09892	0.03969	0.70930	6.21	0.0129
FAT	0.00096	0.00048	0.45675	4.00	0.0458
INC	-0.00454	0.00191	0.64049	5.61	0.0181

Bounds on condition number: 1.471526, 30.51517
All variables in the model are significant at the 0.1500 level. No other variables met the 0.0500 significance level for entry into the model.

Summary of Step wise Procedure for Dependent Variable LPb-BA

Step	Var[21]	No. In	Partial R^2	Model R^2	C(p)	F	Prob > F
1	E1	1	0.0485	0.485	30.7085	45.8441	0.0001
2	INC	2	0.0096	0.0581	23.3882	9.1134	0.026
3	AGE	3	0.0080	0.0661	17.5747	7.6970	0.0056
4	E3	4	0.0065	0.0726	13.2208	6.2961	0.0123
5	FAT	5	0.0041	0.0767	11.1980	3.9996	0.0458

[20] where AGE = age of child; E1 = dummy variable for white ethnicity; E3 = dummy variable for American Indian ethnicity; FAT = number of years of father's education; and INC = family income for census tract of residence.

[21] Variable Entered, Removed

Independent variables that did not improve the model were the model estimate of the blood-lead level (PbB_{mod}), housing age category (HAC), distance from a major highway (DIST), African-American ethnicity (E2 = 1), gender (GEN), number of years of mother's education (MOT), and the measured soil-lead concentration for the census tract (SOIL). The comparison population for ethnicity (E1=E2=E3=0) consists of all ethnic categories except white, African-American, and American Indian. Although the final model is statistically significant ($p > 0.001$), the R^2 value is small, indicating that the selected independent variables do not explain much of the variation in the measured blood-lead levels.

With the exception of the number of years of father's education, FAT, the signs of all regression coefficients in the final model reflect intuitive expectations. The blood-level decreases with age. This is as expected for the overall childhood population range, although within the age strata, blood-lead levels are expected to peak at two and then decrease. The negative association with white ethnicity and increasing family income is also as expected. A positive regression coefficient associated with American Indian ethnicity merely reflects the higher mean blood-lead levels for this ethnic group as a whole.

Only the number of years of fathers education, indicating an increasing blood-lead level with increasing education, is counter to expectations. Further, it would be more logical to have a significant regression coefficient for the mother's education attainment, not the father's, as indicated in the final regression model. Consequently, the inclusion of FAT in the model and the positive sign of the regression coefficient is thought to be a spurious effect.

A change in the ethnicity dummy variable scheme was made to ascertain whether African-American and Hispanic blood-lead values would be statistically significantly higher than values for white children, while controlling for other variables. Variable E1 was changed from E1= white to E1= Hispanic. The change was made to make the comparison population (E1=E2=E3=0) to be white and a small number of ethnic minority children. This comparison population thus excludes Hispanic, African-American, and American Indian children. The final revised model results are provided as Appendix S. The revised final

model is:

Log PbB_{act} = 0.998 -0.0172 AGE + 0.151 E3 + 0.001 FAT - 0.006 INC

Where

PbB_{act}	= Measured blood-lead level from survey (μg/dL)
AGE	= Age of child (years)
E3	= 1 if ethnicity is American Indian, 0 otherwise
FAT	= number of years of father's education
INC	= family income for census tract (thousands of dollars)

5.5. UBK City Results

Based upon the use of ambient air quality data for each city, measured drinking water concentration for the city, and weight averaged soil and dust concentrations for each census tract group, the UBK model was used and city exceedance number developed. Aggregate results are displayed in Table XXIV. Appendix T, provides detailed information by census tract group area.

TABLE XXIV
Numbers of Children Under 7 Years of Age in the Midwest
Expected to Exceed 10 μg/dL Blood-Lead Level in 1988

City	Childhood Population	Total No. Exceeding	African-American Exceeding	Hispanic Exceeding
Rock Island	4,910	461	103	17
Moline	4,379	434	5	37
Chicago	321,585	40,370	18,712	7,888
Kankakee	3,461	289	87	3
Peoria	13,368	1,306	354	24
Bloomington	4,362	330	21	5
Normal	2,430	26	2	0
Champaign	3,979	168	34	2
Urbana	2,359	154	10	3
Rantoul	N/A[22]			
Springfield	9,716	554	76	4
E. St. Louis	8,127	798	768	8
Granite City	3,726	273	4	5
Rockford	14,406	965	198	38
Total State of Illinois	396,809	46,129 (12 %)[23]	20,375	8,034
Gary	20,855	831	652	69
Hammond	10,522	1,059	92	100
E. Chicago	5,073	660	189	275
South Bend	11,441	1,084	207	26
Mishawaka	4,149	225	2	2

[22] N/A not available. Data was not available for these cities.

