United States
Environmental Protection
Agency

National Center for
Environmental Assessment
Research Triangle Park, NC 27711

EPA/600/R-95/139
September 1995
External Review Draft

EPA

Urban Soil Lead Abatement Demonstration Project

Review Draft

(Do Not Cite or Quote)

EPA Integrated Report

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EXTERNAL REVIEW DRAFT
EPA/600/R-95/139

URBAN SOIL LEAD ABATEMENT DEMONSTRATION PROJECT

EPA INTEGRATED REPORT

National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

September 1, 1995

DRAFT-DO NOT QUOTE OR CITE

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LIST OF PARTICIPANTS

Urban Soil Lead Abatement Demonstration Projects

Dr. Ann Aschengrau
Boston University School of Public Health
80 East Concord Street, T-355
Boston, MA 02118

Dr. David Bellinger
Children's Hospital
Gardner House Room 455
300 Longwood Avenue
Boston, MA 02115

Dr. Robert Bornschein
University of Cincinnati
Department of Environmental Health
3223 Eden Avenue #56
Cincinnati, OH 45267-0056

Ms. Dawn Boyer
Inorganic Chemistry Department
Lockheed ESC
1050 East Flamingo
Las Vegas, NV 89119

Ms. Merrill Brophy
MDE/Lead & Soil Project
Maryland Department of the Environment
2500 Broening Highway
Baltimore, MD 21224

Dr. Richard Brunker
U.S. EPA - Region III
Site Support Section MD-3HW26
841 Chestnut Street
Philadelphia, PA 19107

Mr. Barry Chambers
Maryland Department of the Environment
2500 Broening Highway
Baltimore, MD 21224

Dr. Rufus Chaney
U.S. Department of Agriculture
ARC, Building 318 BARC-East
Beltsville, MD 20705

Dr. Julian Chisolm
Kennedy Institute
707 N. Broadway
Baltimore, MD 21205

Dr. Scott Clark
University of Cincinnati
Department of Environmental Health
Mail Stop 56
3223 Eden Avenue
Cincinnati, OH 45267-0056

Ms. Linda Conway-Mundew
University of Cincinnati
Department of Environmental Health
1142 Main Street
Cincinnati, OH 45210

Dr. Robert Elias
U.S. EPA
National Center for Environmental Assessment
Mail Drop 52
Research Triangle Park, NC 27711

Dr. Katherine Farrell
Anne Arundell County Health Department
3 Harry S. Truman Parkway
Annapolis, MD 21401

Ms. Beverly Fletcher
U.S. EPA - Region I
Environmental Services Division
60 Westview Street
Lexington, MA 02173

LIST OF PARTICIPANTS (cont'd)

Urban Soil Lead Abatement Demonstration Projects (cont'd)

Mrs. Barbara Gordon
Cincinnati Health Department
3101 Burnet Avenue, Room 309
Cincinnati, OH 45229

Ms. Jo Ann Grote
University of Cincinnati
Department of Environmental Health
1142 Main Street
Cincinnati, OH 45210

Mr. Bill Hanson
Cincinnati Health Department
UC Soil Project
1142 Main Street
Cincinnati, OH 45210

Mr. Reginald Harris
U.S. EPA - Region III
Site Support Section MD-3HW15
841 Chestnut Street
Philadelphia, PA 19107

Mr. Ronald Jones
Cleveland Department of Public Health
1925 East St. Claire Ave.
Cleveland, OH 44114

Dr. Boon Lim
Environmental Health Program
Maryland Department of the Environment
2500 Broening Highway
Baltimore, MD 21224

Dr. Allan Marcus
U.S. EPA
National Center for Environmental
Assessment
Mail Drop 52
Research Triangle Park, NC 27711

Dr. Tom Matte
Public Health Service - Region II
26 Federal Plaza, Room 3337
New York, NY 10278

Ms. Lisa Matthews
U.S. EPA - OS 230
401 M Street, SW
Washington, DC 20460

Mr. J. Todd Scott
The Cadmus Group
3580 Cinderbed Road
Suite 2400
Newington, VA 22122

Mr. Dave McIntyre
U.S. EPA - Region I
Environmental Services Division
60 Westview Street
Lexington, MA 02173

Mr. William Menrath
University of Cincinnati
Department of Environmental Health
1142 Main Street
Cincinnati, OH 45210

Dr. Winkey Pan
University of Cincinnati
Department of Environmental Health
3223 Eden Avenue
Cincinnati, OH 45267-0056

Dr. Dan Paschal
Centers for Disease Control
1600 Clifton Road, NE
Mail Stop F-18
Atlanta, GA 30333

LIST OF PARTICIPANTS (cont'd)

Urban Soil Lead Abatement Demonstration Projects (cont'd)

Ms. Sandy Roda
University of Cincinnati
Department of Environmental Health
3223 Eden Avenue
Cincinnati, OH 45267-0056

Dr. Charles Rohde
Biostatistics
Johns Hopkins University
615 N. Wolfe Street
Baltimore, MD 21205

Ms. Penny Schmitgen
University of Cincinnati
Department of Environmental Health
3223 Eden Avenue
Cincinnati, OH 45210

Dr. James Simpson
Centers for Disease Control
CEHIC/EHHE, Mail Stop F28
1600 Clifton Road
Atlanta, GA 30333

Dr. Tom Spittler
U.S. EPA - Region I
Environmental Services Division
60 Westview Street
Lexington, MA 02173

Mr. Warren Strauss
MDE/Lead & Soil Project
Maryland Department of the Environment
2500 Broening Highway
Baltimore, MD 21224

Dr. Paul A. Succop
University of Cincinnati
Department of Environmental Health
3223 Eden Avenue
Cincinnati, OH 45267-0056

Dr. Pat VanLeeuwen
U.S. EPA - Region V
Technical Support Unit 5HR-11
230 S. Dearborn Street
Chicago, IL 60404

Dr. Harold A. Vincent
U.S. EPA
Quality Assurance Division
Environmental Monitoring Systems
Laboratory - Las Vegas
P.O. Box 93478
Las Vegas, NV 89193-3478

Dr. Michael Weitzman
Chief of Pediatrics
Rochester General Hospital
1425 Portland Avenue
Rochester, NY 14621

LIST OF REVIEWERS

LIST OF ABBREVIATIONS, ACRONYMS, AND TERMS

AAS	Atomic absorption spectroscopy
ANCOVA	Analysis of covariance
BAL P	Baltimore Study Group with paint intervention
BAL SP	Baltimore Study Group with soil and paint intervention
BOS P	Boston Study Group with paint intervention
BOS PI	Boston Study Group with paint and interior dust intervention
BOS SPI	Boston Study Group with soil, paint, and interior dust intervention
CDC	Centers for Disease Control and Prevention
CIN I-SE	Cincinnati Study Group with interior dust intervention, followed by soil and exterior dust intervention (second year)
CIN NT	Cincinnati Study Group with no treatment
CIN SEI	Cincinnati Study Group with soil, exterior dust, and interior dust intervention
dL	Deciliter; used here as a measure of blood lead in micrograms per deciliter
Double blind	Analytical audit sample where analyst knows neither that the sample is an audit sample nor the concentration
Dust loading	Mass of dust per unit area
ECAO/RTP	Environmental Criteria and Assessment Office/Research Triangle Park (now National Center for Environmental Assessment/Research Triangle Park)
EPA	U.S. Environmental Protection Agency
GLIM	Numerical Algorithms Group software package for a general linear model

GLM	SAS procedure for general linear models approximately equivalent to Systat MGLH
Hand dust	Sample taken by wiping the child's hand thoroughly; a measure estimating the ingestion of lead
HEPA	High-efficiency particle accumulator
ICP	Inductively coupled plasma emission spectroscopy
Lead concentration	Mass of lead per mass of medium (soil, dust, water)
Lead loading	Mass of lead per unit area
MGLH	Systat procedure for general linear models approximately equivalent to SAS GLM
NHANES II	National Health Assessment and Nutrition Examination Survey II
ORD	Office of Research and Development
OSWER	Office of Solid Waste and Emergency Response
P-value	Statistical term for the likelihood that an observed effect differs from zero
Pb	Lead
Project	In this report, "project" refers collectively to the three individual studies that compose the Urban Soil Abatement Demonstration Project.
P-XRF	Field or Portable XRF used in this study for paint measurements
QA/QC	Quality assurance/quality control
Repeated measures analysis	Statistical procedure for analyzing normally distributed responses collected longitudinally
Round	Period of sampling and data collection during study
SARA	Superfund Amendments and Reauthorization Act

SAS	Statistical software package
SES	Socioeconomic status
Single blind	Analytical audit sample where analyst knows sample is an audit sample but doesn't know concentration (see Double blind)
Study	In this report, "study" refers to one of the three individual soil abatement studies that compose the Urban Soil Abatement Demonstration Project.
SYSTAT	Statistical software package
USLADP	Urban Soil Lead Abatement Demonstration Project
XRF	Laboratory scale X-ray fluorescence instrument used in this study for soil and dust analysis (see P-XRF)

1. EXECUTIVE SUMMARY

1.1 BACKGROUND AND OVERVIEW

In the past 25 years, concern for children with lead poisoning has steadily increased with mounting evidence for the subtle but serious metabolic and developmental effects of lead exposure levels previously thought to be safe. Childhood lead poisoning was formerly considered a severe medical problem usually traced to swallowed chips of peeling lead-based paint. Scientific evidence has systematically revealed deleterious effects of lead at lower levels of exposure. Agencies such as the U.S. Environmental Protection Agency (EPA) and the Centers for Disease Control and Prevention (CDC) have repeatedly lowered the level of concern for children's lead burden that recommends environmental or clinical intervention from a blood lead level of 30 μg/dL established in 1978 by CDC to 25 μg/dL in 1985, just prior to the start of this project, then to the present level of 10 μg/dL, which was defined in October 1991 by CDC as a blood lead level that should trigger community-wide prevention activities if observed in many children.

The relationship between soil lead and blood lead is an indirect relationship in the sense that children most commonly do not eat soil directly but ingest small amounts of dust derived, in part, from this soil. In the child's environment, dust is only one of several sources of lead that also include food, air, and drinking water. Likewise, the lead in blood reflects not only recent exposure from these sources but also the biokinetic processes that distribute and redistribute lead between blood and other body tissues, especially bone tissue.

The Urban Soil Lead Abatement Demonstration Project (USLADP), known also as the Three City Lead Study, was authorized in 1986 under Section 111(b)(6) of the Superfund Amendments and Reauthorization Act (SARA), which mandated that EPA conduct soil lead abatement projects in up to three U.S. cities (SMSA's). The purpose of the project was to determine whether abatement of lead in soil could reduce the lead in blood of inner city children. It did not attempt to compare the relative effectiveness of alternative soil abatement methods.

This report, then, is an integrated assessment of data from three coordinated longitudinal studies of children in urban neighborhoods of three cities (Boston, Baltimore, Cincinnati), where intervention into soil lead exposure pathways was expected to reduce the children's blood lead. Many cross-sectional studies of childhood lead exposure have previously shown that differences in soil lead exposure are associated with differences in blood lead concentrations, but they did not evaluate the effectiveness of intervention steps in terms of demonstrating that reductions in external exposure to lead from soil result in reductions in blood lead concentrations. Thus, a unique aspect of this project is that it measures response to intervention, not to contamination. Because of the physiology of lead mobilization in body tissues, there is a difference between the rate of change in a population with increasing lead exposure and in one with decreasing exposure. In other words, the decrease in blood lead concentrations in response to intervention was not expected to be at the same rate as an increase in blood lead concentrations in response to increasing exposure.

The project began in December 1986 with the appointment of an EPA steering committee to develop recommendations for implementing the SARA lead-in-soil demonstration project. A panel of experts was formed in early 1987 to assist EPA in defining a set of criteria for selection of sites and the minimum requirements for a study at each site. The panel also met in mid 1987 to discuss technical issues and study designs and to evaluate technical criteria for selection of urban areas as potential soil-lead abatement demonstration project sites, ultimately leading by the end of 1987 to the selection of Boston, Baltimore, and Cincinnati as the participating cities.

The individual studies were each designed around the concept of participating families within a definable neighborhood. These families and their living units were part of a study group, either a treatment group or a control group. Each study group was sampled during preabatement and postabatement phases of the studies carried out in each city. Prior to and after abatement, blood lead levels were ascertained and the environment of the child was extensively evaluated through measurements of lead in soil, dust, drinking water, and paint, and through questionnaires about activity patterns, eating habits, family activities, and socioeconomic status (SES). The objective of the preabatement phase was to determine the baseline exposure history and status (stability of the blood lead and environmental measures) prior to abatement. During the postabatement phase, samples were taken to confirm effectiveness of abatement actions in reducing lead in the abated media, to measure the duration of the effect of soil abatement, and to detect possible recontamination. Blood lead measurements were also obtained postabatement to ascertain abatement impacts at various postabatement intervals.

Research teams in each city included state and/or local health department personnel, academic researchers from local universities, and/or various other institutions (including in Boston participation by EPA Region I Laboratory personnel). Because of the complex nature of this exposure assessment, intermediate exposure indices, such as street dust, house dust, and hand dust were measured in some study groups. Protocols for these measurements were developed by a Scientific Coordinating Committee composed of representatives from each study, the three EPA regional offices, the CDC, EPA/Office of Solid Waste and Emergency Response, and EPA/Office of Research and Development.

1.1.1 Comparison of Study Hypotheses

The Scientific Coordinating Committee attempted to establish uniformity among the three studies for major aspects of the project. This required a study plan from each city that was discussed and reviewed at several early planning workshops. Although there were differences in form and content, each study plan contained

- a statement of the objectives of the study;
- a testable hypothesis that provided direction and focus to the study;
- protocols for collecting and analyzing the data;
- an array of treatment groups that addressed all features of the hypothesis;
- measures to be taken to ensure that all phases of the study would be conducted as planned; and
- procedures by which the results of the study would be processed, analyzed, and interpreted.

The objectives, protocols for sampling and analysis, quality assurance/quality control (QA/QC) plans, and data processing procedures were nearly identical for all three studies. Elements that differed among the three studies were the hypotheses and the array of treatment groups. The hypotheses differed only slightly, as seen from the following statements.

The central hypothesis of the USLADP is:

> *A reduction of lead in residential soil accessible to children will result in a decrease in their blood lead levels.*

The formal statement of the Boston hypothesis is:

> *A significant reduction (equal to or greater than 1,000 μg/g) of lead in soil accessible to children will result in a mean decrease of at least 3 μg/dL in the blood lead levels of children living in areas with multiple possible sources of lead exposure and a high incidence of lead poisoning.*

The Baltimore hypothesis, stated in the null form, is:

> *A significant reduction of lead (≥1,000 μg/g) in residential soil accessible to children will not result in a significant decrease (3 to 6 μg/dL) in their blood lead levels.*

The Cincinnati hypothesis was separated into two parts:

(1) *A reduction of lead in residential soil accessible to children will result in a decrease in their blood lead levels.*

(2) *Interior dust abatement, when carried out in conjunction with exterior dust and soil abatement, would result in a greater reduction in blood lead than would be obtained with interior dust abatement alone, or exterior dust and soil abatement alone.*

Secondary hypotheses in the Cincinnati study are:

(3) *A reduction of lead in residential soil accessible to children will result in a decrease in their hand lead levels.*

(4) *Interior dust abatement, when carried out in conjunction with exterior dust and soil abatement, would result in a greater reduction in hand lead than would be obtained with interior dust abatement alone, or exterior dust and soil abatement alone.*

The array of treatment groups differed considerably among the three studies (Table 1-1). In each study, the treatment groups had several features in common. The groups were taken from demographically similar neighborhoods. All groups had some prior evidence of elevated lead exposure, usually a greater than average number of public health reports of lead poisoning. Three phases were employed in each study: preabatement

TABLE 1-1. DESCRIPTION OF STUDY GROUPS AND TYPES OF INTERVENTION

Treatment Group Name[a]	Cross-Reference to Individual Study Report	Description of Treatment
	BOSTON	
BOS SPI	Study Group	Soil and interior dust abatement, and interior paint stabilization at beginning of first year, no further treatment
BOS PI	Control Group A	Interior dust abatement and interior paint stabilization at beginning of first year
BOS P	Control Group B	Interior paint stabilization at beginning of first year
	BALTIMORE	
BAL SP	Study Area	Soil abatement and exterior paint stabilization at beginning of first year, no further treatment
BAL P-C1[b]	Study Area Low	Exterior paint stabilization at beginning of first year, no further treatment because soil lead not above cutoff level
BAL P-C2[b]	Control Area High	Exterior paint stabilization at beginning of first year, no further treatment
BAL P-C3[b]	Control Area Low	Exterior paint stabilization at beginning of first year, no further treatment
	CINCINNATI	
CIN SEI	Area A	Soil, exterior dust, and interior dust abatement at beginning of first year, no further treatment
CIN I-SE[c]	Area B	Interior dust abatement at beginning of first year, soil and exterior dust abatement at beginning of second year, no further treatment
CIN NT[c]	Area C	No treatment, soil and interior dust abatement at end of study

[a]The treatment group designation indicates the location of the study (BOS = Boston, BAL = Baltimore, CIN = Cincinnati), the type of treatment (S = soil abatement, E = exterior dust abatement, I = interior dust abatement, P = loose paint stabilization, NT = no treatment).
[b]Treated as one group in the Baltimore report, analyzed separately in this report.
[c]Treated as one group in the Cincinnati report, analyzed as individual neighborhoods in this report.

baseline phase for 3 to 18 mo; abatement or intervention (except for controls) phase, and postabatement follow-up for 10 to 23 mo.

1.1.2 Study Design and Conduct

Table 1-1 describes the study groups and the forms of intervention employed in each of the three cities. The Cincinnati study design used intervention on the neighborhood scale, where the soil in parks, play areas and other common grounds were abated, and paved surfaces in the neighborhood were cleaned of exterior dust. In Boston and Baltimore, only soil on individual properties was abated. Table 1-2 shows the number of subjects participating in different phases of the three studies in relation to the respective participant groups for each city. The general characteristics are that soil lead concentrations are typically high in Boston, where it is also common to find lead in both exterior and interior paint, as well as in drinking water. In the Boston areas studied, housing is typically single and multi-family units with relatively large lot sizes. In the Baltimore neighborhoods, the houses were mixed single and multifamily, and the lots were smaller than Boston lots, with typical yards less than 100 m^2. Nearly every house had lead-based paint. Residential units in Cincinnati were mostly multifamily with little or no soil on the residential parcel of land.

1.1.3 Intervention Procedures

Figure 1-1 illustrates the generalized concept of human exposure to lead, showing the pathways of lead from the several sources in the human environment to four compartments immediately proximal to the individual. In the past decade, dramatic reductions in exposure to lead in air and food have occurred as a result of regulatory and voluntary programs to reduce lead in gasoline and canned food. Figure 1-2 expands the critical dust route to show the complexity of the many routes of dust exposure for the typical child. The strategies for intervention used in this project were designed to interrupt the movement of lead along one or more of these dust pathways.

There were three forms of intervention in this project: (1) soil abatement, (2) dust removal, and (3) paint stabilization. Soil abatement was by excavation and removal. Dust intervention was by vacuuming, wet mopping, and, in some cases, replacement of rugs and upholstered furniture. Cincinnati and Boston performed interior dust abatement, and

TABLE 1-2. NUMBER OF PROJECT PARTICIPANTS BY ROUND[a]

Study						
BOSTON	Round 1	Round 3	Round 4	Round 5		
Middate	10/17/89	4/9/90	9/12/90	7/20/91		
Children[b]	150	146	147	92		
Famlies[c]	125	121	122	77		
Properties[d]	100	96	97	67		
BALTIMORE	Round 1	Round 2	Round 3	Round 4	Round 5	Round 6
Middate	10/25/88	4/1/89	2/17/90	1/27/91	6/7/91	9/3/91
Children[b]	408	322	269	200	196	187
Families[c]	290	226	181	133	128	126
Properties[d]	260	207	160	117	114	112
CINCINNATI	Round 1	Round 3	Round 4	Round 6	Round 7	
Middate	7/6/89	11/14/89	7/1/90	11/17/90	6/16/91	
Children[b]	201	185	219	198	169	
Families[c]	71	67	66	94	82	
Properties[d]	141	129	124	124	124	

[a]Number shown is based on samples taken and does not include individuals enrolled but not sampled. Intervention is shown by the vertical dashed lines.
[b]Based on number of children sampled for blood. Some children may not have been included in the statistical analyses.
[c]Based on number of households sampled for dust.
[d]Based on number of properties (Boston, Baltimore) or soil parcels (Cincinnati) sampled.

Cincinnati also removed neighborhood exterior dust with mechanical sweepers and hand tools. Dust intervention was not expected to be permanent, because dust continually moves through the human environment. Instead, the removal of dust with elevated lead concentrations was to expedite the impact of soil abatement on the child's environment.

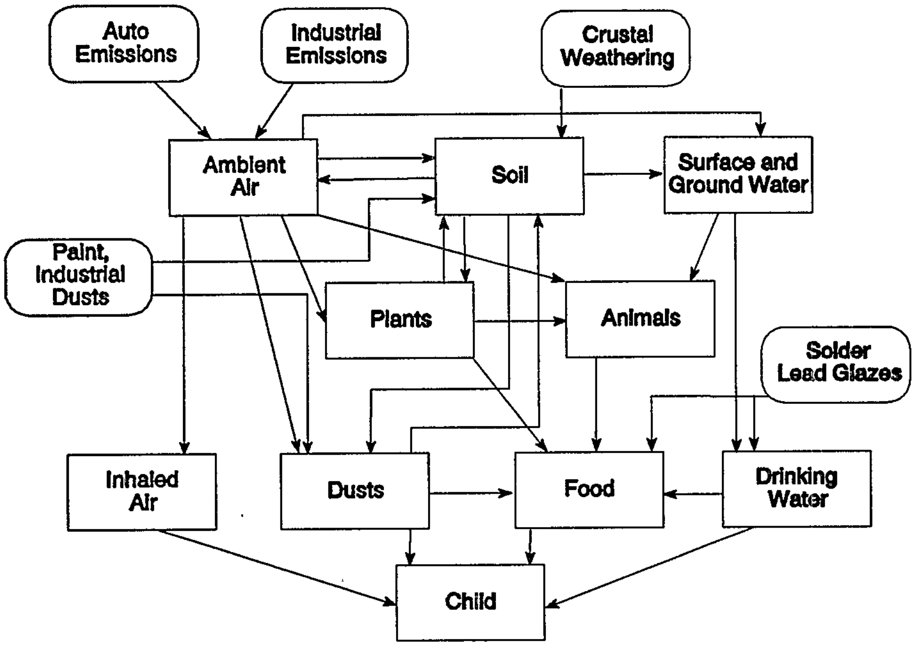

Figure 1-1. Generalized concept of the sources and pathways of lead exposure in humans.

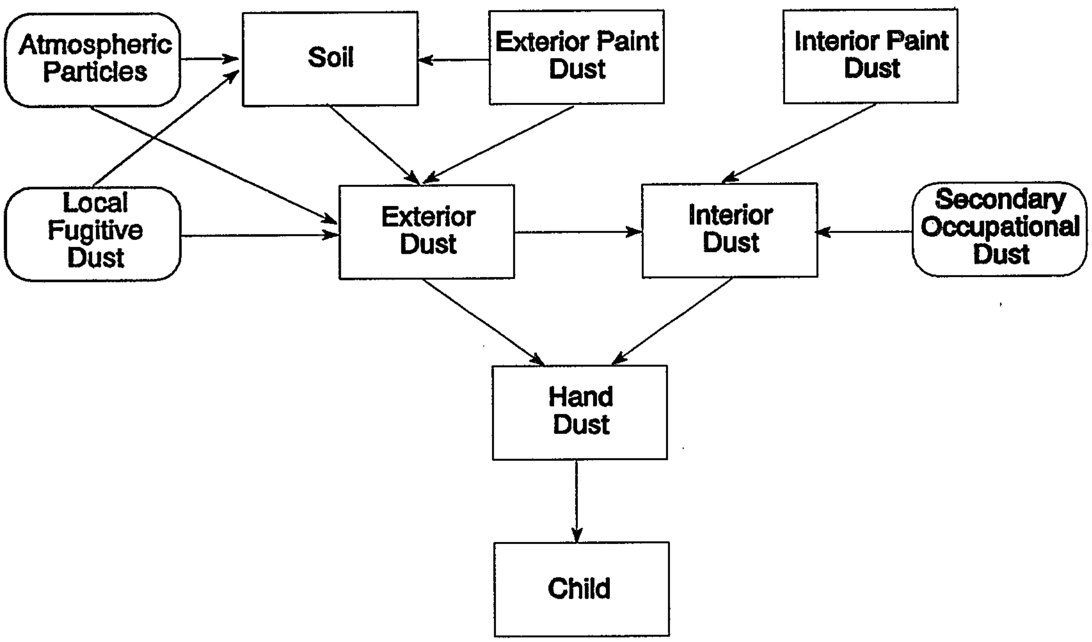

Figure 1-2. Typical pathways of childhood exposure to lead in dust.

In the home, house dust is a mixture of street dust and soil, interior and exterior paint dust, workplace dust carried home by adults, and dust generated from human activities within the household. It is believed that most of the mass of the interior dust originates from soil immediately exterior to the home, but this can vary greatly by the types of family activities and by neighborhood characteristics. Nevertheless, in the absence of lead-based paint inside the home, it would seem reasonable to assume that most of the lead in household dust comes from soil and other sources immediately outside the home.

Many of the Boston and Baltimore households selected for the project had chipping and peeling lead-based paint, both interior and exterior. In order to reduce the impact of this paint, the walls and other surfaces were scraped and smoothed, then repainted. It is important to note that this approach in not a full scale paint abatement and was not designed to permanently protect the child from lead-based paint. Paint stabilization was used on interior surfaces in Boston, and on exterior surfaces in Baltimore. Paint stabilization was not used in Cincinnati because the lead-based paint was believed to have been removed from these homes in the early 1970s as part of a housing rehabilitation project.

In order to accurately measure the effectiveness and persistency achieved by soil abatement and the impact of this abatement on reducing lead exposure for children, the sampling and analysis plans for soil and dust required robust quality control and quality assurance objectives. Protocols were developed to define sampling schemes that characterize the expected exposure to soil for children; collect, transfer, and store samples without contamination; and analyze soil, dust, handwipe, and blood samples in a manner that would maximize interlaboratory comparison. The original design focussed on sampling blood lead during the late summer, as it was known that the seasonal blood lead cycle peaks during this time. Where this schedule could not be adhered to, an effort was made to schedule the follow-up blood lead sampling at a comparable time in the cycle.

Information on area treated and volume of soil removed from each of the three cities properties appears in Table 1-3. A total of 35 Boston properties were abated during the study. In Baltimore, 63 properties in the BAL SP treatment group (see Table 1-3) were abated between August and November 1990. An additional seven properties that did not meet the requirements for abatement were transferred to a control group. Unpaved surfaces

TABLE 1-3. SOIL ABATEMENT STATISTICS FOR THE THREE STUDIES

	Boston	Baltimore	Cincinnati
Number of properties[a]	35	63	171
Surface area (m^2)	7,198	4,100[b]	12,089
Volume soil removed (m^3)	1,212	690	1,813
Surface area/property (m^2)	200	73	71
Volume soil/property (m^3)	34	11[b]	11

[a]Includes only properties abated during the study. Properties abated at the end of the study, where no further sampling was reported, are not included in this analysis, but are included in the individual study reports. In Cincinnati, a property is the location of the soil abatement, not the location of the child's residence.
[b]Surface area not provided by Baltimore report. This was calculated using Boston volume-to-surface ratio, which is equivalent to an average removal depth of 17 cm.

were divided into areas on each property (usually front, back, and one side) and any area with the maximum soil lead concentration above 500 μg/g was abated entirely.

Within each of six neighborhoods, the Cincinnati study identified all sites with soil cover as discrete study sites. The decision to abate was based on soil lead concentrations for each parcel of land, and for the depth to which the lead had penetrated. Lead was measured at two depths, the top 2 cm and from 13 to 15 cm. If the average concentration of the top and bottom samples was greater than or equal to 500 μg/g, the soil was removed and replaced. If the average of the top samples exceeded 500 μg/g, but the average of the bottom samples was less than 500 μg/g, the soil was also abated. Ground cover was reestablished on abated soils and some unabated soils according to protocols described in the Cincinnati report.

Exterior dust abatement was performed in the Cincinnati study only. The approach to this abatement was to clean all types of hard surfaces where dust might collect, using vacuum equipment that they tested and found to remove about 95% of the available dust on the area. The dust surface categories were streets, alleys, sidewalks, parking lots, steps, and porches.

Dust measurements were made in a manner that determined the lead concentration (micrograms of lead per gram of dust), the dust loading (milligrams of dust per square meter), and the lead loading (micrograms of lead per square meter) for the surface measured. This required that a dry vacuum sample be taken over a prescribed area, usually 0.25 to

0.50 m^2. It is important to note that dust abatement is not expected to cause an immediate change in the lead concentration on dust surfaces, only in the dust and lead loading.

Household dust was abated in the Boston and Cincinnati studies, but not in Baltimore. The BOS SPI and CIN SEI groups (see Table 1-1) received interior dust abatement at the same time as soil abatement, the BOS PI group received interior dust abatement without soil abatement, and the three CIN I-SE neighborhoods received interior dust abatement in the first year, followed by soil and exterior dust abatement in the second year.

In Boston, interior dust abatement was performed after loose paint stabilization. Hard surfaces (floors, woodwork, window wells, and some furniture) were vacuumed, as were soft surfaces such as rugs and upholstered furniture. Hard surfaces were also wiped following vacuuming. Common entries and stairways outside the apartment were not abated.

The Cincinnati group performed interior dust abatement after exterior dust abatement. Vacuuming was followed by wet wiping with a detergent. They vacuumed hard surfaces and replaced one to three carpets and two items of upholstered furniture per housing unit. Their previous studies had shown that these soft items could not be cleaned effectively with vacuuming alone.

Most homes in the Cincinnati group had undergone extensive remodeling, believed to have removed the lead-based paint 20 years prior to the project, but in Boston and Baltimore lead-based paint occurred in nearly every home. Because full paint abatement was not within the scope of this project, the alternative was to retard the rate of movement of lead from painted surfaces to household dust to the extent possible. The interior surfaces of all Boston homes and the exterior surfaces of all Baltimore homes received loose paint stabilization approximately one week before soil abatement.

In Boston, loose paint stabilization consisted of removing chipping and peeling paint and washing the surfaces. Window wells were painted with a fresh coat of primer. Baltimore homes were wet scraped over the chipping and peeling surfaces, followed by vacuuming. The entire surface was primed and painted with two coats of latex paint.

1.2 SUMMARY OF INDIVIDUAL STUDY REPORTS

Following the completion of data collection and analyses, the research teams in each city prepared individual study reports characterizing in detail the study design, procedures, and results obtained in their respective cities. Some of the more reliant features of each study and key findings reported by the individual city investigators are summarized next.

1.2.1 Boston Study

The Boston study retained 149 of the original 152 children enrolled, although 22 children moved to a new location while continuing in the study. Children with blood lead concentrations below 7 μg/dL or above 24 μg/dL had been excluded from the study and two children were dropped from some aspects of the data analysis when they developed lead poisoning, probably due to exposure to lead-based paint abatement debris at a location outside of their home.

Baseline characteristics (age, SES, soil lead, dust lead, drinking water lead, and paint lead) were similar for the three study groups (BOS P, BOS PI, BOS SPI). The preabatement blood lead concentration was higher for BOS P. The proportion of Hispanics was higher in BOS P than in BOS PI or BOS SPI, and the proportion of blacks was lower. There was a larger proportion of male than female children in BOS P.

Data were analyzed by analysis of covariance (ANCOVA), which showed a significant effect of intervention for both the BOS PI and BOS SPI groups. These results did not change following adjustment for age, sex, SES, or any other variable except race and paint. When the paint variable was controlled, the blood lead declines were diminished and the results were borderline statistically significant. When the race variable was added, the blood lead declines were also diminished and the results were not statistically significant.

Participants were chosen to be representative of the population of urban preschool children who are at risk of lead exposure. The Boston Childhood Lead Poisoning Prevention Program identified potential participants from neighborhoods with the highest rates of lead poisoning. Because study candidates with blood lead levels below 7 μg/dL or in excess of 24 μg/dL at baseline were excluded from the study, no conclusion about the effect of abating lead contaminated soil for children outside of this range can be made. Similarly, a different effect might have been found for children who had a greater blood lead contribution from soil, such as in communities with smelters or other stationary sources where soil lead levels are substantially higher than those seen in this study, or where differences in soil properties result in differences in bioavailability.

Follow-up blood lead measurements were made in Boston 11 months after intervention and again at 23 months.

1.2.2 Baltimore Study

The Baltimore study recruited 472 children, of whom 185 completed the study. Of those that completed the study, none were excluded from analysis. The recruited children were from two neighborhoods, originally intended to be a treatment and a control group. Because soil concentrations were lower than expected, some properties in the treatment group did not receive soil abatement. The Baltimore report transferred these properties to the control group. In this report, the unabated properties in the treatment group are treated as a separate control group.

Because of logistical problems, there was an extended delay between recruitment and soil abatement that accounted for most of the attrition from the project. In their report, the Baltimore group applied several statistical models to the two populations to evaluate the potential bias from loss of participating children. These analyses showed that the two populations remained virtually identical in demographic, biological and environmental properties.

The Baltimore study provided limited information on the impact of house dust as a part of the change in lead in the child's environment. The study design focused on changes in biological parameters, hand dust and blood lead, over an extended period of time. There were no measurements of exterior dust, no interior paint stabilization, and no interior dust abatement. Except for the abated properties, there were no follow-up measurements of soil lead concentrations.

Including the prestudy screening measurements of hand dust and blood lead in the original cohort of participants, the Baltimore study made six rounds of biological measurements that spanned 20 months, including postabatement measurements made at 2, 7, and 10 months following abatement.

1.2.3 Cincinnati Study

The Cincinnati study recruited 307 children, including 16 children born to participating families during the study, and an additional 50 children who were recruited after the beginning of the study. In their primary data analysis, the Cincinnati group excluded these 66 children who were recruited after the start of the study, plus 31 children who were living in nonrehabilitated housing suspected of having lead-based paint, and four children (in two families) who had become lead-poisoned from other causes. Thus, data for 206 children were analyzed in the Cincinnati report and these 206 children were included in this integrated report along with 7 of the 31 children living in nonrehabilitated housing. The remaining 24 were dropped because of insufficient follow-up data.

The Cincinnati study abated soil on 140 parcels of land scattered throughout six neighborhoods. If soil were the only source of lead in the neighborhoods, exterior and interior dust should have responded to the reduction in soil lead concentrations. However, exterior dust lead loading decreased only slightly following both soil and dust abatement, and returned to preabatement levels within one year. Corresponding changes in house dust, hand lead, and blood lead that paralleled changes in exterior dust. Interior dust returned to preabatement levels about one year after abatement. Because blood lead concentrations also decreased in the control area, the Cincinnati group concluded that there is no evidence for the impact of soil and dust abatement on blood lead concentrations. However, this integrated report concludes, through a more detailed structural equation analysis, that there is a strong relationship between entry dust and interior dust in this subset of the Cincinnati study, where the impact of lead-based paint was minimized.

Postabatement measurements in the Cincinnati were made at 2, 10, 14, and 21 months following abatement in the first year, and at 3 and 10 months following abatement in the second year.

1.2.4 Individual Study Conclusions

The Baltimore group stated their conclusions as follows:

- *"Statistical analysis of the data from the Baltimore Lead in Soil Project provides no evidence that the soil abatement has a direct impact on the blood lead level of children in the study."*

- *"In the presence of lead-based paint in the children's homes, abatement of soil lead alone provides no direct impact on the blood lead levels of children."*

The basis for these statements consisted of an adjusted and unadjusted analysis of selected covariates. The natural log of the blood lead of children in the treatment group showed no significant difference from the natural log of the blood lead of children in the control group, even when adjustments were made for age, SES, hand lead, season, dust, soil, sex, weak mouthing behavior, or strong mouthing behavior. These analyses were made on two sets of data. The first set consisted of all children enrolled in Rounds one and six. The second group consisted only of children enrolled in all six rounds.

In their report following the first phase of their study, the Boston group stated their conclusions as follows:

- *"...this intervention study suggests that an average 1,856 ppm reduction in soil lead levels results in a 0.8-1.6 μg/dL reduction in the blood lead levels of urban children with multiple potential sources of exposure to lead."*

Following the second phase of the study, they concluded (Aschengrau et al., 1994):

- *"The combined results from both phases suggest that a soil lead reduction of 2,060 ppm is associated with a 2.2 to 2.70 μg/dL decline in blood lead levels."*[1]

The basis for their conclusions consisted of an analysis of variance comparing mean blood lead changes among the three intervention groups, paired t-tests for within group effects, and analysis of covariance with one-at-a-time adjustment for age, SES, race, sex, paint, water, and mouthing behavior. The analysis of covariance was performed using no transformation of blood lead data, which appeared to be normally distributed.

The Cincinnati conclusions can be paraphrased from their report as follows:

- Following interior and exterior dust and soil lead abatement, blood lead concentrations decreased (in Area A) from 8.9 to 7.0 (21%) but increased to 8.7 μg/DL at 10 mo postabatement. Following interior dust abatement alone blood lead concentrations decreased from 10.6 to 9.2 (13%) 4 mo postabatement and were 18% below preabatement 10 mo postabatement. With no abatement, blood lead levels decreased by 29 and 6% during these same time periods. Other comparisons also revealed no effects of the soil or dust abatement.

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[1]This value for soil, 2,060 ppm, cited in their published report, was not adjusted by the Boston group with the interlaboratory correction factor of 1.037 in Table 3-6.

- There was no evidence that blood lead levels were reduced by soil lead or dust abatement in Area A (with soil, exterior dust, interior dust abatement). There was a slight reduction (net reduction over control area) of 0.6 μg/dL in Area B that might be attributed to interior dust abatement. This difference is not statistically significant.

The basis for the Cincinnati conclusions was a comparison of geometric mean blood lead concentrations in the three treatment groups between Rounds 1 and 4.

1.3 SUMMARY OF EPA INTEGRATED ASSESSMENT RESULTS AND FINDINGS

The original data sets for each of the three participating cities were submitted to EPA, along with the individual study reports alluded to above. Further analysis of the data were conducted by EPA staff in ORD, specially in the Environmental Criteria and Assessment Office/Research Triangle Park, NC (ECAO/RTP, now the National Center for Environmental Assessment [NCEA-RTP]). The present intergrated report presents information on the additional EPA statistical analyses and their results, as summarized here.

From the perspective of the child's environment, changes in the soil lead concentration are expected to bring about changes in the house dust concentration, the hand dust, and the blood lead concentration. In each of the three studies, the soil lead concentrations were reduced to approximately 25 to 200 μg/g in the study area, and for many treatment groups, there was a reduction of group mean blood leads, although not always statistically significant.

1.3.1 Quality of the Data

In the absence of certified standards for soil and dust, it was necessary to implement a program that would ensure that chemical analyses performed by the three participating laboratories would be internally accurate and externally consistent with similar analyses by other researchers. This program consisted of identifying acceptable analytical and instrumental methods, establishing a set of soil and dust standards, and monitoring the performance of the participating laboratories through an external audit program.

Because chemical extraction of an estimated 75,000 soil and dust samples per study presented a costly burden on the project both in terms of time and expense, and because of the advantage of nondestructive analysis for a project of this nature, the Scientific Coordinating Panel recommended the use of laboratory scale X-ray fluoresence (XRF) for soil analysis on the condition that a suitable set of common standards could be prepared for a broad concentration range and that a rigorous audit program be established to ensure continued analytical accuracy. Two groups, Boston and Baltimore, elected to use laboratory XRF for interior dust analysis also, whereas Cincinnati opted for hot nitric acid extraction with atomic absorption spectroscopy (AAS) for interior dust and XRF for exterior dust. During the study, the Baltimore group recognized problems with analyzing dust by XRF when the sample size was small, less than 100 mg. They reanalyzed the dust samples by AAS and reported both measurements. In Boston, this problem was solved by compositing the floor dust samples for XRF analysis, reporting one floor dust sample per housing unit.

During the project, there were two rounds of soil and dust interlaboratory calibration exercises, one near the beginning and one at the completion of the soil and dust analyses. These exercises, which involved the three participating laboratories and two additional laboratories for each exercise, provided the basis for the evaluation of the performance of each laboratory in the audit sample program, and for the conversion factors used to compare soil and dust data between laboratories.

Each study maintained rigorous standards for database quality. These included double entry, 100% visual confirmation, and standard procedures for detecting outliers. Some errors were found during the preparation of this report and corrected prior to use in this report. None of these errors would have impacted the conclusions drawn by the individual study.

1.3.2 Effectiveness and Persistency of Intervention

Soil abatement reduced soil concentrations in all three studies and there was no evidence of soil recontamination in either Boston or Cincinnati. There were no follow-up measures of soil in Baltimore that would detect recontamination. There was some evidence for exterior dust recontamination in Cincinnati. The Cincinnati group suggests that this might be caused by chipping and peeling lead-based paint from the exterior surfaces of nearby buildings not included in the project.

Interior dust abatement was persistent in both Boston and Cincinnati, even though some recontamination occurred in Cincinnati in response to the exterior dust recontamination. Paint stabilization appeared to have some impact on exposure, but there were no measures of persistency.

1.3.3 EPA Integrated Report Results

This integrated assessment looks at the three individual studies collectively to determine if a broad overview can be taken of the project results when each study is placed in its correct perspective.

