

*Report on the*

***Analysis of Risk Factors for  
Elevated Levels of Blood Lead  
in New Jersey Children, 2000-2004***

*Demonstration Project on*

*Geographic Patterns of Childhood Blood Lead and  
Environmental Factors in New Jersey*

*Program 03074, Environmental and Health Effects Tracking  
National Center for Environmental Health  
Centers for Disease Control and Prevention (CDC)*

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**New Jersey Department of Health and Senior Services  
Consumer and Environmental Health Services  
Environmental Public Health Tracking Project  
and  
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Childhood Lead Poisoning Prevention Surveillance System**

**In cooperation with the  
New Jersey Department of Environmental Protection**

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## **Overview of the Demonstration Project**

The New Jersey Department of Health and Senior Services (NJDHSS) was awarded funding from the Centers for Disease Control and Prevention (CDC) to conduct three demonstration projects under the program, “Environmental and Health Effects Tracking,” in cooperation with the New Jersey Department of Environmental Protection (NJDEP). The purpose of these demonstration projects was to develop and evaluate methods for linking data contained in ongoing, existing health effects and human exposure surveillance systems with existing data on environmental hazards and exposures. The three projects were designed to describe spatial patterns and time trends in public health data on cancer incidence, birth defect prevalence, and childhood lead exposure, and to link these health outcomes with environmental hazard data.

This report describes a study linking childhood blood lead measurements among children aged 6 to 29 months reported to the NJDHSS in the period 2000 through 2004, with environmental data from the NJDEP on potential lead exposure via lead in air and water.

This demonstration project was conducted by the Environmental Public Health Tracking Project (EPHT) in Consumer and Environmental Health Services, NJDHSS, in partnership with the Childhood Lead Poisoning Prevention Surveillance System (CLPPSS) in NJDHSS and the New Jersey Department of Environmental Protection (NJDEP).

## **Study Team**

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## Summary

This report describes a study linking childhood blood lead measurements among children aged 6 to 29 months reported to the New Jersey Department of Health and Senior Services in the period 2000 through 2004, with environmental data from the New Jersey Department of Environmental Protection on potential lead exposure via lead in air and drinking water. The dataset used for this analysis contained 326,047 unique children (89.2% of original dataset). Cases missing sampling date, age at screening, or whose residential address could not be geocoded to a latitude and longitude were excluded from analysis.

Each child was assigned a value for ambient air lead exposure based on the modeled 1999 USEPA NATA estimates of lead concentration for the census tract in which the child's residential address was located. Each child was assigned values for drinking water lead concentration based on the 90<sup>th</sup> percentile and the median lead value for the drinking water system in which the child's residence was located, for the year in which the child's blood lead specimen was collected.

The logistic regression modeling found that several variables are associated strongly with increased risk of elevated blood lead in children. Within the 6-29 month age window, increasing age was associated with elevated blood lead levels. Children screened in the summer and autumn were more likely to have an elevated blood lead test than in those screened in the winter or spring months. These results were consistent with the findings reported from the descriptive analysis of childhood blood lead levels in New Jersey, reported separately.

Several census tract-based variables were also strongly associated with childhood blood lead. The results of this multivariate model are generally consistent with known relationships between elevated blood lead levels and poverty, race and age of housing stock of the census tract in which a child lives. Each of these factors, adjusted for each other, show strong monotonic response relationships with elevated blood lead levels.

Drinking water lead was found to be positively associated with the risk of elevated childhood blood lead. In an attempt to examine whether this observed association is a realistic outcome from lead exposure at the magnitude contributed from drinking water, the study team used the USEPA's Integrated Exposure Uptake Biokinetic (IEUBK) model to predict changes in blood lead distributions at varying drinking water levels, for children aged 6-29 months. The modeled change in risk of elevated blood lead as water lead increases is consistent with the results of the logistic regression analysis.

The findings of this demonstration project should be considered suggestive, and not necessarily causal. Exposure misclassification in linkage studies, as with any epidemiologic study, is an important consideration. There are many sources of error in using the NATA or drinking water data for human exposure assessment. While this epidemiologic study found an association between elevated individually measured childhood blood lead and drinking water system lead estimates, after controlling for

census tract-level demographic, economic and housing characteristics, these findings may be due to alternate explanations. While exposure to lead paint will continue to be the primary source of acute lead poisoning in New Jersey's youngest residents, it is an important public health goal to reduce or eliminate all preventable chronic lead exposures that impact young children.

## Introduction

Exposure to the heavy metal lead may result in harmful effects on the kidneys and nervous system. Effects on the nervous system are particularly serious and can cause learning disabilities, hyperactivity, decreased hearing, mental retardation and possibly death. Lead exposure is particularly hazardous to children less than six years of age, and may cause intellectual and behavioral deficits (ATSDR, 1999).

While blood lead levels in the U.S. continue to decline, 2% of U.S. children less than age 1 year and 4% of children 1 to 5 years are estimated to have blood lead levels  $\geq 10 \mu\text{g/dL}$ , which is the level that CDC considers elevated (CDC, 2005). Nearly 3% of New Jersey children age 6 to 29 months are estimated to have blood lead levels  $\geq 10 \mu\text{g/dL}$  (NJDHSS, 2004).

Lead exposure may result from ingestion or inhalation. Over time, the most common sources of lead exposure have been lead-based paint, leaded gasoline, occupation or hobby, tap water, food stored in lead soldered cans or improperly glazed pottery, and traditional folk remedies and cosmetics containing lead. High exposures in children are typically associated with ingestion of lead paint chips or dusts in housing stock built prior to 1978, and particularly in houses built before 1950 (Clickner et. al, 1995; MMWR, 2003).

**Lead Paint in Older Housing:** Nearly one million housing units in New Jersey (30.2% of the housing units in the state) were built before 1950. Each of New Jersey's 21 counties has more than 9,000 housing units built before 1950. Housing stock, therefore, remains a significant source of lead exposure for New Jersey children. In addition to this important source, however, there is concern about the role of other sources of lead in contributing to the body burdens of lead in New Jersey's children. This data linkage demonstration study examines the role of lead in ambient air and lead in drinking water, while also attempting to account for well-established risk factors such as the age of housing stock.

**Lead in Ambient Air:** Historically, ambient air was a substantial contributor to lead exposure, primarily from the combustion of leaded gasoline. Since the phased removal of lead from gasoline beginning in the 1970s, the amount of lead in ambient air has dropped dramatically. Along with that decrease was a significant drop in average blood lead levels in the population (ATSDR, 1999). While exposure to lead in older residences with peeling lead-based paint has long been established as the cause of the majority of acute lead poisoning in children, the role of other sources of lead – drinking water, diet, proximity to lead-emitting air pollution sources – has also been recognized (ATSDR, 1999). The relative contribution of such sources to lead body burden is likely to vary geographically depending on the mixture of possible environmental lead exposure.