[23] Percentage of total population. Numbers are based upon ambient air, drinking water, and derived soil and dust lead concentrations used in the Uptake Biokinetic Model.

City	Childhood Population	Total No. Exceeding	African-American Exceeding	Hispanic Exceeding
Elkhart	4,616	464	97	7
Goshen				
Ft. Wayne	18,910	1,780	414	55
LaFayette	4,146	243	5	3
Kokomo	5,437	401	27	6
Anderson	6,707	502	110	3
Muncie	6,822	522	56	4
Indianapolis	73,868	5,223	1,740	52
Terre Haute	5,250	797	71	6
Bloomington	2,775	141	7	2
Evansville	12,444	1,248	135	7
New Albany	3,598	258	13	2
Total State of Indiana	196,612	15,439 (8 %)	3,817	619
Saginaw	9,943	935	348	92
Bay City	4,358	564	10	27
Midland	3,834	45	1	1
Muskegon	4,741	603	135	18
Grand Rapids	20,064	1,942	486	99
Lansing	15,251	955	128	75
East Lansing	2,531	115	7	2
Flint	19,923	1,446	581	38
Detroit	134,680	19,142	12,409	555
Ann Arbor	7,819	381	38	9
Battle Creek	4,150	569	129	12
Jackson	4,588	748	119	15
Kalamazoo	7,323	777	181	17
Benton Harbor	N/A			

City	Childhood Population	Total No. Exceeding	African-American Exceeding	Hispanic Exceeding
Total State of Michigan	239,205	28,225 (12 %)	14,571	961
Moorhead	2,401	61	0	1
Duluth	8,299	1,284	9	5
St. Cloud	3,577	206	1	1
Minneapolis	29,884	4,611	379	59
St. Paul	25,357	3,333	194	97
Rochester	5,774	237	1	2
Total State of Minnesota	75,292	9,732 (13 %)	584	165
Toledo	38,143	4,515	1,157	182
Cleveland	61,289	9,396	4,022	360
Akron	23,644	3,161	694	20
Lorain	8,962	465	53	70
Canton	9,739	1,342	264	18
Steubenville	2,002	160	25	1
Wheeling	N/A			
Marietta	N/A			
Youngstown	11,968	1,884	673	64
Warren	5,742	437	69	3
Mansfield	7,180	688	90	7
Lima	5,972	550	116	6
Dayton	22,426	2,206	688	17
Springfield	7,745	914	175	7
Columbus	2,968	432	110	5
Hamilton	7,217	728	77	6
Middletown	4,467	281	31	1
Cincinnati	38,829	5,415	1,939	41

City	Childhood Population	Total No. Exceeding	African-American Exceeding	Hispanic Exceeding
Total State of Ohio	313,139	35,797 (11 %)	11,165	837
Eau Claire	4,250	247	1	1
Wausau	3,017	284	0	1
Green Bay	9,058	483	1	4
Oshkosh	3,992	388	2	2
Neenah	N/A			
Milwaukee	67,871	13,878	4,225	781
Racine	9,626	819	130	56
Kenosha	7,927	494	19	22
Madison	12,294	759	21	11
Janesville	5,655	263	0	1
Beloit	3,982	421	63	5
LaCrosse	3,341	404	1	2
Sheboygan	4,810	443	1	8
Appleton	6,251	286	0	2
Total State of Wisconsin	138,773	18,767 (13.5 %)	4,465	894
Total Six States	1,359,830	154,089 (11.3 %)	55,247	11,513

The highest percentages of children exceeding 10 µg/dL Pb-B in Illinois were derived for Chicago, where the majority of census areas were in double digit percentages, with many in the 15 to 19 percent range. The maximum value was 19 percent. A majority of the total number of children exceeding 10 µg/dL in many of the communities were African-American and Hispanic, reflecting the racial makeup of the neighborhoods. This factor also gives rise to the large number of children under seven years of age at risk of exposure to lead. The high exposure potential reflects primarily soil and dust concentrations

(based upon housing stock age). Drinking water and air concentration values in the city were low.

Two areas in East St. Louis also had high numbers of African-American children with expected exceedances, although the percentages were not high at 11 and 12 percent (597 and 100 African-American children, respectively). Granite City, an area with widespread soil and dust contamination resulting from industrial operations in the city, had an average of 7 percent exceedances using the methodology. The relative small numbers for African-American and Hispanic children reflect the low population concentrations of these two ethnic groups in Granite City. It is clear that the study approach underestimates the risk in Granite City, however, by not using the higher actual soil and dust concentrations that are currently being determined. One area in Peoria is notable, with a 15 percent exceedance estimate corresponding to 224 African-American and 13 Hispanic children. No other Illinois city was notable for large numbers of African-American or Hispanic children under seven years of age, expected to exceed 10 μg/dL Pb-B.