The key findings of this integrated assessment with regard to the Boston study are as follows:

1. The median preabatement concentration of lead in soil was relatively high in Boston, averaging about 2,400 μg/g with few samples below 1,000 μg/g.

2. Abatement of the soil effectively reduced the median concentration of lead in the soil to about 150 μg/g (an average decrease of about 2,300 μg/g).

3. Soil was clearly a part of the exposure pathway to the child, contributing significantly to house dust lead.

4. Other sources of lead, such as interior lead-based paint were minimized by stabilization.

5. The reductions of lead in both soil and house dust persisted for at least two years.

6. Blood lead levels were reduced by approximately 1.6 μg/dL at 10 mo after soil lead abatement.

7. Additional reductions in blood lead of about 1.0 μg/dL (relative to non-abated) were observed at 22 mo postabatement for children in houses where the soil lead was abated and the interior house dust lead was consequently reduced and remained low.

Thus, in the Boston study, the abatement of soil resulted in a measureable, statistically significant decline in blood lead concentrations in children, and this decline continued for at least two years. It appears that the following conditions were present, and perhaps necessary for this effect: (a) a notably elevated starting soil lead concentration (e.g., in excess of 1,000 to 2,000 μg/g); (b) a marked reduction of more than 1,000 μg/g in soil lead consequent to soil abatement accompanied by (c) a parallel marked and persisting decrease in house dust lead.

These conclusions are consistent with those reported by the Boston research team. This integrated assessment found no basis for modifying their conclusions, although we choose not to express these findings as a broadly generalizeable linear relationship between soil and blood, such as change in micrograms of lead per deciliter of blood per change in micrograms of lead per gram of soil, because we believe that such a linear expression of abatement effects is highly site specific for the soil-to-blood relationship. We found evidence that the dust-to-blood relationship is more significant and, perhaps, more linear than the soil-to-blood relationship.

With regard to the Baltimore analyses conducted for this integrated assessment, the participants in the abatement neighborhood that did not receive abatement were treated as a separate control group, rather than combined with the nonabatement neighborhood (as the Baltimore research team did). The reason for this was to establish a control group not influenced by differences between neighborhoods. This alternative approach used in this integrated assessment had little impact on the statistical significance of soil abatement effects as reported by the Baltimore research team.

The key findings of this integrated assessment for Baltimore are:

1. The preabatement concentrations of lead in soil were notably lower (i.e., averaging around 500 to 700 μg/g, with few over 1,000 μg/g) than in Boston.

2. The actual reduction of lead in soil by abatement was small (a change of about 400 μg/g), compared to the Boston study (a change of about 2,300 μg/g).

3. Measurements of blood lead were made for only ten months following abatement; and no significant decreases in blood lead consequent to soil abatement were observed compared to non-abatement control group children.

4. Except for exterior lead-based paint, there was no control of other sources of lead, such as the stabilization of interior lead-based paint (as done in Boston) or abatement of house dust (as done in Boston and Cincinnati).

5. Follow-up measurements of soil (except immediately postabatement) were not made to establish the persistency of soil abatement, and its possible effects on house dust.

Thus, in Baltimore, where starting soil lead concentrations were much lower than in Boston and soil abatement resulted in much smaller decreases in soil lead levels and no interior paint stabilization or dust abatement was performed, no detectable effects of soil lead abatement on blood lead levels were found.

These conclusions are consistent with those reported by the Baltimore research group, and are not inconsistent with those above for the Boston study. At soil concentratons much lower than the Boston study, the Baltimore group would have likely been able to see only a very modest change in blood lead concentrations (perhaps less than 0.2 μg/dL) assuming similarity between the study groups in Boston and Baltimore and the same linear relationship between change in soil concentration and change in blood lead. Furthermore, the interior paint stabilization and house dust abatement performed in Boston perhaps enhanced and reinforced the impact of soil abatement on childhood blood lead, whereas in Baltimore, any possible small impact of soil abatement would have likely been swamped by the large reservoir of lead in the interior paint and the large unabated amounts of lead in interior house dust.

As for the Cincinnati study, because of differences in the neighborhoods, we found that combining neighborhoods into treatment groups often obscures important effects, and chose to analyze each of the six Cincinnati neighborhoods as separate treatment groups. One neighborhood, Back Street, had an insufficient number of participants and was dropped from some analyses. The Back Street group started with nine families, but by Round 5 there was only one participating family in the study. We also found that the two control neighborhoods, Glencoe and Mohawk, were substantially different, and that the three remaining treatment groups, Pendleton, Dandridge, and Findlay, were more comparable, both demographically and in geographic proximity, to Mohawk than to Glencoe.

On this basis, we concluded that, in most cases, the effect of soil abatement could not be clearly determined, and offer the following explanation for this conclusion:

1. Most of the soil parcels in each neighborhood were not adjacent to the living units, and this soil was therefore not the primary source of lead in house dust. Evidence for this statement includes the observation that street dust lead concentrations are much higher than soil concentrations, indicating there is a large source of lead contributing to street dust in addition to soil lead.

2. The preabatement median soil lead concentrations in the three treatment groups were about 300 μg/g in Pendleton, 700 μg/g in Findlay, and 800 μg/g in Dandridge, and the postabatement soil concentrations were less than 100 μg/g, so that the reduction of lead in soil was small, as in Baltimore.

Evidence for the impact of dust abatement or dust and soil abatement consists of a statistically significant difference between changes in blood lead between Rounds 1 and 4, approximately one year apart. Some Cincinnati neighborhoods showed decreased blood lead concentrations in response to dust abatement or dust and soil abatement. The two neighborhoods that received only interior dust abatement in the first year, Dandridge and Findlay, showed a small decrease in blood lead concentrations, compared to large increases in the nearest control group, Mohawk. The treatment group that received soil, exterior dust and interior dust abatement, Pendleton, showed a smaller effect than did the Dandridge and Findlay neighborhoods. After consultation with the Cincinnati research team, we suspect that there was recontamination of street dust in Pendleton during the study, probably caused by demolition of nearby buildings in the neighborhood.

The consistent theme across the outcomes for all three studies is that soil abatement must be both effective and persistent in markedly reducing soil lead concentrations accompanied by a corresponding reduction in house dust lead in order to result in any detectable reduction of blood lead. The location of the soil relative to the exposure environment of the child is important. In this project, the movement of lead from soil or street dust into the home seems to be a key factor in determining blood lead concentrations. Although these USLADP results provide substantial evidence for the link between soil or street dust and house dust lead, there is insufficient information by which to clearly quantify this relationship in terms of the lowest level of soil or street dust lead reduction that will yield a measurable decrease of lead in blood.

1.4 INTEGRATED PROJECT CONCLUSIONS

The main conclusions of this Integrated Report report are two-fold:

(1) *When soil is a significant source of lead in the child's environment, the abatement of that soil will result in a reduction in exposure that will, under certain conditions, cause a reduction in childhood blood lead concentrations.*

(2) *Although these conditions for a reduction in blood are not fully understood, it is likely that four factors are important: (1) the past history of exposure of the child to lead, as reflected in the preabatement blood lead; (2) the magnitude of the reduction in soil lead concentrations; (3) the magnitude of other sources of lead*

exposure, relative to soil; and (4) a direct exposure pathway between soil and the child.

The basis for the first conclusion is: in Boston, where the soil lead concentrations were high and the contribution from lead-based paint was reduced by paint stabilization, there was a measurable reduction of blood lead concentrations. This reduction continued to increase for two years following abatement in Boston.

Conversely, in Baltimore and Cincinnati, where soil was not a significant source of lead relative to other sources, there was no measurable reduction of blood lead except in cases where those sources were also removed or abated. In Baltimore, these sources may have been interior lead-based paint that was not stabilized, or house dust that was not abated. In Cincinnati, the principle source of lead seemed to be neighborhood dust that may have been contaminated with lead-based paint.

The basis for the second conclusion is: in those cases where all important elements of the exposure pathway were available for assessment, the structural equation model analyses showed that preabatement blood lead concentration was a major predictor of postabatement blood lead, suggesting that the remobilization of bone lead is a major component of the measured blood lead.

All other factors being equal, the measurable reduction in blood lead was observed only at higher concentrations of soil lead. In the absence of information about other sources of lead, no clear statement can be made about the possibility of smaller reductions in blood lead at lower soil lead concentrations.

In spite of the recent successes in reducing exposure to lead by removing lead from gasoline and canned food, lead exposure remains a complex issue. This integrated assessment attempts to assess exposure to lead in soil and house dust. Lead in soil and lead-based paint are closely linked in the child's environment. If there is exterior lead-based paint, then soil lead is likely to be elevated with a consequent elevation in house dust lead. If there is interior lead-based paint, then efforts to reduce the impact of soil lead on house dust will be only partially effective. The maximum reduction in lead exposure will not be achieved unless both paint and soil abatement are implemented.

There is evidence from all three studies that lead moves through the child's environment. This means that lead in soil contributes to lead in street or playground dust, lead in exterior paint contributes to lead in soil, and lead in street dust contributes to lead in house dust. A more detailed analysis of the data may show the relative contribution from two or more sources, but the present analyses imply that this transfer takes place.

The analysis of the data from the three studies showed evidence that blood lead responds to changes in house dust lead. There is also evidence for the continued impact of other, independent sources following abatement of one source. This means that abatement of soil or exterior paint does not necessarily reduce the contribution of lead from other sources such as interior lead-based paint.

The conclusions of this report suggest that soil abatement alone will have little or no effect on reducing exposure to lead unless there is a substantial amount of lead in soil and unless this soil lead is the primary source of lead in house dust. At a minimum, when implemented, both soil abatement and interior dust removal should both be performed to be fully effective. Conversely, soil abatement should be considered in conjunction with paint abatement when it is likely that soil will otherwise continue to contaminate house dust after a paint abatement is completed.

From one perspective, decisions about soil abatement should be made on an individual home basis. For an individual home, the owner or renter needs to know that the property is safe for children. This report shows that, on an individual house basis, soil abatement may reduce the movement of lead into the home and its incorporation into house dust. The magnitude of this reduction depends on the concentration of lead in the soil, the amount of soil-derived dust that moves into the home, the frequency of cleaning in the home and the cleanability of the home. The number and ages of children and the presence of indoor/outdoor pets are factors known to increase this rate of dust movement, whereas frequent cleaning with an effective vacuum cleaner, use of entry dust mats, and removing shoes at the door serve to reduce the impact of soil lead on house dust.

From another perspective, soil abatement at the neighborhood level poses problems not pertinent to individual homes. Playground, vacant lot, and other plots of soil may pose an immediate problem if they are accessible to children and there is a direct pathway for dust generated by this soil to enter the home. Likewise, sources of lead other than soil may contribute more to exterior dust than soil itself. The evidence in this report suggests that the key to reducing lead exposure at the neighborhood level is to abate significant sources of lead contributing to exterior dust, in addition to the soil and paint abatement that would be performed on an individual property.

2. BACKGROUND AND OVERVIEW OF PROJECT

2.1 PROJECT BACKGROUND

2.1.1 The Urban Lead Problem

Children are exposed to lead through complex pathways from multiple sources. In the mid 1980s, attention to sources of childhood lead exposure focused on urban environments with high concentrations of lead in soil, where there was an apparent correlation with the incidence of high blood lead concentrations. At that time, there were several other sources of exposure that could potentially account for unusually high blood lead in a population of urban children. Among these were lead in the air (primarily from automobile emissions), lead in food (primarily from canned foods with lead soldered side seams), lead in drinking water (primarily from lead pipes or newly soldered copper pipes), and lead in paint. The lead in the soil was believed to be a mixture of lead from the atmosphere and lead from exterior paint. Regulations were in place that would largely remove lead from gasoline by the end of 1986, and there was a voluntary program among food processors to phase out cans with lead soldered side seams. Renewed public interest in paint abatement emerged in the late 1980's concurrent with the start of this project.

Soil abatement had been performed in many nonurban residential areas with elevated soil lead. The decision to abate soil was usually based in part on the distribution of blood lead within the population of children. There was limited experience on the effectiveness of this abatement and little or no opportunity for follow-up studies of the results. There were little data from controlled evaluations because the intent of abatement was remediation, not experimentation.

2.1.2 Legislative Background

In the mid 1980s, the scientific evidence for a correlation between soil lead and blood lead was sufficient to warrant concern for the health of children, but not strong enough to support a large scale program for soil lead abatement. Consequently, the Urban Soil Lead Abatement Demonstration Project (USLADP), known also as the Three City Study, was authorized in 1986 under Section 111(b)(6) of the Superfund Amendments and Reauthorization Act (SARA).

SARA called for EPA to conduct a "pilot program for the removal, decontamination, or other actions with respect to lead-contaminated soil in one to three different metropolitan areas."

Although not specified in the amendment, the legislative history focused on lead-based paint as the source of lead in soil in urban residential areas. In response to the Superfund mandate, USLADP was designed to evaluate the effectiveness of removal of lead-contaminated soil in urban residential areas as a means to reduce blood lead levels of young, preschool children residing in abated residences or neighborhoods. It did not attempt to evaluate the relative effectiveness of different soil abatement technologies per se, but rather focussed on determining the extent to which the blood lead levels of children less than six years old (as a key risk group for lead health effects) could be reduced by intervention to decrease soil lead concentrations.

The EPA's Office of Solid Waste and Emergency Response (OSWER) had lead responsibility for overall implementation of the project, as a Superfund-mandated activity. Administrative and financial management responsibilities, it was decided, were to be delegated to EPA regional offices for the geographic areas containing those cities selected for inclusion in the project. EPA's Office of Research and Development was asked to provide technical oversight and coordination assistance to help integrate scientific activities across the cities selected. An EPA Steering Committee was set up to oversee site selection and initiation of the project.

In 1987, EPA convened a set of experts to advise on the design of the project and to develop selection criteria for study sites. Six cities submitted proposals, and Boston, Baltimore, and Cincinnati were chosen by the following site selection process.

2.1.3 Site Selection

The three cities were selected based on an evaluation of each proposal in relationship to the following site selection criteria, as recommended by the experts.

A. To be considered for selection, a metropolitan area *must* have:

1. Agreement by the appropriate EPA regional office to provide general project oversight, and to disburse the funds.

2. An established entity, preferably the state, documented as willing to be responsible for removing and disposing of lead contaminated soil. This included identification of an appropriate facility within the state for disposal of the soil, facilitation of permits, community relations and education, and any other activities necessary to expeditiously provide for safe disposal.

3. The administrative infrastructure to carry out a large scale project. This included a key government department with appropriate authority to coordinate the project, and generally included active participation by the state, by community groups, and by all the different metropolitan departments with some responsibility for the project.

4. Access to scientific and medical expertise to ensure that sampling and analysis were properly conducted, and access to medical care needed for any children found to have lead toxicity.

5. Evidence that there are children with elevated blood lead levels (25 μg/dL as defined by the CDC in its 1985 childhood lead screening guidelines), and soil in residential areas with lead levels of 1,500 μg/g or greater.[1] It would be desirable for lead-based paint to be established as a major contributor to the soil lead levels.

B. To be considered for selection, a metropolitan area *should* have:

6. A documented high incidence of children with elevated blood lead levels in the proposed study areas. This meant that the municipality supported an active childhood lead screening program.

7. A pattern of high density population in study areas. The number of children available for evaluation as part of the project was important to the statistical validity of the study.

8. Availability of other sources of funding for portions of the project not funded by SARA. Such items might include de-leading the outside of houses, or intensive interior vacuuming to remove residual leaded dust.

The Steering Committee reviewed proposals from six metropolitan areas: Boston, Baltimore, Cincinnati, Minneapolis, Detroit, and East St. Louis. These were reviewed on

[1] Note that the stipulated soil value of 1,500 μg/g was interpreted as a significant number of soil parcels in which at least one soil measurement exceeded this value. Reports in this document of means or median values below 1,500 μg/g for individual soil parcels or entire treatment groups should not be misinterpreted as failure to meet the original selection criteria.

December 3 and 4, 1987, by the Steering Committee and the set of expert consultants. Boston, Baltimore, and Cincinnati were selected based on the following key points:

1. The Boston investigators proposed to select three groups of families randomly from several neighborhoods known to have soil lead concentrations in the range of 2000 to 5000 μg/g. One of these groups would receive only paint stabilization; a second group would receive paint stabilization and dust abatement, and the third group would receive soil abatement, dust abatement, and paint stabilization.

2. The Boston proposal involved collaboration among Boston City Hospital, Boston University, and the EPA Region I Laboratory (for conduct of analysis of lead in soil, dust, etc.). This collaborative group also had demonstrated experience with collection, analysis, and assessment of soil and blood lead data in inner city neighborhoods of Boston.

3. Cincinnati proposed a neighborhood level abatement study where housing units had been previously gutted and rehabilitated approximately 20 years ago, and were thought to be free of lead-based paint. The Cincinnati sites contained soil lead from 220 to 900 μg/g, exterior surface dust (primarily from paved areas) from 2,000 to 5,000 μg/g, and a number of children with blood lead concentrations above 25 μg/dL.

4. The Cincinnati proposal was prepared by the University of Cincinnati and demonstrated a high degree of organizational infrastructure, with commitments from the City of Cincinnati. There was an established infrastructure of neighborhood associations that was perceived to be a plus for the project.

5. The Baltimore project proposed individual housing units with soil lead concentrations in excess of 1,000 μg/g. Lead-based paint had been abated in some, but not all houses.

6. The Baltimore proposal was prepared by the State of Maryland and showed a satisfactory level of organizational infrastructure and local scientific expertise; problems with the proposed statistical approach were resolved by consultation with the Steering Committee.

With the selection of Boston, Cincinnati, and Baltimore, a Scientific Coordinating Committee was established to provide scientific and technical support for the three studies and to coordinate the exchange of scientific information. This committee was composed of representatives from the research teams of each of the three cities, the three EPA regional offices (Regions I, III, and V), the Office of Solid Waste and Emergency Response, the Environmental Criteria and Assessment Office/Research Triangle Park, NC (now the National Center for Environmental Assessment/RTP), and the Centers for Disease Control and Prevention. The task of organizing, scheduling, and conduct of meetings of the Scientific Coordinating Committee was assigned to ECAO/RTP. Major policy decisions remained with the Steering Committee.

The funding mechanisms were set into place individually through the respective EPA regional offices (Regions I, III, and V). Each of these regional offices set up an independent funding mechanism and oversight plan. The regional project officer became the liaison to the Steering Committee and to the Scientific Coordinating Committee. Each city submitted a work plan, which included the project description, organization, operation plan, and reporting mechanisms, and the Quality Assurance (QA) plan. These work plans required more than one year to complete and acquire Regional approval. In the meantime, the projects were staffed and made operational. Community relations programs were initiated that began the process of recruiting the study participants. Coordination between the three cities was accomplished through a series of workshops, organized and convened by ECAO/RTP, approximately three per year.

This integrated assessment includes a review of the hypotheses and study designs of the individual studies (Chapter 2), a report of the methods intercomparison and quality assurance/quality control program (Chapter 3), a summary of the individual study results and conclusions reported by the three cities (Chapter 4), a description and explanation of the statistical procedures performed as part of this EPA integrated assessment and the results of these procedures (Chapter 5), and a summary of key findings and conclusions derived from this assessment (Chapter 6).

2.2 INTEGRATION OF THE THREE STUDIES

2.2.1 Study Hypotheses

To place this project in perspective, it is helpful to look at the similarities and differences among the three studies. They are similar in that their hypotheses and study designs were drawn from the same general hypothesis, namely, that removing lead from soil will reduce lead exposure.

The central hypothesis of the USLADP is

A reduction of lead in residential soil accessible to children will result in a decrease in their blood lead levels.

Each study chose to develop a specific hypothesis that could be tested by data and observations from their own study design. The formal statement of the Boston hypothesis is

A significant reduction (equal to or greater than 1,000 µg/g) of lead in soil accessible to children will result in a mean decrease of at least 3 µg/dL in the blood lead levels of children living in areas with multiple possible sources of lead exposure and a high incidence of lead poisoning.

The Baltimore hypothesis, stated in the null form, is

A significant reduction of lead (≥1,000 µg/g) in residential soil accessible to children will not result in a significant decrease (3 to 6 µg/dL) in their blood lead levels.

The Cincinnati hypothesis, separated into two parts, is

(1) A reduction of lead in residential soil accessible to children will result in a decrease in their blood lead levels.

(2) Interior dust abatement, when carried out in conjunction with exterior dust and soil abatement, would result in a greater reduction in blood lead than would be obtained with interior dust abatement alone, or exterior dust and soil abatement alone.

Secondary hypotheses in the Cincinnati study are

(3) A reduction of lead in residential soil accessible to children will result in a decrease in their hand lead levels.

(4) Interior dust abatement, when carried out in conjunction with exterior dust and soil abatement, would result in a greater reduction in hand lead than would be obtained with interior dust abatement alone, or exterior dust and soil abatement alone.

2.2.2 General Study Design

The project objective was to measure the relationship between soil lead and blood lead. This is an indirect relationship in the sense that children most commonly do not eat soil directly but usually ingest small amounts of dust derived, in part, from this soil. Likewise, the lead in blood reflects not only recent exposure from all environmental sources, but the remobilization of lead from bone tissue.

Each study was designed around the concept of participating families within a definable neighborhood. There were a total of twelve neighborhoods in the project, six in Cincinnati, four in Boston, and two in Baltimore. Except in Boston, these neighborhoods constituted the treatment and control groups in the study. In Boston, families in the treatment group were randomly assigned from volunteers from each of the four neighborhoods, as were families in the control group. For each treatment group, there was a preabatement, abatement, and postabatement phase. The immediate residential environment of the child was extensively evaluated prior to and after abatement, through measurements of lead in soil, dust, drinking water, and paint, and through interviews about activity patterns, eating habits, family activities, and socioeconomic status. Parallel environmental and biological measurements, as well as interviews, were taken in the control groups, but without abatement. The objective of the preabatement phase was to achieve a clear understanding of the exposure history and status (stability of the blood lead and environmental measures) prior to abatement. During the abatement phase, attention was given to preventing any possible exposure that might result from the abatement activities. During the postabatement phase, the project was designed to determine the duration of the effect of soil abatement and to detect possible recontamination.

The array of treatment groups differed considerably among the three studies. Each treatment group, however, had several features in common. All groups were taken from one to three demographically similar neighborhoods. All groups had some prior evidence of elevated lead exposure, usually a greater than average number of public health reports of lead poisoning. Each group received the same pattern of treatment: baseline phase for 3 to 18 months, intervention (except for controls), and follow-up for 12 to 24 months.

In each treatment group, even the controls, there was an attempt to minimize the impact of chipping and peeling lead-based paint. In Boston, this was done by paint stabilization of interior paint. In Baltimore, only exterior paint was stabilized. Therefore, in these two studies, the effects of soil abatement should be evaluated in the context of some intervention for lead-based paint. In Cincinnati, most of the living units may have been abated of lead-based paint more than 20 years before the start of the study. In the case of those that had lead-based paint, the lead-based paint was measured but not treated prior to the study.

The Boston and Baltimore studies used a parallel intervention scheme, compared to the staggered scheme used in Cincinnati. In other words, intervention in Boston (and Baltimore) took place at the same time for all treatment groups, and the follow-up period was of the same duration. But in Cincinnati, the soil and exterior dust intervention was delayed for three neighborhoods, such that follow-up varied between 12 and 24 months. Throughout all phases of each study, the timing of the blood lead measurements was planned according to a seasonal cycle of blood lead levels that peaks in the late summer and according to an age-related pattern that peaks at 18 to 24 months.

The complex nature of this project required measurement of exposure indices, such as street dust, house dust, and hand dust, that are in the pathway between soil and blood. New sampling and analysis protocols for these measurements, not generally available in the scientific literature, were developed during the initial coordinating workshops.

The studies differ in several respects. The two pathways: (a) soil → exterior dust and (b) paint → house dust differ slightly among the studies, as do the intervention strategies to interrupt the flow of lead along these pathways. Collectively, these differences in study design broaden the scope of the project to cover aspects of lead exposure intervention not possible through the study of a single neighborhood or even a single city.

2.2.3 Study Groups

Variations in the nature and form of intervention were included in the study designs to take advantage of the unique characteristics of the cities and their housing types. For example, soil lead concentrations are typically high in Boston, where it is also common to find elevated concentrations of lead in drinking water and in both exterior and interior paint. In the areas studied, housing is typically multi-unit with some single family units with relatively large soil cover in accompanying yards. In the Baltimore neighborhoods, nearly every house had lead-based paint, the houses were mixed single and multifamily, and the soil areas were smaller, typically less than one hundred square meters. On the other hand, houses in Cincinnati were selected because they were thought to be relatively free of interior lead-based paint, which might obscure the contribution of soil lead to house dust lead. As it happened, these neighborhoods were mostly multifamily housing with little or no soil on the residential parcel of land. The Cincinnati study design used intervention on the neighborhood scale, where the soil in parks, play areas, and other common grounds were abated, and exterior dust on paved surfaces in the neighborhood removed.

Detailed information on study design and methods of analysis can be found in the appended individual reports for each city. Table 2-1 summarizes the study design characteristics for each of the three studies and their respective neighborhood groups. The nonmenclature for these groups has been standardized for this report. With the exception of the Cincinnati control group (CIN NT), all groups received some form of intervention during the study.

For the purposes of consistency, certain descriptive terms that are used differently in the three individual study reports are standardized here and described in the glossary of this document. One example is the use of the terms "study" and "project". In order to avoid confusion, the term "study" refers to one of the three separate community studies, and the term "project" is used in reference to the three studies collectively. Similarly, the terms "treatment group" and "control group" are generally preferred in this report as a "study group".

The names that identify the individual treatment groups have been modified in this report to assist the reader in remembering the type of intervention performed on each group. Table 2-1 lists these names, with a brief description and the corresponding term in the report of each separate study. This nomenclature identifies location of the study and the nature of the intervention. For example, BOS SPI refers to the Boston group that received Soil, Paint, and Interior dust intervention. A hyphen is used to indicate intervention in two different rounds, as in CIN I-SE, where interior dust abatement took place about one year before soil and exterior dust abatement. The reader may want to become familiar with this nomenclature for the ten groups of participants in the project, as the data and results will be presented using these designations without further explanation. One further note: The BOS PI, BOS P and CIN NT groups each received soil abatement at the end of the study. Because no data were reported following this intervention, the designation "-S" was not used.

TABLE 2-1. TREATMENT GROUP NOMENCLATURE WITH CROSS-REFERENCE TO INDIVIDUAL REPORTS

Treatment Group Name[a]	Cross-Reference to Individual Study Report	Description of Treatment
BOSTON		
BOS SPI	Study Group	Soil and interior dust abatement, and interior paint stabilization at beginning of first year, no further treatment.
BOS PI	Control Group A	Interior dust abatement and interior paint stabilization at beginning of first year.
BOS P	Control Group B	Interior paint stabilization at beginning of first year.
BALTIMORE		
BAL SP	Study Area	Soil abatement and exterior paint stabilization at beginning of first year, no further treatment.
BAL P-C1[b]	Study Area Low	Exterior paint stabilization at beginning of first year; because soil was not above cut off level, no further treatment.
BAL P-C2[b]	Control Area High	Exterior paint stabilization at beginning of first year, no further treatment; soil above cut off level.
BAL P-C3[b]	Control Area Low	Exterior paint stabilization at beginning of first year; soil lead was not above cut off level; no further treatment.
CINCINNATI		
CIN SEI (P)	Area A	Soil, exterior dust, and interior dust abatement at beginning of first year, no further treatment. Includes only the Pendleton neighborhood.
CIN I-SE (B,D,F)[c]	Area B	Interior dust abatement at beginning of first year, soil and exterior dust abatement at beginning of second year, no further treatment. Includes the Back St., Dandridge, and Findlay neighborhoods.
CIN NT (G,M)	Area C	No treatment; soil and interior dust abatement following last sampling round. Includes the Glencoe and Mohawk neighborhoods.

[a]The treatment group designation indicates the location of the study (BOS = Boston, BAL = Baltimore, CIN = Cincinnati), the type of treatment (S = soil abatement, E = exterior dust abatement, I = interior dust abatement, P = loose paint stabilization, NT = no treatment).
[b]Treated as one group in the Baltimore report, analyzed separately in this report.
[c]Treated as one group in the Cincinnati report, analyzed as individual neighborhoods in this report.

Other departures here from the terminology of the respective individual study reports are conversion to a common system of units (metric where possible) and standard terms for phases, stages, or rounds of the project. The term "round" refers to a distinct period of time when one or more measurements were made. Other activities, such as soil abatement, occurred between rounds. There is no consistent pattern for when abatement occurred (i.e., after Round 1, Round 3, etc.) for the different individual cities.

The numbers of participating children, families, and properties appear in Table 2-2. Because of attrition and recruitment in Baltimore and Cincinnati, these numbers do not accurately represent the number of participants present for the duration of the study. In this report, subsets of these participants were statistically analyzed for specific purposes and to meet specific statistical requirements, and these subsets may not be the same subsets used by the individual study teams in their statistical analysis described in their respective individual city reports.

2.2.4 Project Activity Schedule

The project activity schedule, shown in Figure 2-1, illustrates the major intervention and measurement activities of the individual studies and the sequence and duration of these activities. The frequency and timing of sampling relative to abatement and seasonal cycles are important issues in the study design. These time lines are the actual occurrence of these events and they differ somewhat from the planned schedule. The original design focused on sampling blood lead during the late summer, as it was known that the seasonal cycle for blood lead reaches a peak during this period.

2.2.5 Environmental and Biological Measurements of Exposure

Figure 2-2 illustrates the generalized concept of the pathways and sources of human exposure to lead, showing the routes of lead from the several sources in the human environment to four compartments (inhaled air, dusts, food, drinking water) proximal to the individual. One of these proximal sources, dust, is the primary route of concern in this project. Figure 2-3 expands this dust route to show the complexity of the many routes of dust exposure for the typical child. The intervention strategies used in this project were designed to interrupt the movement of lead along one or more of these pathways.

TABLE 2-2. NUMBER OF PROJECT PARTICIPANTS BY TREATMENT GROUP AND ROUND[a]

	Treatment Group						
BOSTON		R1 (PRE)	R3 (POST 1)	R4 (POST 2)	R5 (Phase 2)		
Middate of round		10/17/89	4/9/90	9/12/90	7/20/91		
Children[b]	BOS SPI	52	52	52	33		
	BOS PI	51	48	49	33		
	BOS P	47	46	46	26		
		150	146	147	92		
Famlies[c]	BOS SPI	43	43	43	28		
	BOS PI	43	40	41	27		
	BOS P	39	38	38	22		
		125	121	122	77		
Properties[d]	BOS SPI	34	34	34	24		
	BOS PI	36	33	34	24		
	BOS P	30	29	29	19		
		100	96	97	87		
BALTIMORE		R1	R2	R3	R4	R5	R6
Middate of round		10/25/88	4/1/89	2/17/90	1/27/91	6/7/91	9/3/91
Children[b]	BAL SP	212	168	154	112	107	104
	BAL P	196	154	115	88	89	83
		408	322	269	200	196	187
Families[b]	BAL SP	155	121	103	76	71	71
	BAL P	135	105	78	57	57	55
		290	226	181	133	128	126
Properties[b]	BAL SP	141	112	91	66	63	62
	BAL P	119	95	69	51	51	50
		260	207	160	117	114	112
CINCINNATI		R1 (P01)	R3 (P03)	R4 (P05)	R6 (P07)	R7 (P09)	
Middate of round		7/6/89	11/14/89	7/1/90	11/17/90	6/16/91	
Children[b]	CIN SEI (P)	54	52	46	37	31	
	CIN I-SE (B,D,F)	86	81	92	87	77	
	CIN NT (G,M)	61	52	81	74	61	
		201	185	219	198	169	
Families[c]	CIN SEI (P)	31	30	31	31	30	
	CIN I-SE (B,D,F)	58	56	56	74	60	
	CIN NT (G,M)	40	37	35	63	52	
		129	123	122	168	142	
Parcels[d]	CIN SEI (P)	55	39	39	40	40	
	CIN I-SE (B,D,F)	74[e]	121[e]	121	119	121	
	CIN NT (G,M)	86	85	85	84	84	
		215	245	245	243	245	

[a] Round designations (R1, R2, etc.) are not the same as used in the Boston and Cincinnati study reports. Their round designations are shown in parentheses. Some rounds are omitted from this table because blood lead data were not collected. Intervention, shown by the dashed lines, occurred between R1 and R3 in Boston, R3 and R4 in Baltimore, R1 and R3 in the first year of the Cincinnati study, and R4 and R6 in the second year. Middates are the mean blood sampling dates.
[b] Based on number of children sampled for blood.
[c] Based on number of households sampled for dust.
[d] Based on number of soil areas sampled.
[e] Dandridge was added to the Cincinnati study after the soil sampling for R1, but before the completion of all other R1 sampling. This accounts for the sharp increase in the number of soil parcels between R1 and R3, with little change in the number of children or families.

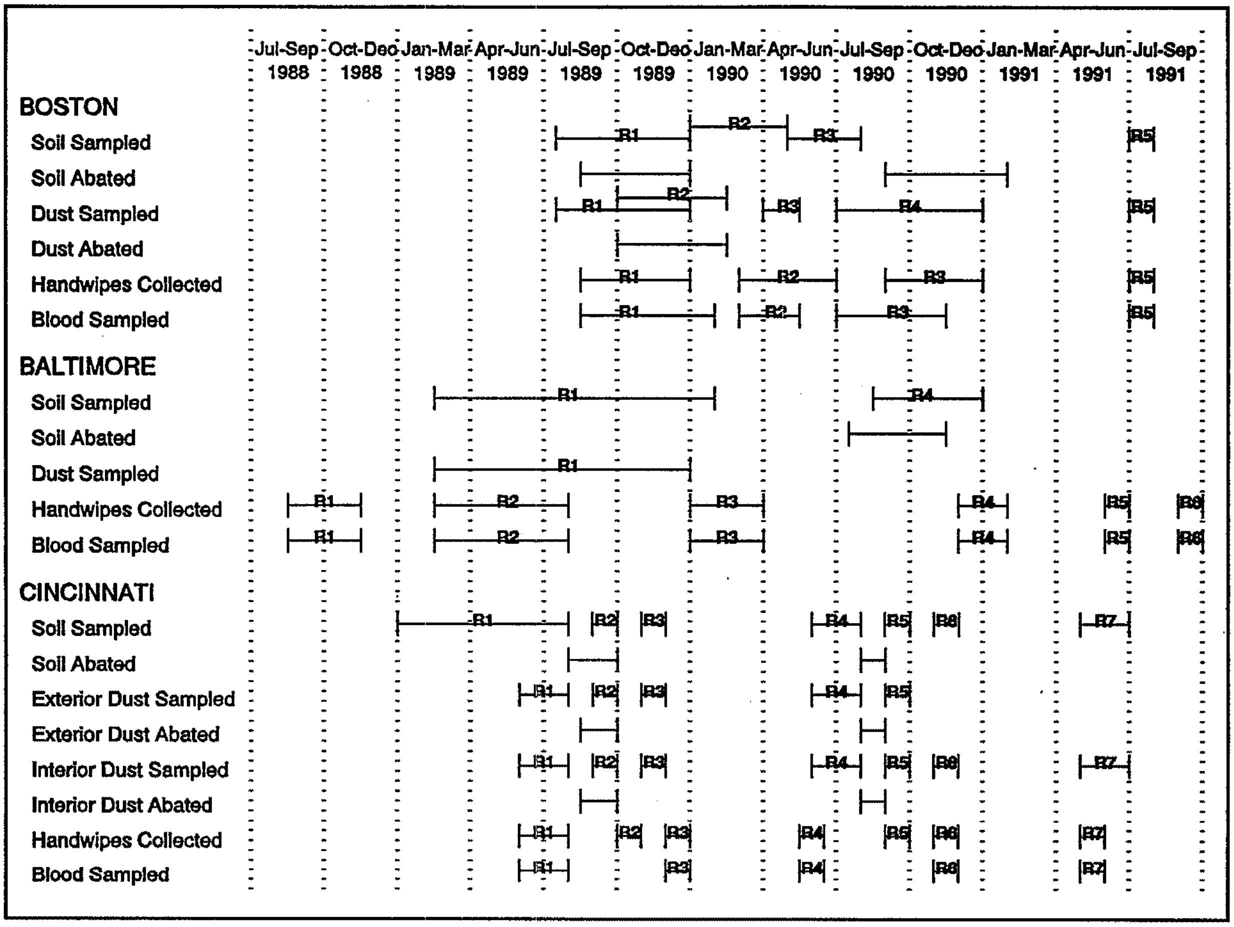

Figure 2-1. Project activity schedule showing the round designations and time periods for sampling and interviewing, and the time periods for soil abatement. Paint stabilization in Boston and Baltimore was performed during the soil abatement period prior to any other intervention. Abatement in Cincinnati that was performed after the final sampling round (as a courtesy to participants) is not shown in this figure.

Exposure is the amount of a substance that comes into contact with an absorbing surface over a specific period of time. In the case of lead, the absorbing surface can be the gastrointestinal tract or the lungs. Exposure is measured in micrograms of lead per day. Thus, an exposure of 10 μg/day represents a total ingestion and inhalation of 10 micrograms of lead from all sources; a fraction of this 10 micrograms would be absorbed into the body. In this project, blood lead was used as an indicator of exposure, and reductions in blood lead concentrations were expected as a result of any combination of the interventions described above. The units for blood are micrograms of lead per deciliter of blood and they are not

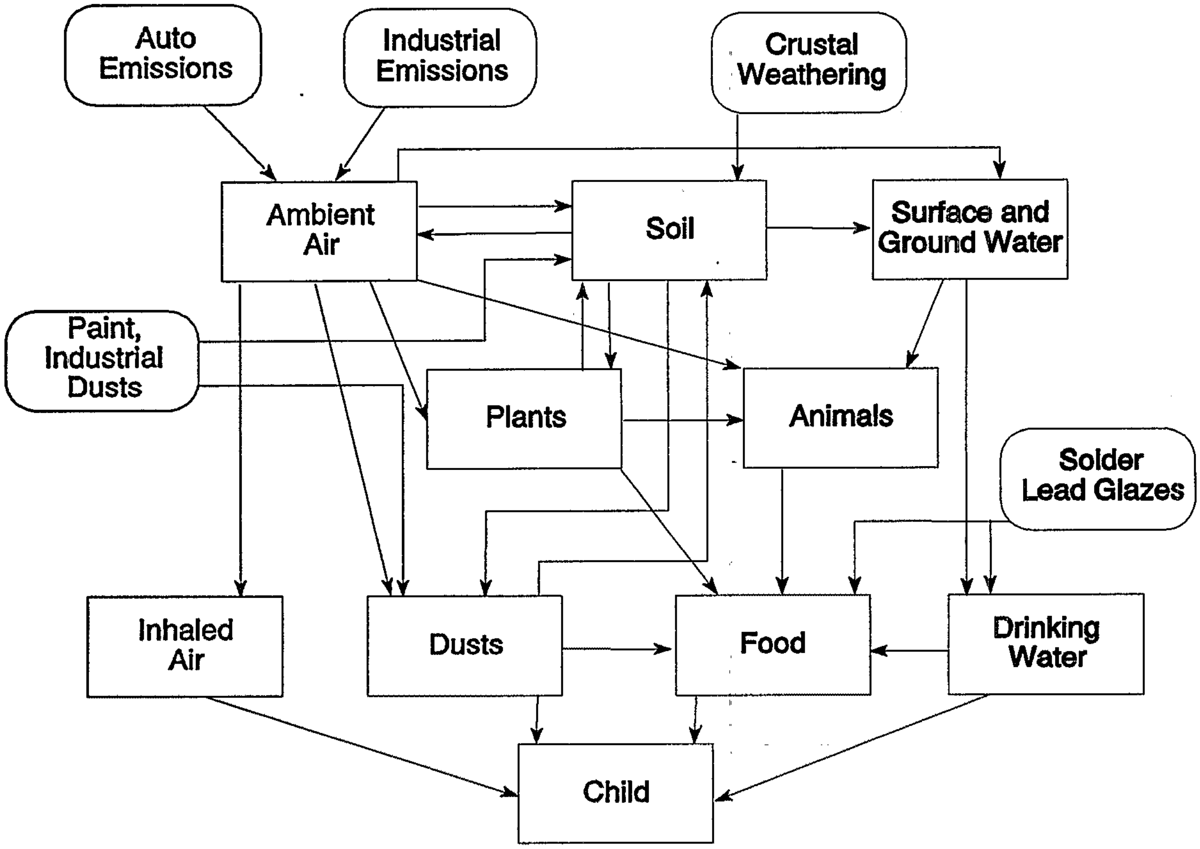

Figure 2-2. Generalized concept of the sources and pathways of lead exposure in humans.

compatible with the normal units of exposure, micrograms of lead per day. This illustrates that lead in one deciliter of blood reflects cumulative exposure for an unknown number of days plus an unknown amount of lead mobilized from bone tissue. Other indicators of potential exposure are hand lead and house dust. The amount of lead on the child's hands is believed to be closely related to the child's blood lead and to the dust lead in the child's environment.

2.2.5.1 Blood Lead

The amount of ingested lead that is actually absorbed in the gastrointestinal tract depends in part on the bioavailability of the particular form of lead. The amount of absorbed lead that reaches specific body tissues depends on the biokinetics of lead in the human body.