As measured by the ambient air monitoring network maintained by the New Jersey Department of Environmental Protection (NJDEP), lead levels in outdoor air have continued to decrease over the past decade. However, a substantial amount of lead is still

used in industry. In 2003, for example, 149 of New Jersey's largest manufacturers reported using 84,922,057 pounds of lead (NJDEP, 2003). These facilities reported the release of approximately 19,000 pounds of lead into the air in 2003. In 2002, New Jersey's releases of lead by industrial category were highest for energy generating facilities (76%), foundries (12%), and refuse and sewerage systems (8%) (NJDEP, 2002).

The U.S. Environmental Protection Agency (USEPA) developed the National-Scale Air Toxics Assessment (NATA) based on a complex modeling of 1996 facility emissions, mobile sources, and area sources of air pollutants. Annual average air toxics levels were estimated for many chemicals, including lead, at the census tract level (using 1990 census tract boundaries) across the U.S. According to the 1996 NATA, no New Jersey census tracts exceeded the National Ambient Air Quality Standard (NAAQS) of 1.5 micrograms per cubic meter ( $\mu\text{g}/\text{m}^3$ ). However, the NJDEP has established a 24-hour reference concentration of 0.1  $\mu\text{g}/\text{m}^3$  that is more protective than the NAAQS. This level is used in the air permitting program to assess potential risks posed by releases of lead into the air. It was established using USEPA's Integrated Exposure Uptake Biokinetic (IEUBK) model for lead in children under age seven who are exposed to environmental lead.

**Lead in Drinking Water:** An additional exposure source examined in this linkage demonstration study was lead in drinking water. Approximately 90 percent of New Jersey's population is served by public water supplies for which water quality data are collected routinely and systematically. The federal Lead and Copper Rule<sup>1</sup> specifies the requirements for lead in community drinking water systems, including lead levels in drinking water and monitoring requirements. The rule establishes an Action Level of 0.015 mg/l based on the 90th percentile value of all tap water samples taken. The number of samples taken is based on the size of the system and varies from 5 samples for systems serving less than 100 residents to 100 samples for large systems serving more than 100,000 residents. First draw samples must be collected at cold water taps in homes that are at highest risk of lead and copper contamination as specified in the federal Safe Drinking Water Regulations. Systems must monitor lead levels every 6 months, unless they qualify for reduced monitoring based on prior sampling results showing that concentrations are below the Action Level. A regulatory violation occurs when more than 10% of samples exceed the action level of 15 micrograms per liter ( $\mu\text{g}/\text{L}$ ).

## Methods

These analyses have been conducted to allow NJDHSS and NJDEP to begin to evaluate the relationships between childhood blood lead levels in New Jersey children and estimates of environmental exposure to lead in air and drinking water. Blood lead levels of New Jersey children were linked with environmental databases to examine whether environmental exposures, in addition to lead-based paint in older housing stock, appear to contribute to the lead body burden of New Jersey's children.

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<sup>1</sup> 56 FR 26460 – 26564 June 7, 1991, 40 CFR 141.80 – 141.90



### ***Study Population***

The study population consisted of all New Jersey children aged 6 months through 29 months at time of specimen collection, with at least one blood lead measurement reported to the NJDHSS in the period 2000 to 2004.

### ***Database Preparation***

The Childhood Lead Poisoning Prevention Surveillance System (CLPPSS) in NJDHSS prepared a dataset specifically for this EPHT demonstration project. This dataset was de-duplicated such that it contained only one record per individual child. De-duplication was based on the child's name and date of birth. For individuals with more than one record in the period, the record with the highest blood lead measurement for that child was included, and all others were excluded.

The CLPPSS transferred the de-duplicated study dataset to the CEHS study team staff via CD. The dataset contained blood lead measurements in micrograms of lead per deciliter of blood ( $\mu\text{g}/\text{dL}$ ) and limited demographic information for the New Jersey resident children. Each record contained the following variables: street address; city; ZIP code; municipality code; county code; census tract suffix code; census tract code; latitude; longitude; blood lead measurement ( $\mu\text{g}/\text{dL}$ ); date of birth; age in months; age in years; date of sample; date of analysis; gender; and race code. Latitude and longitude were coded to the ZIP+4 centroid. As a result of the de-duplication process, the child's residential address was the one at the time of the highest blood lead measurement.

Initially, 365,524 de-duplicated records were provided by the CLPPSS to the CEHS for the 5-year period. Cases without a known sampling date, missing demographic information, or those with addresses that could not be geocoded to a latitude and longitude were excluded from further analysis. The final dataset used for linkage analyses contained 326,047 children (89.2%).

### ***Exposure Assessment***

#### **Lead in Ambient Air**

Each child was assigned a value for ambient air lead exposure based on the modeled 1999 USEPA NATA estimates of lead concentration for the census tract in which the child's residential address was located. NATA is a state-of-the-science national-scale screening tool intended to help regulatory agencies and communities to assess air toxics priorities. The assessment includes four steps: 1) compiling a national emissions inventory of air toxics emissions from outdoor sources, 2) estimating ambient concentrations of air toxics across the United States, 3) estimating population exposures across the United States, and 4) characterizing potential public health risk due to inhalation of air toxics including both cancer and non-cancer effects.

Estimates of annual average ambient air concentrations of 177 air toxics are predicted for each census tract based on census tract shapes used in the 2000 U.S. Census. NATA estimates are derived from an annual emissions inventory of major and other point sources, estimates of area and mobile source contributions, and “background” concentrations. Non-road mobile sources, which include airplanes, trains, construction vehicles and others not found on roads, are estimated to contribute the most to lead air exposure, accounting for approximately 56 % of the lead in higher exposure areas. Major stationary air sources, including chemical facilities, battery manufacturers and electric generating units are also significant sources, accounting for approximately 26 % of the lead in high exposure areas. The USEPA used the Assessment System for Population Exposure Nationwide (ASPEN) dispersion model to estimate ambient air concentrations from these emissions.

### **Lead in Drinking Water**

Mandatory testing for lead in residential tap samples taken from homes identified as high risk for lead and copper contamination has been conducted by all community water systems (CWS) in New Jersey since 1993. A CWS is defined as a system with  $\geq 15$  service connections or  $\geq 25$  persons in residence more than 6 months of the year. The NJDEP Bureau of Safe Drinking Water provided the NJ EPHT project with a database consisting of over 52,000 records of test results from CWSs over the interval 1998 through 2004. Data were reviewed by the EPHT team to identify quality assurance issues. Several unrealistically high results were found in the database. All 142 results exceeding 0.5 mg/L (more than three standard deviations higher than the mean) were deleted, since it was felt that lead levels of this magnitude are unlikely from valid tap samples. Some (67) of the unrealistically high lead data were found to be copper data misreported to the NJDEP as lead, and reasons for the other values are not known.