Compared to other states in the Midwest, the community areas of most cities in Indiana have low percentile values for expected exceedances, and low numbers of potentially exposed African-American and Hispanic childhood populations. This is due to not only generally low derived-exceedance-percentages, but also to smaller city populations and relatively low population density for both minority ethnic groups. Five cities, East Chicago, Evansville, Ft. Wayne, Indianapolis, and Terre Haute, had community areas in the 15 to 19 percent range. The largest numbers of African-American and Hispanic children with expected exceedances of 10 μg/dL Pb-B were in Indianapolis, with 1,740 and 52 children, respectively, followed in quantitative rank by Gary, with 652 and 69 children, respectively. This ranking is generally indicative of the relatively large population size of these two cities. It is noted, in particular, that the community area percentages for the City of Gary were all less than 10 percent.

Detroit closely resembles Chicago in having a number of areas with expected percentages ranging from 15 to 20 percent, with corresponding high numbers of African-American and Hispanic children

reflecting the ethnic makeup of the communities. A total of 19,142 children, including 12,409 African-American and 556 Hispanic children, are the expected exceedance numbers. For other State of Michigan cities, aside from Detroit, community areas in Ann Arbor, Battle Creek, Flint, Jackson, and Kalamazoo had percentile values in the 15 to 19 percent range. None of these areas, however, had very high numbers of African-American or Hispanic children with expected exceedances of 10 μg/dL Pb-B.

As expected, for the State of Minnesota, both the highest percentages and the greatest number of African-American and Hispanic children with exceedances were derived for Minneapolis and St. Paul. The Twin Cities expected numbers of African-American and Hispanic children with exceedances were, respectively, 379 and 59 for Minneapolis, and 194 and 97 for St. Paul. For the State of Minnesota, only Duluth, aside from the Twin Cities, had community areas with percentile ranges of 15 to 18 percent.

The community area in Ohio with the largest expected exceedance percentile was located in the City of Toledo, with a value of 20 percent, corresponding to 431 African-American and 45 Hispanic children expected to exceed 10 μg/dL Pb-B. For the city as a whole, 1,157 African-American and 182 Hispanic children are expected to exceed 10 μg/dL Pb-B. Two cities in Ohio have higher numbers of children with exceedances. Cleveland's numbers are 4,022 African-American children and 360 Hispanic children, and Cincinnati's numbers are 1934 and 41, respectively. Although several other cities had community areas with percentage values in the 15 to 19 percent range, the only other city with more than 1,000 children potentially exceeding the criterion value was Columbus, with 1,094 African-American and 33 Hispanic children. For the State of Ohio, the highest percentile of 23 percent was derived for a low population density community area in Youngstown.

Wisconsin is set apart somewhat from the other states and community areas by having several communities with levels of lead in drinking water at measured levels, above the level of detection. This factor, combined with soil and dust concentrations associated with older housing stock, resulted in the higher estimates of exceedance for four Wisconsin cities.

On a community total basis, Milwaukee is high both in percentile (20 percent) and in numbers (13,878 total, including 4,225 African-American and 781 Hispanic children). Several areas were in the 28 to 30 percent range, making the city the highest overall of all cities assessed, and resulting in large estimated numbers of children with exceedances. Milwaukee's drinking water concentration also measured comparatively high, at 25 ppb for 1988. Aside from estimated percentages of 17 percent for areas in both La Crosse and Racine, neither the percentiles nor the numbers of African-American and Hispanic children exceedances were exceptional for all other communities in Wisconsin.

Seven cities are in the top 10 by virtue of both overall percent exceeding and number of children exceeding 10 μg/dL. Those cities are Milwaukee, Wisconsin; Detroit, Michigan; Minneapolis and St. Paul, Minnesota; and Cincinnati, Akron, and Cleveland, Ohio. The top 10 cities, by percentile and total number of children, are shown in TABLES XXV and XXVI, respectively.