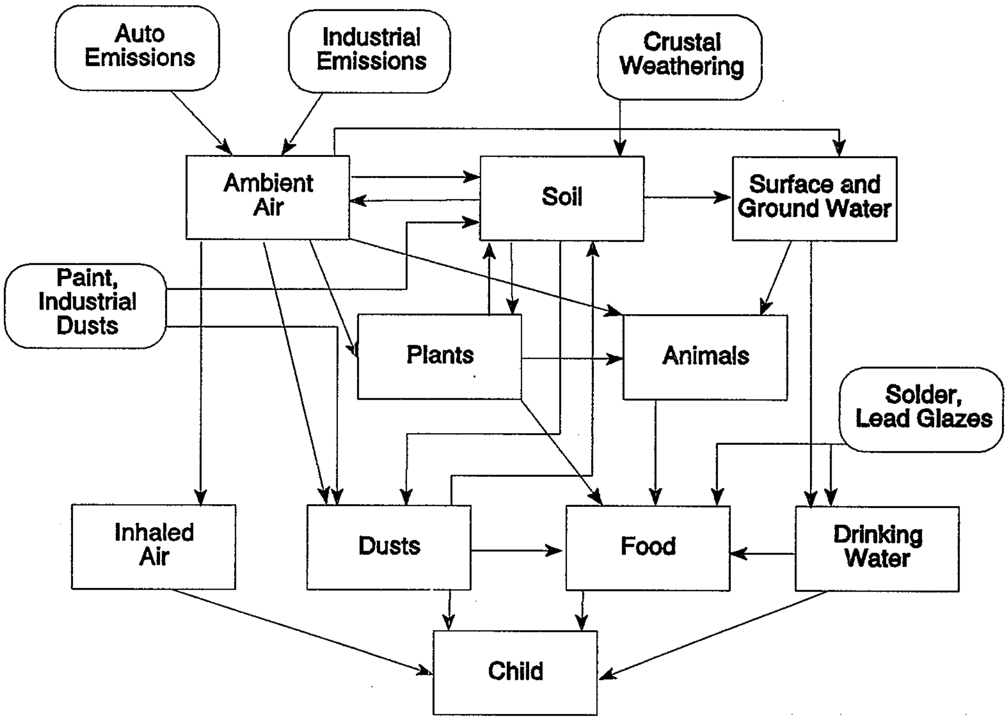

Figure 2-3. Typical pathways of childhood exposure to lead in dust.

Blood tissue is in dynamic equilibrium with all other body tissues, including bone tissue, where the lead is stored for longer periods of time. The relationship between blood lead and the onset of health effects of lead, depends largely on the distribution of lead to the target tissues, including the red blood cells themselves. Blood lead, then, is a convenient indicator of both exposure and potential health risk to the child. This situation becomes important when measuring the rate at which blood lead concentrations might decline following abatement. For a child with lead stored in bone tissue following a long history of high lead exposure, the decline in blood lead might be expected to be slower than for a child with low previous exposure.

2.2.5.2 Hand Lead

Because blood lead reflects exposure to lead from all environmental sources, a second exposure indicator, hand lead, was used to focus directly on the immediate pathway of dust into the child. The units of measure are micrograms of lead per pair of hands, and like blood lead, this measure does not reflect the rate at which lead moves into the body in units of micrograms of lead per day. Instead, this hand dust is a measure of lead loading on the hand. It is a measure of the "dirtiness" of the hand in the same sense that dust loading is a measure of the dirtiness of the floor. Hand dust loading could possibly be converted to micrograms of lead per day if there were a measure of the area of the hand mouthed by the child and the frequency of hand to mouth activity during each day.

2.2.5.3 House Dust

House dust is a mixture of lead from many sources, including soil, street dust, interior paint, and biological sources such as insects, pets, and humans. The units of measurement are μg Pb/g (lead concentration), μg Pb/m^2 (lead loading), and mg dust/m^2 (dust loading). When expressed as micrograms of lead per gram, the measurement can be converted to an exposure measurement by assuming a specific amount of dust ingested per day, usually about 100 mg/day for preschool children. Exposure to household dust then becomes micrograms per day:

$$\textit{Pb Concentration} \times \textit{Ingestion} = \textit{Exposure}$$

$$\frac{\mu g Pb}{g\, dust} \times \frac{g\, dust}{day} = \frac{\mu g Pb}{day}. \tag{2-1}$$

In a similar manner, exposure to food, drinking water, and inhaled air can be expressed as μg/day, and these three sources, circa 1990, normally account for about 5, 1, and 0.1 μg Pb/day respectively. If the lead concentration in household dust is 200 μg/g and dust ingestion is 0.1 g/day, the exposure is 20 μg/day or much more than the other sources combined. In this project, the maximum lead concentration in household dust was 107,000 μg/g.

By a different calculation, childhood lead exposure may be expressed as a function of dust lead loading. In this case, the ingestion parameter is in units of m^2/day:

$$\textit{Pb Loading} \times \textit{Ingestion} = \textit{Exposure}$$

$$\frac{\mu g Pb}{m^2} \times \frac{m^2}{day} = \frac{\mu g}{day}. \tag{2-2}$$

The ingestion parameter estimates the effective contact area for the child's hands (assuming all dust is ingested by hand-to-mouth activity). Literature reports of childhood lead exposure based on contact area are not known.

2.2.6 Intervention Strategies

Intervention is defined here as the interruption of the flow of lead along an exposure pathway. Soil abatement is one form of intervention. If done correctly, this abatement should establish an effective and persistent barrier to the movement of lead through the child's exposure pathways. Other forms of intervention used in this project were exterior dust abatement, interior dust abatement, and paint stabilization. Because dust is a very mobile constituent of the human environment, exterior and interior dust abatement would not be expected to form a permanent barrier to lead unless other sources of lead, such as soil, were also abated. Likewise, the form of paint stabilization used in Boston and Baltimore, where chipping and peeling paint was removed and the walls repainted, was not intended to be permanent lead-based paint abatement.

The strategy for soil abatement was to remove all soil with concentrations above a specific level (500 μg/g for Baltimore and Cincinnati, 1,000 μg/g for Boston), and replace this soil with clean soil in the range of 25 to 100 μg/g lead concentration. This method, called excavation and removal, was used in all three studies. In some cases, repair and maintenance of ground cover was used where the soil concentrations did not warrant excavation and removal.

To further interrupt the flow of lead along the exposure pathways, entire neighborhoods in Cincinnati were cleaned of exterior dust using street cleaning vacuum equipment and hand tools.

Interior house dust is believed to be a major direct lead exposure pathway for children. Because household dust typically contains a mixture of lead from several sources (e.g., soil, interior/exterior paint, air, etc.), abating house dust temporarily separates such sources from the child's environment. Their recontamination of house dust and consequent impact on the child's lead exposure can be evaluated by comprehensive measurements of the household dust that include changes in lead concentration, lead loading, and dust loading. Understanding the expected impact of abatement on these three parameters is critical to interpreting the observed changes in blood lead concentrations. Following dust abatement, there should be an immediate decrease in the dust loading, with no change in the lead concentration for those groups that did not receive soil, exterior dust, or paint intervention. The rate at which this dust loading returns to preabatement levels reflects the rate of movement of dust from other sources into the home, the frequency of cleaning, and the "cleanability" of the home. (Many inner city homes have surfaces that are cracked, pitted, or in disrepair and are difficult to clean effectively.)

The effectiveness of both paint stabilization and soil and dust abatement can be observed by changes in the lead concentrations of house dust. In the presence of lead-based paint, the concentration of lead in house dust is expected to be greater than 1,500 to 2,000 μg/g, whereas without the influence of lead-based paint, the house dust is expected to be comparable to external dust and soil (U.S. Environmental Protection Agency, 1986).

House dust is a mixture of dusts from many sources within and outside the home. In the absence of lead-based paint inside the home, it would seem reasonable to assume that most of the lead in household dust comes from soil and other sources external to the home. Therefore, to enhance the impact of soil abatement, interior dust abatement was carried out for some treatment groups in Boston and Cincinnati.

Many of the Boston and Baltimore households selected for the project had chipping and peeling paint, both interior and exterior. In order to reduce the impact of lead-based paint, the walls and other surfaces were scraped and smoothed, then repainted. It is important to note that no attempt was made to remove all lead-based paint, nor to isolate intact paint from the child. Paint stabilization was used on interior surfaces in Boston and on exterior surfaces in Baltimore. Paint stabilization was not used in Cincinnati because most of the lead-based paint was believed to have been removed from these homes in the early 1970s.

2.3 EXTERNAL FACTORS THAT COULD INFLUENCE PROJECT RESULTS AND INTERPRETATION

The Scientific Coordinating Panel recognized that several extraneous factors might influence the outcome of the project and that these factors were generally beyond the control of the investigators. Among these are seasonal cycles and time trends of childhood blood lead concentrations, unexplained or unexpected sources of lead in the children's homes or neighborhoods, changes in public perception and avoidance of lead exposure hazards, and movement of lead in soil either down the soil column or laterally with surface runoff or as fugitive dust.

2.3.1 Cycles and Trends in Environmental Lead Concentrations

Figure 2-4 illustrates a pattern of childhood blood lead concentrations for Chicago during the 1970s, showing a seasonal cycle and a downward trend throughout the decade. The National Health Assessment and Nutrition Examination Survey II (NHANES II) data for the entire country and all age groups (Figure 2-5) show a similar seasonal cycle and downward trend during the last half of that decade. (Seasonal patterns from the NHANES III data of 1988 through 1991 are not yet available.)

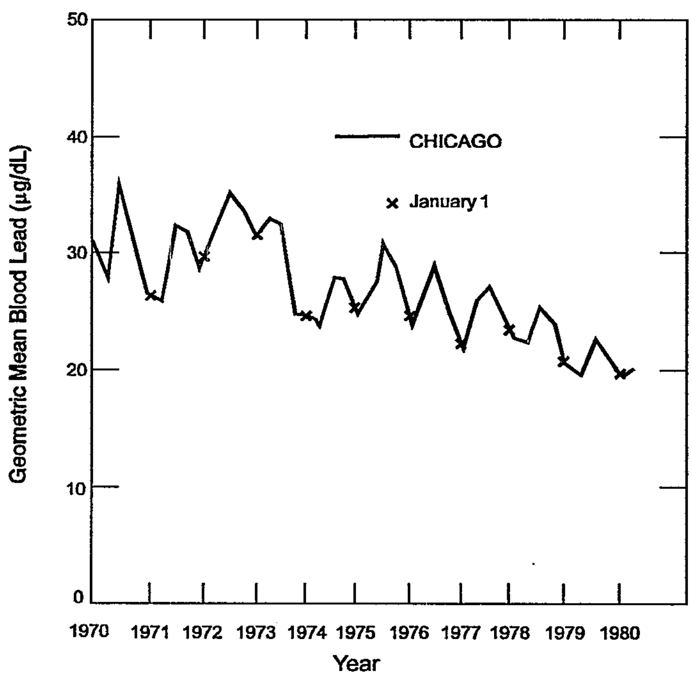

Figure 2-4. Literature values for seasonal patterns for childhood blood lead (age 25 to 36 mo).

Source: U.S. Environmental Protection Agency (1986).

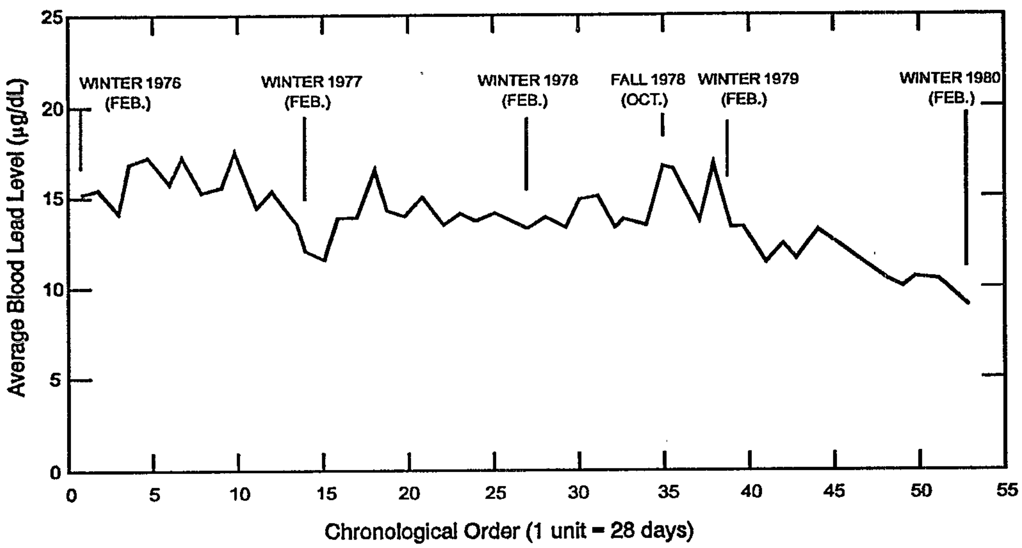

Figure 2-5. Literature values for seasonal patterns for blood lead in children and adults (NHANES II, age 6 mo to 74 years).

Source: Annest et al. (1983).

Investigators have known about this seasonal pattern for some time. Most epidemiological studies are planned so that measurements can be taken at the peak of this cycle, generally during the late summer. Studies of large numbers of children show a sinusoidal pattern, even when the measurements do not include sequential measurements for the same child. During the development of the study designs, it was apparent that understanding of the seasonal cycles and temporal trends in blood lead would play an important part in the interpretation of data collected over several years.

There is a question as to whether the seasonal cycle for blood lead concentrations is caused by fluctuations in exposure or by physiological processes that regulate the biokinetic distribution of lead within the body. Some investigators have attributed fluctuations in blood lead concentrations to changing environmental lead concentrations or changing activity patterns. During the late summer months, the child may eat food or dust with high lead concentrations or ingest more dust during outdoor play. This project was designed to measure changes in lead concentrations in soil and dust, but not changes in activity patterns. The observations made on these fluctuations and the interpretation of these observations are reported in Section 5.2.5.

Although this project was designed to maximize the measurements of blood lead during the late summer for each of the three studies, measurements were made during other times of the year in order to observe changes immediately after abatement. These sequential measurements show a similar cycle when all children are grouped together.

Two other patterns, long-term time trends and early childhood patterns dependent on age, are applicable to this project. Little is known about age related patterns, but one study in Cincinnati, prior to the project, showed a pattern of blood lead changes during early childhood growth patterns (Figure 2-6).

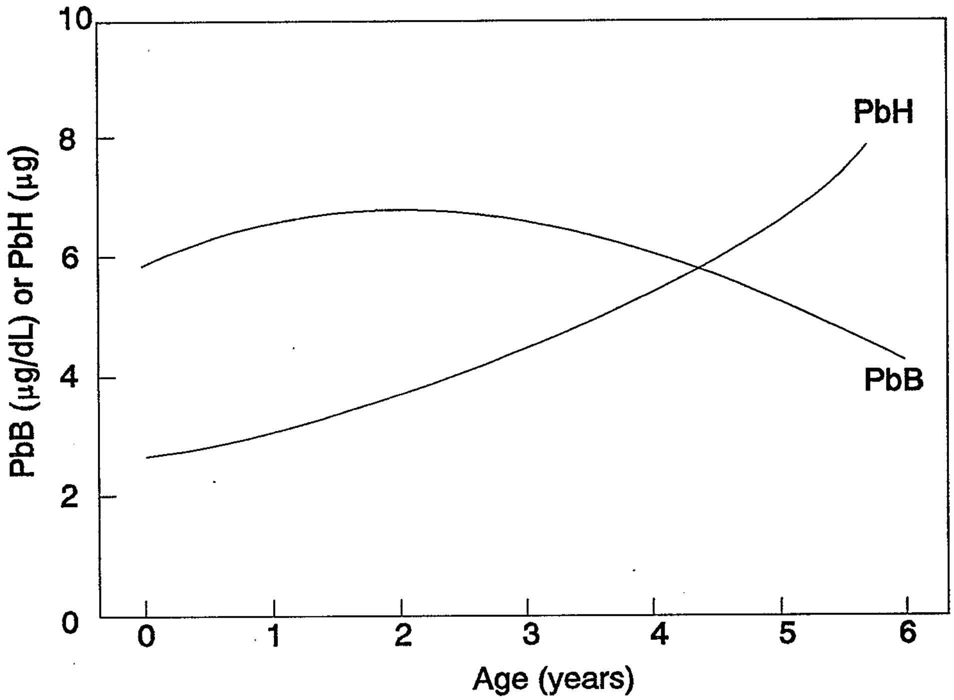

Figure 2-6. Predicted differences in blood lead (PbB) and hand lead (PbH) during early childhood, based on empirical data.

Source: Bornschein et al. (1988).

Long-term downward trends were documented for child blood lead concentrations during the 1970s and 1980s and have been attributed to decreasing concentrations of lead in food and air. Data for this project were analyzed for decreasing concentrations of lead in soil or dust and the results are reported in Chapter 5. The QA/QC measures reported in detail in Chapter 4 rule out the possibility of this trend being caused by a measurement artifact such as analytical drift.

2.3.2 Unexplained and Unexpected Sources of Lead

Occasionally, measurements of environmental lead are higher than expected and difficult to explain. Atmospheric deposition can be a reasonable explanation, because this route can change much more abruptly than soil, dust, food or drinking water. This section discusses the possibility that the observed fluctuation in street dust and house dust can be attributed to changes in air concentration alone. Because this project began after the national phasedown of lead in gasoline, the air concentrations of lead in these cities had decreased to about 0.1 $\mu g/m^3$ by the start the project.[2] The following is a theoretical calculation of the amount of lead that could be transferred to soil or dust at this concentration and from this source alone.

Atmospheric deposition during the project was assumed to be typical for air concentrations that averaged 0.1 $\mu g/m^3$ (1.0×10^{-7} $\mu g/cm^3$). At a deposition rate of 0.2 cm/s, this would accumulate 0.6 $\mu g/cm^2 \cdot$year at the soil surface. Assuming that this lead would be retained in the upper 1 cm of soil surface (therefore 1 cm^2 of soil surface equals 1 cm^3 of soil), then the annual increment would be 0.6 $\mu g/cm^3$. Because 1 cm^3 of soil weighs about 2 g, the annual incremental increase in lead concentration would be 0.3 μg Pb/g soil, an insignificant annual contribution for soils that average several hundred micrograms per gram. The calculation for annual deposition to a surface is

$$1 \times 10^{-7} \frac{\mu g\ Pb}{cm^3} \times 0.2 \frac{cm}{s} \times 3.15 \times 10^7 \frac{s}{year} = 0.6 \frac{\mu g\ Pb}{cm^2\ year}. \quad (2\text{-}3)$$

For the accumulation of dust on hard surfaces, however, the same calculation indicates a potentially greater influence of atmospheric lead. Converting to units of lead loading, the 0.6 $\mu g/cm^2 \cdot$year becomes 6,000 $\mu g/m^2 \cdot$year, or 16 $\mu g/m^2 \cdot$day. Therefore, 0.1 $\mu g/m^3$ in air concentration could account for a change of 16 μg Pb/m^2 per day in the dust lead loading to

[2] The 1989 maximum quarterly average air lead concentration for the metropolitan statistical areas of Boston, Baltimore, and Cincinnati were 0.08, 0.11, and 0.11 $\mu g/m^3$, respectively (U.S. Environmental Protection Agency, 1991a).

a surface. An accumulation of 160 $\mu g/m^2$ over 10 days is in the range of the observed changes in surface dust loading in this project.

2.3.3 Movement of Lead in Soil and Dust

There are several reasons why localized soil lead fluctuations might occur. Changes in soil lead concentration independent of intervention that might increase lead concentration are: atmospheric deposition (relatively minor as discussed above), exterior paint chipping and chalking, and human activity such as household waste dumping (motor oil, etc). Soil lead concentrations might decrease if lead leaches downward into the lower soil horizon, or if surface dust shifts by reentrainment. The downward leaching of lead through the soil profile mass occurs at a very slow rate, approximately a few millimeters per decade (Grant et al., 1990). The reentrainment of dust at the soil surface is usually in equilibrium with the local environment, such that inputs would equal outputs by this pathway. This would not be the case if there is flaking or peeling lead-based paint within the neighborhood or an industrial source of fugitive dust in the vicinity of the neighborhood. A limited effort was made to monitor and control the impact of lead-based paint on soil concentrations. In Baltimore, buildings with exterior lead-based paint were stabilized by removal of the chipping and peeling paint, done in a manner to avoid contaminating the soil. In Boston, homes were selected with less then 30% exterior chipping and peeling paint, by area. In Cincinnati, neighborhoods with mostly rehabilitated houses were selected. There were no attempts in any of the studies to control the introduction of lead to the soil by human activity such as household waste dumping.

Lead in household dust is a mixture of dust brought into the house from outside and dust generated from within the home. Studies have shown that as much as 85% of the mass of dust comes from outside the home and much of this is apparently brought in on the feet of children and pets (Roberts et al., 1991). Household dust lead concentrations are usually similar to the soil concentration in the immediate vicinity of the house, unless there are internal sources of lead, such as lead-based paint. Thus, changes in soil concentrations are likely to be reflected by changes in household dust concentrations within a few days and probably reach equilibrium within a few months, depending on the relative contribution from soil and other sources, the frequency and efficiency of house cleaning, and the cleanability of the house.

2.3.4 Other Factors

In the following chapters, this report discusses several issues that identify possible limitations of the studies. This detailed assessment: (1) examines measurement methods used and related QA/QC data to ascertain that adequate measures were taken to produce data of good quality that can be compared across the three studies; (2) examines the study designs to determine if the individual study groups are comparable within each study and if comparisons are possible across the three studies; and (3) performs rigorous statistical analyses that attempt to quantify differences between study groups and identify specific exposure factors that may be responsible for the differences.

With respect to the QA/QC data, it should be noted that there are no estimates of sampling reproducibility for any of the environmental or biological measurements. This would have required collecting duplicate samples for a specified percentage of the samples. In retrospect, the following observations are worth noting:

1. Duplicate soil samples would not have been informative unless the entire soil parcel was sampled in duplicate. In this report, the reproducible number is the arithmetic mean of all soil samples from the parcel;

2. Duplicate sampling of house dust would have identified reproducibility of lead concentration, but probably not lead loading, which changes on a daily basis. Duplicate sampling of house dust may also have impacted the child's environment if a substantial amount of the targeted play areas were sampled.

Nevertheless, this report recognizes the limitations of statistical analysis due to the absence of an estimate of sampling error.

There are several exposure-related factors other than those measured by environmental sampling that must be taken into account during the statistical analyses. Among these are seasonal patterns in weather (especially rainfall as it affects dust loading and mobility), activity patterns (which affect indoor/outdoor play patterns), and possible physiological growth cycles (which affect remobilization of lead from bone tissue). Age of the child may also impact exposure by differences in activity patterns, body size, and parental supervision.

For the most part, this report is only able to ascertain that all groups within a study were impacted equally by these and other confounding factors during the study.

3. METHODS INTERCOMPARISON AND QUALITY ASSURANCE/QUALITY CONTROL

Specific details on measurement methodology employed in each study may be found in the appended individual reports. This chapter describes the initial evaluation of several methods for soil, dust, hand wipe, and blood sampling and analysis that were considered by the Scientific Coordinating Committee, and the basis for selection of these methods by the participating research teams.

Soil sampling methodology was determined by agreement that a 2-cm core would be taken according to a prescribed pattern about a randomly selected point, and that this point would be selected based on the size and shape of the plot of soil. These procedures are described in the individual reports, and no further assessment was made here of the representativeness of this sampling procedure.

Interior dust sampling methods were evaluated based on the desirability of dust load information. This required that a dry sample be taken (as opposed to a wet wipe) in order to determine the mass of dust collected as a function of area (dust load). Although the sampling devices differed, the basic protocol called for a vacuum pump that collected the dust sample on a filter pad at a prescribed flow rate and using a prescribed pattern of moving the pump nozzle over the sample area. No further attempt was made to calibrate the collection devices between the individual studies.

Hand wipe samples were taken according to procedures developed by the Cincinnati group in previous studies. Field blanks and lot blanks were determined by each group. There were some differences in the timing of the hand wipe sample as reported by the individual study teams.

Blood samples were taken according to methods prescribed by CDC in their blood lead certification program. The analysis of blood for health indicators other than lead differed among the three groups. Blood data other than lead concentration were not used in this integrated assessment.

The procedures and results of interlaboratory comparisons of analytical methodology and the results of the QA/QC plan for the individual studies are described in the following sections. These procedures and their results were reviewed and evaluated throughout the project at the scheduled workshops and during monthly teleconference calls.

The research team for each study prepared a sampling and analysis plan that included rigorous QA/QC objectives. These plans included protocols that: defined sampling schemes designed to characterize the expected exposure to soil for children; described how to collect, transfer, and store samples without contamination; and described how to analyze samples with the maximum degree of accuracy and precision. Throughout the project, several intercalibration exercises were performed to guarantee that the analytical results for measurements of soil, dust, handwipes, and blood would be accurate and that the data would be comparable.

3.1 INTERCOMPARISON OF LABORATORY METHODS FOR SOIL AND DUST MEASUREMENTS

The objective of the laboratory intercomparison and QA/QC program was to ensure that the three studies could achieve a high standard of expertise in the analysis of soil and dust samples, and that each of the three laboratories would be expected to get reasonably similar results when analyzing the same soil sample. The framework for the intercomparison effort was two round robin calibration exercises, one at the beginning and one near the end of the project. In each calibration exercise, two additional laboratories were invited to participate in order to determine some measure of comparability with other studies reported in the scientific literature. All laboratories reported their results independently. In the time period between these two calibration exercises, the effectiveness of the individual QA/QC programs was also monitored by inserting double blind audit samples into the sample stream of each study to measure the persistency of analytical precision throughout the study and to monitor analytical drift.

The participating cities recognized the need for standardizing the sampling and analytical protocols so that data from each study could be compared. This standardization was accomplished for soil and dust by measuring the analytical difference between each of the three labs. Common standards were prepared and a program for assuring data quality was put into place. A three step program was agreed to that involved: (1) a round robin calibration study of soil samples to measure differences between laboratories and differences between analytical methods and instrumentation; (2) a double blind audit system for soil and dust to monitor the performance of each laboratory during the project; and (3) a second round robin calibration study to determine the arithmetic correction factor that would normalize dust and soil data to a common project basis. This program ensured that analyses performed by each of the three participating laboratories would be internally accurate and externally consistent with similar analyses by other research laboratories.

Intercalibration exercise I was conducted prior to the beginning of each study using soil and dust samples collected from representative neighborhoods in each city. Intercalibration exercise II was conducted near the end of the sampling phase of the project using aliquots of soil and dust samples collected at the beginning of the sampling phase, some of which were used for QA/QC monitoring during the project.

3.1.1 Round Robin Intercalibration Exercise I

At the beginning of this project, the methods proposed by each study for soil and dust analysis were reviewed by the Scientific Coordinating Panel. The preferred method, hot nitric acid digestion followed by atomic absorption spectroscopy (AAS), was time consuming and expensive. The number of samples was expected to exceed 75,000 per study, so more rapid and less expensive methods were evaluated. Laboratory scale X-ray fluorescence (XRF) spectroscopy and inductively coupled plasma (ICP) emission spectroscopy were proposed, and a cold nitric acid extraction method for AAS was also considered.

In May 1988, prior to the beginning of each study, each of the three laboratories collected ten soil samples from areas similar to those that would be included in their study. One of the samples from Cincinnati was a street dust sample of very high lead concentration. The other 29 samples were selected from soils with lead concentrations expected to range from 250 to 8,000 μg/g. The samples were dried and sieved according to the study protocols. Approximately 200 g of each sample were sent to the other two laboratories and to an outside lab at Georgia Tech Research Institute (GTRI). Table 3-1 shows the instrumentation and method of analysis used by each laboratory. In making these analyses, each laboratory used its own internal standards for instrumental calibration and shared a

TABLE 3-1. WET CHEMISTRY AND INSTRUMENTAL METHODS USED FOR THE FIRST INTERCALIBRATION STUDY

	Participating Laboratories				
Method[a]	Boston	Baltimore	Cincinnati	GTRI[b]	USDA[c]
Hot HNO_3/AAS		X	X		
Cold HNO_3/AAS			X		X
Hot HNO_3/ICP		X			
XRF	X			X	

[a]HNO_3 = Nitric acid; AAS = Atomic absorption spectroscopy; ICP = Inductively coupled plasma emission spectroscopy; XRF = X-ray fluorescence.
[b]GTRI = Georgia Tech Research Institute.
[c]USDA = U.S. Department of Agriculture.

common set of five standards provided by Dr. Rufus Chaney at the U.S. Department of Agriculture. The intercalibration exercise successfully established a baseline for cross study comparison of soil and dust results.

In summary, the test conditions were that each laboratory would be provided with instructions for preparing the samples (drying, sieving, and chemical extraction) but would use their own internal standards and instrumental settings. They would have access to a set of external standards (from U.S. Department of Agriculture) with known values from which they could make corrections if necessary.

Each of the three study laboratories sent aliquots of 10 samples to the other two participating laboratories and to two external laboratories. One of the samples from Cincinnati was a street dust sample with a lead concentration in excess of 15,000 μg/g. The other 29 samples were soils. The samples were subdivided by sieving during preparation to a "total" and "fine" fraction. Thus there were 30 samples, each with two size fractions analyzed by each of five laboratories using either one or two analytical methods. The analytical and wet chemistry methods used are shown in Table 3-1, and the results of the analyses appear in Table 3-2.

The cold nitric acid extraction method was found to be essentially equivalent to the hot nitric acid extraction method for soils with lead concentrations up to 8,000 μg/g (Figure 3-1) for the samples analyzed in this study. The AAS method used by Cincinnati and Baltimore

TABLE 3-2. ANALYTICAL RESULTS OF THE FIRST INTERCALIBRATION STUDY: LEAD CONCENTRATION (μg/g) IN THE TOTAL AND FINE FRACTIONS OF 10 SOILS FROM EACH STUDY

	Boston	Baltimore		Cincinnati		GTRI[a]	USDA[b]
Sample Fraction[c]	XRF	Hot HNO_3 AAS	Hot HNO_3 ICP	Hot HNO_3 AAS	Cold HNO_3 AAS	XRF	Cold HNO_3 AAS
1T	1,200	1,418	1,324	1,552	1,215	1,174	1,338
2T	1,750	2,893	2,544	2,868	2,211	1,912	2,695
3T	400	492	389	387	466	400	417
4T	550	619	462	423	415	500	464
5T	1,100	1,058	882	964	854	980	988
6T	1,450	2,323	1,955	1,876	1,722	1,524	1,808
7T	1,000	1,359	1,098	1,383	990	651	1,473
8T	500	683	535	491	725	400	726
9T	550	608	485	455	417	261	605
10T	1,450	1,649	1,330	1,679	1,228	1,660	1,764
11T	250	484	365	316	348	180	304
12T	800	1,069	878	1,850	1,103	900	1,944
13T	100		53	63	45	100	73
14T	700	2,200	1,701	2,068	1,713	652	1,710
15T	550	1,754	1,410	747	785	505	825
16T	220	264	200	253	295	187	286
17T	220	126	62	59	58	30	83
18T	75	106	48	74	61	100	111
19T	50	9	7	2	3	20	13
20T	4,800	15,792	12,030	14,593	8,147	4,817	14,733
21T	500	496	372	387	378	383	
22T	950	850	698	837	739	717	1,120
23T	1,700	1,559	1,298	1,567	1,368	1,390	1,761
24T	2,400	2,260	1,880	2,284	2,003	2,021	2,561
26T	2,800	2,484	2,119	2,754	2,401	2,331	2,472
27T	3,800	3,846	3,440	4,337	3,835	3,500	4,983
28T	5,200	5,092	4,667	5,454	4,747	4,460	3,184
29T	4,000	5,097	4,510	5,586	4,700	3,280	6,473
30T	6,500	7,995	6,560	8,467	7,502	4,704	10,042
1F	1,500	1,545	1,421	1,560	1,404	1,223	1,569
2F	2,650	3,540	2,921	3,335	3,127	2,263	3,273
3F	500	625	507	478	508	440	515
4F	1,600	1,814	1,554	1,678	1,595	1234	1,824
5F	1,700	1,793	1,475	1,689	1,971	1,290	1,683
6F	2,400	3,137	2,387	2,835	2,009	2,134	2,682
7F	1,200	1,344	1,105	1,306	1,184	815	1,297
8F	600	723	598	595	298	490	672
9F	650	686	558	593	601	375	630
10F	2,200	2,398	1,946	1,808	1,116	1,980	
11F	220	356	244	267	277	180	280
12F	1,800	2,707	2,220	2,683	2,683	1,680	2,610
13F	100	96	68	68	64	100	89
14F	800	100	779	926	818	693	895
15F	620	796	616	635	642	600	664
16F	300	3,200	236	237	239	236	242
17F	100	118	73	73	66	100	80
18F	100	142	85	91	87	100	92
19F	50		10	3	2	30	20
20F	5,100	7,866	6,000	8,109	7,432	4,780	8,451
21F	550	606	506	480	467	505	470
22F	1,100	1,118	916	1,069	944	980	904

TABLE 3-2 (cont'd). ANALYTICAL RESULTS OF THE FIRST INTERCALIBRATION STUDY: LEAD CONCENTRATION (μg/g) IN THE TOTAL AND FINE FRACTIONS OF 10 SOILS FROM EACH STUDY

	Boston	Baltimore		Cincinnati		GTRI[a]	USDA[b]
Sample Fraction[c]	XRF	Hot HNO_3 AAS	Hot HNO_3 ICP	Hot HNO_3 AAS	Cold HNO_3 AAS	XRF	Cold HNO_3 AAS
23F	1,700	1,679	1,424	1,710	1,431	1,320	1,640
24F	2,200	2,331	2,014	2,328	2,010	1,940	
25F	2,200	2,372	2,000	1,665	2,089	2,005	2,492
26F	2,800	2,899	2,402	2,946	2,568	2,249	3,156
27F	4,000	4,833	3,969	4,531	4,130	3,739	4,979
28F	3,100	3,087	2,616	3,073	2,720	2,445	6,194
29F	4,500	5,896	4,717	5,606	4,869	4,240	6,680
30F	8,000	8,555	7,443	8,679	7,789	6,015	9,754

[a]GTRI = Georgia Tech Research Institute.
[b]USDA = U.S. Department of Agriculture.
[c]T = Total fraction, F = Fine fraction.

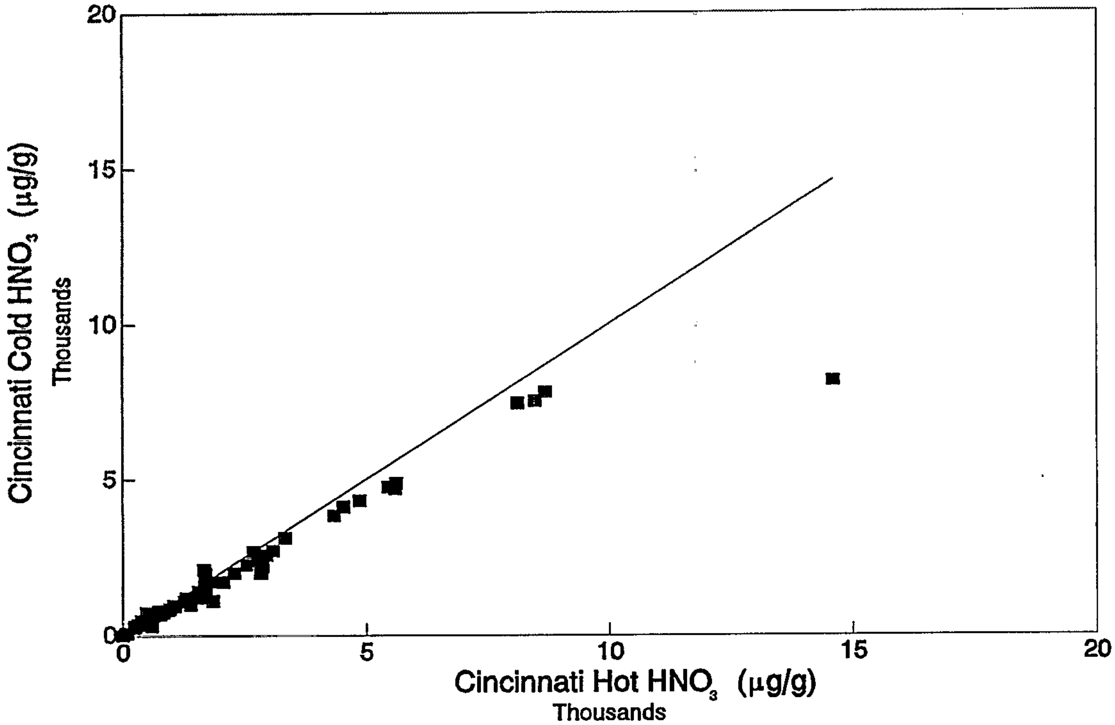

Figure 3-1. Comparison of uncorrected data for two wet chemistry methods of soil analysis showing the comparability of hot and cold nitric acid for the Cincinnati laboratory. The straight line indicates a slope of 1.

was also equivalent (Figure 3-2), showing a high degree of comparability between these two laboratories under these test conditions.

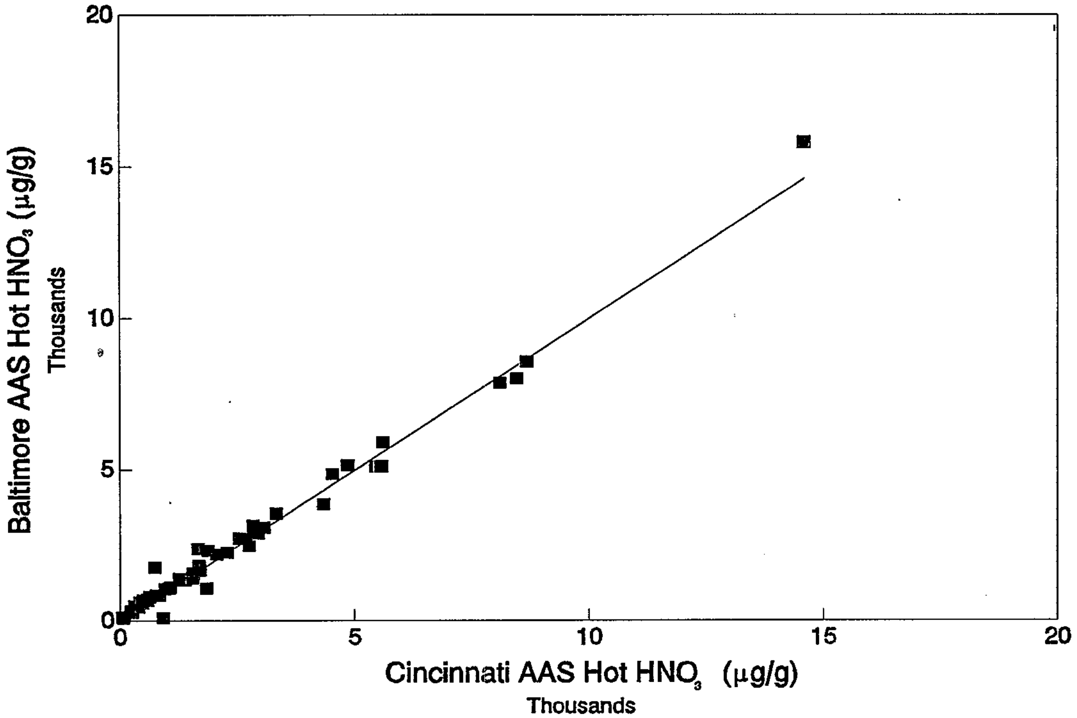

Figure 3-2. Comparison of uncorrected data for atomic absorption spectroscopic analysis by two laboratories (Baltimore and Cincinnati) using the hot nitric acid method of soil analysis. The straight line indicates a slope of 1.

The interlaboratory comparison of XRF between the Boston and GTRI Laboratories showed the method was acceptable, although not fully linear above 5,000 µg/g. There were no soil standards available above 2,000 µg/g, so the analysts had some difficulty calibrating their XRF instruments above this level. The data of Figure 3-3 suggest a systematic difference between the two laboratories that could be corrected with a more uniform calibration. Both interlaboratory (Cincinnati and Baltimore in Figure 3-4) and intralaboratory (Baltimore in Figure 3-5) comparisons of AAS versus ICP demonstrated equivalency between these two instrumental methods. These comparisons showed that there is likewise a systematic difference that can be statistically corrected.