For each CWS, the NJDEP and NJDHSS computed median and 90<sup>th</sup> percentile drinking water lead from among all remaining lead data collected during each calendar year in the period. For years with no lead testing data, values were extrapolated or interpolated in order to have water lead concentration estimates for each year. For years when no samples were required, data from the preceding year were used. If no data were available for a water system prior to 2004, all previous years were assigned the 2004 water lead concentration. These values were also related to a geographic information system (GIS) map of water system boundaries in the state.

Each child was assigned values for drinking water lead concentration based on the 90<sup>th</sup> percentile and the median lead value for the drinking water system in which the child’s residence was located, for the year in which the child’s blood lead specimen was collected.

A key hurdle in connecting the blood lead data to the drinking water data is to be able to link each child’s address to the water system serving the residence. The development of a water systems boundary map was initiated by NJDEP in 1998 and

revisions were completed by NJDHSS in 2006 for the purposes of this NJ EPHT demonstration project. Boundary data were developed using:

- direct outreach to CWSs
- review of select systems by NJDEP/Bureau of Safe Drinking Water staff
- a prior GIS map prepared by NJDHSS representing system boundaries in the 1980s (Cohn et al., 1998)
- a NJDEP database on system sources and infrastructure
- hard copy commercial maps of counties (especially for mobile home parks)
- aerial photographs (especially for mobile home parks)

One of the critical challenges in mapping water system boundaries is differentiating between franchise areas, which represent current and future expansion, and actual physical service areas. In certain instances the population serviced, as reported by the CWS, was compared to 2000 U.S. Census block group populations in the immediate area in order to determine whether the mapped extent of the system was of the appropriate size. Further confirmatory information was then obtained from the DEP, the water utility or the local health department.

### **Housing Stock and Other Variables**

Since the age of house was not available for each individual child, the proportion of houses built before 1960 in the census tract in which the child resided was used as a surrogate. The information was available from the 2000 U.S. Census. Similarly, the following U.S. Census 2000 variables were assigned on the basis of the census tract of residence: percentage of people living in poverty; percent of population that is White; and percent of population that is Hispanic.

### ***Data Analysis***

The CEHS study team constructed an analytical data set in which the individual child was the unit of analysis. A “case” was defined four ways for these analyses. A child was a case if the blood lead level exceeded the following cut-points: 8 µg/dL; 10 µg/dL; 15 µg/dL; or 20 µg/dL). All other children were considered “controls” in each analysis.

Blood lead exceedence of each cut-point was analyzed separately using the logistic regression model. Exposure variables of interest were the air and drinking water exposure metrics described above. Other independent variables were age of the child at specimen collection (in months), season of sample collection, age of housing stock in the child’s census tract (i.e., percent of homes built before 1960), percent of the population in the child’s census tract living in poverty, percent of population in the child’s census tract that was White, and percent of the population in the child’s census tract that was Hispanic.

Water lead measurements using the 90<sup>th</sup> percentile results were categorized by quartile (0-0.004 mg/l; 0.004-0.008 mg/l; 0.008-0.011 mg/l and >0.011 mg/l). Children residing in areas not served by public water systems were excluded from analyses in which water lead was included in the model. In addition, water lead was categorized by equal interval (0-0.003 mg/l; 0.003-0.006 mg/l; 0.006-0.009 mg/l; 0.009-0.012 mg/l; 0.012-0.015 mg/l and > 0.015 mg/l). It should be noted that the action level for lead in drinking water is 0.015 mg/l. NATA air lead data for New Jersey were categorized into tertiles (0-0.0035 µg/m<sup>3</sup>; 0.0035-0.0069 µg/m<sup>3</sup> and >0.0069 µg/m<sup>3</sup>).

Child's age was categorized as follows: < 9 months, 9-15 months, 16-20 months, 21-27 months, and > 27 months. Seasons were defined as Winter (December through February), Spring (March through May), Summer (June through August) and Autumn (September through November). U.S. Census variable data were categorized into quintiles: percent of population living in poverty (0-2.7%; 2.7-4.3%; 4.3-7.9%; 7.9-17.4%; >17.4%); percent White population (0-43%; 43-72%; 72-85%; 85-93%; >93%); percent Hispanic population (0-3%; 3-5%; 5-9%; 9-28%; >28%); and percent housing in each census tract for homes constructed prior to 1960 was categorized into quintiles (0-23%; 23-46%; 46-63%; 63-75%; >75%).

Epidemiologic analyses was conducted using Stata and SPSS statistical software. Mapping of data was conducted using Geographical Information System software (ArcView 9).

### ***Protection of Human Subjects***

The study protocol developed for this demonstration study was reviewed and approved under the NJDHSS and University of Medicine & Dentistry of New Jersey--Newark Campus (UMDNJ) Institutional Review Board Authorization Agreement, whereby the UMDNJ-Newark Campus' Institutional Review Board (IRB) serves as the NJDHSS's IRB.

The NJDHSS has long-standing mechanisms in place to safeguard the use of confidential health data by qualified NJDHSS scientists and researchers. All potentially identifying childhood lead information obtained from the CLPPSS is kept confidential by CEHS. Although no names were obtained by CEHS staff from the Childhood Lead Poisoning Prevention Program, address information is treated as strictly confidential. Data sets with confidential data elements were transferred between CLPPSS and CEHS by hand-carrying of password-protected compact discs. All computerized data are kept by CEHS in password-protected files on a secure local area network (LAN). Paper files at CEHS are kept in locked file cabinets which can only be accessed by authorized CEHS personnel who have a need to access the files.