TABLE XXV
Top Ranked Cities by Percentile
of Children Exceeding 10 μg/dL Pb-B

	City	%	Expected Total No. of Children < 7 Years Old	Expected No. of African-American Children < 7 Years Old	Expected No. of Hispanic Children < 7 Years Old
1	Milwaukee, WI	20.4	13,878	4,225	781
2	Jackson, MI	16.3	748	118	15
3	Duluth, MN	15.5	1,284	9	5
4	Minneapolis, MN	15.5	4,611	379	59
5	Cleveland, OH	15.3	9,396	4,022	360
6	Terre Haute, IN	15.2	797	71	6
7	Detroit, MI	14.2	19,142	12,409	556
8	Cincinnati, OH	13.9	5,415	1,934	41
9	Battle Creek, MI	13.7	569	129	11
10	Akron, OH	13.4	3,161	694	20

TABLE XXVI
Top Ranked Cities by Number of
Children Exceeding 10 µg/dL Pb-B

	City	%	Expected Total No. of Children < 7 Years Old	Expected No. of African-American Children < 7 Years Old	Expected No. of Hispanic Children < 7 Years Old
1	Chicago, IL	13	40,370	18,712	7,888
2	Detroit, MI	14	19,142	12,409	555
3	Milwaukee, WI	20	13,878	4,225	781
4	Cleveland, OH	15	9,396	4,022	360
5	Cincinnati, OH	13	5,415	1,939	41
6	Indianapolis, IN	7	5,223	1,740	52
7	Minneapolis, MN	15	4,611	379	59
8	Toledo, OH	12	4,515	1,157	182
9	St. Paul, MN	13	3,333	194	97
10	Akron, OH	85	3,161	694	20

The six Midwest states ranged from 8 percent exceedance estimates in Indiana to 13 percent in Minnesota, although it is noted that these percentages are not particularly meaningful at the state level. The States of Illinois and Michigan had the largest numbers of African-American and Hispanic children under seven years of age expected to exceed 10 µg/dL Pb-B, including 28,000 and 16,000 minority children, in the respective states. Every state has community areas where elevated blood-lead levels are of concern.

For the six states, all cities combined, the total childhood population (children under seven years of age) was 1,359,000 in 1988. The analysis indicates that 154,000 children, or 11 percent of the total, would have blood-lead levels exceeding 10 µg/dL. This includes 55,000 African-American and 12,000

Hispanic children. These numbers are presented for illustrative purposes, and are not a prediction of numbers of children. It is noted, however, that these numbers are conservative compared to other estimates (refer to Section 2.5).

6. DISCUSSION

The population comparative risk methodology developed for this study contributes to the understanding of the extent to which low-level environmental sources of lead add to elevated blood-lead levels in childhood populations. The methodology provides an assessment of the relative numbers of children at risk to the adverse health effects due to environmental lead exposure. Previous studies have been at the national level. The methodology fills a gap in research efforts on the extent of childhood lead poisoning. It provides city specific estimates to highlight possible areas of high numbers of children with elevated blood-lead levels. Indeed, the methodology provides comparative numbers within cities.

A key value of the methodology is as a ranking tool to guide public health officials to cities and areas within cities having the highest potential for childhood exposure to lead. More definitive data would need to be obtained to confirm the initial characterization of areas as high risk. Rather than investing resources in areas found in retrospect to be low risk areas, however, high risk areas could be targeted and addressed on a priority basis. Further, the environmental pathways of exposure developed in this study, provide a clear indication of whether to gather further information on air quality, drinking water quality, or soil and dust concentrations in a given city. Such measured environmental data, together with any blood-lead data available for a community, is a fundamental step towards primary intervention actions. Removing lead from the environment will avoid the need for clinical intervention for the individual child. That is, of course, the desired outcome.

6.1. Demographics

Although the demographic and associated data (housing age, income) was obtained for each census tract, there were inherent imprecision in the data. The results of the 1990 census was not yet available; 1988 data (estimated from the 1980 census) were utilized. The numbers derived from these estimated data therefore have, inherently, the same level of inaccuracy.

Beyond data estimation, a larger problem concerns how the data were categorized, as reported in the census data base. Because the age categories did not match the study design (e.g., childhood age strata were zero to five, six to 13 years, etc., while the study design focused upon children less than seven years of age), an approximation was derived to reflect the number of children in the study design strata.

A more problematic concern is that children in each age band were not totally disaggregated by ethnicity. Derived numbers were thus underestimates of the actual numbers of minority children in an area and, consequently, determined to exceed the criterion blood-lead value. This resulted from a procedure that calculated the number of children in a census tract by a proration of the relevant ethnic group's portion of the total population. Such an approach is accurate only for mono-ethnic populations. As ethnic diversity increases, this method of estimation becomes more and more imprecise. In particular, it results in an underestimate when the number of children in minority families (i.e., family size) exceed the number of children for the community as a whole. Similarly, the demographic data obtained at the census tract level did not include ethnicity-specific birth rates. Because African-American and Hispanic birth rates are often higher than the general population, application of a city's overall birth rate to the ethnicity-specific population, to estimate numbers of fetuses at risk, results in an underestimate of fetuses at risk.