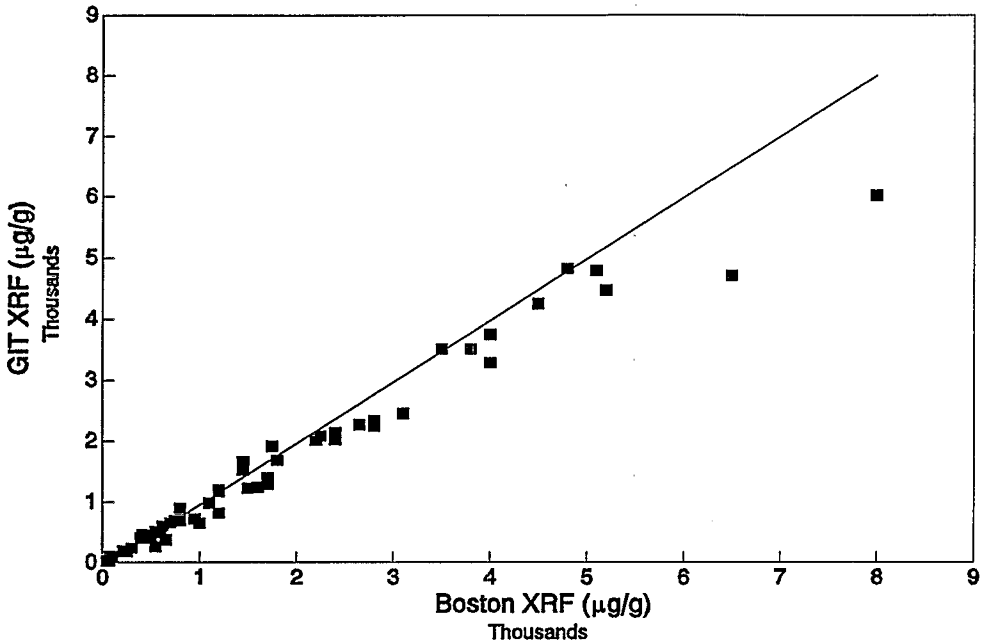

Figure 3-3. **Interlaboratory comparison of uncorrected data for the X-ray fluorescence method of soil analysis showing the comparability of the Boston and Georgia Institute of Technology laboratories. The straight line indicates a slope of 1.**

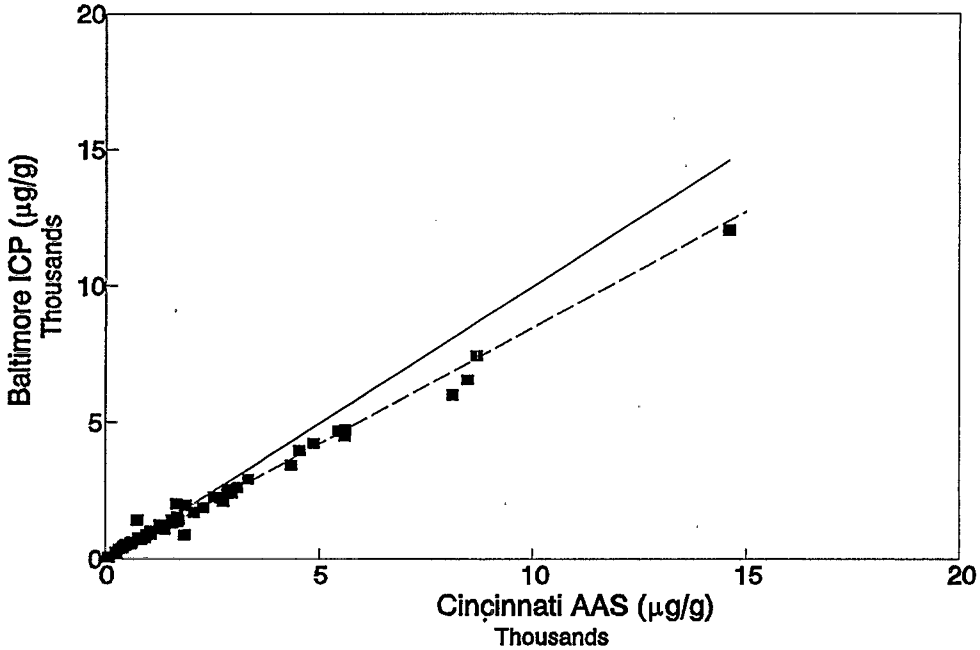

Figure 3-4. **Interlaboratory comparison of uncorrected data for soil analysis showing the comparability of inductively coupled plasma emission spectroscopy and atomic absorption spectroscopy for the Baltimore and Cincinnati laboratories. The straight line indicates a slope of 1.**

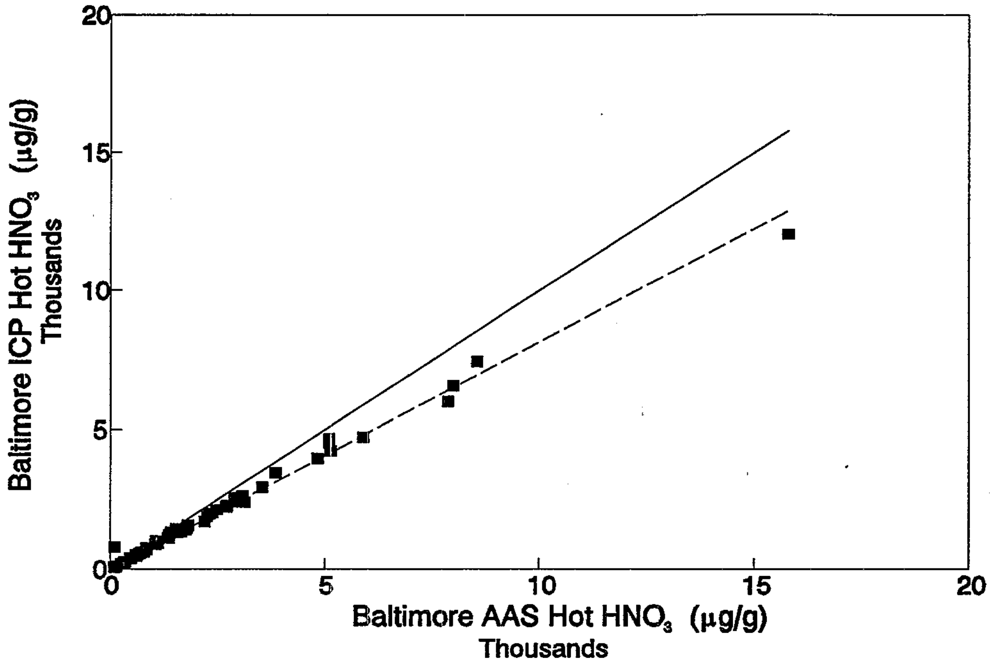

Figure 3-5. Comparison of uncorrected data for soil analysis showing the comparability of inductively coupled plasma emission spectroscopy and atomic absorption spectroscopy within the Baltimore laboratory. The straight line indicates a slope of 1.

Finally, the interlaboratory comparison of XRF versus AAS (Boston and Cincinnati in Figure 3-6, and Boston and Baltimore in Figure 3-7) led to the conclusion that, if suitable soil standards at higher concentrations could be made available, XRF would be an acceptable alternative method to AAS for soil analysis.

The Scientific Coordinating Panel recommended the use of XRF for soil analysis on the condition that a suitable set of common standards could be prepared for a broader concentration range and that a rigorous audit program be established to ensure continued analytical accuracy. This recommendation was based on the interlaboratory comparison study, the awareness that chemical extraction of a large number of soil samples presented a costly burden on the project both in terms of time and expense, and the value of nondestructive analysis in preserving the samples for reanalysis. The Round Robin I calibration exercise also revealed the need for a broader scale calibration exercise to determine the arithmetic correction factor for converting the data to a common basis.

For routine analyses, two groups, Boston and Baltimore, elected to use XRF for interior dust analysis also, whereas Cincinnati opted for hot nitric extraction with AAS for

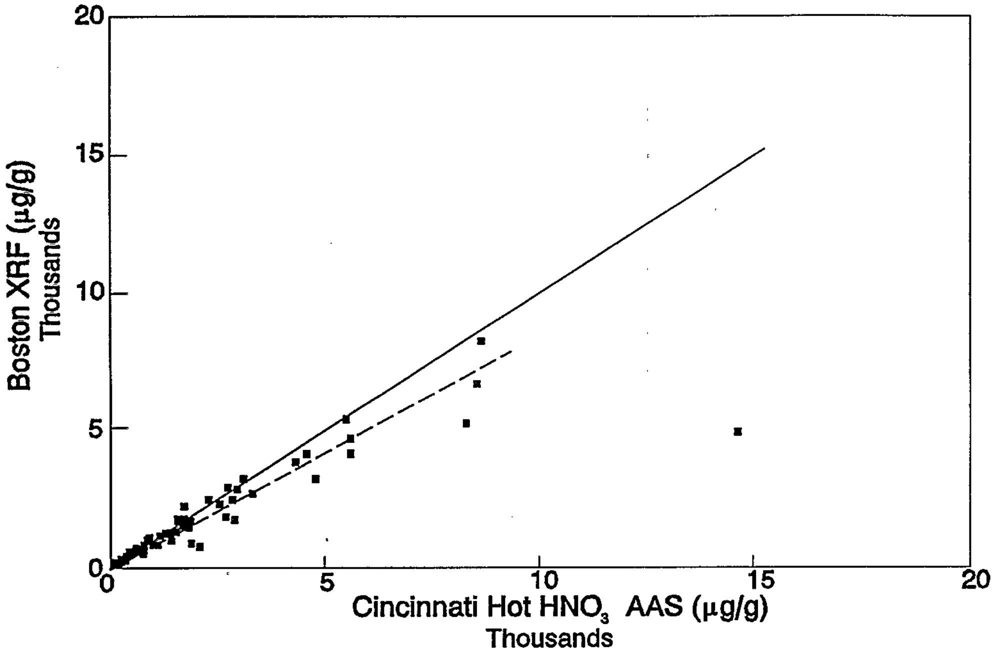

Figure 3-6. Interlaboratory comparison of uncorrected data for soil analysis showing the comparability of X-ray fluorescence and atomic absorption spectroscopy for the Cincinnati and Boston laboratories. The straight line indicates a slope of 1.

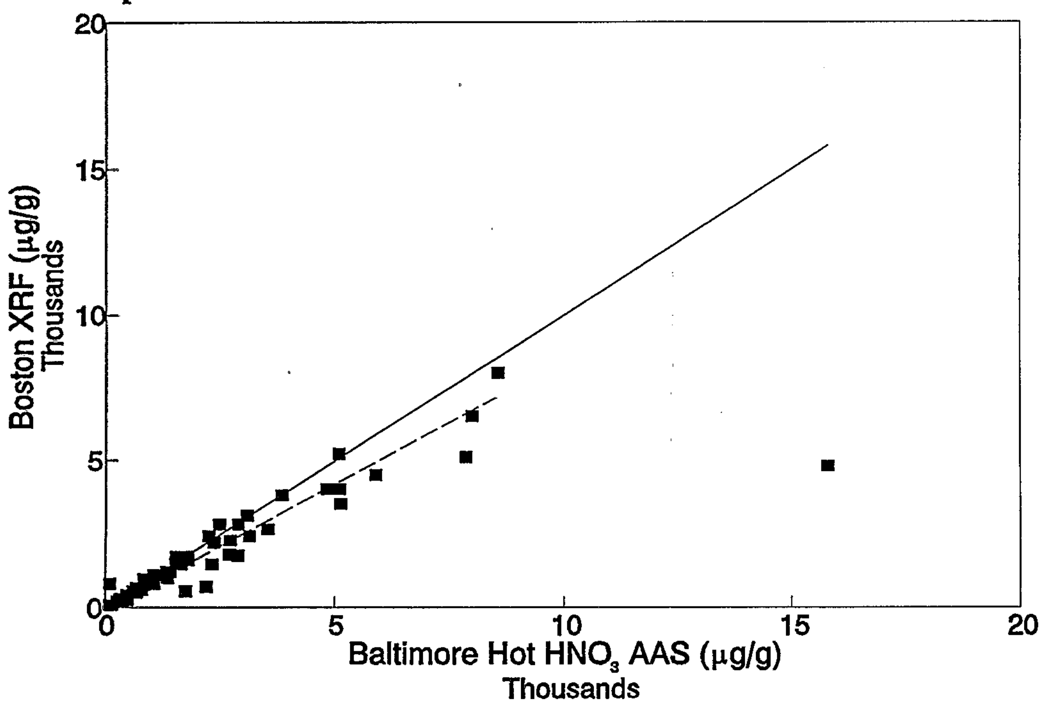

Figure 3-7. Interlaboratory comparison of uncorrected data for soil analysis showing the comparability of X-ray fluorescence and atomic absorption spectroscopy for the Baltimore and Boston laboratories. The straight line indicates a slope of 1.

interior dust and XRF for exterior dust. During the study, Baltimore recognized problems with analyzing dust by XRF when the sample size was small, less than 100 mg. They reanalyzed the dust samples by AAS and reported both measurements. In Boston, this problem was solved by compositing the floor dust samples for XRF analysis, reporting one floor dust sample per housing unit.

3.1.2 Quality Assurance/Quality Control Standards and Audits

After the first intercalibration exercise, a set of nine interlaboratory standards was prepared to monitor the QA/QC performance of soil and dust analysis throughout the project. These were prepared from three soil samples and two dust from each of the three studies, collected in bulk (about 30 kg), in a range thought to be high, medium, and low for that area. Seven of the soil samples and five of the dust samples were dried, sieved, and analyzed at the EPA Environmental Monitoring Systems Laboratory in Las Vegas, NV (EMSL/LV). Following homogenization, approximately fifty aliquots of each of the samples were analyzed by laboratory scale XRF at the EMSL/LV laboratory to estimate the acceptable range for a single laboratory. Three of the nine soils were distributed to the participating cities for use as interlaboratory reference standards. The remaining six were used as double blind external audits.

Each city appointed a QA/QC officer who was not directly involved with the analysis of the soil samples, but who had access to the soil sample preparation stream on a daily basis. This person mailed prelabeled soil sample containers with typical sample numbers to the EMSL/LV laboratory. Approximately 20 g samples from one of the six external audit materials typical for each city were placed in the sample containers fully disguised as field soil samples and returned to the QA/QC officer in lots of 20 to 30. The identification numbers and soil concentration values were monitored by the project QA/QC officer at ECAO/RTP. Each city's QA/QC officer inserted the double blind samples into the sample stream on a random basis at a frequency that would ensure about four QA/QC samples per analytical day. These were occasionally placed as duplicates in the same batch to provide information about replication within the batch.

The preliminary acceptance range for the double blind audit samples was established using the original 50 XRF analyses by the Las Vegas laboratory discussed above. As the analytical results were reviewed by the study QA/QC officer, the audit sample results were sent to the project QA/QC officer at ECAO/RTP. If the audit samples were outside the acceptable range, the study QA/QC officer was informed and could recommend either reanalysis or flagging the data for that entire batch. The initial acceptable range for the six audit samples was based on analyses by a single laboratory (EMSL/LV). This range was adjusted for interlaboratory variation after the Intercalibration Exercise II. Final decisions on the disposition of the audit sample anomalies were deferred until the completion of the second intercalibration exercise near the end of the study.

The results of the double-blind audit program are given in Table 3-3 based on the final biweight distributions in Table 3-4. The preliminary biweight distributions, shown also in Table 3-4, contained no measure of interlaboratory variability because the preliminary analyses were performed by only the EMSL-LV laboratory. These values could only be used in a preliminary assessment of the audit program to identify and flag batches of soil samples that might need to be reanalyzed pending the determination of the final biweight distributions.

The laboratories were found to be systematically low or high. This was not of major concern, as these discrepancies could be resolved by a more detailed intercalibration exercise and statistical correction at the end of the study. The Cincinnati group elected to make a midcourse change in instrumental parameters that reduced this difference, and they described this procedure in their report. Occasionally, the measured audit sample was sporadically high or low, in which case the laboratory investigated the problem and resolved it. Most of these discrepancies occurred for dust samples where the sample size for XRF analysis was below 200 mg. The Boston group found, but did not report in detail, that a calibration curve for XRF analysis using standards that were also less than 200 mg would provide a suitable correction to the original data. They elected, however, to composite their floor dust samples.

3.1.3 Round Robin Intercalibration Exercise II

Near the end of the project, aliquots of the nine soil and six dust audit samples used during the project were redistributed to the three study laboratories for single blind analysis. The analyst was aware that the samples were audit samples, but did not know their

TABLE 3-3. SOIL AND DUST AUDIT PROGRAM RESULTS

Study/Audit Sample	Number of Samples	Mean (µg/g)	Range (µg/g)	Percent Within Final Biweight Distribution[a]
BOSTON DUST (XRF)				
BAL 03	N/A[b]	1,232	980-1,441	92
CIN 01	N/A	2,671	2,075-3,228	100
CIN 02	N/A	331	115-461	65
BOSTON SOIL (XRF)				
BOS M	N/A	6,786	6,015-7,549	100
BAL H	N/A	1,044	747-1,244	73
CIN L	N/A	399	207-570	61
CIN H	N/A	14,074	11,407-16,592	50
BALTIMORE DUST (XRF)				
BAL 02	8	218	159-281	100
CIN 01	10	3,280	800-3,660	90
BOS 01	10	14,444	14,080-14,920	100
BALTIMORE SOIL (XRF)				
BOS M	15	5,046	4,800-5,200	100
BAL H	15	838	433-916	60
CIN L	15	286	266-307	100
CIN H	15	11,290	10,100-12,500	53
CINCINNATI DUST (AAS)				
BAL 03	34	1,727	1,322-2,687	N/A
BOS 01	35	24,104	20,266-27,962	N/A
CIN 01	38	2,683	2,070-3,163	100
CIN 02	26	259	200-393	100
CINCINNATI SOIL (XRF)				
BOS M	32	5,580	4,759-6,107	100
BAL H	49	885	822-1,012	100
CIN L	130	263	244-310	100
CIN H	31	12,304	9,838-13,632	N/A

[a]These percentages include audit samples for which analyses were outside the biweight distribution range and for which the action required by the QA/QC plan, such as reanalysis of the entire batch, was implemented.
[b]N/A = Not available.

TABLE 3-4. PRELIMINARY AND FINAL BIWEIGHT DISTRIBUTIONS FOR SOIL AND DUST AUDIT PROGRAM

Sample Type	Audit Sample	Preliminary Values ($\mu g/g$)			Final Values ($\mu g/g$)		
		Mean	Low	High	Mean	Low	High
Dust	BAL01	78	58	99	84	4	163
Dust	BAL02	331	288	374	309	138	480
Dust	BAL03	1,480	1,346	1,613	1,438	1,091	1,786
Dust	CIN01	2,851	2,660	3,042	2,617	1,422	3,812
Dust	CIN02	252	216	288	233	93	372
Soil	BOS L	3,131	2,858	3,405	3,101	2,283	3,919
Soil	BOS M	6,090	5,748	6,431	6,219	4,742	7,696
Soil	BOS H	14,483	13,071	15,895	13,369	11,980	14,754
Soil	BAL L	639	555	724	626	468	783
Soil	BAL H	923	850	997	1,017	847	1,187
Soil	CIN L	303	284	322	315	204	426
Soil	CIN H	13,585	12,872	14,297	12,729	11,361	14,096
Soil	REF5				413	258	568
Soil	REF6				936	738	1,134
Soil	REF7				1,042	758	1,326
Soil	REF8				2,354	1,950	2,759
Soil	REF9				3,913	2,943	4,888
Soil	REF10				735	615	854

concentrations. These measurements were the basis for establishing the final range of acceptability for the audit samples, and for adjusting the soil and dust measurements in each study to values common to the project.

3.1.4 Biweight Distribution and Final Interlaboratory Calibration

The nine soil and five dust samples that were used for external standards and audit samples were reanalyzed in a more detailed round robin exercise near the end of the project.

The purpose of this exercise was to determine the correction factor for statistically converting the soil and dust data from each study to a common basis and to revise the biweight distribution values for the audit samples to reflect the multilaboratory variance and systematic differences between laboratories. Additional analyses by AAS were performed by Baltimore and Cincinnati for soil and dust, even though only dust was analyzed by AAS during the study. Boston and Las Vegas analyzed the samples by ICP for the purposes of obtaining a broader perspective on the application of this method. The data from this exercise are in Table 3-5. They are the basis for determining the consensus values and correction factors that appear in Table 3-6.

The data evaluation subcommittee of the Scientific Coordinating Panel was appointed to determine the consensus values and methods of statistical interpretation of the intercalibration results. Several methods were discussed in great detail. Tests were made for outliers using the method of Barnett and Lewis (1984), and none were found. The data were of good quality and were highly linear. The r^2 values ranged from 0.997 to 0.999 using a consensus based on the simple arithmetic means of the reported values. The subcommittee chose to explore alternatives to the arithmetic mean and eventually settled on a multiplicative model weighted for within-laboratory variance. The model was run with GLIM statistical software, Version 3.77, Update 2, and gave consensus values and correction factors shown in Table 3-6. Although great care was taken to evaluate several alternatives to simple regression, the consensus values produced by the GLIM procedure differed only slightly from those of a simple linear regression. The correction factors on Table 3-6 were used by the three studies to convert their soil and dust data to a common project basis. A plot of the dust (Figure 3-8) and soil (Figure 3-9) reported values versus the consensus means derived from the GLIM analysis illustrates the reliability of this method.

3.1.5 Disposition of Audit Data

Based on the results of the second intercalibration exercise, a consensus value was determined for each dust and soil sample, biweight distributions were determined for those that had been used in the audit program. This new distribution incorporated interlaboratory variation. When the correction factor is applied to the reported results, the revised number should lie between the upper and lower boundaries of the biweight distribution. Table 3-3

TABLE 3-5. RESULTS OF THE FINAL INTERCALIBRATION STUDY (μg/g)

	XRF					AAS		ICP	
Sample	BOSK	BOSX	BAL	CIN	LV	BAL	CIN	BOS	LV
DUST1	120		121	92	78	15	66	94	72
DUST2	320		482	329	288	201	236	284	307
DUST3	1,430		1,686	1,307	1,288	1,363	1,581	1,428	1,346
DUST4	2,000		3,771	2,924	2,456	2,335	2,451	2,109	2,296
DUST5	280		267	233	212	150	273	244	191
SOIL1	450	510	388	441	310	383	452	401	379
SOIL2	900	910	808	1,033	833	1,001	1,013	850	912
SOIL3	1,050	1,100	961	1,080	923	1,100	1,120	972	1,006
SOIL4	2,200	2,300	2,100	2,555	2,264	2,468	2,502	2,230	2,286
SOIL5	3,800	4,000	3,486	4,227	3,974	4,044	4,251	3,748	3,843
SOIL6	710	770	640	789	611	741	798	699	660
SOIL7	650	930	559	675	532	567	650	597	626
SOIL8	950	930	896	1,036	798	1,032	1,067	944	998
SOIL9	2,800	2,900	2,514	3,126	2,972	3,401	3,263	3,148	3,158
SOIL10	5,600	5,300	5,200	6,493	5,956	6,861	6,937	5,932	6,360
SOIL11	12,500	13,000	11,000	15,963	15,984	13,175	13,955	12,652	12,608
SOIL12	310	290	283	305	286	321	379	300	294
SOIL13	12,000	12,000	10,500	14,156	13,530	13,000	13,195	13,167	11,440
SOIL14	810	850	793	929	763	875	986	907	900
SOIL15	1,450	1,600	1,400	1,705	1,509	1,731	1,766	1,631	1,650

lists the percentage of these audit sample values that fell within these new boundaries. Most of the discrepancies were resolved by the corrective measures taken by the laboratories.

When the audit sample values fell outside the boundaries of the final biweight distribution, the batches were flagged. The options could then be to exclude these data from the statistical analysis, reanalyze the samples, or use the original data based on other evidence that the data are correct. The quality of soil and dust analysis in this project was equal to or greater than the generally acceptable standards for reporting soil and dust data in the scientific literature.

TABLE 3-6. CONSENSUS VALUES AND CORRECTION FACTORS FROM THE FINAL INTERCALIBRATION PROGRAM

	XRF	AAS	ICP
	Interlaboratory Consensus Values for Dust (μg/g)		
Sample			
DUST1	92.8	54.2	81.7
DUST2	342.7	221.9	283.4
DUST3	1,319.0	1,492.2	1,362.3
DUST4	2,943.4	2,378.1	2,133.4
DUST5	228.3	232.4	206.2
	Interlaboratory Correction Factors[a]		
Study			
BOS	1.1527		1.0707
BAL	0.7803	1.0416	
CIN	1.0074	0.9616	
	Interlaboratory Consensus Values for Soil (μg/g)		
Sample			
SOIL1	460.2	430.5	426.6
SOIL2	960.7	1,002.1	909.6
SOIL3	1,140.5	1,106.2	1,018.8
SOIL4	2,493.5	2,474.2	2,342.1
SOIL5	4,139.3	4,164.1	3,706.1
SOIL6	761.0	776.9	736.1
SOIL7	664.1	623.3	656.0
SOIL8	1,062.3	1,049.4	1,005.4
SOIL9	2,987.8	3,272.6	3,274.9
SOIL10	6,175.2	6,863.2	6,411.5
SOIL11	13,120.7	13,645.4	13,224.7
SOIL12	335.3	361.5	323.6
SOIL13	12,498.5	13,041.6	13,080.0
SOIL14	941.3	949.5	923.3
SOIL15	1,663.2	1,744.1	1,716.8
	Interlaboratory Correction Factors for Soil[a]		
Study			
BOS	1.0370		1.0166
BAL	1.1909	1.0166	
CIN	0.8698	0.9839	

[a] The correction factor is the value that the reported soil or dust measurement should be multiplied by in order to adjust each value to a common basis among all three studies.

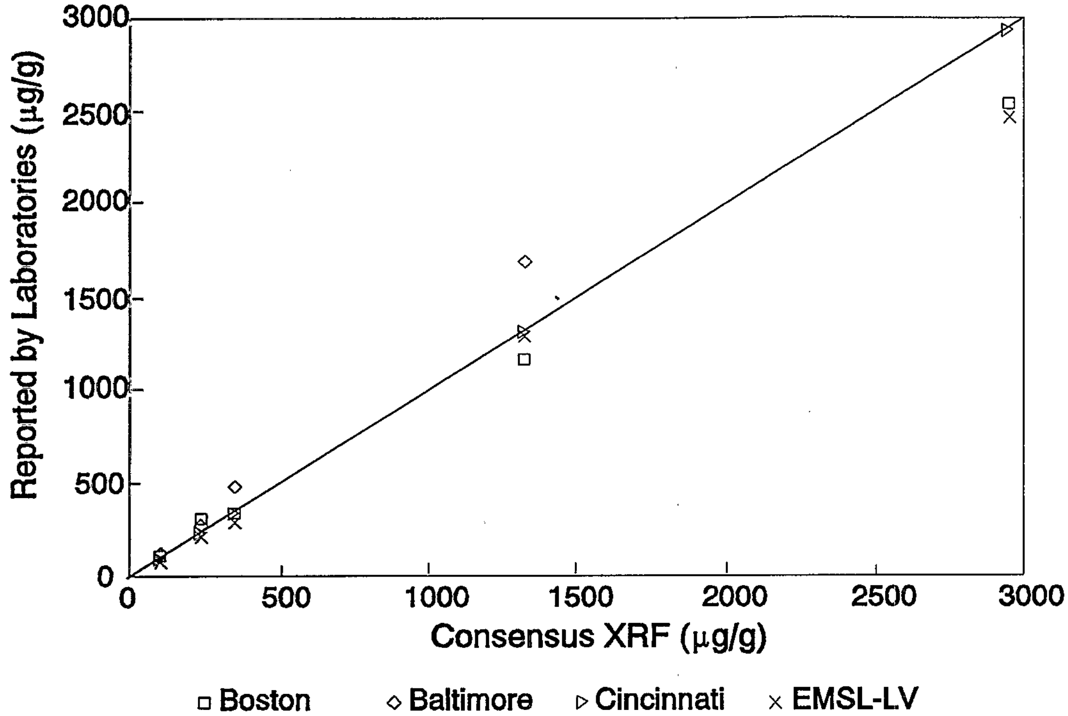

Figure 3-8. Departures from consensus dust values for each of the three studies.

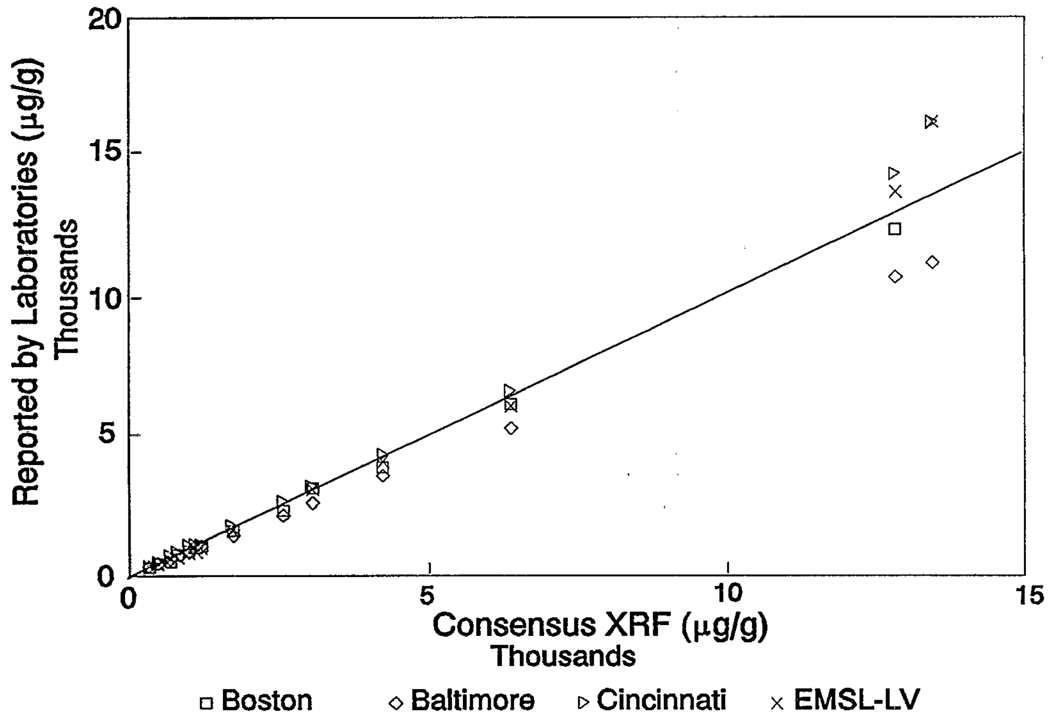

Figure 3-9. Departures from consensus soil values for each of the three studies.

3.2 QUALITY ASSURANCE AND QUALITY CONTROL FOR HAND DUST

The collection and analysis of hand wipes is an innovative procedure developed just prior to the beginning of the project. There were few published reports of the measurement techniques, no certified standards, no internal standards, and little information on which to base decisions for acceptable analytical precision. Double blind audit samples were provided to the study QA/QC officer as an external control for hand wipe analysis. These were prepared as simulated samples by placing a known amount of an appropriate solution of lead nitrate onto the blank hand wipe at the EMSL/LV laboratory, wrapping and labeling according to the field protocol and returning to the participating laboratory for insertion into the sample scheme. There was no attempt to determine interlaboratory variance or to calculate correction factors. The study QA/QC officer was responsible for reporting problems to the laboratory director.

3.3 QUALITY ASSURANCE AND QUALITY CONTROL FOR BLOOD LEAD

The QA/QC program for blood analysis was directed by Dr. Dan Paschal of the Centers for Disease Control and Prevention (CDC) using the protocols developed for the CDC blood lead certification program. Each laboratory received double blind bovine blood samples from CDC Blind Pool 1 and Blind Pool 2. The data from this QA/QC program are in Table 3-7. These data report the number of exceedances to be zero for all three studies. An exceedance occurs when the mean of two replicates exceeds the range established by CDC. The data also report the probability of analytical drift during the period of analysis. There was evidence for drift in the Boston Blind Pool 2 and marginal evidence in Cincinnati Blind Pool 1.

3.4 DATABASE QUALITY

Each study maintained rigorous standards for database quality. These included double entry, 100% visual confirmation, and standard statistical procedures for detecting outliers.

TABLE 3-7. QUALITY CONTROL RESULTS FOR CENTERS FOR DISEASE CONTROL AND PREVENTION BLIND POOL BLOOD LEAD ANALYSES

		Blind Pool 1			Blind Pool 2		
Study	Dates	n	Number of Exceedances[1]	Drift[2]	n	Number of Exceedances[1]	Drift[2]
Boston	Jul 89 - Aug 91	123	0	0.2092	112	0	0.0389
Baltimore	Aug 88 - Oct 90	66	0	0.6382	59	0	0.4748
Cincinnati	Aug 88 - Oct 90	53	0	0.0672	48	0	0.4732

[1]Number of samples that exceeded the range established by CDC for each batch of QC blood analyses within a pool.

[2]The drift test probability is a P-value for the test of the hypothesis that the slope of the difference between the reported values and the CDC accepted value is significantly greater than zero. A P-value less than 0.05 indicates this slope may be greater than zero and that some analytical drift may have occurred over time, but the direction of this possible drift is not indicated by this statistic.

In reviewing the data for statistical analyses contained in this Integrated Report, some errors were found, confirmed, and corrected prior to use in this assessment. None of these errors would have impacted the conclusions drawn by the individual study reports.

This evaluation of the QA/QC data shows that the three studies were comparable in their ability to meet the requirements of their QA/QC program. Furthermore, their performance on the audit program and intercalibration exercises suggests that the data are comparable among the three studies, with the appropriate correction factors shown in Table 3-6. While the QC data for Boston blood lead analyses suggest the possibility of analytical drift for part of the period where blood lead data were being corrected, the statistical methods for evaluating abatement effectiveness used by the investigators and by this assessment would compensate for any possible analytical drift.

4. INDIVIDUAL STUDIES

4.1 INDIVIDUAL STUDY INTERVENTION STRATEGIES AND SAMPLE PLANS

4.1.1 Boston Study

The pathway intervention scheme for Boston is shown in Figure 4-1. The approach to soil abatement was to remove the top 15 cm of soil, apply a synthetic fabric, and cover with a layer of about 20 cm of clean topsoil. The new soil was covered with sod or seeded with grass and watered through dry months. Areas not resodded were covered with a bark mulch. Some driveways and walkways were covered with 5 cm soil and 15 cm gravel or crushed bank (stone with dust). On four properties, the driveway and yard were capped with 7.5 cm asphalt without soil removal, at the owner's request. A total of 93 Boston properties, including those abated at the end of the project, were abated in this manner. The information on area treated and volume of soil removed from these properties appears in Table 4-1. The method of excavation was by small mechanical loader (Bobcat) and hand labor, for the most part. Initially, six properties were abated with a large vacuum device mounted on a truck, but this proved unsatisfactory due to the size and lack of maneuverability. During one extreme cold spell, it was necessary to remove large blocks of frozen soil, often greater than 15 cm thick, by loosening with a jackhammer.

Interior dust abatement was performed after loose paint stabilization. Families spent the day off-site during interior dust abatement. Hard surfaces (floors, woodwork, window wells, and some furniture) were vacuumed with a High-Efficiency Particle Accumulator (HEPA) vacuum, as were soft surfaces such as rugs and upholstered furniture. Hard surfaces were also wiped with a wet cloth (an oil treated rag was used on furniture) following vacuuming. Common entries and stairways outside the apartment were not abated.

In Boston, loose paint stabilization consisted of removing chipping and peeling paint with a HEPA vacuum and washing the surfaces with a trisodium phosphate and water solution. Window wells were painted with a fresh coat of primer.

Although subsequent measurements of lead-based paint were made, no measurements were made of the movement of lead from paint to house dust that would reflect the

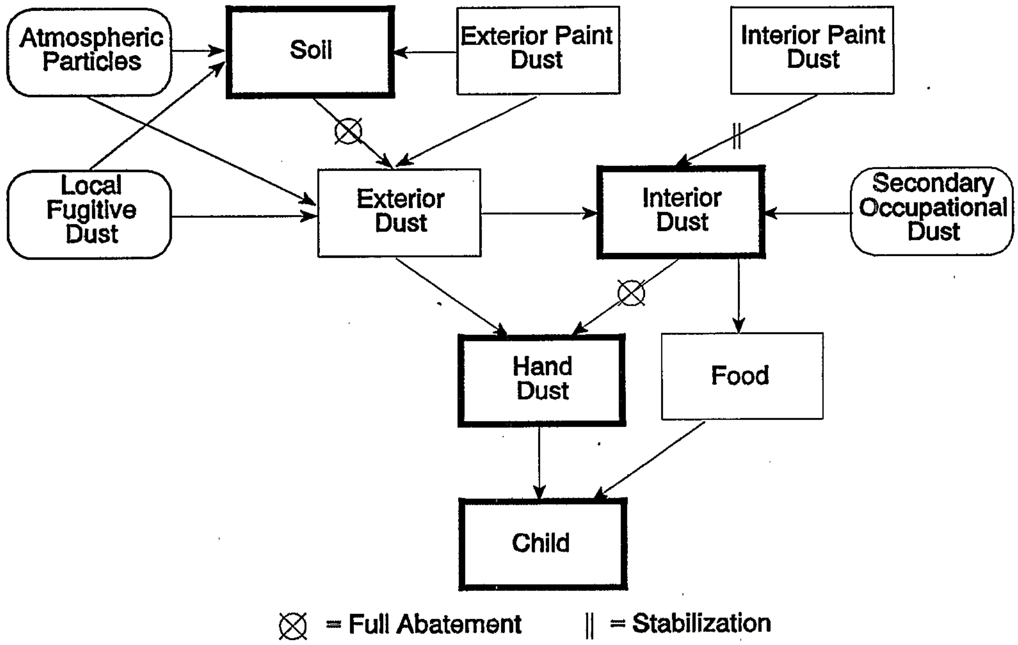

Figure 4-1. Pathway intervention scheme for dust exposure (Boston Soil Abatement Study). Bold-line rectangles indicate pathway components monitored by sequential sampling.

TABLE 4-1. SOIL ABATEMENT STATISTICS FOR THE THREE STUDIES

	Boston	Baltimore	Cincinnati
Number of properties[a]	36	63	171
Surface area (m^2)	7,198	4,100[b]	12,089
Volume soil removed (m^3)	1,212	690	1,813
Surface area/property (m^2)	200	73	71
Volume soil/property (m^3)	34	11	11

[a]Includes only properties abated during study. Properties abated at the end of the study, where no further sampling was reported, are not included in this analysis, but are included in the individual study reports. In Cincinnati, a property is the location of the soil abatement, not the location of the child's residence.
[b]Surface area not provided by Baltimore report. This was calculated using Boston volume-to-surface ratio, which is equivalent to an average removal depth of 17 cm.

effectiveness or persistency of paint stabilization. It was believed that any contamination from lead-based paint would be readily apparent in the dust samples.

The Boston study retained 149 of the original 152 children enrolled. Twenty-two of the 149 children moved to a new location but were retained in the study. Children with blood lead concentrations below 7 μg/dL or above 24 μg/dL had been excluded from the study and two of the 149 children were dropped from the data analysis when they developed lead poisoning, probably due to exposure to lead-based paint outside their home.

Baseline characteristics (age, SES as derived from the Hollingshead Index, soil lead, dust lead, drinking water lead, and paint lead) were similar for the three Boston study groups (BOS P, BOS PI, BOS SPI). The preabatement blood lead concentration was higher for BOS P. The proportion of Hispanics was higher in BOS P than in BOS PI or BOS SPI, and the proportion of Blacks was lower. There was a larger proportion of male children in BOS P.

Data were analyzed by comparison of group means using analysis of covariance (ANCOVA), which showed a significant effect of group assignment (intervention) for both the BOS PI and BOS SPI groups. These results did not change with age, sex, socioeconomic status, or any other variable except race and paint loading (P-XRF measurement). When the paint loading was controlled, the blood lead declines were diminished; when the race variable was added, the blood lead declines were also diminished and the results were not statistically significant.

The Boston study has some limitations. Participants were chosen to be representative of the population of urban preschool children who were already at risk of lead exposure. The Boston Childhood Lead Poisoning Prevention Program was used to identify potential participants from neighborhoods with the highest rates of lead poisoning. Because no study subjects had blood lead levels below 7 μg/dL or in excess of 24 μg/dL at baseline, extrapolation of the effect of lead contaminated soil abatement for children above or below this range is difficult.

Follow-up blood lead measurements were made in Boston eleven months after intervention and again at 23 months.

4.1.2 Baltimore Study

In Baltimore, 63 properties in BAL SP were abated between August and November 1990. An additional seven properties that did not meet the requirements for abatement were transferred to the control group (BAL P). The pathway intervention scheme is shown in Figure 4-2. Soil surfaces were divided into parcels on each property, usually front, back, and one side; and any parcel with soil lead concentrations above 500 μg/g was abated entirely. Soil and ground cover were removed down to 15 cm and replaced to the original level with soil having a lead concentration less than 50 μg/g. These areas were sodded or reseeded as appropriate. Bare areas were prepped and reseeded even if soil lead concentrations did not warrant excavation. Additional abatement statistics appear in Table 4-1.

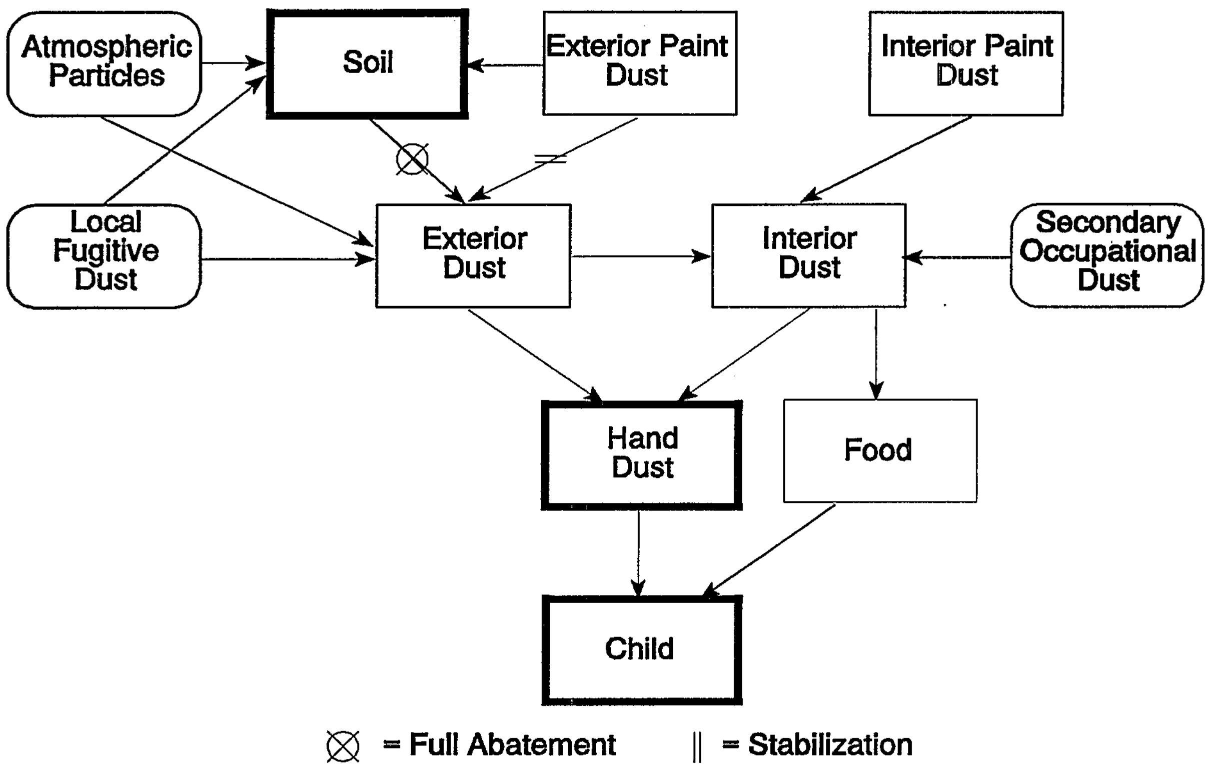

Figure 4-2. Pathway intervention scheme for dust exposure (Baltimore Soil Abatement Study). Bold-line rectangles indicate pathway components monitored by sequential sampling.