## Results

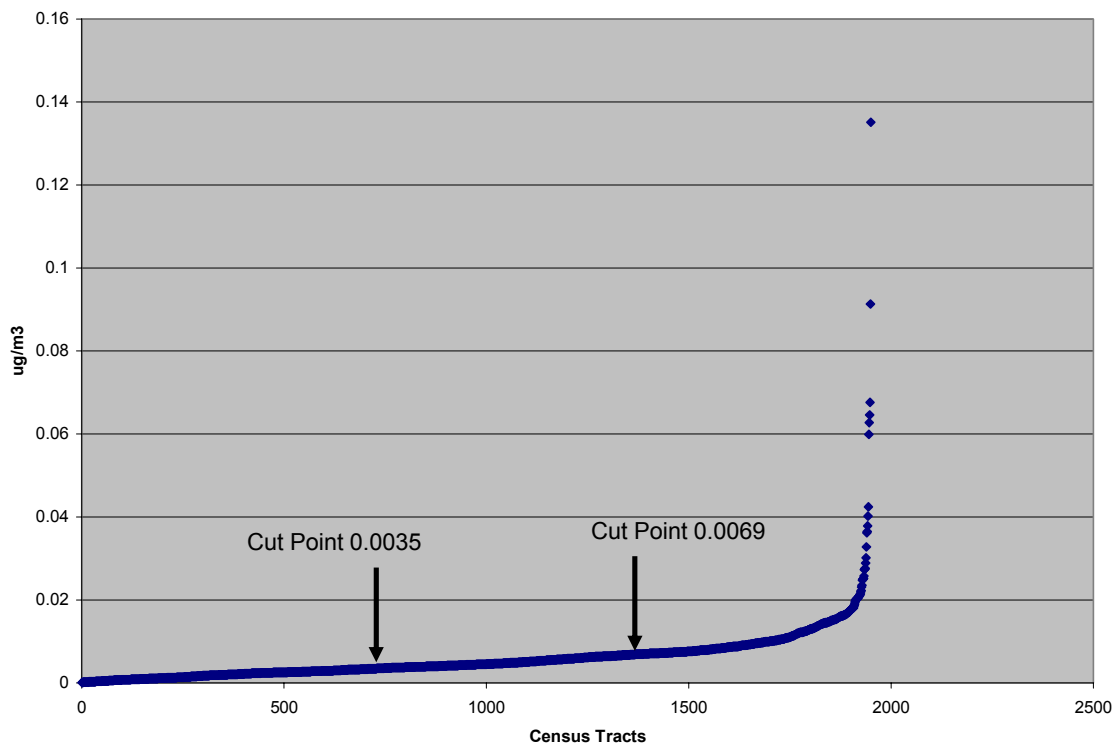
### *NATA Estimates of Lead in Ambient Air*

Descriptive statistics summarizing the NATA estimates for annual average ambient air lead concentrations among the 1,944 census tracts are presented in Table 1. The cumulative frequency distribution is plotted in Figure 1, and the NATA estimates are mapped in Figure 2.

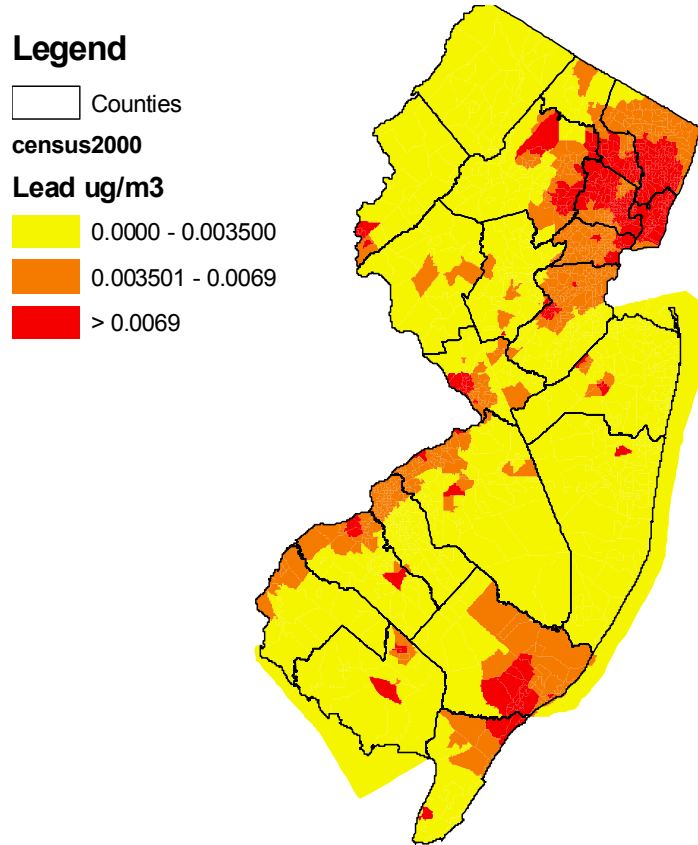
**Table 1. Descriptive statistics for 1999 NATA census tract estimates for lead in ambient air.**

Descriptive Measure	Lead Concentration ( $\mu\text{g}/\text{m}^3$ )
Mean (Std. Dev.)	0.0059 (0.0064)
Median	0.0044
Minimum	0.000075
Maximum	0.135

**Figure 1. Cumulative frequency distribution for 1999 NATA lead estimates for New Jersey census tracts.**



**Figure 2. Map of 1999 NATA estimates for annual average concentrations of lead in ambient air.**

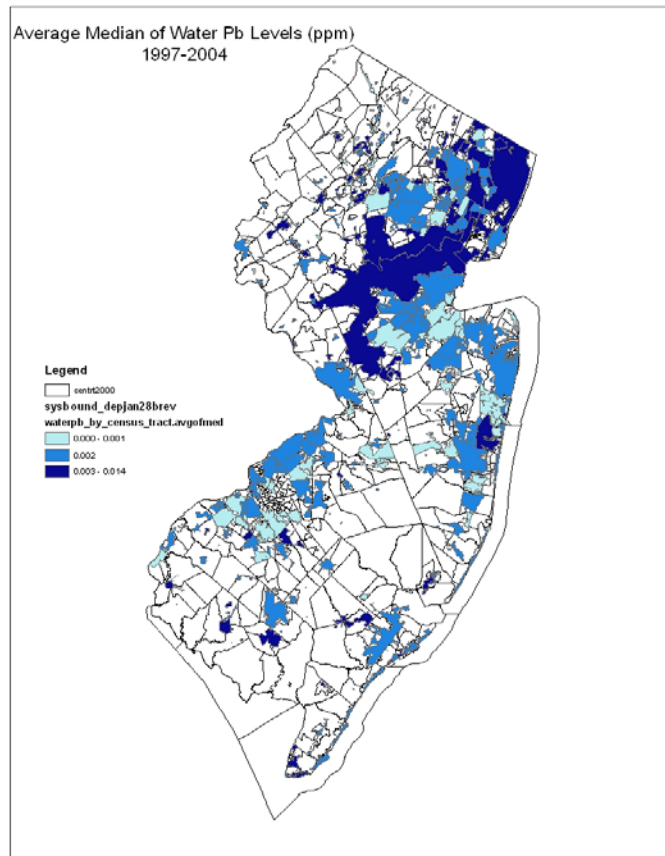


No census tract exceeded the NAAQS of  $1.5 \mu\text{g}/\text{m}^3$ , although one tract exceeded the reference concentration of  $0.1 \mu\text{g}/\text{m}^3$  used by NJDEP. The maximum lead concentration was estimated to be  $0.135 \mu\text{g}/\text{m}^3$ . The distribution is skewed, with few census tracts having estimated concentrations above approximately  $0.015 \mu\text{g}/\text{m}^3$ . Most of the elevated ambient lead levels were found in the northeast region of the state (Essex, Hudson, Bergen, Union, Passaic and Morris counties), with scattered isolated higher estimates located throughout many part of the state.