Problems of matching housing age categories were similar. The census data provided strata beginning with housing stock built prior to 1949. For purposes of the study, age strata for housing stock built before 1920 and before 1940, associated with lead pipes and higher concentrations of lead-based paint, respectively, would be more pertinent. Some precision in estimating soil and dust values, in particular, was sacrificed by assuming houses built before 1949 were also expected to have lead-water supply pipes and higher lead-based-paint concentrations.

A key finding, well into the study, was that the geographic information systems software platform was not the most expedient in manipulation of demographic housing stock age, and other census bureau obtained information. Indeed, extraction of relevant data (census tract information only for selected cities

within each of the six states) proceeded rapidly when processed using standard data base management software on a personal computer platform.

6.2. Environmental Data

Several aspects of the usefulness, or lack thereof, of environmental data became readily apparent as the study progressed. The air route of exposure was determined to be of minor consequence as a contributor to the estimated blood-lead levels. The measured air-lead concentrations were found to be very low in an overwhelming number of cases. Even the modeled major sources, for which air concentrations were derived, proved to have little impact, beyond relative close proximity to the source. Consequently, at the large scale for which the algorithm was applied (aggregations of census tracts), those concentrations could not be included in the UBK model results, nor, consequently, accounted for in the estimated numbers of childhood exceedances. It is also noted that many of the sources are located distant from the central city populations of concern. Based upon these findings, it is apparent that the relatively small numbers of children affected by such point sources, would not change the substantive results of the comparative population (by city) risk analysis. Surprisingly, the Toxic Release Inventory data base proved to be valuable in assessing the relative importance of point sources of emissions, while the Aerometric Information and Retrieval System Facility Subsystem, which also provided emission information, was not useful for the study. The only other category of air emissions, municipal waste combusters, appeared to be of minor import as a source of exposure.

The results for abandoned hazardous waste sites were also unexpected, in that the great majority (of lead contaminated sites) are located outside the central cities, and thus away from populations of concern. As was the case for air stationary sources, contamination of soils and dusts at a site would be a local phenomenon affecting only closely proximate populations. None were accounted for in the UBK modeling. An assessment of sites, however, indicated the need for little concern for the category as a whole, except as noted in the results section concerning Granite City, Illinois.

Operating hazardous waste facilities proved to be the most difficult to assess. This was primarily because there is no central data base for which to list Resource Conservation and Recovery Act facilities that dispose of lead and lead compounds. Generally, for the facilities assessed, the categorical source was not deemed to be a factor in the study area cities.

Drinking water contamination was found generally to not be a problem, and, except for the cities noted in the results section, the categorical source is not a major contributor to estimated elevated blood-lead levels. It is noted, however, that brass plumbing fixtures and lead contamination associated with new home construction is not addressed.

The procedure, utilization of the UBK model, did not include lead-based paint concentrations. That was beyond the scope of the study, which addressed environmental sources of lead. There was also a dearth of information upon which to estimate lead-based paint contribution, for purposes of this methodology. The study focused upon environmental sources of lead to estimate chronic effects. Lead-based paint, historically, has been associated more with acute effects. (This is because at high blood-lead levels, signs and symptom are more readily discernable.) Consequently, soil and dust concentration values, derived from age of housing stock, generally predominates as the source of estimated elevated blood-lead levels. The percent exceedances and corresponding numbers of children exceeding the criterion value are driven by housing age (dust and soil concentrations) with adjustments for drinking water and air concentrations pertinent to each community assessed.

6.3 Correlation Analysis

The original correlation analysis for the Minneapolis and St. Paul areas, upon application, was determined not to be adequate for testing the validity of the algorithm. A fundamental problem is in the use of the UBK model to derive modeled blood-lead values for comparison to actual values. The model calculates a geometric mean blood-lead value for a population. The study data was of individual measurements. An individual child, even having the same exposure concentrations used in the UBK

model run, could, of course, have a Pb-B level on either side of the mean value, and could very well be two or more standard deviations from the mean. Consequently, the derived (mean) value cannot be expected to correlate well with the actual (individual) value. Further, a different scale for the two values was used. For the actual Pb-B value, data for the child was measured specific to the child and the home. For the UBK model, information (soils data) was available only at the (much larger and consequently very much more varied) census tract level. The great deal of variability in soil concentrations at residential yards, as further varied by choice of sampling location, is also noted. Moreover, nothing in the model could account for what was undoubtedly also occurring, i.e., potential for contaminated paint and dust exposure, home habits (such as frequency of dusting), occupational exposure, cigarette smoking in the home, and other factors. These factors are all known to affect Pb-B levels. Consequently, the procedure was not deemed to be robust, in that statistical power was lost due to each of these factors.

A second derived approach, using the geometric mean values for all actual blood-lead values, yielded a better result, although most of the same problems are inherent in that approach as well.