The exterior painted surfaces of Baltimore homes were wet scraped over the chipping and peeling surfaces, followed by HEPA vacuuming. The entire surface was primed and painted with two coats of latex paint.

The Baltimore study recruited 472 children, of whom 185 completed the study. Of those that completed the study, none were excluded from analysis. The recruited children were from two neighborhoods, originally intended to be a treatment and a control group. Because soil concentrations were lower than expected, some properties in the treatment group did not receive soil abatement. In their analysis, the Baltimore group transferred these properties to the control group.

Because of logistical problems, there was an extended delay between recruitment and soil abatement that accounted for most of the attrition of the participating families from the study. In their report, the Baltimore group applied several statistical models to the two populations to evaluate the potential bias from loss of participating children. These analyses showed the two populations remained virtually identical in demographic, biological and environmental characteristics.

The Baltimore study design focused on changes in biological parameters, hand dust and blood lead, over an extended period of time. The study provided limited information on changes in the movement of lead in the child's environment in response to intervention. Repeat measurements of soil were on abated properties only, to confirm abatement. There were no abatement measurements of exterior dust, no interior paint stabilization, and no interior dust abatement.

Including the prestudy screening measurements of hand dust and blood lead in the original cohort of participants, the Baltimore study made six rounds of biological measurements that spanned twenty months.

4.1.3 Cincinnati Study

The pathway scheme for the Cincinnati study is shown in Figure 4-3. Within each of six neighborhoods, the Cincinnati study identified all sites with soil cover as discrete study sites. The decision to abate was based on soil lead concentrations for each parcel of land, and for the depth to which the lead had penetrated. Lead was measured at two depths, the

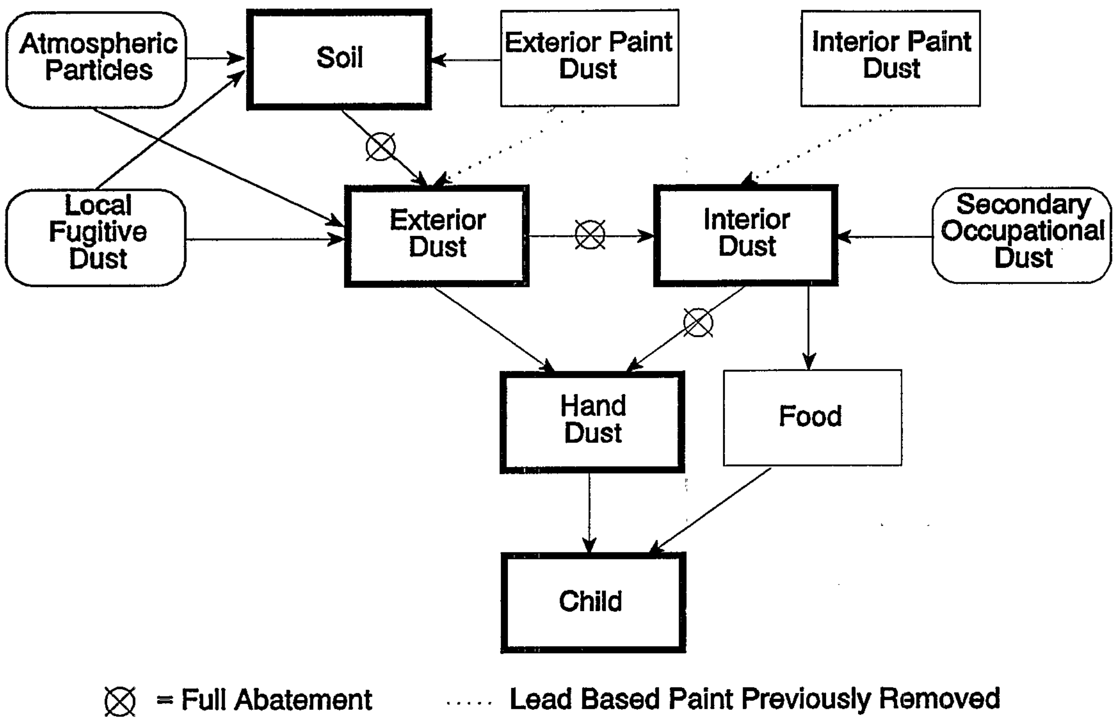

Figure 4-3. Pathway intervention scheme for dust exposure (Cincinnati Soil Abatement Study). Bold-line rectangles indicate pathway components monitored by sequential sampling.

top 2 cm and from 13 to 15 cm. If the average concentration of the top and bottom samples was 500 μg/g or greater, the soil was removed and replaced, regardless of the adequacy of the top cover. If the average of the top samples exceeded 500 μg/g, the soil was also abated. Initially, there was an option to cultivate by roto-tilling, but this approach was abandoned as not feasible in this study. For areas where the top concentration was greater than or equal to 300 μg/g, and the average concentration of the top and bottom samples was less than 500 μg/g and the cover was inadequate, the soil was resodded. Excavation was by front end loader, backhoe, and hand tools down to 15 cm, and the replacement soil lead concentration was less than 50 μg/g. Further abatement statistics can be found in Table 4-1.

The approach to exterior dust abatement was to identify all types of exterior hard surfaces in the neighborhood where dust might collect, to obtain permission to sample and abate these areas, and to clean them once with vacuum equipment, suitable for the area. This vacuum equipment had previously been tested and shown to remove about 95% of the available dust on the area. The groups of surfaces selected were streets, alleys, sidewalks, parking lots, steps, and porches. For data analysis in the Cincinnati report, these were grouped as (1) targeted areas adjacent to the exterior of the buildings where children lived, such as steps, porches, and sidewalks; (2) streets, sidewalks, and alleys throughout the study neighborhoods; and (3) parking lots and other paved areas throughout the study neighborhoods.

The exterior dust measurements in the Cincinnati study (and the interior dust measurements of all three studies) were made in a manner that determined the lead concentration (μg Pb/g dust), the dust loading (mg dust/m^2), and the lead loading (μg Pb/m^2) for the surface measured. This required that a dry vacuum sample be taken over a prescribed area, usually 0.25 to 0.5 m^2. It is important to note that dust abatement is not expected to cause an immediate change in the lead concentration on dust surfaces, only the dust and lead loading.

The Cincinnati group performed interior dust abatement after exterior dust abatement, moving the families off-site during this activity. Vacuuming of noncarpeted areas, which was done two times, at a prescribed rate of 1 m^2/min, was followed by wet wiping with a detergent. They replaced one to three carpets and two items of upholstered furniture per housing unit. Their previous studies had shown that these soft items could not be cleaned effectively with vacuuming alone. Where carpets could not be replaced, these were vacuum cleaned three times at a rate of 1 m^2/min, recognizing the limitations of this method.

The Cincinnati study recruited 307 children, including 16 children born to participating families during the study, and an additional 50 children who were recruited after the beginning of the study. In their main data analysis, the Cincinnati group excluded these children who were recruited after the start of the study, plus 31 children who were living in nonrehabilitated housing suspected of having lead-based paint, and four children (in two families) who had become lead-poisoned from other causes. Thus, data for 206 children were analyzed in the Cincinnati report.

The Cincinnati study abated soil on 140 parcels of land scattered throughout the neighborhoods. In CIN SEI, where soil abatement was performed in the first year, the arithmetic mean concentration dropped from 680 μg/g down to 134 μg/g. In the two groups where soil abatement occurred in the second year, CIN I-SE-1 and CIN I-SE-2, the soil lead concentration dropped from 262 to 125 μg/g and 724 to 233 μg/g, respectively.

If soil were the only source of lead in the neighborhoods, exterior and interior dust should have responded to the reduction in soil lead concentrations. Exterior dust lead loading decreased only slightly following soil and dust abatement, but returned to preabatement levels within one year. The analysis of exterior dust should provide a measure intermediate between external sources, such as soil, and house dust. In the case where the soil was abated, then abatement of external dust should speed up the rate at which the impact of this soil abatement can be observed on the interior dust of homes. But soil is not the only source of exterior lead, especially if the distance between the soil and the living unit entry way is more than a few hundred feet. In this case, the recontamination of exterior dust from sources other than soil complicates the interpretation of the movement of soil lead into the home or to exterior play areas.

Household dust was abated in the Boston and Cincinnati studies, but not in Baltimore. The BOS SPI and CIN SEI groups received interior dust abatement at the same time as soil abatement, the BOS PI received interior dust abatement without soil abatement, and the CIN I-SE received interior dust abatement in the first year followed by soil and exterior dust abatement in the second year.

4.2 DESCRIPTION OF THE DATA

This section focuses on the actual data that formed the basis for the conclusions reached by the individual study reports. These data consist of measurements of soil, exterior dust (sometimes referred to as street dust), interior dust (house dust), hand dust, blood lead, exterior paint, interior paint, and drinking water. The age of the child and the date of collection were also included in some analyses. Tables 4-2, 4-3, and 4-4 summarize key data for all three studies. For the most part, these data are the bases for the results and conclusions presented in the individual city reports, and also for the statistical analyses in Chapter 5 of this integrated assessment.

TABLE 4-2. SUMMARY OF BOSTON STUDY DATA

	Round 1	Round 2	Round 3	Round 4	Round 5
Median Soil Pb Conc. (μg/g)					
BOS SPI	2,396	125	115	-	193
BOS PI	2,307	-	2,084	-	278
BOS P	2,275	-	2,212	-	220
Median Floor Dust Pb Conc. (μg/g)					
BOS SPI	2,100	1,040	845	760	726
BOS PI	2,240	1,105	1,150	1,030	806
BOS P	2,200	-	950	1,300	862
Median Floor Dust Load (mg/m^2)					
BOS SPI	24	36	23	15	31
BOS PI	24	19	26	17	31
BOS P	40	-	28	19	37
Median Floor Dust Pb Load ($\mu g/m^2$)					
BOS SPI	52	40	23	16	24
BOS PI	59	24	27	18	28
BOS P	75	-	27	21	37
Median Window Dust Pb Conc. (μg/g)					
BOS SPI	13,240	9,967	11,217	21,125	8,780
BOS PI	19,667	2,400	10,000	15,650	6,870
BOS P	17,400	-	15,500	12,667	12,350
Median Window Dust Load (mg/m^2)					
BOS SPI	293	104	474	373	919
BOS PI	304	31	380	570	500
BOS P	239	-	239	504	797
Median Window Dust Pb Load ($\mu g/m^2$)					
BOS SPI	7,005	1,392	4,728	5,735	5,402
BOS PI	7,196	88	4,624	5,697	2,553
BOS P	4,179	-	4,441	5,559	6,018
Median Hand Pb Load (μg/pair)					
BOS SPI	6.75	4.0	3.5	-	12.5
BOS PI	6.75	5.5	2.0	-	7.15
BOS P	5.75	3.5	4.5	-	9.2
Median Blood Pb Conc. (μg/dL)					
BOS SPI	13	10	10	-	10
BOS PI	12	8	11	-	8
BOS P	12	9	11.5	-	10
GM Blood Pb Conc. (μg/dL)					
BOS SPI	12.36	9.11	9.90	-	9.07
BOS PI	11.70	8.01	10.74	-	7.11
BOS P	11.49	9.19	10.75	-	8.85

TABLE 4-3. SUMMARY OF BALTIMORE STUDY DATA

	Round 1	Round 2	Round 3	Round 4	Round 5	Round 6
Median Soil Pb Conc. (μg/g)						
BAL SP	440	-	-	22	-	-
BAL P	409	-	-	-	-	-
Median Floor Dust Pb Conc (μg/g)						
BAL SP	1,600	-	-	1,068	-	-
BAL P	1,850	-	-	1,150	-	-
Median Floor Dust Load (mg/m^2)						
BAL SP	40	-	-	37	-	-
BAL P	37	-	-	38	-	-
Median Floor Dust Lead Load ($\mu g/m^2$)						
BAL SP	73	-	-	38	-	-
BAL P	72	-	-	41	-	-
Median Hand Pb Load (μg/pair)						
BAL SP	10.7	12.9	7.4	8.5	12.6	14.9
BAL P	13.6	14.8	9.5	6.0	17.3	13.0
Median Blood Pb Conc. (μg/dL)						
BAL SP	12.4	11.0	9.8	8.8	9.9	10.4
BAL P	10.6	10.2	9.2	7.4	8.0	8.0
GM Blood Pb Conc. (μg/dL)						
BAL SP	11.0	9.9	9.7	8.6	9.6	9.7
BAL P	10.9	10.5	9.1	7.8	8.1	8.4

Each study produced similar information about the occurrence of lead in the environment. The data sets among the studies are not perfectly comparable, however, in that they differed in the timing of the collection relative to intervention (see Figure 2-1), the spatial distribution of the sampling points relative to the expected exposure to the child, and the manner in which the data were reduced to a central tendency.

Data were collected in rounds. That is, during a specific period of time, samples were taken of soil, dust, etc., for a specific objective, such as establishing the concentration of lead prior to intervention. Usually a round lasted for several weeks, perhaps three to

TABLE 4-4. SUMMARY OF CINCINNATI STUDY DATA

	Round 1	Round 2	Round 3	Round 4	Round 5	Round 6	Round 7
Median Soil Pb Conc. (μg/g)							
CIN SEI	680	134	142	103	122	166	132
CIN I-SE	237	247	240	262	125	182	138
CIN NT	339	346	330	256	331	267	266
Median Street Dust Pb Conc. (μg/g)							
CIN SEI	3,937	3,398	2,118	2,559	3,231		
CIN I-SE	3,665	3,416	3,411	2,275	3,040		
CIN NT	1,583	1,156	891	968	1,086		
Median Street Dust Load (mg/m^2)							
CIN SEI	454	242	363	452	310		
CIN I-SE	649	561	326	420	126		
CIN NT	624	755	481	477	654		
Median Street Dust Pb Load ($\mu g/m^2$)							
CIN SEI	1,162	789	641	968	808		
CIN I-SE	2,364	1,618	1127	943	371		
CIN NT	1,005	957	498	587	442		
Median Floor Dust Pb Conc. (μg/g)							
CIN SEI	362	346	325	474		158	
CIN I-SE	395	388	408	431		163	
CIN NT	229	224	209	213		162	
Median Floor Dust Load (mg/m^2)							
CIN SEI	418	134	135	197			
CIN I-SE	167	38	117	392			
CIN NT	147	126	161	200			
Median Floor Dust Pb Load ($\mu g/m^2$)							
CIN SEI	158	76	54	130	76		
CIN I-SE	69	18	58	243	108		
CIN NT	35	32	32	34	92		
Median Window Dust Pb Conc. (μg/g)							
CIN SEI	1,509	1,287	922	1,920	502		
CIN I-SE	2,000	1,572	1,306	2,017	592		
CIN NT	983	816	548	1,399	302		
Median Window Dust Load (mg/m^2)							
CIN SEI	710	433	254	4,524	966		
CIN I-SE	1,258	380	269	9,860	615		
CIN NT	2,170	2,534	324	8,573	648		
Median Window Dust Pb Load ($\mu g/m^2$)							
CIN SEI	983	426	242	15,385	397		
CIN I-SE	2,548	360	286	26,364	358		
CIN NT	1,782	1,111	172	12,849	227		

TABLE 4-4 (cont'd). SUMMARY OF CINCINNATI STUDY DATA

	Round 1	Round 2	Round 3	Round 4	Round 5	Round 6	Round 7
Median Mat Dust Pb Conc. (μg/g)							
CIN SEI	109	738	549	767	659	-	-
CIN I-SE	132	939	702	722	889	-	-
CIN NT	100	373	349	405	332	-	-
Median Mat Dust Load Incremental Increase Per Day (mg/m^2/day)							
CIN SEI	-	6.5	7.7	4.4	28.2	-	-
CIN I-SE	-	18.7	4.7	4.9	16.6	-	-
CIN NT	-	1.8	2.0	2.7	12.2	-	-
Median Mat Dust Pb Load Incremental Increase Per Day (μg/m^2/day)							
CIN SEI	-	6.54	7.62	2.38	9.80	-	-
CIN I-SE	-	7.65	5.14	3.20	8.02	-	-
CIN NT	-	3.30	4.67	0.99	5.29	-	-
Median Entry Dust Pb Conc. (μg/g)							
CIN SEI	334	606	433	491	211	382	488
CIN I-SE	425	492	468	632	102	598	615
CIN NT	290	367	317	286	84	317	284
Median Entry Dust Load (mg/m^2)							
CIN SEI	386	113	230	590	12,671	97	301
CIN I-SE	272	70	142	1,394	17,889	161	513
CIN NT	348	238	294	373	14,509	148	1,080
Median Entry Dust Pb Load (μg/m^2)							
CIN SEI	112	104	167	250	2,502	56	150
CIN I-SE	95	38	70	588	2,700	103	302
CIN NT	157	80	88	106	1,714	58	264
Median Hand Pb Load (μg/pair)							
CIN SEI	6.0	5.0	5.0	12.0	12.5	-	-
CIN I-SE	7.0	7.0	5.0	10.0	8.0	-	-
CIN NT	3.0	4.0	3.0	5.5	7.0	-	-
Median Blood Pb Conc. (μg/dL)							
CIN SEI	9.2	-	7.0	8.0	-	7.9	8.3
CIN I-SE	10.8	-	9.2	8.9	-	8.0	8.8
CIN NT	9.0	-	5.9	6.8	-	6.4	7.8
GM Blood Pb Conc. (μg/dL)							
CIN SEI	8.8	-	6.9	8.8	-	8.2	8.7
CIN I-SE	10.8	-	9.3	8.6	-	7.6	8.9
CIN NT	8.3	-	5.7	6.8	-	7.2	7.8

four months. It may be important to know when a sample was taken during a round, especially following intervention, in order to evaluate the impact on exposure. Consider the pathway from soil ⇒ street dust ⇒ house dust ⇒ hand lead ⇒ blood lead. One would expect, if soil alone (not house dust) were abated and the exposure were mainly through house dust, there would be a lag in time between abatement and response, and the impact of intervention might become greater with increasing time. Conversely, the impact of intervention might be reduced with time if there were recontamination, as would be expected if house dust were abated but soil or other sources were not.

Data linkages are important to the interpretation of the results. Specifically, it is important to know how well the data link (e.g., between soil concentration measurements and house dust concentration measurements) actually represent the hypothesized pathway between soil and house dust. Through these data linkages, it is ultimately possible to construct a simple exposure scenario for the individual child and to analyze these scenarios by structural equation modeling. For example, a young child may spend most of the time indoors, whereupon the exposure scenario becomes the lead that is available to the child through food, drinking water, air, and dust (see Figure 2-1). Each of these proximal sources of lead is influenced by one or more other sources of lead more remote from the immediate exposure of the child.

Data are also linked by a primary identifier or index. Some data are linked to the individual child, such as blood lead and hand lead. Some are specific for the living unit or family, and some are specific for the property. It is important to be aware of this distinction because of the duplication effect that can occur when there are several siblings in a family and several families in a dwelling. This means that a single numerical value for soil such as a mean or median for the premises could be heavily weighted if there were, for example, five children living on the same property.

4.2.1 Measures of Central Tendency for Property Level Soil and Dust

For soil and dust, there is a need to reduce multiple measurements within a round to a single representative data point for each property or living unit. In order to determine the appropriate central tendency for this measurement, the participating groups discussed several alternatives at great length without reaching a consensus. Therefore, different measures of central tendency were reported in each of the three studies. The following is an extended discussion of each of these measures, followed by an argument for the use of the arithmetic mean as the best measure in these circumstances.

The procedures for selecting a representative soil sample were based on the statistical distribution of data in each study. The Boston study used the median, giving no weight to extreme values. The Cincinnati study used the geometric mean, a method that is often used when the measured values are lognormally distributed, because it gives lesser weight to extreme values. The geometric mean is always lower than the arithmetic mean for any set of positive values and therefore may be an underestimate of the exposure to the child.

The distribution problem was approached differently in Baltimore, where the tri-mean was calculated as the weighted average of the first, second, and third quartiles:

$$X = \frac{Q_1 + 2Q_2 + Q_3}{4}, \qquad (4\text{-}1)$$

where

X = tri-mean, and

Q_n = nth quartile (Q_2 = median).

The tri-mean approach gives some consideration to the uneven distribution of values without unduly weighting the extremes. The tri-mean is equivalent to the arithmetic mean if the distribution is perfectly symmetric.

All three approaches assume that the sampling pattern is random and that exposure to soil is spatially random. Neither condition is strictly true in all three studies. One-third to one-half of the soil samples were taken 1 m from the foundation of the home, where concentrations are known to be higher than elsewhere. Because of playtime interests, parental instructions, or other influences, the child tends to play in specific areas that may represent less than 25% of the total soil area.

It would seem reasonable that the ideal method for selecting a representative value should focus on the relationship between the soil and the child. The ideal measurement of central tendency is one that perfectly represents exposure to the child. This means that outside play activity patterns and exterior dust traffic patterns into the home must both be evaluated. In the case of outside play activities, a sample would be taken at each location where the child played and this sample would be weighted according to factors such as the time spent playing there and the frequency of hand-to-mouth activity during that time. Because this information is not available, a simplifying assumption is that weight should be given to the location of the sample rather than concentration. Location, not lead concentration, is the basis of choice for the child's play environment. An exposure weighted mean of the soil samples would seem to be the most direct approach. This would be an arithmetic mean of soil values corrected for the degree of exposure to the child. For example, a sample taken from bare soil in an area observed to be a play area would be given a high weighting factor for exposure. Grass covered areas with limited accessibility would be weighted on the low end of exposure. Although cumbersome, this method is feasible because such information was collected at the time of sampling in each study. The drawback is that the method emphasizes the direct, outdoor playtime contact between the child and the exterior dust, and does not consider other routes of dust exposure, such as soil ⇒ household dust.

An alternative solution is to consider that the child has equal exposure to the entire surface of the soil. In this case, the perfect sample would be to scrape up this upper 2 cm of soil, homogenize it and take a sample. Theoretically, this is equivalent to sampling in a random pattern and taking the arithmetic mean of these samples. In this project, random locations were taken along lines specifically selected to represent the expected high- and low-concentration areas of the plot of soil. In this sense, the arithmetic mean is the best measure of the central tendency of soil data for a property, and is the statistic used in this report. For populations of children at the neighborhood or higher level, the median or geometric mean is often the preferred measure of central tendency.

4.2.2 Adjustments and Corrections to the Data

4.2.2.1 Subjects Dropped from Study

During the analysis of their data, the Boston group discovered that two children of the same family had apparently become exposed to lead-based paint abatement debris while staying at a house outside their neighborhood during a time when it was being remodeled. Both siblings had blood lead concentrations that had tripled in less than five months, between

Rounds 1 and 3, from 10 to 35 μg/dL and 17 to 43 μg/dL. The Boston group analyzed their data with and without these children, eventually excluding these data from the analyses used to test their hypothesis. This Integrated Report accepts the conclusion that the data are outliers and also dropped them from further analysis.

4.2.2.2 Unit Conversion

All data were converted to common units, usually metric. No further corrections were made for analytical blanks or similar analytical adjustments, other than as reported by each individual city research team.

4.3 DESIGN DIFFERENCES

Table 4-5 describes the design differences among the three studies. While considerable effort was made to coordinate the study designs so as to assure the highest possible degree of comparability among study results, the investigators in the three cities faced different design issues that precluded carrying out completely identical or equivalent studies. Thus, although participant recruitment and certain other aspects were similar across the three cities, some salient differences are also worth noting.

The first difference was that there were different levels of remediation or treatment among the cities. Boston used two comparison or reference groups in addition to the soil abatment group, whereas Baltimore used only one such group. In the Cincinnati study, there were three levels of intervention. Also, the trigger level for soil lead removal varied somewhat across the cities. In the Baltimore and Cincinnati, a maximum level of 500 ppm or greater in the parcel or residential property triggered soil removal. In contrast, all Boston yards from which soil was removed initially had soil lead much higher than 500 ppm, most in excess of 1,000 to 2,000 ppm. Properties recruited in the Boston study were scattered across four large neighborhoods or urban areas, although households were assigned at random to the treatment group for soil removal and not specifically limited to any given neighborhood. The Baltimore study was carried out in two large neighborhoods, with soil lead removal restricted to only one of the neighborhoods (Lower Park Heights). Most houses above the soil lead trigger level in the Lower Park Heights neigborhood in the

TABLE 4-5. DESIGN DIFFERENCES BETWEEN THE THREE STUDIES

Design Feature		Boston	Baltimore	Cincinnati
Number of treatment groups		3	2	3
Number of rounds with blood Pb measurement		4	6	5
Interval between abatement and final blood Pb measurement (months)		22	10	20
Soil removal trigger level (μg/g)		1,000	500	500
Paint stabilization		Interior	Exterior	None
Number of neighborhoods		4	2	6
Participant recruitment		Volunteer	Volunteer	Volunteer
Treatment assignment to participants		Random	By Neighborhood	By Neighborhood
Control groups with no intervention		No	No	Yes
Age structure of participants (%)	0-1	2.7	8.6	29.9
	1-2	24.0	17.6	17.2
	2-3	34.0	18.1	17.6
	3-4	34.7	18.4	15.8
	4-5	4.7	20.3	14.0
	5-6		14.5	5.4
	6→		2.5	
Ethnicity (%)				
Black		51	100	97
Hispanic		15	0	0
White		7	0	2
Other		27	0	1
Male/female ratio		47/53	48/52	44/56
Blood sample collection	R1	1-2 mo preabate	24 mo preabate	1-2 mo preabate
	R2	3-4 mo after R1	12 mo preabate	
	R3	10 mo after R1	5-8 mo preabate	3-4 mo after R1
	R4		8-10 mo after R3	11 mo after R1
	R5	22 mo after R1	14-16 mo after R3	
	R6		18-20 mo after R3	16-18 mo after R1
	R7			22-24 mo after R1

Baltimore had yard soil removed, but some did not, and no house in Walbrook junction had soil removed. The Cincinnati study was carried out in six smaller neighborhoods, with soil and exterior dust removal only carried in the Pendleton neighborhood. In the Cincinnati study, all parcels in Pendleton above the soil lead trigger level had soil removed.

Paint was stabilized inside all Boston houses and outside all Baltimore houses, but not in Cincinnati where it was believed that only gut-rehab houses had been recruited into the study. No Baltimore residence received interior abatement, either of dust or lead paint, whereas as the majority of the residences in the Boston and Cincinnati studies received interior dust abatement whether or not they were in the soil removal treatment group.

Demographic differences among study populations should also be noted. The age distribution of children at the time of abatement differed among the three studies. The Baltimore group had more children of age at least four years, since many of the children had been initially recruited up to 2 years earlier. Almost all of the children initially recruited in the Baltimore study were of African-American ancestry; by the final phase of the study, 100 percent of the study group was African-American. The Cincinnati study group was slightly more diverse, with a small percentage of Caucasians of Appalachian origin. The Boston group was the most diverse, with substantial subgroups of white and Cape Verdean children, and also with a large percentage of African-American children. Percentages of male and female children differed somewhat among the cities. While all of these inner city households tended to be economically disadvantaged, the majority of the households in Baltimore were occupied by the property owner, which was uncommon in the other two cities.

Lastly, as for biological measurements indexing changes in lead exposure, each study involved collection of preabatement and postabatement blood samples and their analyses. However, the numbers of sampling points varied across the studies. The studies had four to six rounds of blood lead collection, with one to three pre-abatement rounds, a short-term post-abatement round (about two or three months), and two to three rounds up to two years post-abatement.

4.4 INDIVIDUAL STUDY CONCLUSIONS

In their report following the first phase of their study, the Boston group stated their conclusions:

> *"...this intervention study suggests that an average 1,856 ppm reduction in soil lead levels results in a 0.8-1.6 μg/dL reduction in the blood lead levels of urban children with multiple potential sources of exposure to lead."*

Following the second phase of the study, they concluded (Aschengrau et al., 1994):

"The combined results from both phases suggest that a soil lead reduction of 2,060 ppm[1] is associated with a 2.2 to 2.70 μg/dL decline in blood lead levels."

The basis for their initial conclusions consisted of an analysis of variance comparing mean blood lead changes among the three intervention groups, paired t-tests for within group effects, and analysis of covariance with one-at-a-time adjustment for age, SES, race, sex, paint, water, and mouthing behavior. The analysis of covariance was performed using no transformation of blood lead data, which appeared to be normally distributed.

The conclusions from the second phase of the study are based on additional analyses of phase one and phase two data using two-way analysis of variance (ANOVA) with repeated measures. Soil was abated for the two original control groups (BOS PI and BOS P) at the beginning of phase 2. The reduction in blood lead is based on pre- and postabatement measurements of all three groups.

The Baltimore group stated their conclusions as follows:

"Statistical analysis of the data from the Baltimore Lead in Soil Project provides no evidence that the soil abatement has a direct impact on the blood lead level of children in the study."

"In the presence of lead-based paint in the children's homes, abatement of soil lead alone provides no direct impact on the blood lead levels of children."

The basis for these statements consisted of an adjusted and unadjusted analysis of selected covariates. The natural log of the blood lead of children in the treatment group showed no significant difference from the natural log of the blood lead of children in the control group, even when adjustments were made for: age, SES, hand lead, season, dust, soil, sex, weak mouthing behavior, or strong mouthing behavior. These analyses were made on two sets of data. The first set consisted of all children enrolled in rounds one and six. The second group consisted only of children enrolled in all six rounds.

The Cincinnati conclusions can be paraphrased as follows based on their individual report:

Following interior and exterior dust and soil lead abatement, blood lead concentrations decreased (in Area A) from 8.9 to 7.0 (21%) but increased to 8.7,

[1] This value for soil, 2,060 ppm, cited in their published report, was not adjusted by the Boston group with the interlaboratory correction factor of 1.037 in Table 3-6.

10 months postabatement. Following interior dust abatement alone blood lead concentrations decreased from 10.6 to 9.2 (13%) four months postabatement and were 18% below preabatement 10 months postabatement. With no abatement, blood lead levels decreased by 29 and 6% during these same time periods. Other comparisons also revealed no effects of the soil or dust abatement.

There was no evidence that blood lead levels were reduced by soil lead or dust abatement in Area A (with soil, exterior dust, interior dust abatement). There was a slight reduction (net reduction over control area) of 0.6 μg/dL in Area B that might be attributed to interior dust abatement. This difference is not statistically significant.

The basis for the Cincinnati conclusions was a comparison of environmental and blood lead data for the three treatment groups from Rounds 1, 3, 4, 6, and 7 and of additional environmental data from Rounds 2 and 5.

5. RESULTS OF INTEGRATED ANALYSES

5.1 BASIC STRATEGY FOR EVALUATING ABATEMENT EFFECTIVENESS

Abatement effectiveness is assessed by comparing changes in critical measurements before and after abatement. Changes in blood lead levels, in hand lead levels, and in household dust lead levels are expected to occur in response to abatement but may also occur even without environmental interventions. Blood lead concentrations in young children often increase up to ages 2 or 3 years, which are peak ages for ingestion of soil and dust during play, and then decrease slowly in older children (U.S. Environmental Protection Agency, 1986; Clark et al. 1988). Hand lead loadings increase steadily with age (Bornschein et al., 1988). House dust lead levels may increase as changes in sources or exposure pathways cause change in house dust lead levels to occur.

Each individual report reached its conclusion based partially or entirely on linear regression using analysis of covariance. With this statistical method, when either or both the measurement error or sampling error of the independent or predictor variable are unknown, then the estimated regression effect (reduction of blood lead per unit reduction in soil lead) may be reduced or attenuated. Part of the potential attenuation attributable to "simultaneous equation bias" is addressed in this integrated report by the use of structural equation models so that effects size estimates derived by that method are likely more accurately characterized.

This integrated assessment also addresses the question of whether there are effects of intervention other than soil abatement that might reduce childhood lead exposure. Some of these intervention strategies, such as paint stabilization, interior dust abatement, and neighborhood level exterior dust abatement, were used in this project and an evaluation of their effectiveness is also reported below.

Finally, this report contains some information on the reliability of childhood lead exposure measures other than blood. In this respect, data on handwipes and house dust are interpreted as predictors of childhood lead exposure.

5.1.1 General Discussion of Conceptual Approaches

5.1.1.1 Basic Strategies for Evaluating Abatement Effectiveness

Childhood blood lead concentrations are, to some extent, a measure of the recent history of lead exposure and may respond to environmental changes in lead within a time frame of a few months. Reductions in blood lead due to reductions in exposure might be somewhat attenuated by the remobilization of lead in bone tissue as shown in Figure 5-1. This figure shows the complexity of biokinetic translocations of lead when the total body burden is decreasing. If the total lead exposure of the child decreases, there seems to be no doubt that the blood lead concentrations would decrease, but measurements of this decrease would be complicated by the remobilization of bone tissue lead, and interpretation of these measurements would be complicated by the uncertainty that the reduction in exposure might not be fully attributable to reductions in soil lead exposure.

Changes in blood lead must be interpreted in the context of four time-dependent effects that are independent of each other as follows:

(1) the typical seasonal changes in children's blood lead concentrations, found in virtually every longitudinal study, that usually indicate a peak in concentration during the late summer months;

(2) the changes that occur with age during early childhood that usually peak between 18 and 27 months;

(3) long-term changes in national baseline levels of exposure, believed to be mostly from reductions of lead in gasoline and in food, that are reflected in a downward trend for childhood blood lead levels observed since 1978; or

(4) changes that can be attributed to interventions of this project.

Several different analytical strategies may be used to evaluate the effectiveness of lead abatement or intervention methods: comparison of simple changes for different treatment groups; comparison of adjusted changes among different treatment groups where the adjustment normalizes the preabatement treatment and control groups; and comparison of adjusted changes among different treatment groups where the adjustments both normalize the groups to a common starting point and account for different rates of change during the study. These strategies could be applied to any of the lead measurements used to compare abatement

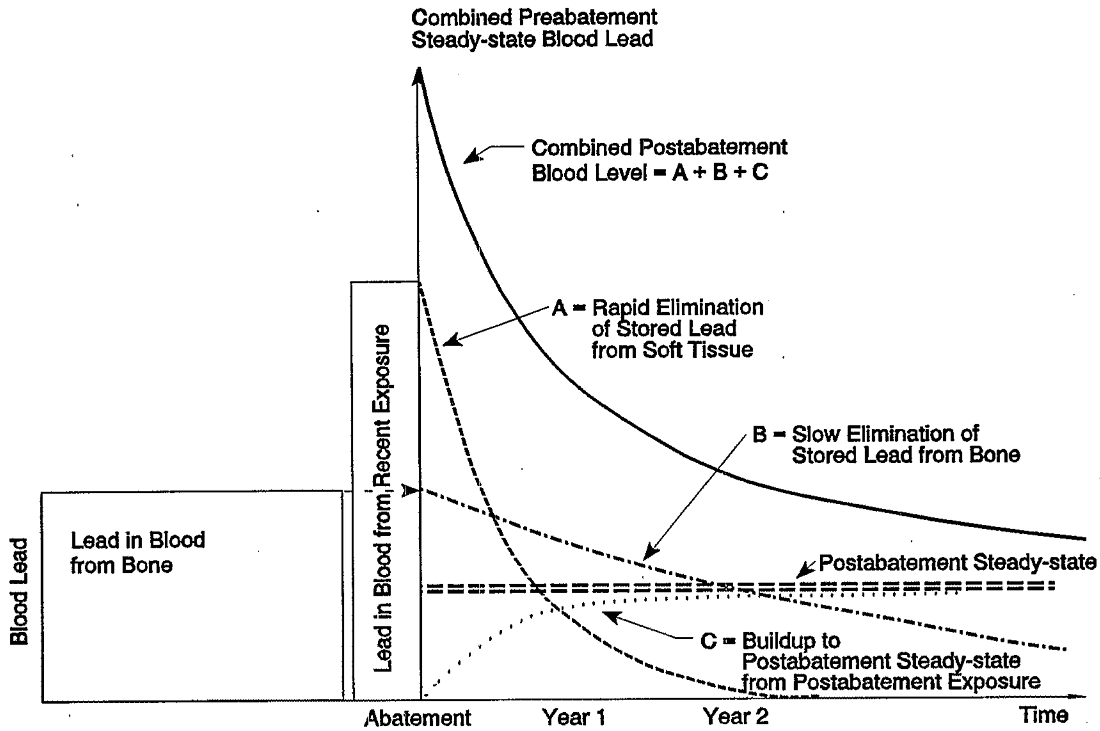

Figure 5-1. Hypothetical representation of the expected decrease in blood lead, (solid curved line) following abatement. This rate of decrease is less than might be expected from exposure reduction alone. This is because blood also contains lead recently released from storage in bone and soft tissue.

effectiveness: blood lead concentration, hand lead loading, dust lead concentrations, dust lead loading, or soil lead concentration. Each of these three analytical strategies represents a different perspective on the importance of the components of the entire exposure pathway and on the possible changes that may occur, either as a consequence of intervention or because of other unplanned changes during the course of the study.

In the simplest approach, the best comparisons are the lead variables before and after the abatement was carried out. In general, the lead levels would be expected to be different, with or without abatement, so that it is necessary to compare the changes that occurred in the soil or dust abatement groups with the change that occurred in the nonabatement groups. The statistical methods that would commonly be used here are paired-sample tests, looking at the difference between the lead levels or logarithms of lead levels before and after abatement.

If the lead levels are measured at more than two time points or phases, then a simple repeated measures analysis of some sort would be used.

The second analytical strategy recognizes that the treatment groups may not be entirely equivalent to each other. It would therefore be necessary to adjust the "starting line" for different groups to a common baseline so that all subsequent comparisons could be made as if everything else were equal, except for the experimental interventions or treatments. Some of the initial adjustment factors could also be lead related variables. For example, the comparison of blood lead concentrations may need to be adjusted for differences in soil lead concentrations in different yards, because one would expect (everything else being equal) that children who live in houses with higher soil lead would start with higher blood lead concentrations than children who started in houses with lower soil lead. Similarly, it may be useful to adjust for other nonlead factors such as the child's age. Repeated measures analyses with adjustments for covariates (multiple regression or multivariate general linear model) are appropriate statistical methods for carrying out the second strategy.

The Boston study offers the fewest complications in using the second strategy, because treatments were randomly assigned to houses and there is little reason to believe that there may be some intrinsic confounding effect between treatment group and either blood lead or environmental lead. Adjustments for environmental lead as covariates should therefore clarify comparisons of the effectiveness of different treatments for individual children in the Boston study. The Baltimore and Cincinnati studies are more difficult to interpret, because the treatment groups were assigned by geographical area or location, not randomly selected from within the same group. There were substantial differences in soil lead and dust lead concentration between neighborhoods.

Several comparisons could be carried out using the second strategy. These include: comparisons of treatment group effect on blood lead concentration, adjusted for initial hand lead, dust lead, and soil lead; comparisons of treatment group effect on hand lead, adjusted for initial differences in dust lead and soil lead; comparisons of treatment group effect on dust lead, adjusted for initial differences in soil lead; and even comparisons of soil lead before and after treatment, to determine whether soil lead in the soil lead abatement group remained at reduced levels or was recontaminated.

The third strategy uses structural equation modeling to combine the seemingly unrelated tests of the changes in blood lead and other lead variables. The basis for testing the changes simultaneously is the assumption that current blood lead and environmental lead levels reflect recent lead exposure, and that changes in exposure will lead to changes in lead levels further along the pathways from source to child. The appropriate statistical methodology for this strategy involves testing group differences in models with simultaneous equations for different environmental lead variables. Separate model equations would be needed for dust lead concentration and for total dust loading.

Key characteristics of each of the three strategies are illustrated graphically in Figures 5-2 through 5-4. Figure 5-2 shows four separate models for blood lead, hand lead, dust lead, and soil lead, as they would be tested using Strategy 1. Figure 5-3 extends each of these to models with covariate adjustments as the most detailed implementation of Strategy 2. The third strategy is illustrated in Figure 5-4. The interconnected nature of the lead measurements over time is shown explicitly, reflecting the hypothesis that changes in dust lead, hand lead, and blood lead are quantifiable effects of changes in lead source terms such as lead in soil and lead in paint.

In their individual reports, all three research teams used Strategy 1 as their primary statistical tool and the main basis for their conclusions. The Boston and Baltimore teams also reported results of statistical analyses using Strategy 2, and the Cincinnati group used structural equation modeling to report some of their results.