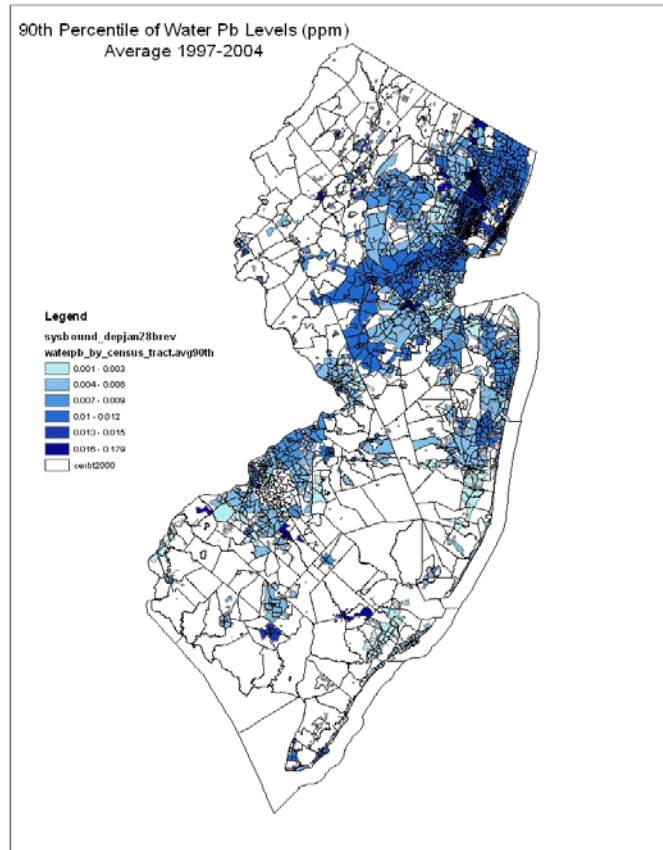
### *Drinking Water Lead Estimates*

The average annual median and 90<sup>th</sup> percentile drinking water lead levels in the period 1997-2004 are mapped in Figures 3 and 4, according to CWS boundaries in New Jersey. It can be seen that the highest levels of water lead are in the northeast and central regions of the state, but that areas of high lead are found throughout the state.

**Figure 3. Average Median Drinking Water Lead, 1997-2004.**



**Figure 4. Average 90<sup>th</sup> Percentile Drinking Water Lead, 1997-2004.**

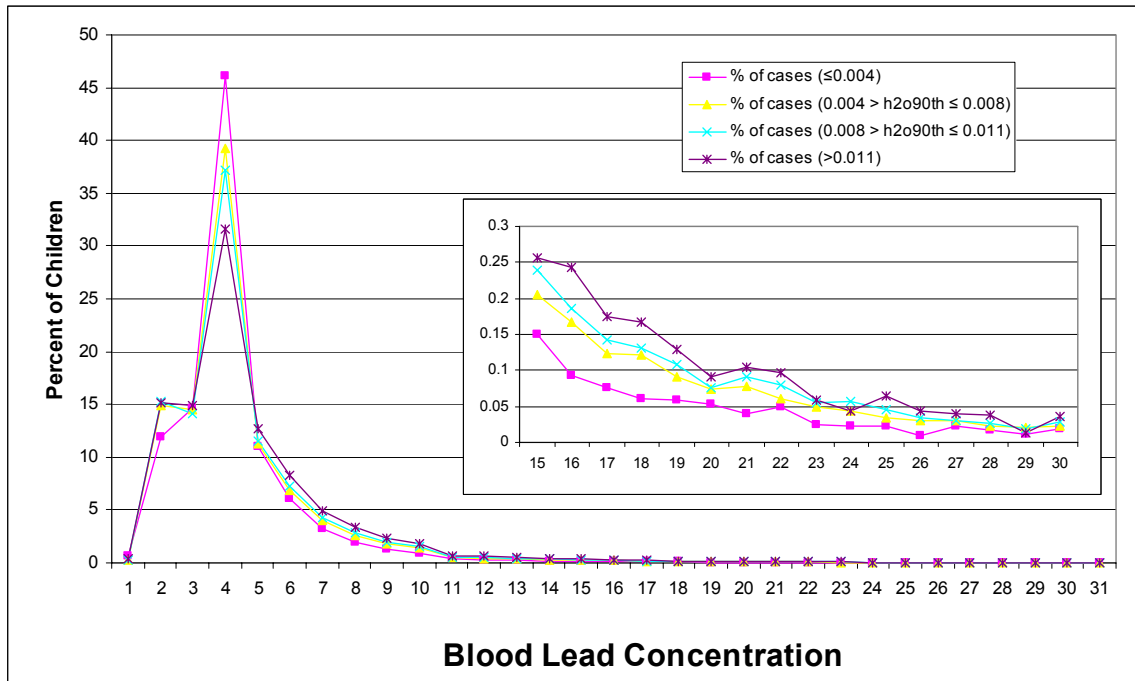


***Distribution of Blood Lead Levels by Drinking Water Lead Concentration***

Figure 5 shows the distribution of children’s blood lead levels by quartile of 90<sup>th</sup> percentile water system lead concentration. The inset magnifies the portion of the distribution above 15  $\mu\text{g}/\text{dl}$ . The general shapes of the distributions were similar in all exposure categories. The mode of each distribution is 4  $\mu\text{g}/\text{dl}$ , with a marked tail toward higher values. However, there was a higher percentage of children with higher blood lead levels among children in the higher water lead exposure categories.



**Figure 5. Distribution of blood lead levels by quartiles of 90<sup>th</sup> percentile water system lead concentration. Inset magnifies portion of distribution above 15 µg/dL.**



### ***Logistic Regression***

Four multivariate logistic regression models are presented in Table 2, for each of the four case definitions ( $\geq 8$  µg/dL;  $\geq 10$  µg/dL;  $\geq 15$  µg/dL; or  $\geq 20$  µg/dL). Each model contains the same set of independent variables; child’s age, season, 90<sup>th</sup> percentile water lead, ambient air lead, percent of population living in poverty, percent White population, percent Hispanic population, and percent of homes built before 1960. Results from the four models are generally similar. Therefore, the remaining discussion will focus on the model in which “case” is defined as having a blood lead level  $\geq 10$  µg/dL.

**Ambient Air Lead:** Children living in areas with higher ambient air concentrations of lead, as estimated by NATA, had a lower risk of having a blood lead level above 10 µg/dL.

**Drinking Water Lead:** Only the model with water lead categorized by quartile is presented here. Results of the model using water lead categorized by equal interval were similar. Children living in areas served by community water systems with 90<sup>th</sup> percentile drinking water lead concentrations above 0.011 mg/l were about 1.3 times as likely to have an elevated blood lead level (Table 2). The adjusted risk of elevated blood lead increased with increasing drinking water lead concentration.