6.4 Regression Analysis

Distance from a major highway was not found to be associated with blood-lead levels. The finding of statistical significance is not particularly relevant given the small R^2 value calculated for the final model. The analysis failed to find an association between blood-lead levels and distance from either an interstate highway, or with soil. The latter finding is consistent with that of the State of Minnesota study (MPCA and MDH, 1987) (i.e., the relationship between blood-lead levels and soil concentrations is weak). Given that weakness, the lack of a relationship with distance from a highway is also an expected result.

With the exception of the number of years of father's education, FAT, the signs of all regression coefficients in the final model reflect intuitive expectations. The blood-level decreases with age. This is as expected for the overall childhood population range, although within the age strata, blood-lead levels

are expected to peak at two and then decrease. The negative association with white ethnicity and increasing family income are also as expected. A positive regression coefficient associated with American Indian ethnicity merely reflects the higher mean blood-lead levels for this ethnic group as a whole. Thus compared to minority children (other than African-American), white children have statistically significantly lower blood-lead level. American Indian children, however, have statistically significantly higher blood-lead levels.

Only the number of years of fathers education, indicating an increasing blood-lead level with increasing education, is counter to expectations. Further, it would be more logical to have a significant regression coefficient for the mother's education attainment, not the father's, as indicated in the final regression model. Consequently, the inclusion of FAT in the model and the positive sign of the regression coefficient is thought to be a spurious effect.

The revised final regression model, substituting Hispanic for white as dummy variable E1, yielded unexpected results. After controlling for other variables, neither variable E1 (Hispanic) nor E2 (African-American) were associated with the actual blood-lead levels at statistically significant levels. Thus the higher mean blood-lead levels for African-American and Hispanic children compared ton white children, controlling for other variables, is not statistically significant. Compared to white children, American Indian children have statistically significant higher mean blood-lead levels. Compared to (predominately) white childhood population in the revised model, or other ethnic minorities as in the original regression model, American Indian children have blood-lead levels that are statistically significantly higher.

6.5 City Estimates of Exceedance

The derived values are thought to be minimal estimates for several reasons. As discussed under demographics, the numbers of children estimated in the ethnicity categories of interest are low. The procedure, further, focuses upon chronic exposure from environmental sources, and does not account for additional numbers of children due to exposures to contaminated paint, including any resultant acute

exposures. The latter, in an area of deteriorating older housing stock, can greatly increase the numbers of affected children.

Moreover, a significant concern in the procedure is that the UBK model does not account for ethnicity or socioeconomic status (nor was it designed to do so). It is well documented that such factors increase the relative risk for many in the study population. By point of comparison, for large numbers of African-American children, ATSDR (1988) postulates that fully two-thirds in the lowest socio-economic stratum would exceed 15 μg/dL blood-lead. This is well above the percentages derived from the algorithm. Further, a 10 μg/dL criterion level would result in greater than two-thirds of the pertinent population exceeding the value.

6.6 Uncertainties

As with most screening methodologies, there are a number of areas in the methodology that introduce uncertainty into the results. Due to the wide range of data that the methodology uses, mixing actual data with postulated data and then using a model, it is impossible to calculate an uncertainty in the traditional sense (derivation of a confidence interval with an associated level of statistical significance for the numbers of children cited for each city). It is not the intent of the study, however, to predict numbers of children exceeding 10 μg/dL blood-lead. Rather, it is to compare cities in order to make reasoned judgements on which geographic areas appear to have children at highest risk of exposure to environmental sources of lead. Actual measurements would then be necessary to ascertain childhood exposure. Nonetheless, it is useful to discuss uncertainties in the methodology, discussed throughout this document, in one section. That is the purpose of this discussion, to summarize uncertainties inherent in this population comparative risk screening methodology.

The quality of ambient air quality data is judged to be excellent. The data is from an ongoing ambient air quality network administered by each state agency under a rigorous quality assurance program. The program is prescribed by U.S. EPA regulations. Monitors are generally sited to ascertain peak spatial

concentrations, however, not to determine representativeness of air quality in a city, per se. It is, nevertheless, often used for that purpose. Further, the limited numbers of monitors does raise concern about how representative the data are.

Drinking water data were taken from data generated by drinking water suppliers, as provided to state agencies, under a quality assurance program prescribed by U.S. EPA regulation. A limited number of samples, taken at the supply, is used to characterize exposure for the entire community serviced. Variations in samples taken over a course of time during the year indicates that actual exposure, in some instances, may be difficult to determine. There is no readily discernable pattern of variation, where lead was found at detectable levels. It is also noted that most of the supplies consistently measured non-detectable levels of lead.