The statistical analyses conducted as part of this EPA integrated assessment were aimed at addressing the following questions:

- DID THE ABATEMENT OR INTERVENTION HAVE AN EFFECT? This hypothesis is tested statistically by the interaction between the intervention group and the phase or year. If the statistical significance or P value of the interaction terms is larger than a conventional value such as 0.05, one would conclude that there is no effect of the abatement or intervention (parallel group mean profiles not significantly different).

- WAS THE EFFECT IN THE EXPECTED DIRECTION? Abatements and other interventions are expected to reduce blood lead, hand lead, or dust lead levels more than in nonabatement or control groups. That is, if group 1 is the control group and group 2 is the intervention group, one would expect pre- versus postabatement differences in the treatment (intervention) group to be larger than the pre- versus postabatement difference in the control group.

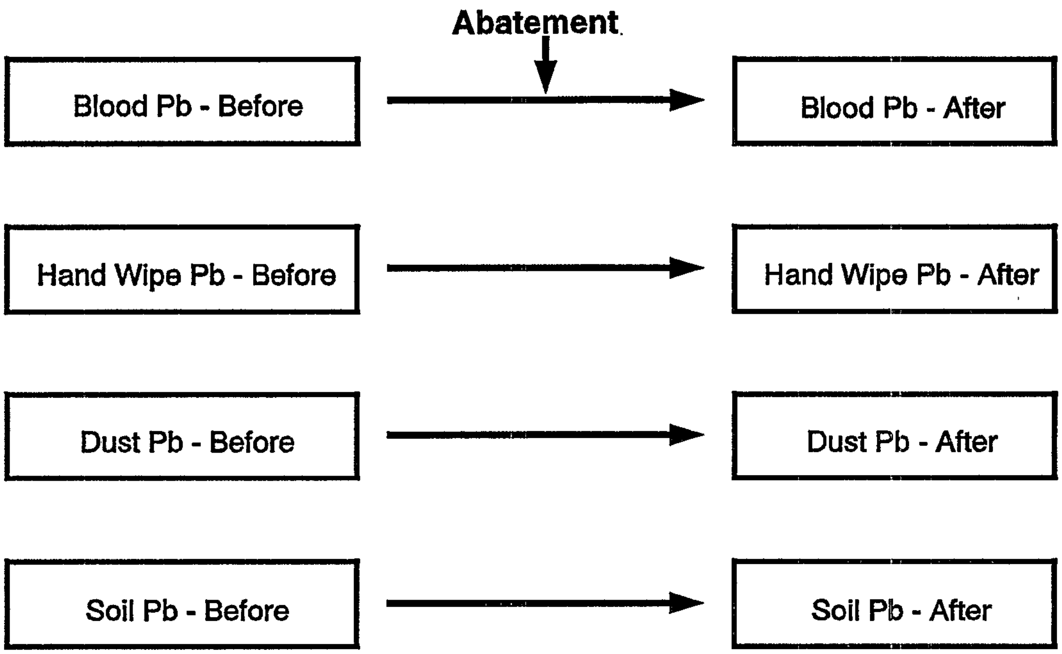

Figure 5-2. A simple approach that compares lead variables before and after abatement comparable to Strategy 1.

• WAS THERE AN OVERALL DIFFERENCE BETWEEN PHASES? This hypothesis is tested statistically by the mean within-subject difference between the preabatement and postabatement groups averaged across all intervention groups. If the statistical significance or P value of the phase term is larger than a conventional value such as 0.05, one would conclude that there is no difference in overall level over time. As noted above, lead levels are expected to change over time with or without interventions.

• WAS THERE AN OVERALL DIFFERENCE BETWEEN GROUPS? This hypothesis is tested statistically by the mean between-group differences averaged across preabatement and postabatement groups. If the statistical significance or P value of the phase term is larger than a conventional value such as 0.05, one would conclude that there is no difference in overall group mean levels. Group mean lead levels are expected to differ when different interventions are associated with different neighborhoods, as in Baltimore and Cincinnati.

• WAS THERE A CHANGE IN THE RELATIONSHIP BETWEEN THE RESPONSE VARIABLE AND THE COVARIATES AFTER ABATEMENT? Many factors affect blood lead, hand lead, dust lead, dust loading, and other indicators of lead exposure. Blood lead depends on hand lead and on environmental lead exposure indices, dust lead depends on lead in soil and paint, and so on. Blood lead may also depend on child age, on behavioral variables such as the frequency of outdoor play, on

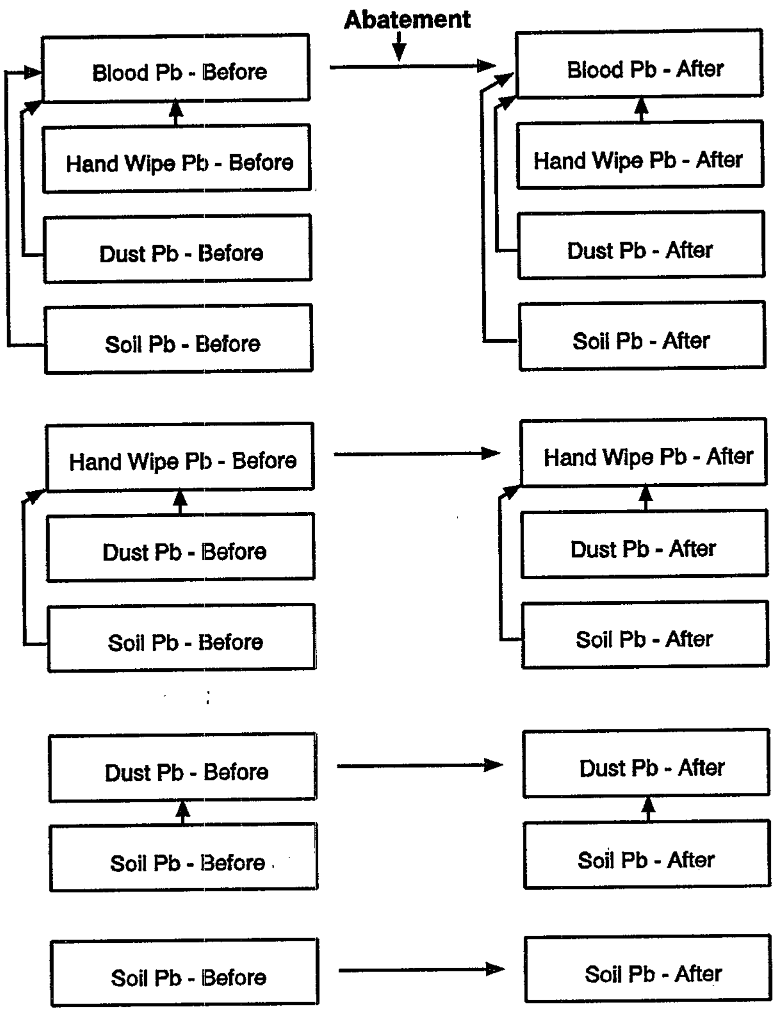

Figure 5-3. A more complex approach that uses covariate adjustments with repeated measures analysis, comparable to Strategy 2.

household socioeconomic indicators such as parental education, and on demographic factors such as race or ethnicity. These factors may modify the effectiveness of abatement. One way to test for this is to include the covariate in the analysis as an adjustment factor so that the baseline levels can be tested as if all children started out at the same level. A similar argument may apply to adjustments of postabatement blood lead. The effect of the covariate may be assumed to have changed over the course of abatement (possibly as a consequence of abatement) if the three-way interaction between the treatment group, the phase of the study, and the covariate is statistically significant.

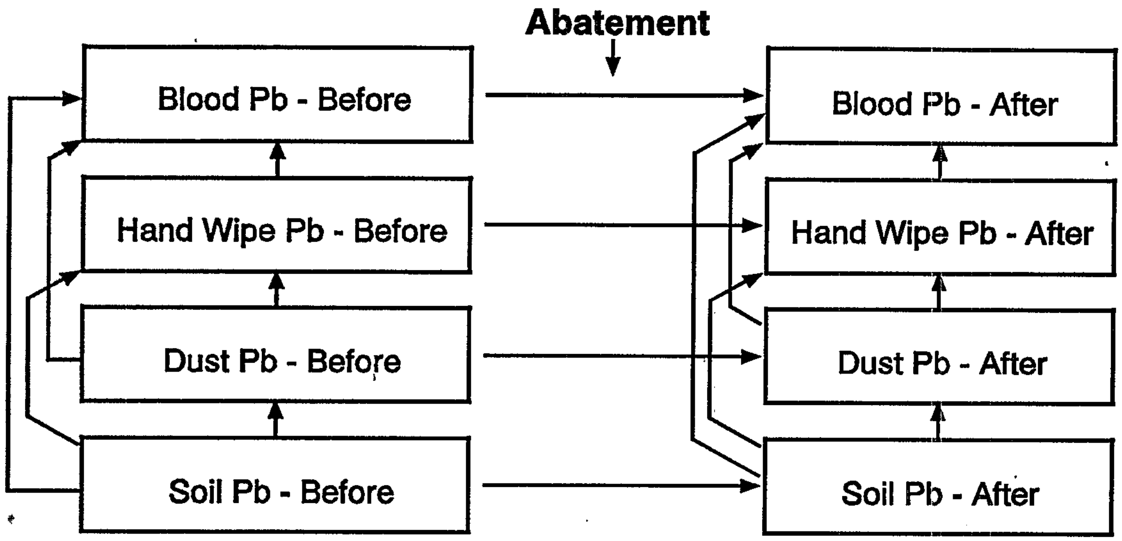

Figure 5-4. A structural equation modeling approach comparable to Strategy 3.

5.1.1.2 Expected Impact of Intervention

Impact of Soil Abatement on Exterior and Interior Dust

The key to understanding the impact of soil (and exterior dust) abatement on interior dust is to observe changes in the three components of the interior dust measurement: lead concentration (micrograms of lead per gram of dust), lead loading (micrograms of lead per square meter), and dust loading (milligrams of dust per square meter). Where there was no interior dust abatement, the lead concentration in interior dust should decrease gradually over time, provided that the influence of lead-based paint has been minimized. Also, the lead loading should decrease if the dust loading remains constant or the lead loading is normalized to dust loading. This normalization is believed to correct for differences in housekeeping efficiency. If interior dust abatement has occurred, the lead concentration should decrease markedly and remain low where the influence of lead-based paint is minimal, and the lead loading and dust loading should decrease and then increase in tandem.

The impact of lead-based paint can be minimized in three ways: (1) observe only cases where there is no lead-based paint; (2) stabilize the paint so that the rate of incorporation to house dust is minimized; and (3) compare measurements where the influence of lead-based paint is probably high relative to soil to areas where the influence of soil is high. A crude measure of the rate of recontamination of house dust from lead-based paint can be observed from the changes in window well dust lead concentrations following interior dust abatement, for units with and without lead-based paint.

The analysis of three types of internal dust measurements, (1) entry, (2) floor, and (3) window well, can provide additional information about the impact of soil abatement. The entry measurement probably shows the greatest influence of exterior lead from soil and dust. If the entryway to the housing unit is somewhat removed from the building entrance, such as an apartment on the second or third floor, then a comparison of these two measurements should demonstrate the effect of soil lead on multifamily houses. Likewise, where interior dust abatement has taken place, the rate of recontamination of interior dust should be entry > floor > window well.

Exterior dust was measured and abated in Cincinnati only. In this study, the results suggest a recontamination rate for exterior dust of less than two weeks, and that the source of this recontamination is not the soil. With a neighborhood level perturbance of this type, it is not possible to measure the impact of soil abatement on house dust directly. However, if abatement is considered on the broader scope, where neighborhood cleanup would include soil, external dust, and any other sources of lead external to the home, then the house dust measurements made immediately inside the homes can be used as a measure of this "total neighborhood abatement". For those cases in the Cincinnati study where there was no immediate recontamination of this entryway dust, this measurement may sometimes be used as a surrogate for soil abatement. To make this determination, it is also necessary to evaluate the fraction of exposure that would derive directly from soil or from playground dust, which would not be included in the interpretation of house dust alone.

Impact of Soil and Dust Abatement on Hand Lead Loading

It was expected that hand dust would serve as an surrogate measure of changes in exposure following abatement to augment information about blood lead changes. Hand dust reflects the child's recent exposure (since the latest hand washing), but is a measure only of lead loading, not lead concentration or dust loading, because the total amount of dust is not measured. Consequently, it is not possible to determine the source of lead (soil or paint) by differences in concentration, nor is it possible to correct for housekeeping effectiveness by observing changes in dust loading, as with house dust. It seems plausible that the amount of dust (not mud or dirt) on the hand reaches equilibrium after a short period of time, perhaps 30 min to 2 h. The dustiness of the house would affect only the rate at which this equilibrium is reached, not the total amount of dust at equilibrium.

Impact of Soil and Dust Abatement on Blood Lead Concentrations

Blood lead concentrations should respond to soil and dust abatement through the impact of abatement on two routes of exposure: (1) hand-to-mouth activity, reflecting the impact of interior house dust and exterior play area dust on exposure; and (2) food contamination, reflecting the incorporation of house dust in food during kitchen preparation. There was no measure of the incorporation of house dust into food during this project. Intuitively, the impact of interior dust abatement should be the same, or at least comparable, for food and hand dust. In some homes, however, lead-based paint is more common in kitchens and bathrooms, and the rate of return of dust from lead-based paint following stabilization would have a greater impact on food than hand dust. There is a limited amount of data, not yet analyzed, where kitchen floor dust can be compared to bedrooms and other living areas, and likewise for window wells. Most of these data, however, are from the Cincinnati study, where there was a minimum influence of lead-based paint.

The Baltimore study showed no influence of soil abatement on blood lead concentrations. The Baltimore study did not measure the impact of soil abatement in the absence of interior lead-based paint, and it is possible that soil abatement would be swamped by the presence of paint lead in the house dust. This negative result is an important finding of this study and the integrated project that suggests, in the absence of interior dust abatement and interior paint stabilization (or abatement), soil, exterior dust, and exterior paint abatement will have little impact on childhood lead exposure.

The Cincinnati study showed no effect of soil abatement alone on the blood lead concentrations, but showed a positive effect of interior dust abatement and a marginal effect of total abatement when the interior-entry dust immediately inside the home was used as a surrogate of neighborhood lead abatement. The importance of these findings is that when the sources of lead that recontaminate exterior dust can be identified and abated, the impact of neighborhood-level abatement will be greater than single dwelling unit abatement alone.

Effect of Lead Abatement or Intervention on Blood Lead Over Time

One of the most important limitations in carrying out a longitudinal lead abatement or intervention study over time is that reductions in blood lead are limited to some fraction of the total amount of lead stored in the child's body prior to abatement. Even if lead-burdened children were completely removed from lead exposure, a significant amount of lead would still be present in the child's blood due to the slow release of lead from the large amounts stored in the body, mostly in the bones. Autopsy data show that as much as 60 to 70% of the lead in a child's body is stored in the skeletal system, especially in the hard (or cortical) part of long bones such as the femur and the tibia (Barry, 1981). In adults this percentage is even larger, 90 or 95%. Lead is retained in cortical bone for many years, and even though bone remodeling in young children is very rapid, these large body burdens contained in the bone constitute a significant internal source of lead exposure for several years after exposure has stopped.

The persistence of elevated blood lead concentrations has some important public health implications. No matter how effective the environmental intervention, children can be expected to retain a fairly high fraction of their initial blood lead concentration for a period of several years. Because the health effects of lead exposure are believed to be cumulative, increasing as the total internal dose (years of exposure times micrograms per deciliter of blood lead), there may be substantial postremediation internal exposure and consequent health effects even after a successful intervention.

Reduction of environmental lead exposure should not be expected to produce a complete reduction of elevated blood lead levels attributable to the preabatement exposure. Blood lead levels are expected to be more persistent when there is long-term exposure to higher preabatement environmental lead from any source or medium. Much of the lead in the blood is distributed to other tissues before being eliminated from the body. Lead is avidly accumulated in the child's skeletal tissues, along with calcium needed for further growth and development. However, lead is released only very slowly from skeletal tissues, and this skeletal lead burden may become an internal source of blood lead even after the source of the lead exposure has been removed. Therefore, the postabatement blood lead level will not only reflect exposure to the new postabatement environmental lead levels, but will also in part reflect retention of skeletal lead from historical preabatement exposure. The long-term stability of blood lead levels in a stationary exposure environment has been noted by a number of authors (David et al., 1982; Rabinowitz, 1987).

Persistence of elevated blood lead after abatement has both biological and environmental components. The biological component is the resorption of skeletal lead. In adults, recent stable lead isotope studies (Smith et al. 1995) suggest that 30 to 65% of the circulating lead in adults is due to skeletal lead, which is consistent with other estimates. Although a somewhat lower percentage may be appropriate for children rather than adults, it is clear that even in children a substantial fraction of blood lead has a skeletal origin.

The environmental component of persistence is the child's remaining exposure to other nonremediated lead media, such as lead in diet, drinking water, or air. This was illustrated in Figure 5-1, which shows a blood lead profile (for an individual, or possibly as a population mean) before and after a hypothetical lead abatement. The steady-state blood lead concentrations are shown as flat curves, although in reality there may be substantial age-dependent changes during the course of abatement even when environmental lead concentrations remain constant. Assuming that environmental concentrations remain constant after abatement (they may not; see below), the child's blood lead would eventually reach a new steady-state concentration at a much lower level. At any given time after abatement, the child's blood lead is a mixture of three components, denoted "A", "B", and "C" in Figure 5-1. Component A shows the relatively rapid decrease in blood lead from elimination of preabatement lead deposits in blood and soft tissues. Component B shows the contribution of preabatement skeletal lead to post-abatement blood lead, which is much slower because the large skeletal burden in cortical bone is eliminated on a time scale of several years. Almost all of the stored lead will eventually be eliminated. However, the contribution of preabatement deposits of lead now stored as an internal source of exposure may be quantitatively significant compared to remaining postremediation environmental exposure media.

The combination of persistent internal exposure and persistent baseline external exposure amounts to a post-abatement blood lead contribution of about 50 or 60% of the preabatement blood lead starting value at 8 to 12 months after abatement. This means that *any environmental abatement or intervention can achieve at most a 40 to 50% reduction in child blood lead concentrations within a year after abatement (see Figure 5-1).*

Soil Lead Remediation Effects Modeled by Environmental Pathways for Lead

Soil lead remediation in residential yards is expected to have both direct and indirect effects on childhood lead exposure. The direct effect of removing lead contaminated soils is to deny access to the lead in the soil. However, most children do not eat large quantities of soil. Some children may regularly ingest a large amount of soil (a condition known as pica for soil), and some adults are known to experience geophagia, but these are untypical conditions and are not appropriate for assessing soil risks for the majority of children. For most children, direct exposure to lead in soil is likely to come from fine particles of loose soil or exterior surface dust that adhere to the child's hands and are transferred to the child's face and mouth during hand-to-mouth contact that is part of normal behavior for preschool children and infants.

The larger part of the contribution of lead in soil is as a source of lead in household dust. Soil in the residential yard may be tracked into the house by its occupants (including pets), and fine exterior dust particles may become re-entrained and carried into the house as micro-scale air contaminants. Fine dust particles may adhere to the child's hands, and may contaminate food during its preparation. Dust is usually a more important medium of lead intake than is soil. This is an indirect soil lead exposure pathway, from soil to house dust to the child's blood.

It is therefore necessary to model lead exposure through multiple pathways or exposure media in order to accurately characterize the complete effects of soil abatement. Time-dependent modeling of changes in environmental media and exposure pathways is a parallel process to time-dependent modeling of blood lead changes as noted in the preceding subsection.

5.1.2 Conceptual Approach to Differences in Group Means

The basis for simple analyses of abatement effectiveness is comparison of changes in mean blood lead in groups of children who received different interventions. The basis for interpreting such tests will be discussed before any formal statistical techniques are applied. Figure 5-5 sketches the probable outcomes of a soil abatement study (in general, any intervention study). All of the studies assigned a control group who received no soil lead

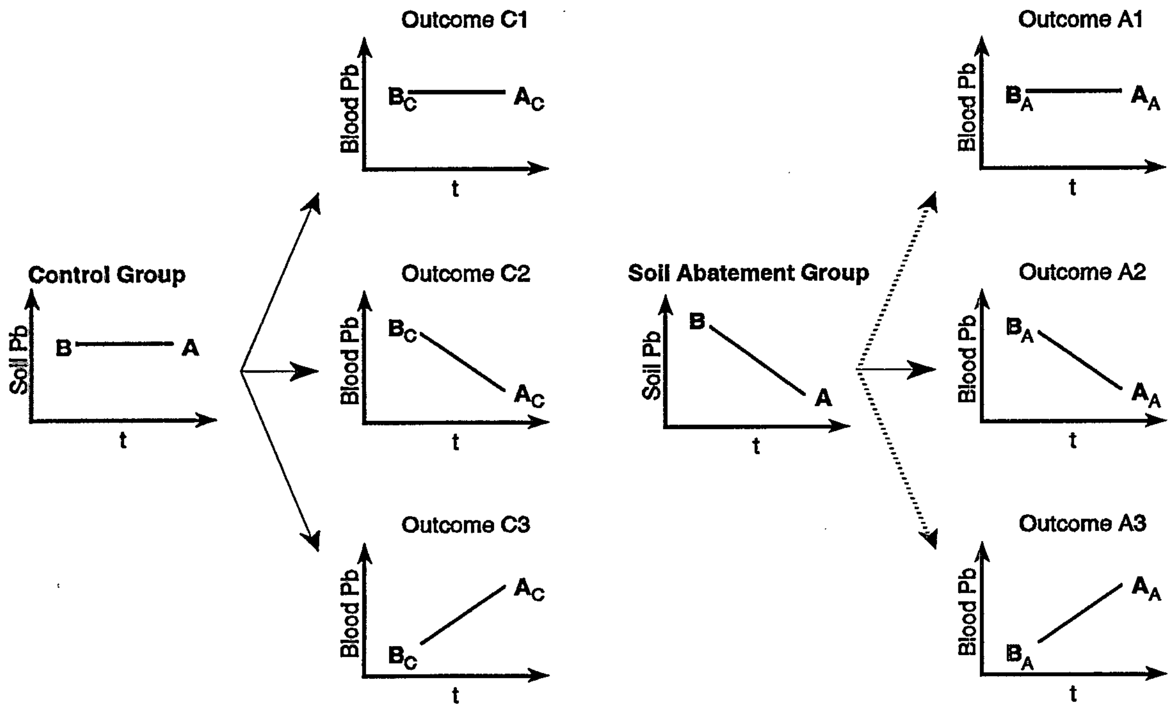

Figure 5-5. Schematic representation of expected outcomes for treatment and control groups.

abatement during the first year of the study. This is shown by a flat line connecting soil lead measured before (denoted B) and after (denoted A) the abatement period, because soil lead concentrations are expected to show little decrease during a year or two of study. The probable responses of blood lead are either no change in blood lead (denoted outcome C1) or a measurable decrease in blood lead (denoted outcome C2). The straight lines in outcomes C1 and C2 connect mean blood lead measured in the control group before (denoted B_C) and after abatement (denoted A_C). Similar results could conceivably occur in the soil abatement group, whose outcomes are denoted A1 and A2, and whose observed mean blood lead before and after abatement are denoted B_A and A_A respectively.

Figure 5-6 shows all possible combinations of outcomes for the control group and the abatement group that could lead to different conclusions. The preabatement blood lead concentrations of these groups are shown as possibly different, because in the Baltimore and Cincinnati studies the soil abatement group was in a distinctly different neighborhood from the intended control group and had a different mean blood lead. Outcomes C1 and A1 occurring together show that blood did not change in either the soil abatement group or the control group, suggesting that there was no effect of the abatement. Outcomes C1 and A2 occurring together show that blood decreased in the soil abatement group and did not change in the control group, suggesting that there was a beneficial effect of the abatement. Outcomes C2 and A1 occurring together show that blood lead did not decrease in the soil abatement group and did decrease in the control group, suggesting that there might be a possible negative effect of the abatement compared to doing nothing that was not done for the control group. Outcomes C2 and A2 occurring together show that blood decreased in both the soil abatement group and in the control group, but the nature of the effect depends on the magnitude of the changes between the two groups, which are denoted as Types 1, 2, and 3 changes. In Type 1, blood decreased by the same amount in both groups, suggesting no effect of abatement. In Type 2, blood decreased by a greater amount in the abatement group than in the control group, suggesting a beneficial effect of abatement. In Type 3, blood decreased by a greater amount in the control group than in the abatement group, suggesting a possible negative effect of abatement. Again, these are hypothetical outcomes that illustrate the possibilities in interpreting the results of a longitudinal study. It is clearly not adequate to look at changes in blood lead in a single treatment group in the absence of an appropriate reference group or control group.

5.1.3 Conceptual Approach to Pre- and Postabatement Differences in Individuals

A potential problem arises in simple comparisons of group mean values during a longitudinal study when different individuals are present at different phases of the study. For example, some individuals in the preabatement phase of the study may have dropped out by the time of the postabatement phase, whereas other individuals who were not in the preabatement phase may have been recruited into the postabatement phase (e.g., infant siblings who reached enrollment age status during the study). Although it would be reassuring to think that attrition and recruitment do not depend on the treatment group, and that children lost or gained during the progress of the study are no different from those

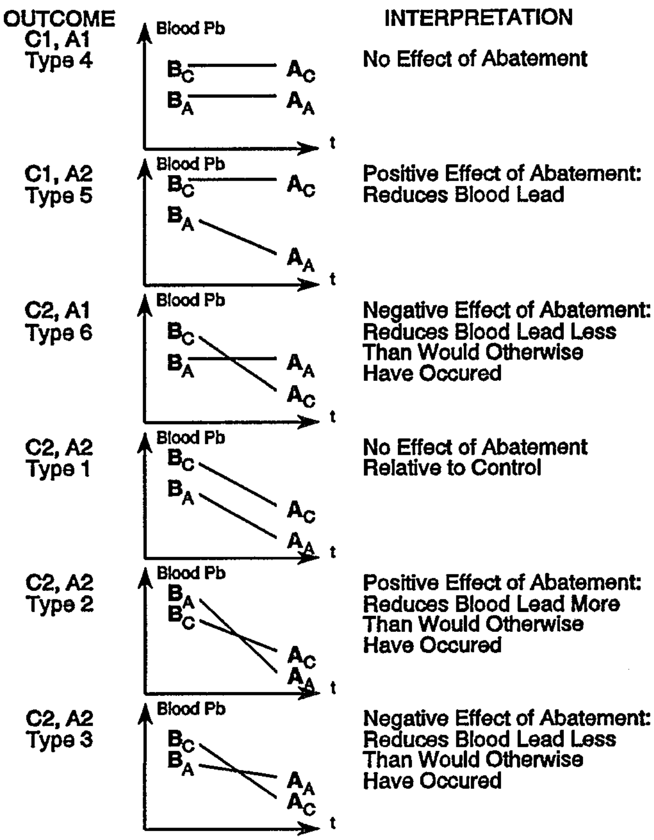

Figure 5-6. Schematic representation of the potential interpretations that might be reached from the various abatement outcomes.

enrolled throughout the study, this cannot be guaranteed. One of the simplest solutions is to limit the analyses to children who were present during all phases of the study.

When the analyses are restricted to subjects with both pre- and postabatement data, then abatement effectiveness may be assessed by simply taking differences of blood lead concentrations or differences of their logarithms. Unfortunately, blood lead differences ignore the intrinsic persistence of blood lead concentrations over time. The only part of the preabatement blood lead concentration that can be reduced by intervention is the nonpersistent part,

$$\text{removable blood lead} = \text{fraction of preabatement blood lead}$$

where the fraction for one year postabatement may be about 50%. The difference between preabatement and postabatement blood lead cannot be larger than the amount of removable blood lead. In other words,

preabatement − postabatement blood lead < fraction of preabatement blood lead.

This suggests that a better index for abatement effectiveness might be a partial difference:

postabatement − (1 − fraction) preabatement blood lead > 0.

Unfortunately, the value of this fraction is not known well enough to define a priori the partial difference for use as an index of lead effectiveness, because the value of the retained fraction of lead depends on the time since abatement and the child's age, and probably on other factors as well.

5.1.4 Conceptual Approaches to Repeated Measures Analyses

The simple comparison of typical values of blood lead concentrations among treatment groups at different phases of these longitudinal studies has certain limitations that may not be obvious to the reader. These limitations are the same whether blood leads are characterized by the group mean, geometric mean, median or other percentile values. The first is that some of the children in any treatment group are probably not exactly the same children at one phase of the study as at a subsequent phase. Some children will almost certainly be lost to follow-up by moving or by refusal to participate (normal processes of attrition in longitudinal studies), whereas other children may be added by recruitment (such as at Round 3 in the Baltimore study) or as additional members of households where other children are already enrolled in the study. Since children who are lost to follow-up or who are added to the study may differ in some systematic ways from children who were retained throughout the study, it may be prudent to analyze data from these children who were not present separately from those who were present at all relevant phases. On the other hand, if study results are restricted only to children who were present at certain specific pre- or postabatement phases of the study, then repeated measurements on the same child at different phases of the study are not statistically independent of each other. Although data from one treatment group at a given phase are independent of data from a different group, data on the same group at a different phase are not independent of data from an earlier phase.

Data from the same individual at different phases of a study can be analyzed as "repeated measurements" techniques. "Repeated measurements analyses" is a statistical term usually applied to a certain kind of mixed model multivariate analysis of covariance in which it is assumed that there are several distinct kinds of predictors for the response variable (such as blood lead):

(i) *Repeated observation phases* (for example, pre- and postabatement rounds);

(ii) *Within-individual non-random differences or fixed effects attributable to specific covariates* (for example, hand lead or dust lead loading at each round);

(iii) *Within-individual random differences not attributable to specific covariates or treatment groups* (random error at each round);

(iv) *Between-individual non-random differences (fixed effects) attributable to specific treatment groups or between-group covariates* (for example, the treatment group could be a control group or soil abatement group or neighborhood, and the average soil lead concentration or percentage of non-gut-rehab houses within a neighborhood could be a numeric covariate);

(v) *Between-individual random differences attributable to other factors* (for example, being in different households or families, when there are some households with multiple children enrolled in the study);

(vi) *Between-individual random differences not attributable to specific covariate or other factors (a random intercept term).*

Let us provide an explicit mathematical model to illustrate these points. This model will be a linear model of the sort that could be fitted using SAS PROC MIXED or similar statistical programs. We will first define the subscripts corresponding to each case:

g = group index, such as neighborhood or treatment group (treatment groups are often denoted RGP for remediation group in the models we used);

h = household or other "nested" unit within each treatment group (often denoted FMID in the models we used);

I = individual index or identifier (denoted KDID in the models we used);

j = round or phase of the study.

The generic form of the model is defined as follows:

$$Y_{ij} = G_{gj} + H_{h(g)} + I_{i(gh)} + X_{ij} B_{gj} + e_{ij}.$$

In the above sequence of effects, the response variable for child I at round j is denoted Y_{ij}, and the other terms are identified as follows:

(i) *Repeated observation phases*, denoted j;
(ii) *Within-individual non-random differences or fixed effects attributable to specific covariates* (for fixed effect of predictor X in child I at round j, denoted $X_{ij} B_{gj}$);
(iii) *Within-individual random differences not attributable to specific covariates or treatment groups* (denoted e_{ij} for child I at round j);
(iv) *Between-individual non-random differences (fixed effects) attributable to specific treatment groups or between-group covariates* (denoted G_{gj} for treatment group g at round j in this example);
(v) *Between-individual random differences attributable to other factors* (denoted $H_{h(g)}$ for household h in group g in this example);
(vi) *Between-individual random differences not attributable to specific covariate or other factors* (denoted $I_{i(gh)}$ for child I in group g, household h, in this example).

Hypotheses about treatment group effects could be formulated in terms of *contrasts*, which are pre-specified linear combinations of group effect estimates, for example:

Difference in group g between rounds j = 1 and j = 2
$= G_{g1} - G_{g2}$;

Difference between groups g=1 and g=2 at round j
$= G_{1j} - G_{2j}$;

Effect of treatment g=2 relative to treatment g=3 between rounds 1 and 4
$= G_{21} - G_{24} - (G_{31} - G_{34})$
also $= G_{21} - G_{31} - (G_{24} - G_{34})$;

Effect of treatment g=2 relative to average of treatments g=1 and g=3 between rounds 1 and 4
$= G_{21} - G_{24} - 0.5\,(G_{11} - G_{14}) - 0.5\,(G_{31} - G_{34})$;

Difference in effect of covariate adjustment at round 4 between groups 1 and 2
$= B_{14} - B_{24}$ per unit of X.

Several approaches are evaluated for analyzing the longitudinal data from the three cities using "repeated measures" models. Several convenient computer implementations of the method are available. We tried three versions and found that in many cases, the ability to identify differences among interventions was greatly improved by including covariates in the analyses. For example, child blood lead is known to change with age. When age is included as a covariate, some of the variation in blood lead differences before and after abatement can be attributed to the age of the child when the abatement was carried out. This increases the ability to estimate the relationship between blood lead and other variables, such as soil lead. Similarly, the effect of abatement may depend on changes in proximate exposure variables such as house dust lead. The effects of changes in house dust lead may be different at different ages, however, so that other covariates that may be useful in the analyses include interactions between age, house dust lead, and treatment group.

The use of baseline preabatement environmental or demographic measurements as covariates allows one to proceed as if all groups had the same starting values. The use of differences in environmental measurements before and after abatement allows one to proceed as if individuals responded similarly to similar changes in lead exposure, which is a fundamental assumption in a remediation and intervention program. It might even be useful to evaluate treatment effects adjusted only for the final postabatement values of the covariates if one assumed that blood lead differences reflected only the final post-abatement lead exposures. In general, differences in environmental indices before and after abatement were found to be more predictive of blood lead changes than the absolute baseline or final values.

Repeated measures analyses can be carried out using standard statistical programs for analyses of general linear models. PROC GLM in the SAS statistical package (SAS, 1990) and the MGLH procedure in the SYSTAT statistical package (SYSTAT, 1990) were used for most of the analyses. Analyses of repeated measures models with time-varying covariates cannot be conveniently carried out using these programs, so some analyses were therefore done using the P2V and P5V programs in the BMDP (BMDP, 1993) statistical package. Repeated measures models with more than two phases or time points may require specific assumptions about time correlation structure in some programs, which can be done using generalized estimating equation (GEE) approaches such as that used in some of the Baltimore analyses, but no such assumptions are needed when comparing outcomes at only two time points, pre- and postabatement.

5.1.5 Conceptual Approach to Structural Equation Modeling

Even though statistical models could be based on the partial differences of blood lead levels between pre- and postabatement phases, the environmental exposure variables are themselves more or less correlated with earlier measurements of the exposure variables. This violates one of the most important assumptions about linear regression models, and generally about linear models such as the analysis of variance and the analysis of covariance. That assumption is that the predictor variables or regressors are known without statistical error. Although the statistical error is usually called "measurement error" (Fuller, 1987), the errors include many other kinds of variability. In environmental epidemiology, the most common measurement errors in exposure include behavior or activity pattern variability, repeat sampling variability, sampling location variability, as well as analytical error. That is, the observed value of the predictor, such as floor dust lead loading, may not perfectly reflect the activity of the child and the child's actual exposure to dust lead over time.

One way to deal with this is to predict the precursor exposure variables in an environmental model. For example, suppose that blood lead is predicted by hand lead, soil and dust lead, and by a preceding value of the blood lead. Hand lead may then be predicted by current dust and soil lead levels, and dust lead by current soil lead, so that in addition to the direct effect of soil lead on blood lead, there are indirect effects from soil to dust to hand to blood, and from soil to hand to blood. This approach allows estimation of the measurement error variance in the precursor lead exposure variables in terms of residual deviations between the observed exposure variable and its best estimate from its own precursors. If the model is correct, this approach will essentially eliminate the bias introduced by measurement errors. The usual bias in estimating a regression coefficient or effect size of intervention will be to deflate or attenuate the estimate (i.e., to shrink the estimate towards 0, which reduces both its magnitude and its statistical significance). However, with multiple correlated predictors such as lead soil and dust variables for a single residential premises used in these analyses, this attenuation may not occur (Klepper et al., 1993).

Structural Equation Modeling is a computational approach that allows estimation of sets of inter-related linear or nonlinear models (Buncher et al., 1991). This has been widely used for cross-sectional environmental pathway modeling (Bornschein et al., 1985, 1988, 1990; Marcus, 1991, 1992). Applications to longitudinal lead studies have recently been developed (Marcus, 1991; Menton et al., 1994; Marcus and Elias, 1994). PROC MODEL program in the SAS ETS computer package (SAS, 1992) allows estimation of either linear or nonlinear models. This procedure is believed to result in unbiased or less biased estimates of regression coefficients than other estimation procedures that do not include fitting simultaneous equations for blood lead to predictor variables such as lead in paint, soil, or dust.

The most complete and technically correct evaluation of these studies requires a simultaneous assessment of changes in blood lead levels and changes in environmental lead pathways following soil lead or dust lead abatement. Underlying any analysis of time-dependent relationships are the following assumptions:

(1) Both preabatement and postabatement blood lead levels reflect, in part, contemporary environmental lead exposures that can be characterized by measurements of lead levels in soil, dust, paint, and other media;

(2) Postabatement blood lead levels may also reflect, in part, preabatement blood lead levels due to the contribution of preabatement body burdens of lead (principally in the skeleton) from earlier exposures;

(3) Postabatement dust lead levels may also reflect, in part, preabatement dust lead levels due to mixing of incompletely abated or unidentified sources of lead in dust for which preabatement dust lead levels are a surrogate indicator;

(4) Postabatement soil lead levels may also reflect, in part, preabatement soil lead levels due to mixing of incompletely abated or unidentified sources of lead in soil for which preabatement soil lead levels are a surrogate indicator;

(5) Even when lead-based paint has been stabilized, lead paint levels measured by P-XRF may also help to predict postabatement soil and dust lead levels from incompletely abated or unidentified sources of lead in soil and dust for which lead-based paint levels are a surrogate indicator.

These models were fitted using indicator or "dummy" variables for different study or treatment groups. Sometimes these indicator variables were used as "switches", for example when postabatement soil lead concentration is modeled as a fraction of preabatement soil lead for soil nonabatement groups, but as a new replacement value for the soil abatement groups. At other times, indicator variables were used when the data suggested that the effect of abatement was to modify the regression coefficient for the predicted variable (for example, floor dust lead concentration) for a pathway. In that case, separate coefficients were fitted to the product of the treatment group indicator and the predictor variable (for example, entry dust lead concentration) as well as separate intercept terms for each treatment group. Apart from this, the underlying assumptions in the Structural Equation Model approach are that abatement effects can be characterized by concentrations or loadings of appropriate environmental lead exposure variables, a concept that allows inferences about effects of hypothetical abatements at other levels of lead exposure.

5.1.6 Comparison of Interventions Across Studies

There were substantial differences among the three studies that complicated a direct comparison of intervention effectiveness. The differences included:

(1) different levels of soil lead abatement and intervention. Although all three studies excavated soil associated with child exposure, the Baltimore and Boston studies removed soil in the yard surrounding the child's home, usually a single detached dwelling unit. The Cincinnati study had most children in multi-family units, and removed soil and exterior dust from common play areas and accessible areas in the neighborhood. The Baltimore study did not include exterior dust abatement, whereas the Boston and Cincinnati studies were accompanied by substantial interior dust abatement.

(2) different "control" groups. The Baltimore control group used homes in a different distant neighborhood than the soil abatement homes. These homes had exterior paint stabilized in order to avoid further soil contamination, and the soil abatement group houses also had exterior paint stabilization. There was also a de facto control group in the soil abatement neighborhood, because houses with soil lead below 500 ppm were not abated. The Boston control group consisted of houses in the same neighborhoods as the houses that received soil and dust abatement. The Cincinnati control group houses received no treatment of any sort, and were located in neighborhoods that were some distance away from the abated neighborhoods.

Other conditions will facilitate comparison of the studies:

(1) all three studies have blood lead measurements that were made in late summer or early autumn (July to October) during the peak blood lead season, at least 8 months after abatement but not more than 15 months afterward;

(2) all studies have baseline or preabatement blood lead levels taken not more than 18 months before the summer-fall postabatement blood lead level in the same child, so that individual pre- and postabatement differences may be compared;

(3) all studies have hand lead data that were taken at or about the same time as the blood lead data, and may be used as proximate indicators of actual environmental soil and dust lead exposure or contact;

(4) all studies have preabatement residential dust lead levels linked to each child, and preabatement soil or entry-area dust lead levels as indicators of environmental exposure for each child;

(5) all studies have used the same or nearly identical protocols for blood lead and hand lead sampling and analyses;

(6) soil sampling and analysis protocols are very similar across studies; and

(7) dust lead sampling and analysis were done by somewhat different methods, but were calibrated to produce comparable dust lead and soil lead concentrations across all studies.

The application of many hypothesis tests to the same set or subset of data may greatly distort the overall significance level of the entire decision-making process. This problem of multiple comparisons can be controlled by testing only hypotheses that are specified in advance. Because tests of the across-study hypotheses depend on the results of preceding tests on the pooling of certain groups within studies, the exact number of times that each data set is used in a test cannot be stated, but is not more than six tests. An extremely conservative approach is to assign experiment-wise significance at level alpha (for example, alpha = 0.05) only to those tests whose individual test-wise significance is at level alpha / (number of tests). That is, to assert that all of the results of six tests involving the same data set are significant at level 0.05, each test should be carried out at level 0.05 / 6 = 0.0083. Some authors argue that this adjustment, which is called the Bonferroni correction, is exceptionally conservative and that no adjustments are needed for multiple comparisons (Rothman, 1990). P levels are provided for each test to assist the reader who wishes to form his or her own judgements of the meaning of the results of the analyses. The decision level alpha of any statistical test is a subjectively chosen number. For most users of these tests, the conventional choice of alpha = 0.05 with the conservative decision to use an experiment-wise Bonferroni adjustment based on five tests per group per variable would suggest a test-wise level of 0.01 in order to decisively reject the hypothesis of no change, difference, or effect.