**Table 2. Logistic Regression Final Model**

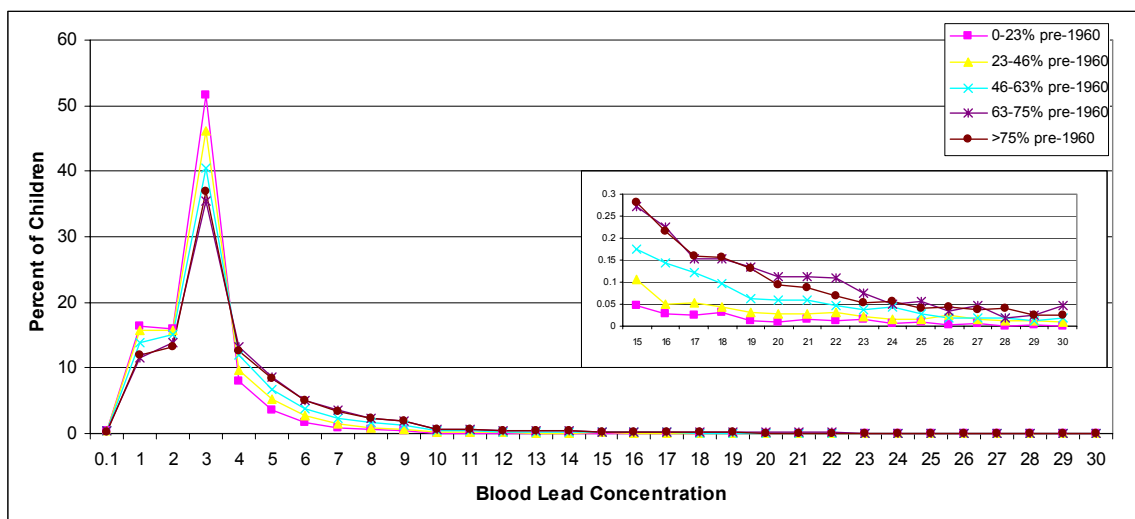
MODEL INPUT VARIABLE	CATEGORIES	ODDS RATIO BPb≥20			ODDS RATIO BPb≥15			ODDS RATIO BPb≥10			ODDS RATIO BPb≥8		
		C.I.	Sig @ 0.05		C.I.	Sig @ 0.05		C.I.	Sig @ 0.05		C.I.	Sig @ 0.05	
Child's Age (months)	<9	0.36	0.23, 0.56	<0.0001	0.36	0.27, 0.48	<0.0001	0.36	0.29, 0.43	<0.0001	0.41	0.36, 0.47	<0.0001
	9to15	1			1			1			1		
	16to20	2.40	2.08, 2.76	<0.0001	2.44	2.21, 2.68	<0.0001	2.28	2.14, 2.43	<0.0001	2.14	2.05, 2.25	<0.0001
	21to27	2.62	2.32, 2.95	<0.0001	2.54	2.34, 2.76	<0.0001	2.51	2.37, 2.65	<0.0001	2.37	2.28, 2.46	<0.0001
	>27	3.80	3.16, 4.56	<0.0001	3.07	2.68, 3.52	<0.0001	2.82	2.57, 3.1	<0.0001	2.55	2.37, 2.73	<0.0001
Season	Winter	1			1			1			1		
	Spring	1.06	0.89, 1.26	0.5174	0.99	0.88, 1.12	0.8665	1.01	0.93, 1.09	0.8862	0.97	0.92, 1.02	0.2776
	Summer	2.15	1.85, 2.5	<0.0001	2.03	1.83, 2.25	<0.0001	1.74	1.62, 1.86	<0.0001	1.50	1.43, 1.58	<0.0001
	Autumn	1.45	1.24, 1.71	<0.0001	1.51	1.35, 1.68	<0.0001	1.53	1.43, 1.64	<0.0001	1.43	1.36, 1.51	<0.0001
Water Lead 90th Percentile (mg/l)	0to0.004	1			1			1			1		
	0.004to0.008	1.17	1, 1.36	0.0484	1.17	1.05, 1.3	0.0040	1.13	1.06, 1.21	0.0003	1.10	1.05, 1.16	0.0001
	0.008to0.011	1.31	1.13, 1.53	0.0004	1.27	1.14, 1.41	<0.0001	1.16	1.08, 1.24	<0.0001	1.15	1.1, 1.21	<0.0001
	>0.011	1.35	1.15, 1.58	0.0002	1.35	1.21, 1.51	<0.0001	1.29	1.2, 1.39	<0.0001	1.28	1.22, 1.35	<0.0001
Lead in ambient air (ug.m3)	0to0.0035	1			1			1			1		
	0.0035to0.0069	0.95	0.79, 1.14	0.5706	0.86	0.76, 0.97	0.0180	0.85	0.78, 0.92	0.0001	0.89	0.84, 0.94	0.0001
	>0.0069	0.92	0.77, 1.1	0.3596	0.81	0.71, 0.91	0.0006	0.79	0.73, 0.85	<0.0001	0.82	0.78, 0.87	<0.0001
Percent of pop. living in poverty	0to2.7	1			1			1			1		
	2.7to4.3	1.05	0.79, 1.4	0.7433	1.22	1, 1.49	0.0533	1.24	1.09, 1.4	0.0012	1.25	1.14, 1.36	<0.0001
	4.3to7.9	1.56	1.19, 2.04	0.0014	1.73	1.42, 2.09	<0.0001	1.70	1.5, 1.92	<0.0001	1.70	1.57, 1.85	<0.0001
	7.9to17.4	2.47	1.88, 3.25	<0.0001	2.62	2.16, 3.18	<0.0001	2.60	2.3, 2.95	<0.0001	2.54	2.33, 2.77	<0.0001
	>17.4	3.32	2.5, 4.42	<0.0001	3.77	3.08, 4.62	<0.0001	3.79	3.32, 4.32	<0.0001	3.66	3.34, 4.01	<0.0001
Percent of pop. that is White	0to43	3.95	2.93, 5.34	<0.0001	4.17	3.37, 5.16	<0.0001	3.38	2.96, 3.86	<0.0001	3.13	2.86, 3.43	<0.0001
	43to72	2.38	1.76, 3.22	<0.0001	2.45	1.98, 3.03	<0.0001	1.96	1.72, 2.24	<0.0001	1.85	1.69, 2.03	<0.0001
	72to85	1.69	1.26, 2.25	0.0004	1.74	1.42, 2.14	<0.0001	1.37	1.2, 1.55	<0.0001	1.34	1.23, 1.46	<0.0001
	85to93	1.20	0.89, 1.62	0.2410	1.37	1.11, 1.69	0.0033	1.16	1.02, 1.31	0.0280	1.16	1.07, 1.27	0.0007
	>93	1			1			1			1		
Percent of pop. that is Hispanic	0to3	2.19	1.75, 2.75	<0.0001	1.99	1.69, 2.33	<0.0001	1.87	1.65, 2.04	<0.0001	1.72	1.59, 1.86	<0.0001
	3to5	1.82	1.51, 2.18	<0.0001	1.72	1.51, 1.96	<0.0001	1.69	1.55, 1.85	<0.0001	1.67	1.56, 1.78	<0.0001
	5to9	1.78	1.52, 2.08	<0.0001	1.64	1.46, 1.83	<0.0001	1.49	1.38, 1.61	<0.0001	1.44	1.36, 1.52	<0.0001
	9to28	1.23	1.07, 1.42	0.0041	1.25	1.14, 1.38	<0.0001	1.21	1.14, 1.3	<0.0001	1.24	1.18, 1.3	<0.0001
	>28	1			1			1			1		
Percent of homes built prior to 1960	0to23	1			1			1			1		
	23to46	2.26	1.66, 3.08	<0.0001	2.08	1.68, 2.59	<0.0001	1.94	1.68, 2.24	<0.0001	1.69	1.54, 1.85	<0.0001
	46to63	2.48	1.83, 3.36	<0.0001	2.64	2.1, 3.25	<0.0001	2.95	2.58, 3.38	<0.0001	2.48	2.27, 2.71	<0.0001
	63to75	3.71	2.76, 5	<0.0001	3.59	2.92, 4.4	<0.0001	3.67	3.21, 4.2	<0.0001	3.07	2.81, 3.35	<0.0001
	>75	3.49	2.6, 4.69	<0.0001	3.78	3.08, 4.63	<0.0001	4.30	3.75, 4.9	<0.0001	3.52	3.23, 3.85	<0.0001