The soil and dust values used in the UBK model were those estimated from the ages of housing stock for each area. No measured values were used. Consequently, there is substantial uncertainty in the derived concentrations. Further, the data base from which the estimates were derived, the National Housing Survey Data discussed in Section 4.6.3., had a range of values for each housing age category. For houses built between 1960 and 1979, dust-lead concentration values ranged from 0 ppm to 1520 ppm, with a mean value of 20 ppm and a standard deviation of 145. For older homes, those built prior to 1940, the range was even greater, from a minimum of 0 ppm dust-lead to a maximum of 33,130 ppm, with a mean of 565 ppm and a standard deviation of 3,780. Comparable soil-lead concentrations for pre-1940 housing stock were a minimum soil-lead concentration of 1 ppm, a maximum of 6,260 ppm, a mean of 565 ppm, and a standard deviation of 1,060. Thus there is uncertainty in the values chosen to represent soil and dust values in the UBK model, based upon age of the dwelling.

There are a number of routes for introduction of uncertainty via use of the UBK model. The model uses assumptions regarding behavioral and physiological parameters that affect the results (discussed in Section 4.7). Behavioral patterns assumed for each age group, for example, could miss the mark.

Section 4.7.1. discusses the high dependence of the model on the selection of the geometric standard deviation (GSD) assumed to be applicable a modeled population. For a given set of concentrations, changing the GSD from 1.42 to 1.8 results in an estimated percentage of childhood exceedance of 10 μg/dL of 0.05 percent for the lower GSD, to 2.47 percent for a GSD of 1.8. This, consequently, would introduce a great amount of uncertainty, if the UBK model were being used as a predictive tool. It is not being used for that purpose here. Consequently, because any uncertainty introduced by selection of a GSD value is in the same direction for all cities, the uncertainty introduced via this mechanism is of less concern (when comparing populations).

Additional uncertainty is introduced via this new use of the model. It has not been validated for use at the census tract level. Rather, it was developed for use at specific sites, for which environmental concentrations have been more readily obtainable.

Finally, the correlation analysis, comparing the mean values of blood-lead values for groups of children in a census tract, to the UBK modeled values for the tract, resulted in a correlation coefficient of 0.3 at $p > 0.10$. While this result indicates a relatively weak correlation, it appears to be quite reasonable given the myriad of uncertainties associated with the methodology. In particular, it is reasonable given the use of the methodology as a population comparative risk screening tool, as opposed to as a predictive methodology.

7. CONCLUSIONS

Central city residents, particularly African-American and Hispanic children, are subject to low-level exposure of environmental sources of lead. Differential exposure exists amongst the cities. The population screening methodology provides a viable method for estimating where the greatest numbers of children at highest risk reside. Clearly soil and dust concentrations predominate as sources of lead contamination. Drinking water quality contributes in a few cities.

The screening methodology is based upon using existing environmental and demographic information. Consequently, not all desired information was attainable. Several assumptions were made in order to proceed with the study. To test the impact of the assumptions (for example, the use of model default concentrations when measured environmental data were unavailable), a sensitivity analysis was conducted. That analysis indicated that soil and dust concentrations, at higher concentrations, predominated as contributing to higher blood-lead levels. The dust concentration value, however, was unreliable. Nevertheless, the analysis indicated that use of the calculated dust concentration had minimum effect upon the numbers of children calculated to exceed 10 μg/dL Pb-B.

An inability to account for ethnicity and socioeconomic status resulted in an underestimate of the at-risk population in lower socioeconomic minority communities. Nevertheless, the approach is considered to be valid, even though there was only a weak correlation between Pb-B modeled and Pb-B measured, due to the factors discussed. A fundamental factor of the analysis is that the UBK model used to derive modeled blood-lead levels is not, nor was it intended to be, applicable and appropriate for use to discern a blood-lead level for an individual child. The model is only appropriate for estimating the affects on populations of children. That is the use for which the methology uses the UBK model. Consequently, its use in this population comparative risk analysis is thought to be appropriate. Accordingly, the methodology should prove to be useful in identifying "hot spot" areas where there may be sizable numbers of children at higher relative risk to environmental lead exposure. The study approach estimates that

significant numbers of children under the age of seven years are exposed to environmental sources of lead at levels exceeding 10 μg/dL. The population comparative risk number of children is 163,000 in 83 cities in the six states assessed, including 56,000 African-American and 12,000 Hispanic children. The actual numbers exceeding 10 μg/dL cannot be ascertained by the population screening methodology. Additional blood-lead elevations due to lead-based paint exposure is not accounted for in the methodology. Consequently, the calculated numbers are believed to be conservative.