5.2 DIFFERENCES IN GROUP MEANS

5.2.1 Changes in Mean Soil Concentrations

Differences in group means are presented in the following set of figures. The subsets of participants in these figures are not necessarily the same as in comparable presentations in the individual reports. Therefore, the number of participants may also differ. In the Boston study analyses, we used the same subset of children as in the Boston report, excluding the same two children who had become lead-poisoned. For the Baltimore data, we chose to assign the small group of participants from the treatment group whose properties were not abated to a separate control group, rather than merge them with the main control group. We also report data for all children for a specific round, rather than all children in round one or children in all six rounds, as Baltimore reported. We treat the Cincinnati neighborhoods as individual treatment groups and include all children recruited, except for the four children were undergoing treatment for lead poisoning.

The presentation of these group mean data uses a similar format for all of the figures in this series. Each treatment group is represented in each round by a box and whisker plot. Each box has a mark approximately midway that shows the median value for the group and these medians are connected by a line between boxes. The upper and lower ends of the box mark the 3rd and 1st quartiles (75th and 25th percentiles) respectively. The tick marks on the upper and lower whiskers show the location of the 84th and 16th percentiles, respectively. (These two statistics are useful in estimating geometric distributions.) The diamond on the line or in the box shows the location of the arithmetic mean. These statistical parameters are shown in Figure 5-7, expanded for clarity. The data for these plots are given in Appendix A, Table A-1.

In order to form an effective, permanent barrier between the source of lead and the human environment, soil abatement must reduce the concentration of lead in the soil in a manner that is persistent for a period of years. In each of the three studies, measurements were made prior to abatement and immediately after abatement (within three months). Followup measurements were made periodically until the end of the study in Cincinnati and Boston. The results of these soil analyses are graphically illustrated in Figures 5-8 and 5-9. These data show, for all three studies, a substantial reduction in the amount of lead in abated soil areas. In Boston and Cincinnati, where follow-up soil measurements were taken, this reduction persisted for the duration of the study. In Baltimore, the postabatement measurements were made only in the locations where soil had been excavated and removed.

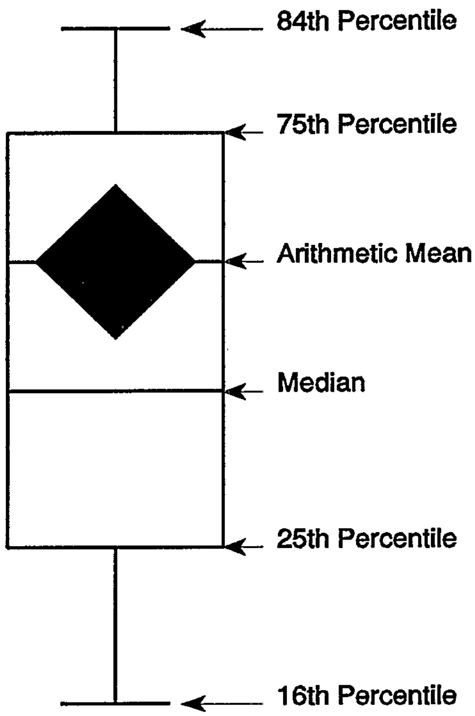

Figure 5-7. Hypothetical representation of common statistical parameters for a single group and a single round.

Each study was able to achieve the targeted concentration for abated soil. The median soil concentrations following abatement are not substantially higher than the specifications for clean soil. The amount of soil lead reduction actually achieved directly influences the expected changes in dust lead and blood lead. In Section 5.3, an attempt will be made to evaluate the treatment/response relationship for each step of the pathway of lead in the human environment.

To determine the effectiveness and persistency of soil abatement, the mean for each parcel of land was taken for each round where soil measurements were made. The median of these parcel means for the Boston and Cincinnati studies show that abated soil concentrations (BOS SPI and CIN SEI) dropped significantly after abatement (Figures 5-8 and 5-9) whereas unabated soil (BOS PI, BOS P, and CIN NT) appear to decrease only slightly, if at all. The Cincinnati groups CIN I-SE(B) and CIN I-SE(D), and CIN I-SE(F), which received soil and exterior dust abatement later (during the second year), showed a

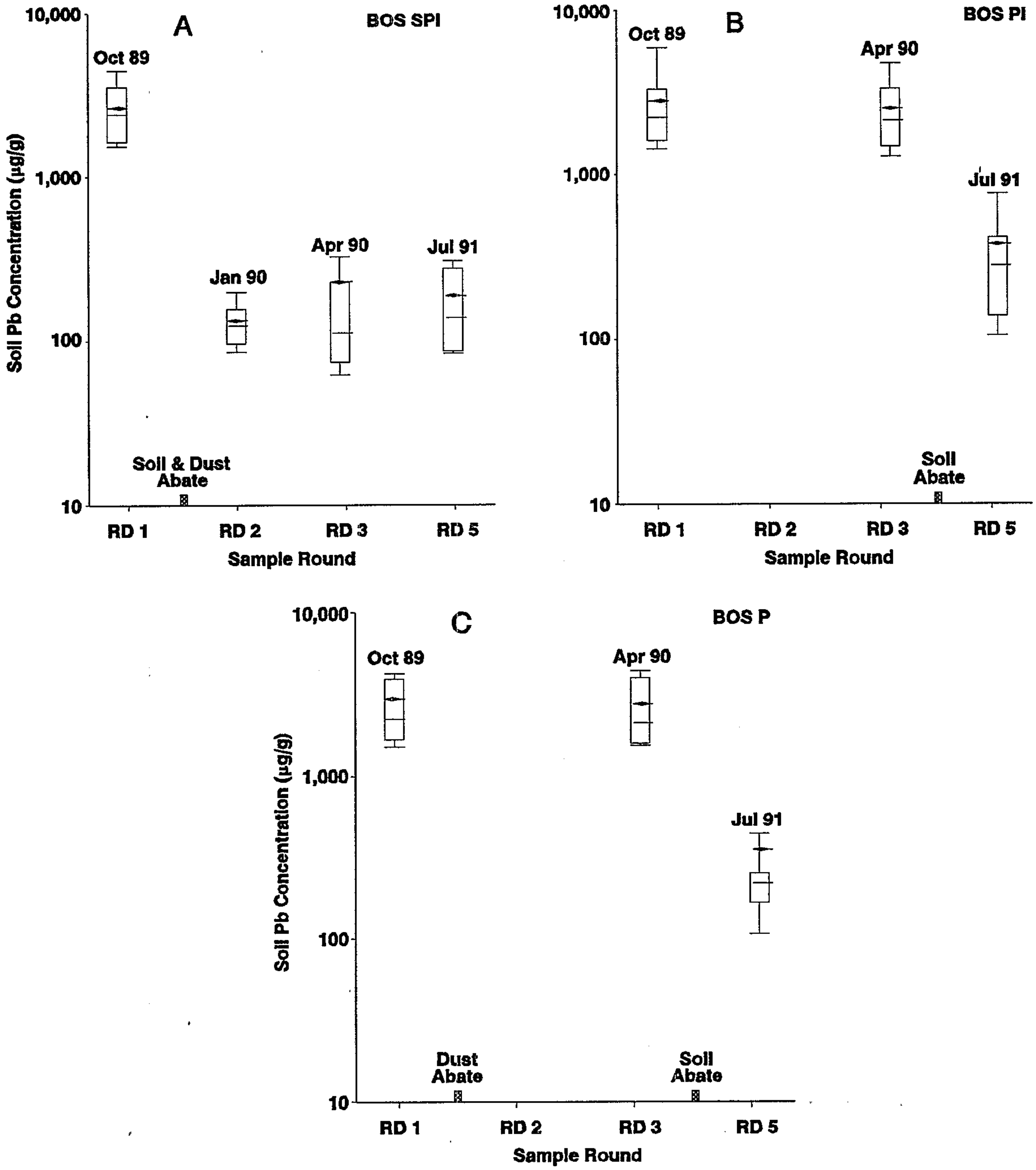

Figure 5-8. **Boston soil lead concentrations (on a log scale) by study group show the effectiveness and persistency of soil abatement. Note the decrease in soil lead concentrations (RD 2) immediately post soil abatement and persisting through RD 2, RD 3, and RD 5 for BOS SPI Group (Panel A); no soil lead sampling in RD 2 for other two groups (BOS PI and BOS I); RD 3 values for those two groups similar to their RD 1 soil lead concentrations; and the later marked decrease in their RD 5 soil lead values following soil abatement after RD 3.**

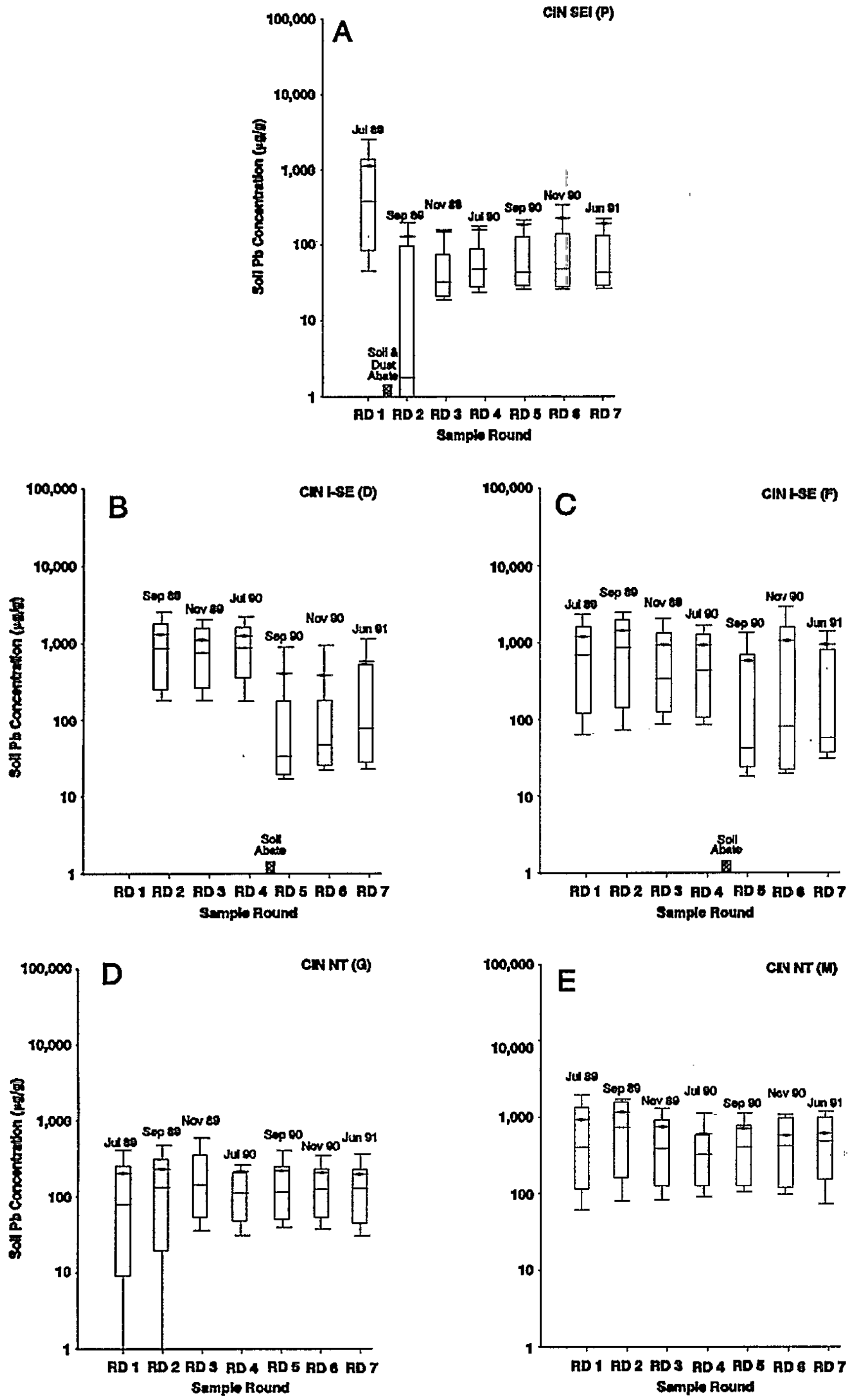

Figure 5-9. Cincinnati soil lead concentrations (log scale). Data are shown by neighborhood and reflect abatement in the first or second year of the study. There were no soil samples taken in the Dandridge neighborhood (Panel B) during round 1.

postabatement decrease in the range expected. Follow-up measurements of exterior dust after this second year abatement were limited to targeted entry areas.

There appears to be a general downward trend of soil lead concentrations. Although not statistically significant for any individual group, the fact that all treatment groups where the soil remained unabated show this phenomenon lends some credence to this observation. Analysis of QA/QC audit samples shows this trend cannot be attributed to analytical drift (see Section 3.1). Soil lead concentrations vary widely over relatively small distances. Because it was not feasible to return to the exact spot for sequential soil samples, two sequential samples may vary widely.

5.2.2 Changes in Exterior Dust Concentrations and Loadings

In Cincinnati, exterior street and sidewalk dust concentrations remained relatively constant throughout the study (Figures 5-10 and 5-11). This indicates that even though the relative contribution of lead from other sources may have changed over time, exterior dust abatement did not seem to be impacted by the contribution from these sources.

If the major source of the lead in exterior dust is soil and the soil parcels are abated prior to or at the same time as external dust abatement, then the lead concentration of dust on the streets and sidewalks should slowly decrease to a level comparable to the new soil concentration. This does not appear to be the case. Furthermore, the exterior dust lead concentrations in Cincinnati are much higher than the soil concentrations, suggesting a source or sources with higher lead concentrations than soil that mix with leaded dust from soil to form exterior dust. A possible conclusion is that sources of lead in exterior dust other than soil impacted each neighborhood differently. This is reasonable because the neighborhoods are geographically separated. Interpretation of the spatial distribution of the Cincinnati data is not possible without more information on the location of the dust samples.

For Boston and Baltimore, the question arises that there may also be external sources of lead other than soil that contribute to household dust and to the exposure of children during outside activities. Because there were no measurements of exterior dust in these studies, little evidence is available to accept or reject this hypothesis. However, in the context of exposure pathways, the parcels of soil in Boston and Baltimore were on the individual

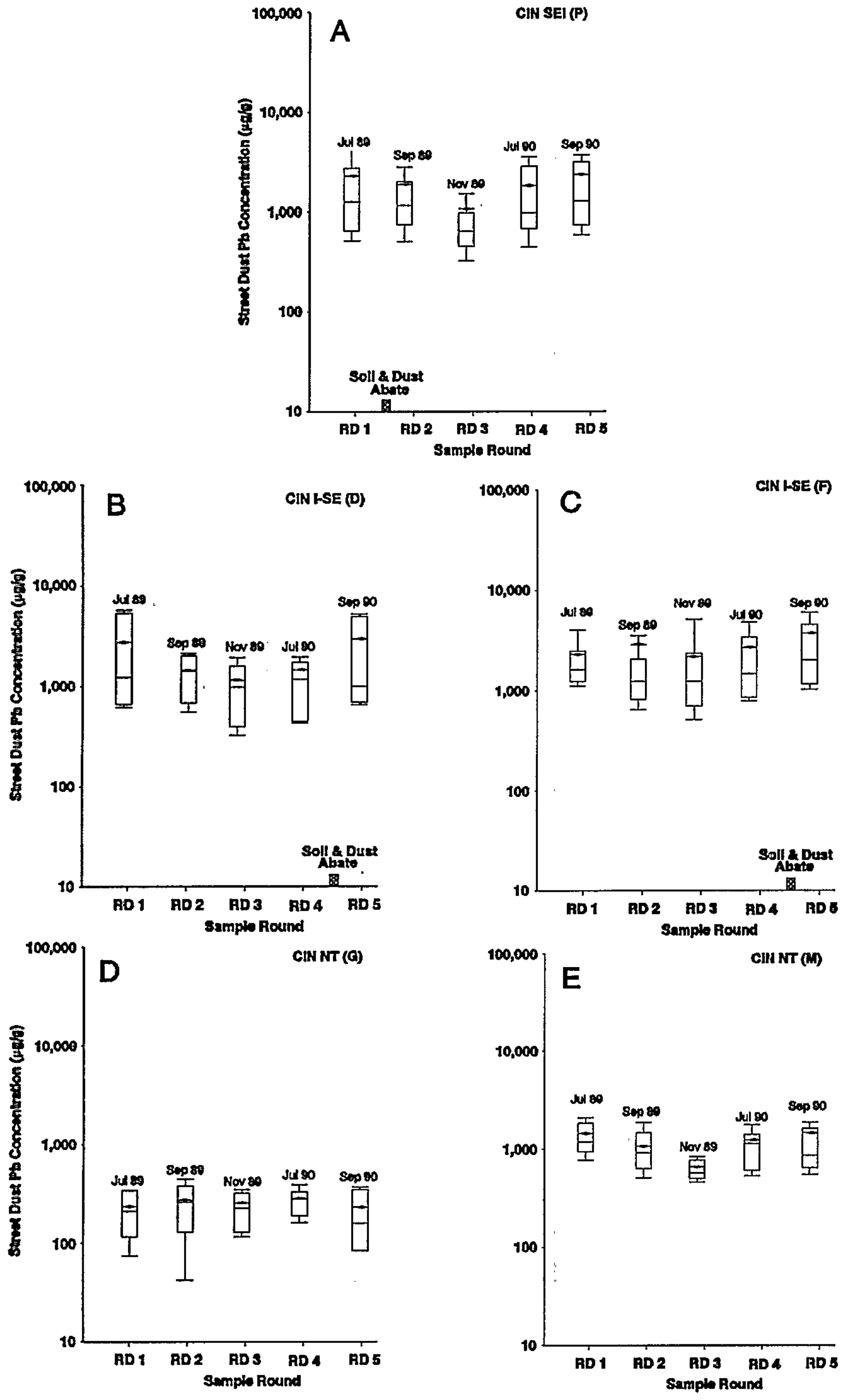

Figure 5-10. Exterior dust lead concentrations (log scale) from the street samples in the Cincinnati study. Data are by neighborhood. Exterior dust samples were not reported for rounds 6 and 7.

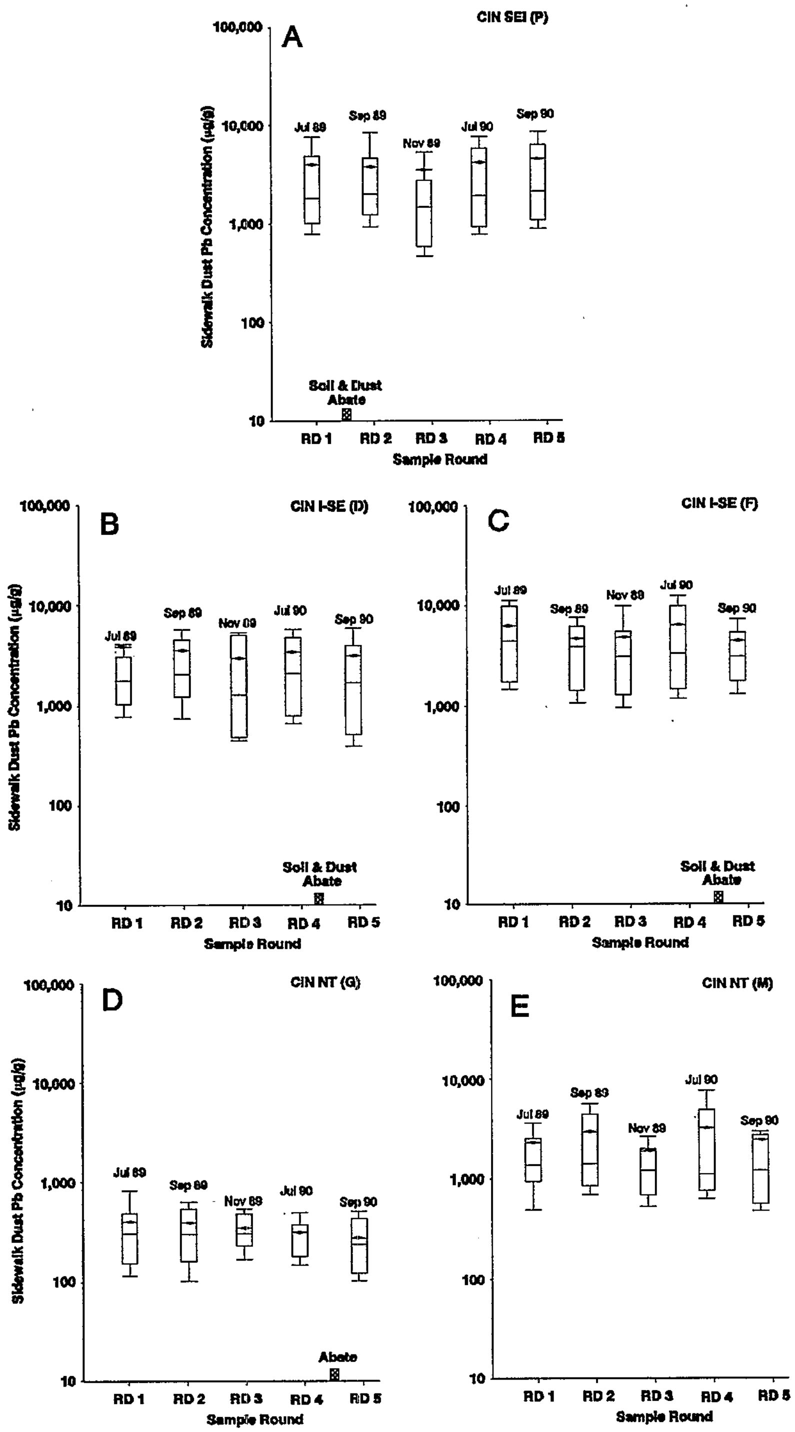

Figure 5-11. Exterior dust lead concentrations (log scale) from the sidewalk samples in the Cincinnati study. Data are by neighborhood. Exterior dust samples were not reported for rounds 6 and 7.

properties, whereas in Cincinnati, most soil parcels were in areas separated spatially from the living units, such as parks and vacant lots.

5.2.3 Changes in Interior Dust Concentrations and Loadings

Interior dust is measured in both concentration and surface loading. Concentration is measured in micrograms of lead per gram of dust, whereas loading is measured in milligrams of lead per square meter. When dust abatement is performed, the amount of dust changes, but the concentration of lead in the dust does not. Therefore, there should be no change in dust lead concentration unless the source of the dust changes. Where soil abatement has been performed in connection with dust abatement, the dust lead concentration should also decrease abruptly if the soil is the major component of the dust. If there is a mixture of dust sources and only one has been abated, the lead concentration would change less abruptly, according to the contribution from each source.

The data for the Boston study interior dust are shown in Figures 5-12 through 5-17. In both BOS SPI and BOS PI, there was a general decrease in the floor dust lead loading following interior dust abatement, as shown in Figure 5-14, and further decreases were observed at 7 to 12 months after abatement. In the window wells, however, the lead loading decreased immediately after dust abatement (Figure 5-17) persisted for a few months, then returned to original levels by 12 months after abatement. The high concentrations of lead in individual measurements of window well dust (5,000 to 22,000 μg/g) indicate lead-based paint was present (Figure 5-15).

The Cincinnati study (Figures 5-18 through 5-20) found an immediate reduction in floor dust lead loading that persisted for at least 5 months, followed by an increase by 12 months to 70% of the preabatement level in CIN SEI, where soil abatement had taken place, and to nearly twice the preabatement interior dust level in CIN I-SE-1 and CIN I-SE-2, where soil had not yet been abated. Similar patterns were observed in the window wells (Figures 5-21 through 5-23) and entry ways (Figures 5-24 through 5-26). The window well concentrations were lower in Cincinnati (1,000 to 2,300 μg/g) than in Boston, suggesting a minimum influence of lead-based paint.

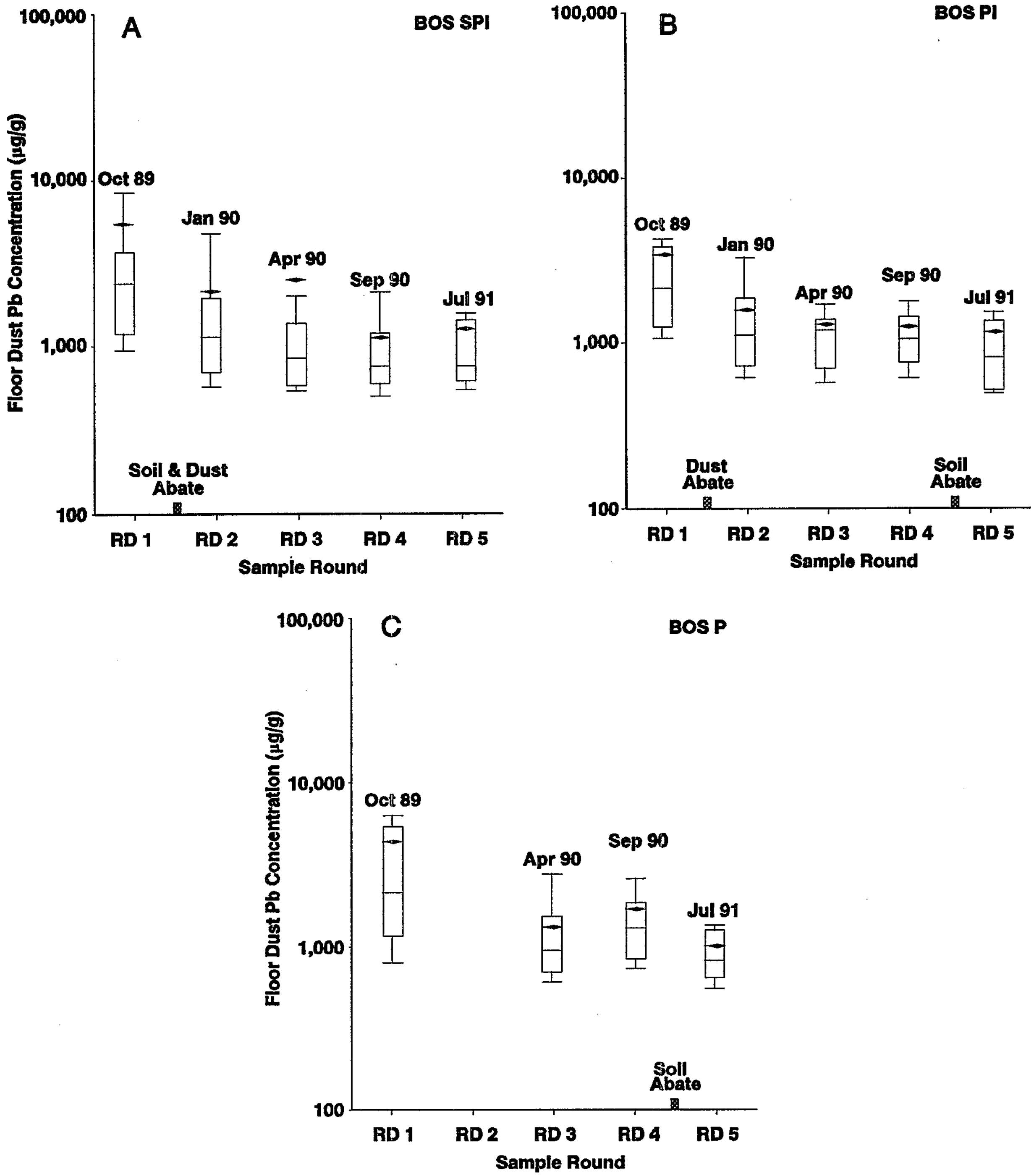

Figure 5-12. Boston floor dust lead concentration. While dust abatement alone may temporarily reduce the total dust lead loading (see Figure 5-14), it may not change the concentration of lead in any remaining dust.

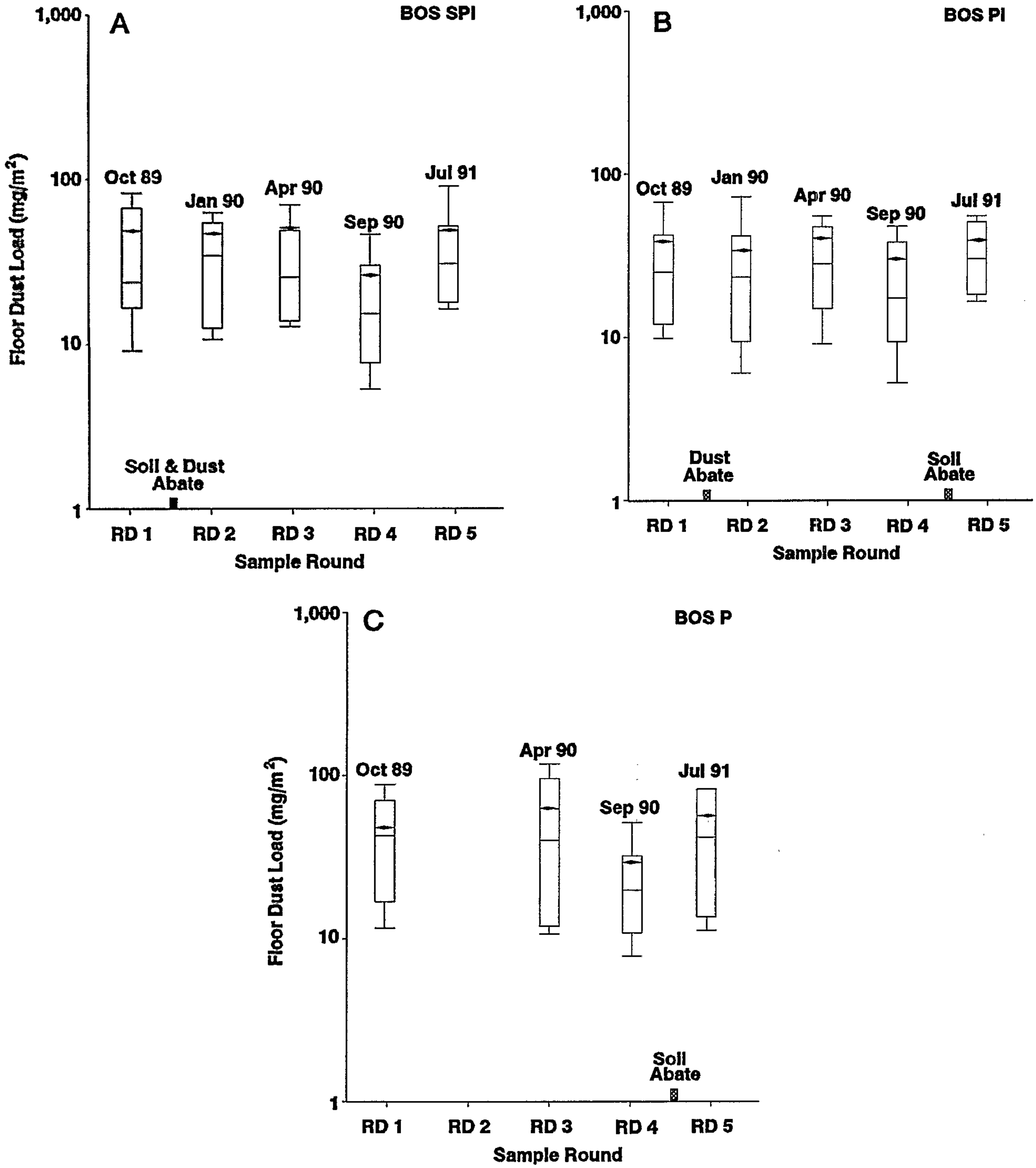

Figure 5-13. Boston floor dust load (log scale). The absence of a decrease following interior dust abatement in the BOS SPI and BOS PI groups suggest that house dust loadings may be replenished back to preabatement levels in a time period shorter than the interval between Round 1 and Round 2.

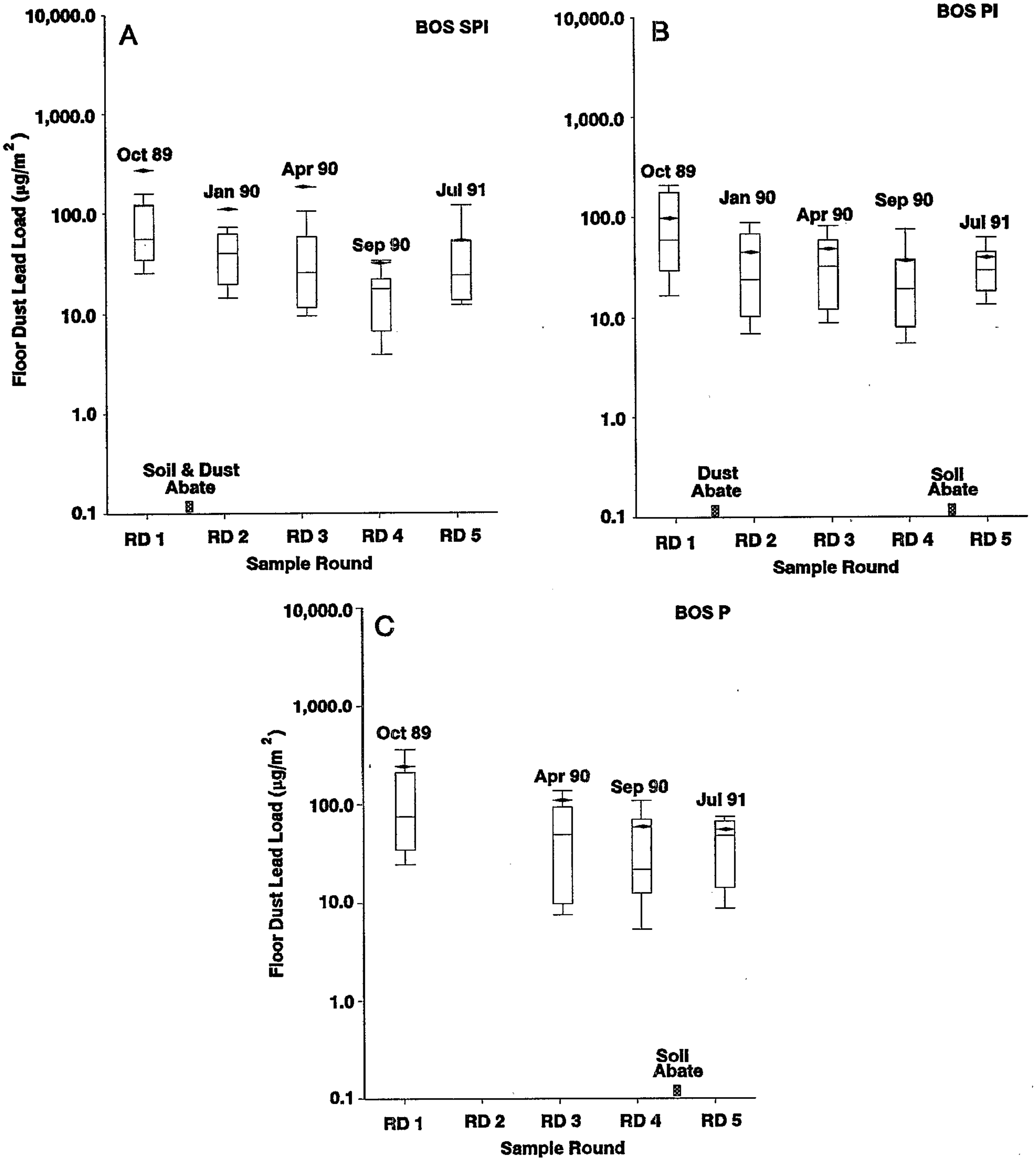

Figure 5-14. Boston floor dust lead load (log scale). Even though the dust load in Figure 5-13 indicates a quick recovery, the lead load did not recover immediately, indicating that the source of the lead was cut off, at least temporarily.

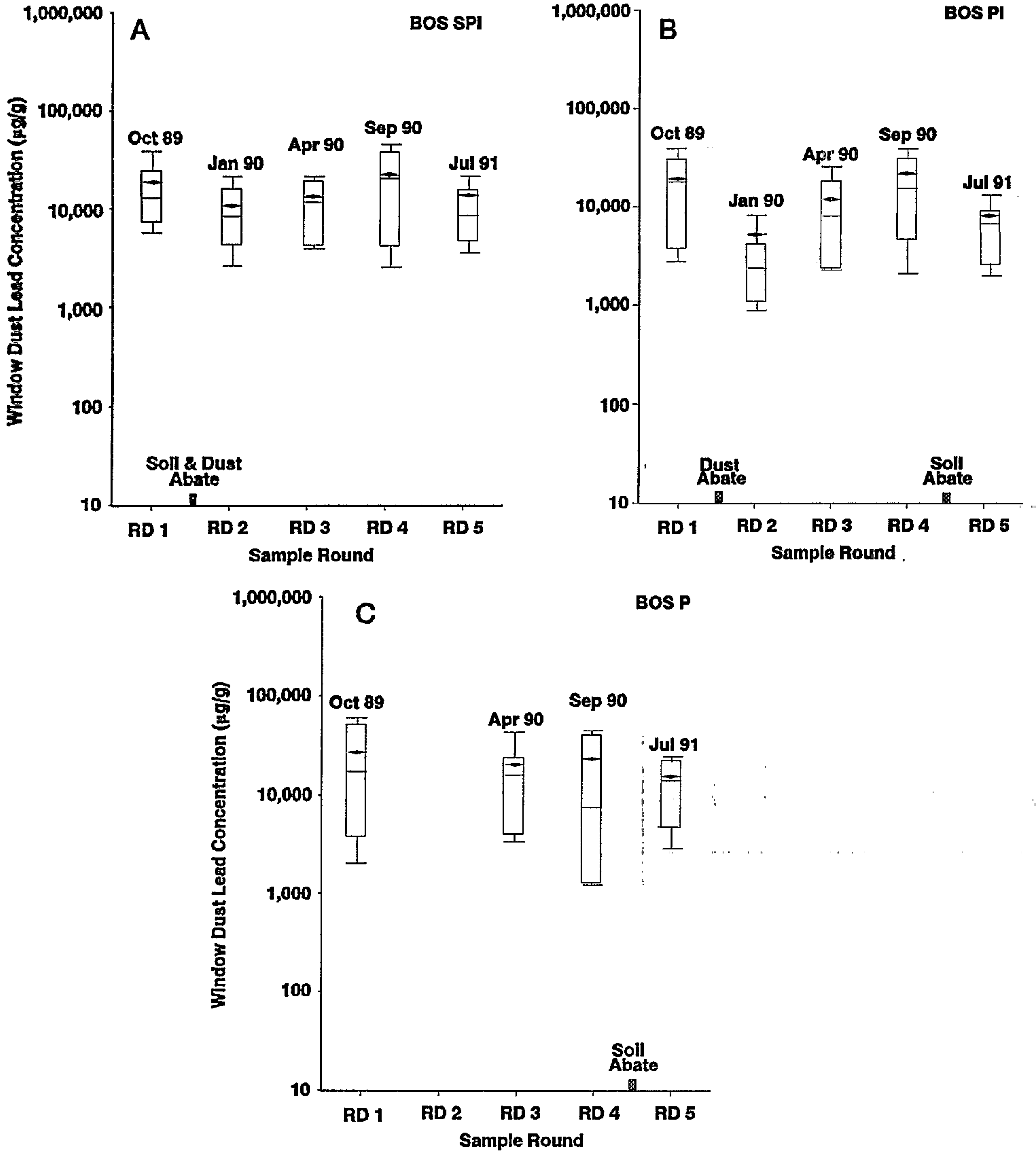

Figure 5-15. Boston window dust lead concentrations (log scale). Paint stabilization and soil abatement appear to have been effective and persistent for several hundred days, similar to floor dust. The recovery observed between April and July 1990 was not observed for the floor dust load data.