**Child’s Age, and Season of Year:** Table 2 shows that elevated childhood blood lead level was associated with child’s age at testing and season of testing. Children older than 27 months of age were 2.8 times as likely to have a blood lead concentration  $\geq 10$   $\mu\text{g/dL}$  than children 9-15 months old. There was an apparent monotonic increase in risk with increasing age in months. Children screened in summer were 1.7 times more likely to have a blood lead test  $\geq 10$   $\mu\text{g/dL}$ , compared to winter. These findings are consistent with those found in the descriptive analysis of childhood blood lead reported separately from this demonstration project.

**Poverty, Race and Ethnicity in Census Tract:** Living in poverty was associated with an increased risk of elevated blood lead, with risk increasing with increasing poverty. Children living in a census tract in the highest quintile of percent poverty ( $> 17.4\%$ ) were 3.8 times more likely to have a blood lead level  $\geq 10$   $\mu\text{g/dL}$ . Living in a census tract with a lower proportion of White population was monotonically related to an increase in risk of having a blood lead level  $\geq 10$   $\mu\text{g/dL}$ . Children living in a census tract with a 0-43% White population were 3.4 times more likely to have elevated blood lead compared to children living in a tract with  $> 93\%$  White population. Similarly, children living in a region with a low proportion of Hispanic population were at higher risk of an elevated blood lead concentrations when compared to regions with a larger Hispanic population.

**Age of Housing in Census Tract:** Living in an area with a large percentage of older homes built prior to 1960 increased the child’s risk of having an elevated blood lead measurements (Figure 6 shows the distribution of blood lead levels by census tract age of housing category). From the regression analyses (Table 2), children living in a census tract with  $> 75$  percent of homes built before 1960 were 4.3 more likely to have a blood lead level  $\geq 10$   $\mu\text{g/dL}$ , compared to children living in a census tract where 0-23 percent of the homes were built before 1960.

**Figure 6. Distribution of Blood Lead Level by Quintile of Age of Housing in Census Tract. Inset magnifies portion of distribution above 15  $\mu\text{g/dl}$ .**



## Discussion

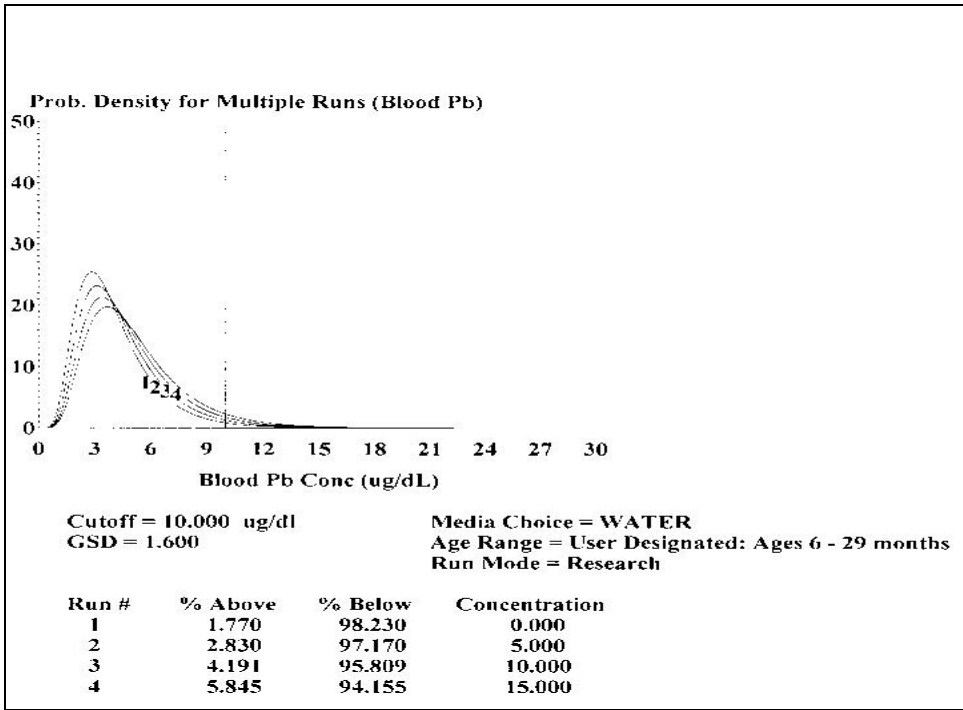
### *Interpretation of Findings*

The logistic regression modeling found that several variables are associated strongly with increased risk of elevated blood lead in children. Within the 6-29 month age window, increasing age was associated with elevated blood lead levels. Children screened in the summer and autumn were more likely to have an elevated blood lead test than in those screened in the winter or spring months. These results were consistent with the findings reported from the descriptive analysis of childhood blood lead levels in New Jersey, reported separately.

Several census tract-based variables were also strongly associated with elevated childhood blood lead. The results of this multivariate model are generally consistent with known relationships between elevated blood lead levels and poverty, race and age of housing stock of the census tract in which a child lives. Each of these factors, adjusted for each other, show strong monotonic response relationships with elevated blood lead levels.