Although soil and dust are the most important determinants of modeled Pb-B levels, there is a paucity of information about the extent of lead-contamination caused by operating and abandoned hazardous waste facilities that could cause such contamination. Generally, off-site soil, dust, and air sampling for lead has not been conducted. Nonetheless, there may be a relatively small number of residents potentially exposed. Except for Granite City, Illinois and Lansing, Michigan, this category of sources does not appear to warrant significant concern for most areas. Extensive sampling, however, around each site, would be required to make a definitive finding. Unless there is strong indication of contamination, however, such sampling is not generally deemed to be prudent or cost effective.

Major air sources are of concern only for residences near emission sources. Municipal waste combusters, as a whole, do not appear to constitute a serious concern. More information is required, however, to make that judgement. Modeling of the air sources did not add value to the methodology, aside from confirming the lack of wide-spread impact.

The ambient air, drinking water supply, and toxic release inventory data were useful in development of the methodology and, for the former two data bases, for calculation of mean blood-lead values.

One would expect to find a stronger correlation for distance from a major highway and the actual blood-lead measurements only if there were a strong correlation for soil concentration and distance from a major highway. That association, although statistically significant, was weak. The conclusion is that,

for the population (recall that high soil-lead values were the criterion for selection of tested children for the blood-lead survey), the distance from a major highway does not correlate with actual blood-lead levels (or soil concentrations). Consequently, other factors (e.g., lead-based paint) appear to contribute more to the elevated Pb-B found, in the survey. It is noted, however, that the average distance from the center of the census tracts to an interstate is greater than one km, and that the maximum distance exceeds five km. Accordingly, most of the children appear to be too far distant to be exposed via the mobile source route. These results are applicable only for the Minneapolis/St. Paul area studied, and can not be generalized to the study area as a whole.

The majority of data analysis should be conducted on a personal computer, in lieu of manipulation using geographic information systems. Data manipulation on the personal computer proceeds with relative ease. The latter computer platform should be utilized to obtain the census bureau data, as well as to map the results of the analysis.

The demographic and housing information was adequate for purposes of developing the methodology and for estimating the spatial and numerical dimensions of minority children at risk for low-level exposure from environmental sources of lead. Precise population and housing estimates for optimal stratification were not available. Nevertheless, that factor was not deemed crucial to the study results, in that estimated and representative environmental exposures were used. Further, the UBK model itself is an approximation. Consequently, for population risk screening purposes, the data were satisfactory.

8. RECOMMENDATIONS

Repeat the derived methodology for other EPA regions and states, or for smaller geographic areas where targeting is desired to rank, prioritize, and better characterize the numbers and extent of at risk minority populations exposed to lead. The derived methodology recognizes the efforts that did not contribute to the screening approach. For example, account for abandoned and operating waste sites, municipal waste combusters, and stationary sources of air emissions spatially and qualitatively, with follow-up if a facility is located within a high percentage exceedance area, as identified via the methodology. It is not worthwhile, however, to include the modeled air-lead concentrations as input to the UBK model.

Include the contribution of lead-based paint to elevated blood-lead levels by using procedures as derived for soil- and dust-lead concentrations, based upon age of housing stock. This would better estimate expected blood-lead values. Calculated values would be based upon better knowledge of the association of lead-based paint contributions to daily intake, with housing age.

Select areas within the top 10 cities with the highest numbers of children at risk, for on-site sampling and investigation, in order to determine the actual extent of residential lead contamination. Develop and implement a public outreach and awareness strategy, pertinent to African-American and Hispanic communities, in particular, but inclusive of any population at high risk of exposure, in selected cities. Work with public health departments to coordinate outreach and education efforts to targeted communities.

Determine if census tract level data are available from the Bureau of the Census, stratified by ethnicity for children under seven years of age, for ethnicity specific birth rates, and housing age categories more relevant to lead usage in residential areas.

Further investigate hazardous wastes sites in Granite City, East St. Louis, Lansing, Michigan, and any city

where a site falls within an area with large numbers of children expected to exceed 10 μg/dL blood-lead. Complete the remedial design work for the NL/Taracorp Corp. site in Granite City, Illinois, pursuant to on-site abatement and replacement of contaminated soil in the 55 square block residential area.

Review major sources with high (relatively) high modeled air values to ensure nearby residents are not exposed to excessive air-lead concentrations. Obtain results of stack test information, when available, for the Chicago, Illinois, North Montgomery County, Ohio, and South Montgomery County, Ohio, municipal waste incinerators, to ensure that lead emissions do not pose an unacceptable risk to local residents.

Ascertain the current drinking water lead concentrations for the Cities of Wausau, Milwaukee, and Madison, Wisconsin, and Youngstown, Ohio, and consider whether additional education or other action is warranted.

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