Drinking water lead was found to be positively associated with the risk of elevated childhood blood lead. In an attempt to examine whether this observed association is a realistic outcome from lead exposure at the magnitude contributed from drinking water, the study team used the USEPA's Integrated Exposure Uptake Biokinetic (IEUBK) model to predict changes in blood lead distributions at varying drinking water levels, for children aged 6-29 months. The model used default parameters but varied the water concentration at 0, 0.005, 0.010, and 0.015 mg/l. The result from these models is shown in Figure 7. The risk of blood lead  $\geq 10$   $\mu\text{g}/\text{dl}$  was 5.8% for water lead at 0.015 mg/L, and 1.8% for water lead at 0 mg/L. The modeled change in risk of elevated blood lead as water lead increases is consistent with the results of the logistic regression analysis.

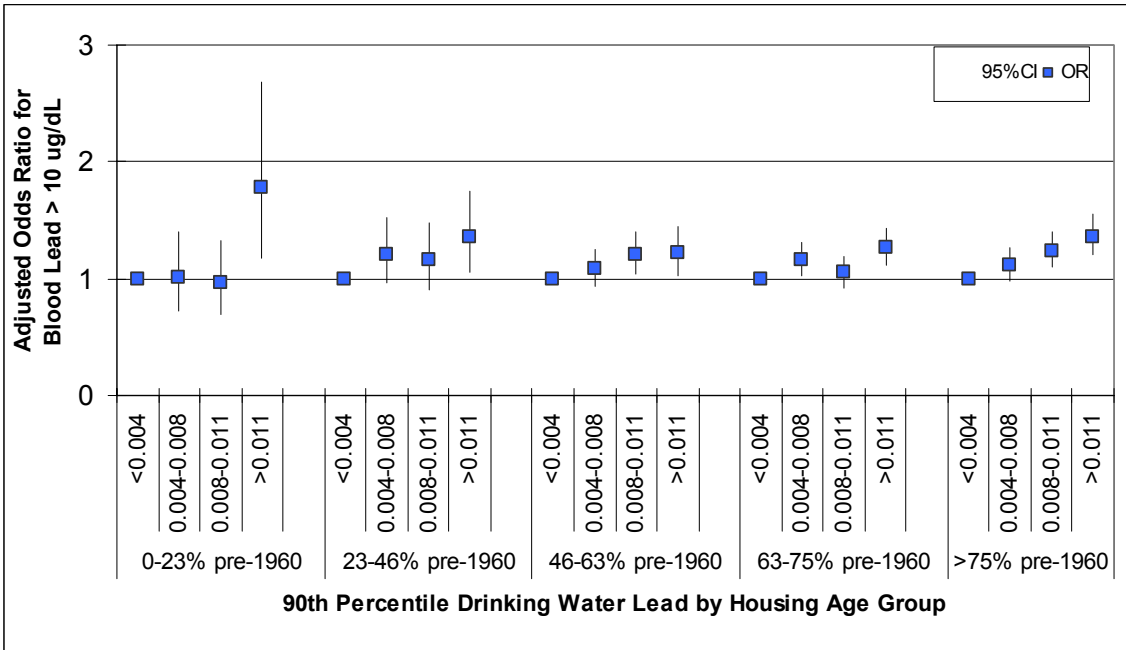
**Figure 7. Results of IEUBK model at Varying Drinking Water Lead Concentrations**



There are alternative explanations for the association between elevated childhood blood lead and drinking water lead. First, children with elevated blood lead levels may live in areas where drinking water lead is systematically overestimated compared to other children. The regulatory requirement for drinking water sampling for lead is to target housing at risk of having high water lead due to known lead service lines or plumbing. Thus, some water system lead estimates are based on “worst case” rather than “typical” exposure scenarios. It is possible that areas in which children are at risk of elevated blood lead due to deteriorated older housing are more likely to be served by water systems that have lead estimates based on “worst case” exposures. This differential misclassification would result in a spurious association between water system drinking water lead and risk of elevated blood lead.

A second alternative explanation is that confounding by age of housing is incompletely controlled in the analysis, since it is an ecological-level measure based on the census tract in which the child resides, rather than the actual age of the child’s house. An analysis of the relationship between drinking water lead and risk of elevated blood lead, stratified by age of housing category, is shown in Figure 8. Across all age of housing strata, there is a consistent exposure-response between drinking water lead and risk of elevated blood lead, suggesting that the relationship may be real.

**Figure 8. Odds Ratios for Elevated Blood Lead by Water Lead Concentration, Stratified by Age of Housing in Census Tract.**



No positive association was found between childhood blood level and ambient air lead levels, as estimated by USEPA from the 1999 NATA. In fact, risk of elevated blood lead was lower in areas estimated to have higher air lead concentrations. This may not be surprising, since the estimated levels of lead in air are low, and would not be expected to contribute substantively to overall lead exposure. Using the USEPA’s Integrated Exposure Uptake and Biokinetic (IEUBK) pharmacokinetic model, the EPHT project estimates exposure at the maximum modeled air lead level from NATA ( $0.135 \mu\text{g}/\text{m}^3$ ) would not result in an increase in the proportion of children with elevated blood lead levels, in comparison to an air lead concentration of  $0.0 \mu\text{g}/\text{m}^3$ .

***Lessons Learned from the Demonstration Project***

This demonstration project resulted in a successful collaboration among staff of the EPHT project in CEHS, the CLPPSS, and NJDEP. Through the development of a protocol, agency representatives were able to define the questions and to design the exposure assessments and analytical approaches used in the study. The project created to a more complete understanding of the strengths and limitations of New Jersey databases regarding both childhood blood lead and environmental lead in ambient air and drinking water among the EPHT Study Team.

The findings of this demonstration project should be considered suggestive, and not necessarily causal. Exposure misclassification in linkage studies, as with any epidemiologic study, is an important consideration. There are many sources of error in using the NATA or drinking water data for human exposure assessment. While this

epidemiologic study found an association between elevated individually measured childhood blood lead and 90<sup>th</sup> percentile lead levels in community water systems, after controlling for census tract-level demographic, economic and housing characteristics, these findings may be due to alternate explanations. It will be important to try and confirm these findings by linking individual level blood lead assessments, and individual assessment for poverty, race, and age of residence. These findings are particularly important as they create an opportunity to potentially limit lead exposure to very large populations of children by reducing lead exposure through drinking water. While exposure to lead paint will continue to be the primary source of acute lead poisoning in New Jersey's youngest residents, it is an important public health goal to reduce or eliminate all preventable chronic lead exposures that impact young children.

This demonstration project identified several opportunities to improve future childhood lead surveillance and tracking/linkage. Several data quality and data completeness issues were identified. New Jersey childhood blood lead measurements generally lack individual information on sex of child, race of child, and Hispanic ethnicity of child. Additionally there is nothing currently known about the loss of study population due to geographic coding loss, or incomplete data. Future tracking and linkage projects might also want to consider trying to obtain individual age of residences if available from local data sources.

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