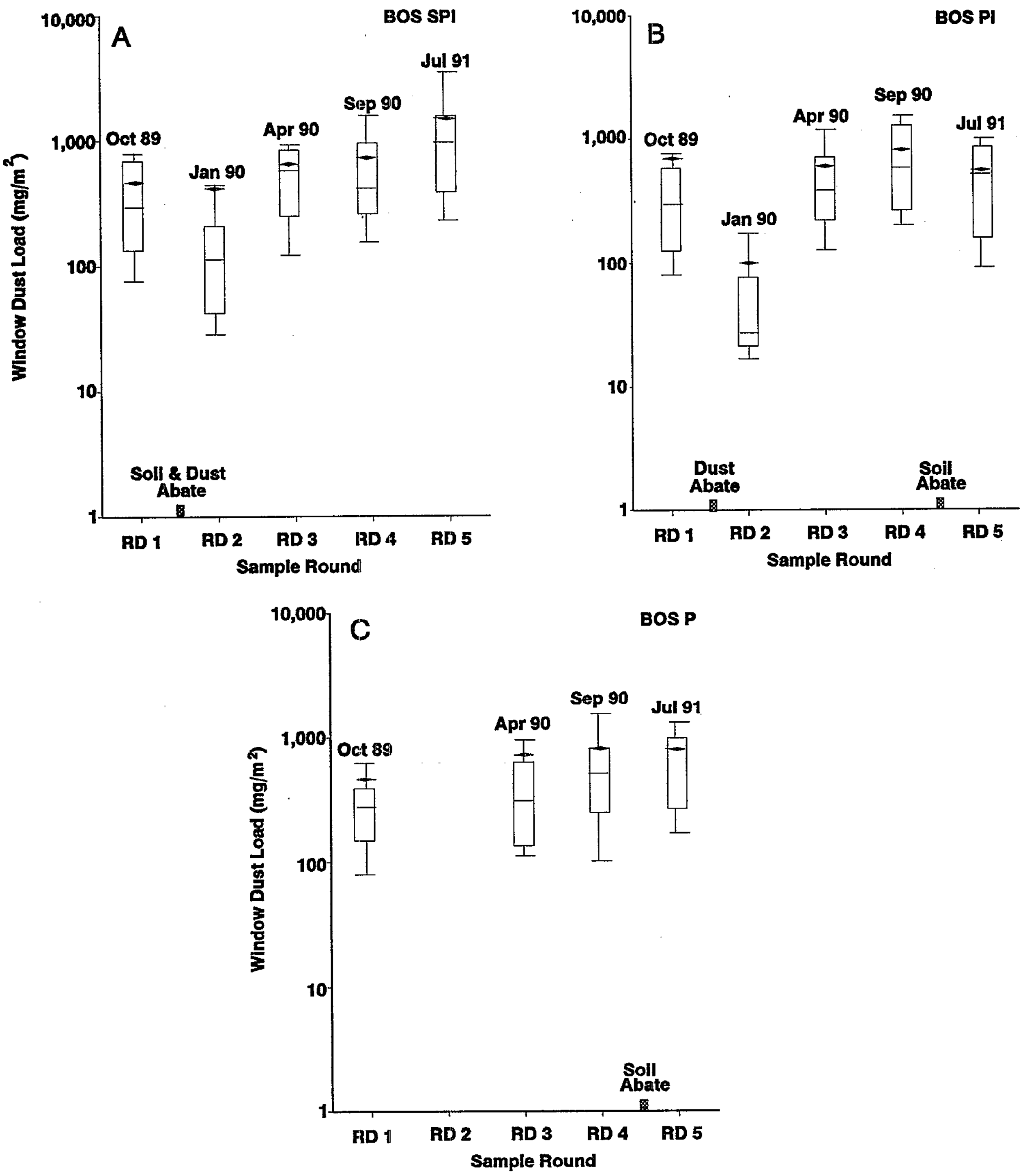

Figure 5-16. Boston window dust load (log scale). These data show the effectiveness of window dust abatement, which appears to recover after about 150 days, similar to floor dust loads observed in Figure 5-13.

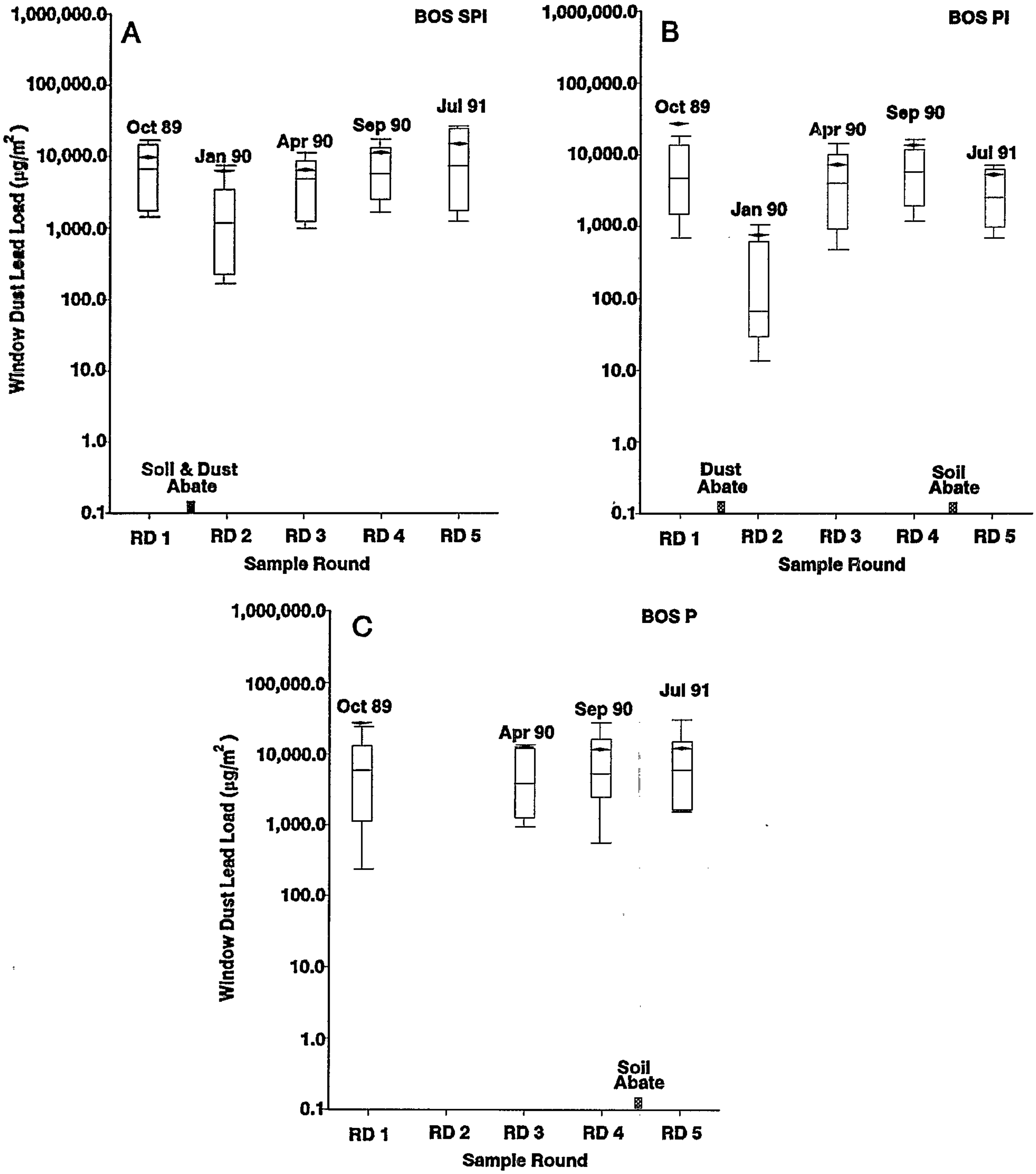

Figure 5-17. Boston window dust lead load (log scale). As with floor dust lead loads, the window data indicate that both paint and soil sources of lead were interrupted, at least temporarily. The data appear to be consistent with Figure 5-14.

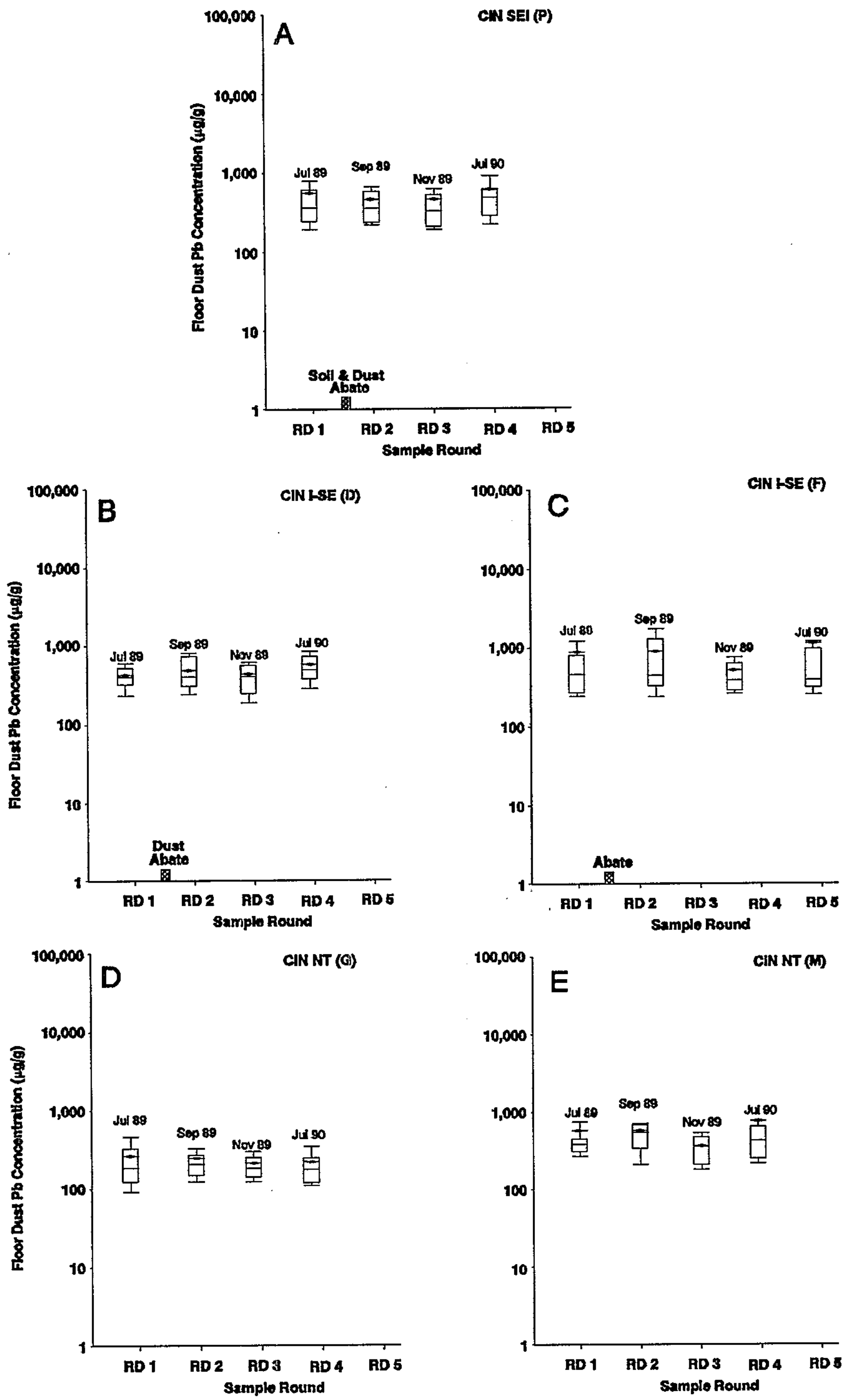

Figure 5-18. **Cincinnati floor dust lead concentrations (log scale). The small changes in lead concentrations across all sampling points suggest that the sources of lead and their relative contributions to housedust lead did not change as a result of the abatement activities.**

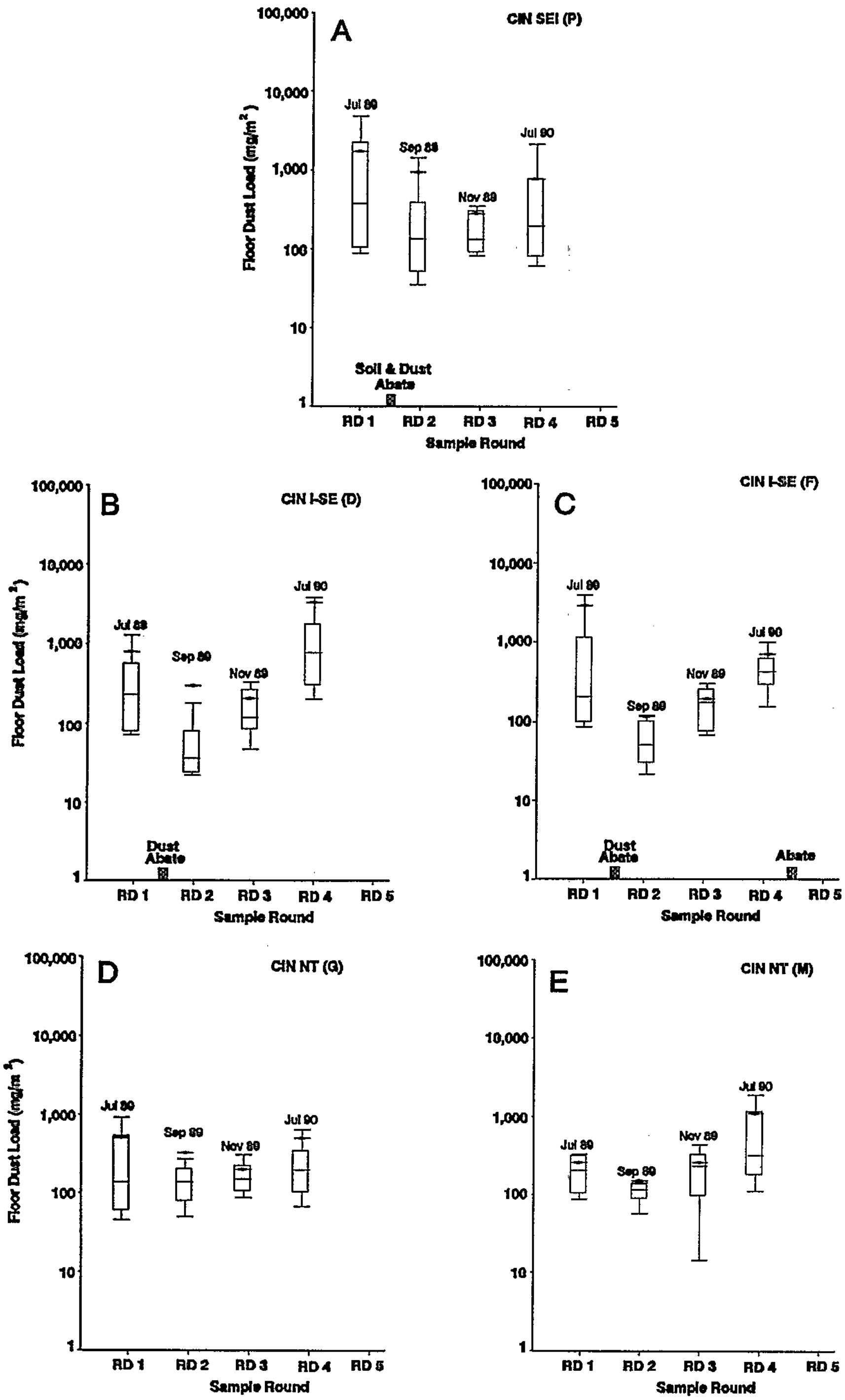

Figure 5-19. Cincinnati floor dust load (log scale). These data confirm the effectiveness of the household dust abatement and show that this reduction was persistent for as much as 60 days.

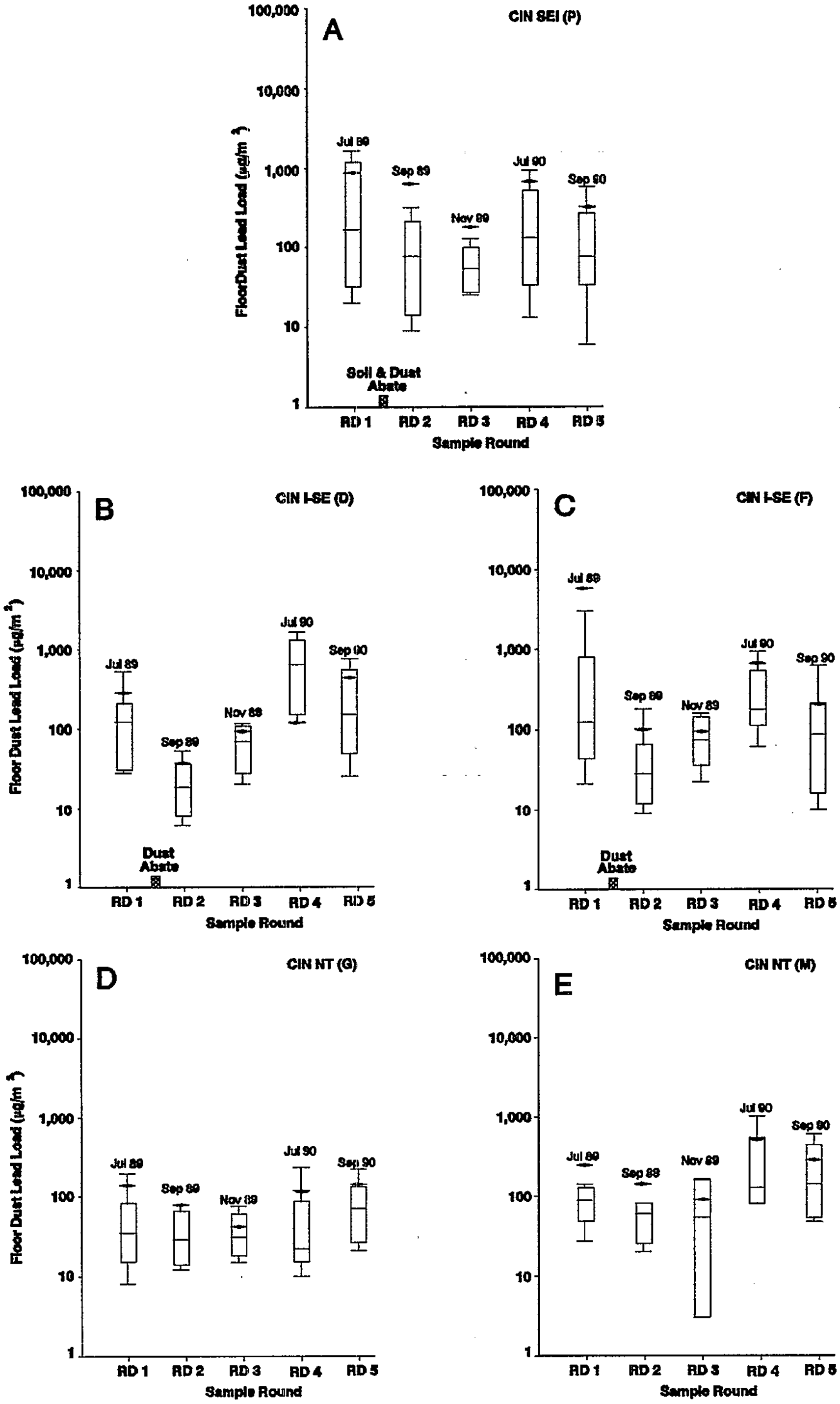

Figure 5-20. Cincinnati floor dust lead load (log scale). The data suggest that the sources of lead were interrupted by the abatement activities, but that at least one source recovered after November 1989.

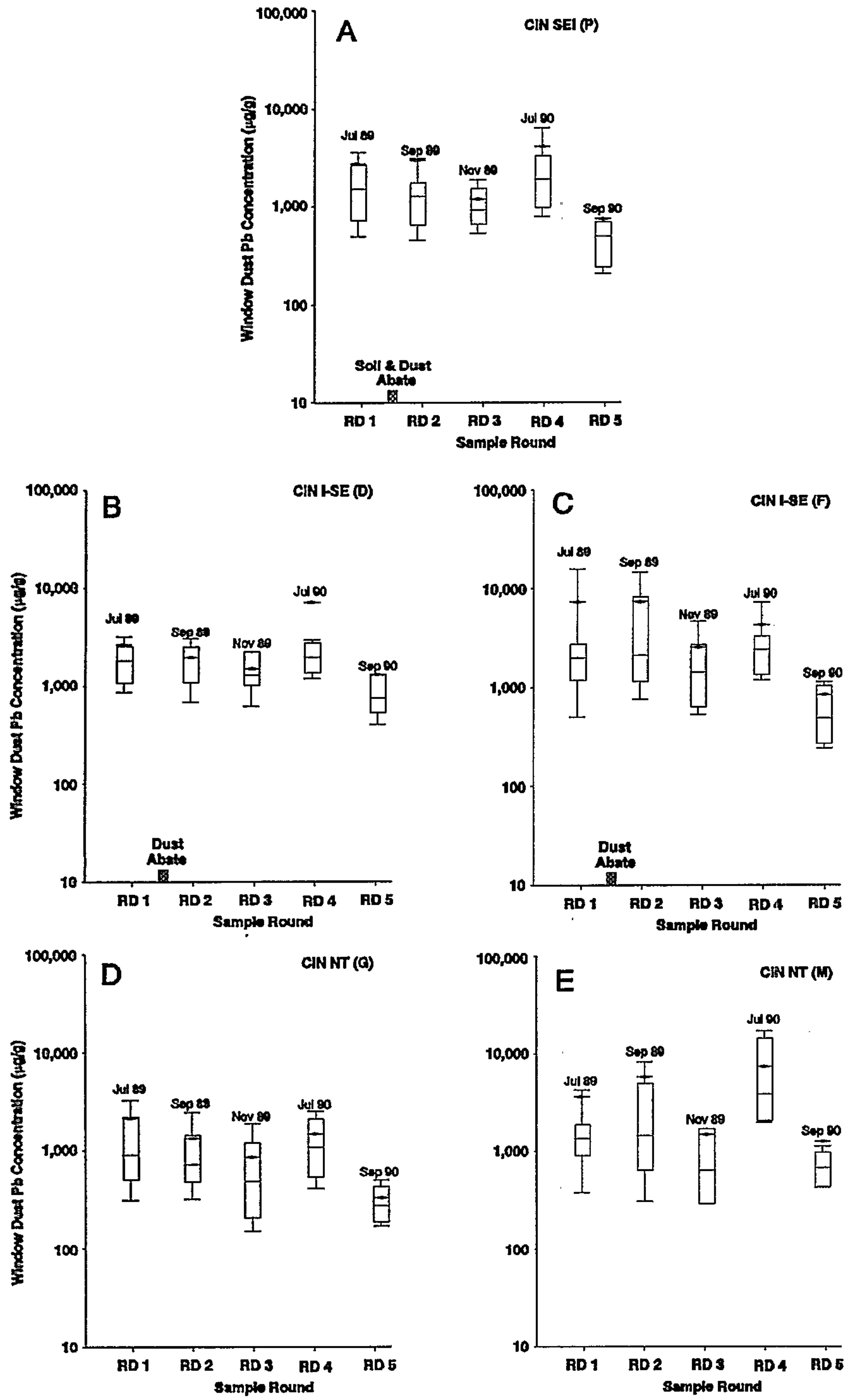

Figure 5-21. Cincinnati window dust lead concentration (log scale). The small response in lead concentration to soil and/or dust abatement appears to be consistent with the observations of the floor dust in Figure 5-18.

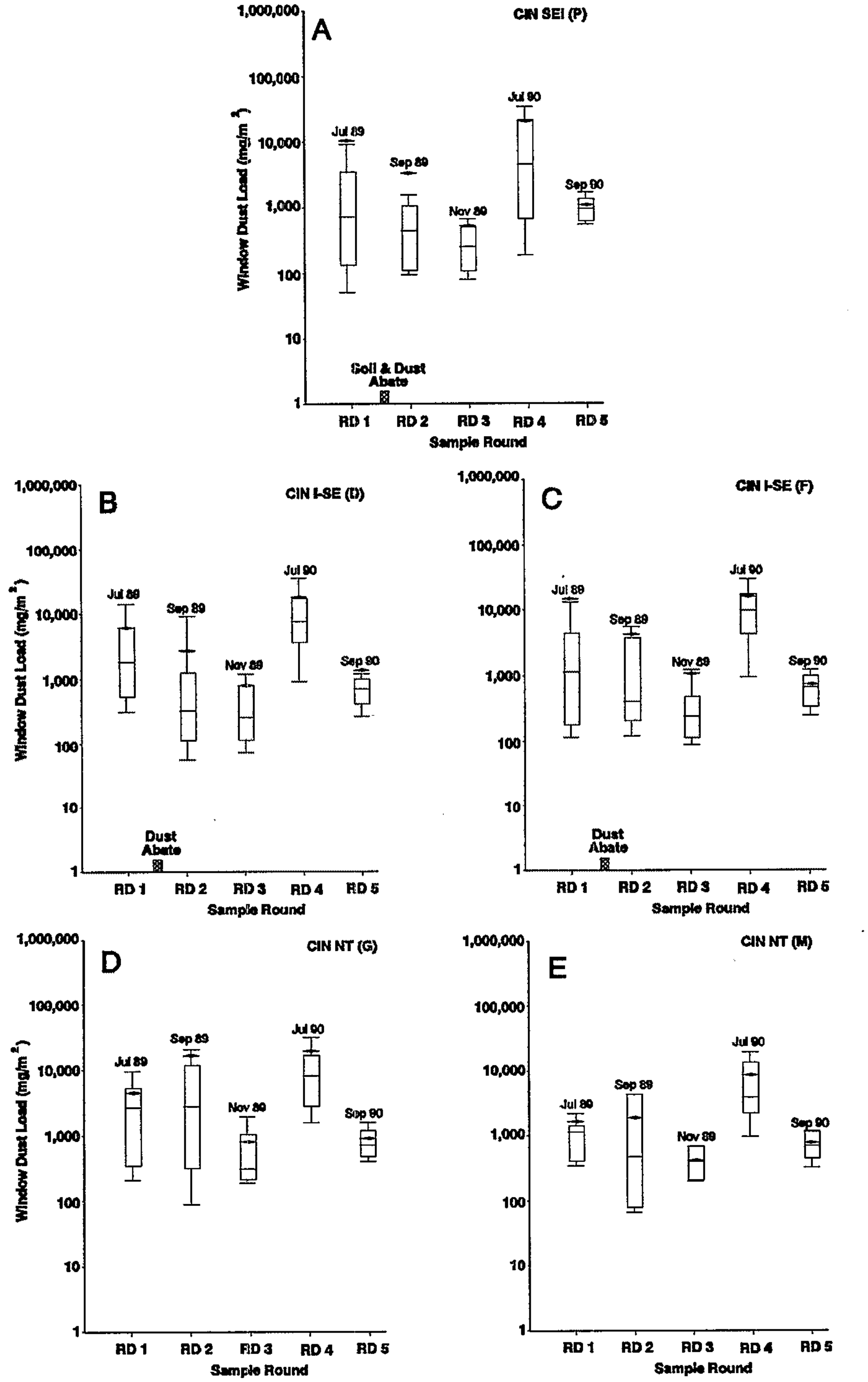

Figure 5-22. Cincinnati window dust load (log scale). The impact of abatement and the changes in the CIN NT groups are consistent between floor dust load (Figure 5-19) and window dust load as shown here.

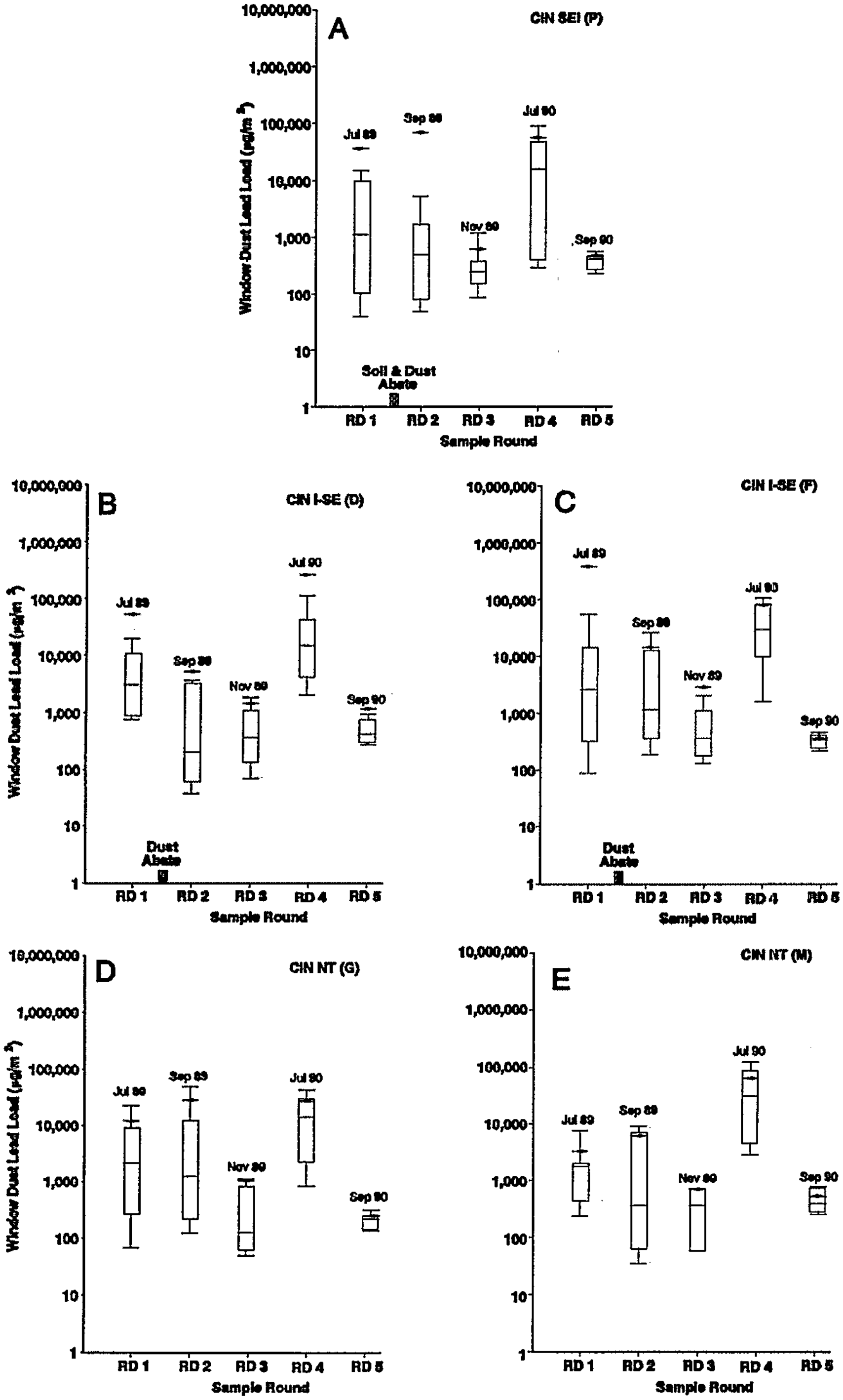

Figure 5-23. Cincinnati window dust lead load (log scale). The sharp increase between RD 3 and RD 4 may be due more to an increase in overall dust load (Figure 5-22) than in dust lead concentration (Figure 5-21).

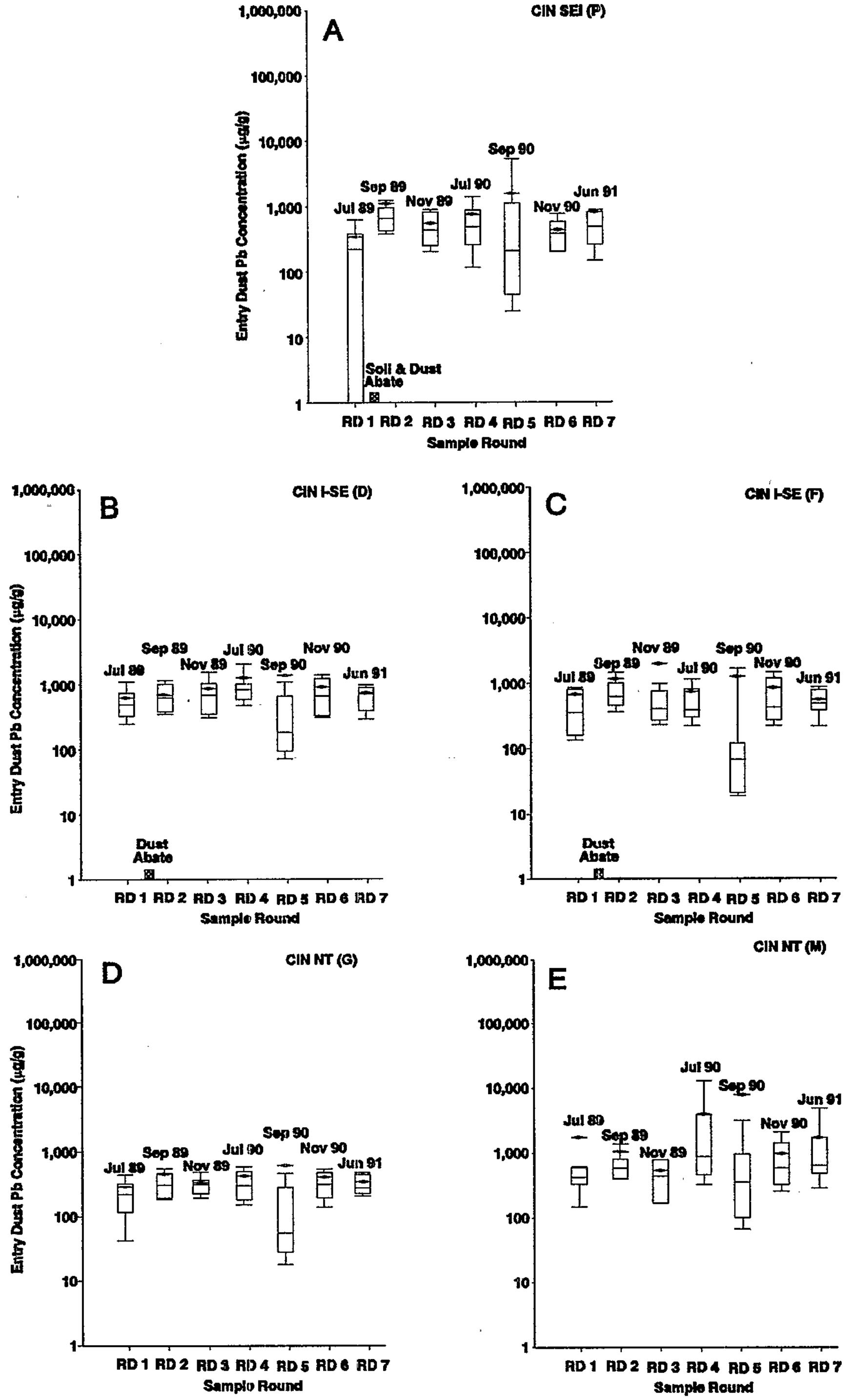

Figure 5-24. Cincinnati entry dust lead concentration (log scale). The entry way subset of the floor dust shows a pattern different from the complete floor dust data of Figure 5-18. Note the three additional rounds, September 1990, November 1990, and June 1991.

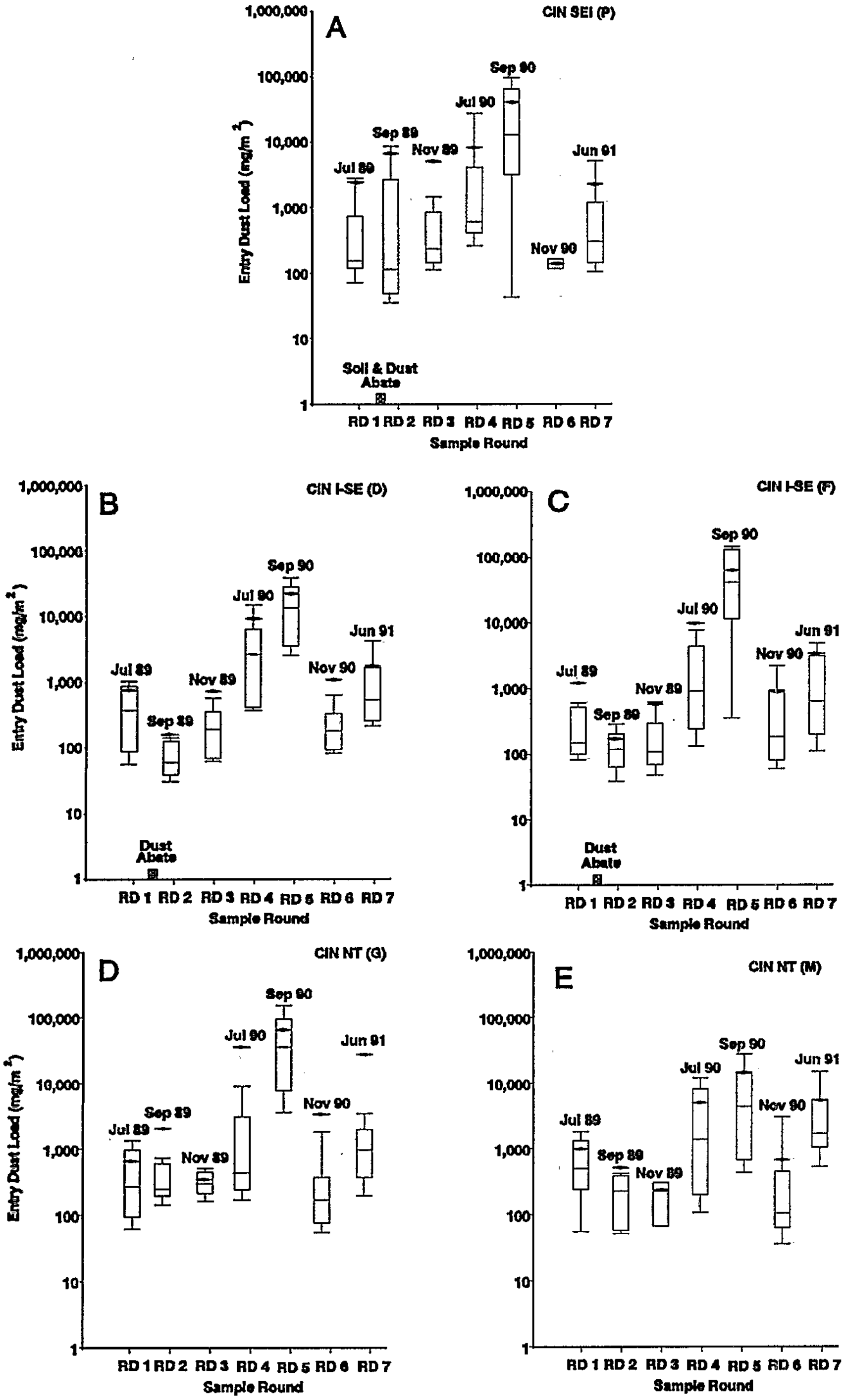

Figure 5-25. Cincinnati entry dust load (log scale). Similar to Figure 5-19, dust abatement at the entry appears to have been effective and persistent through November 1989.

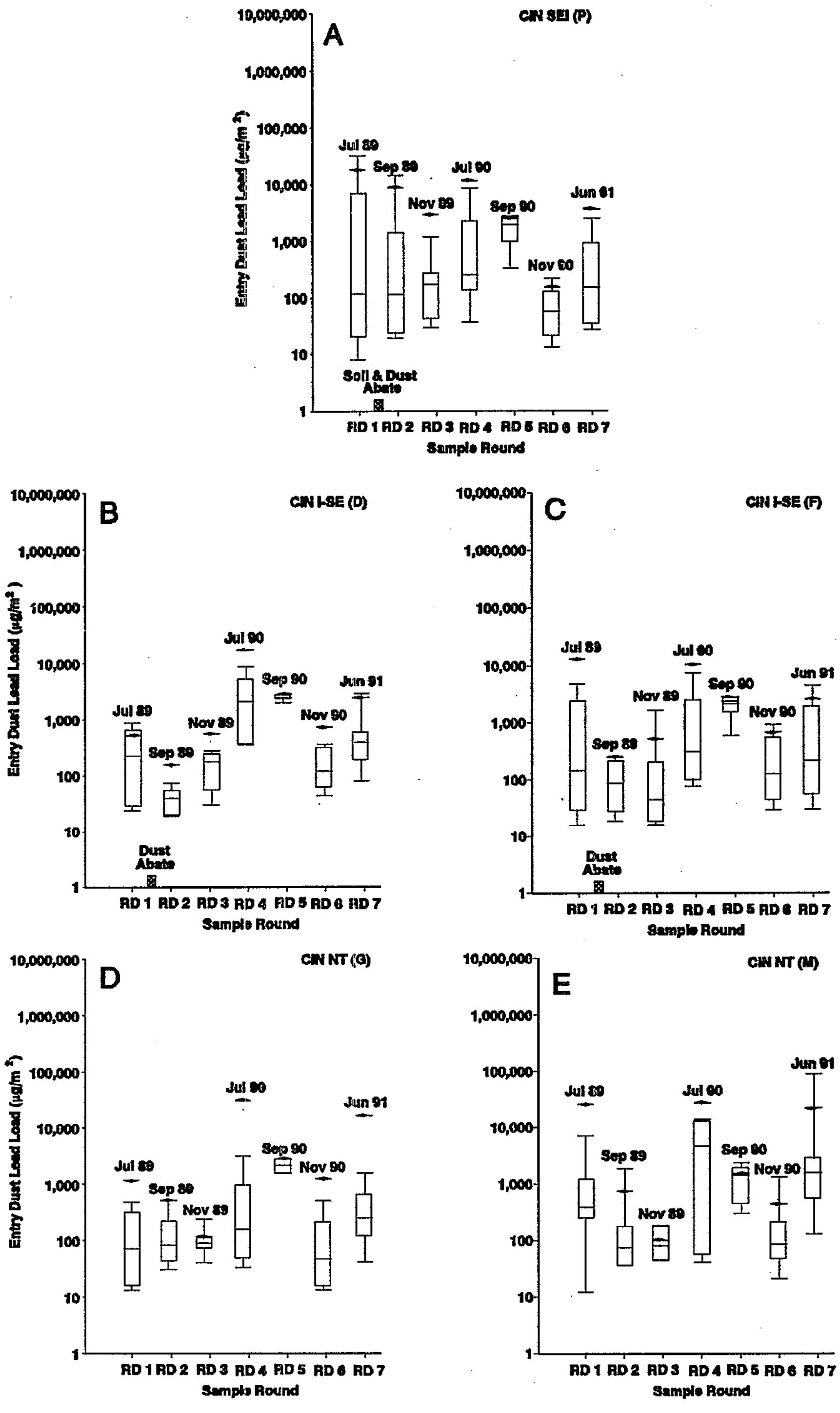

Figure 5-26. Cincinnati entry dust lead load (log scale).

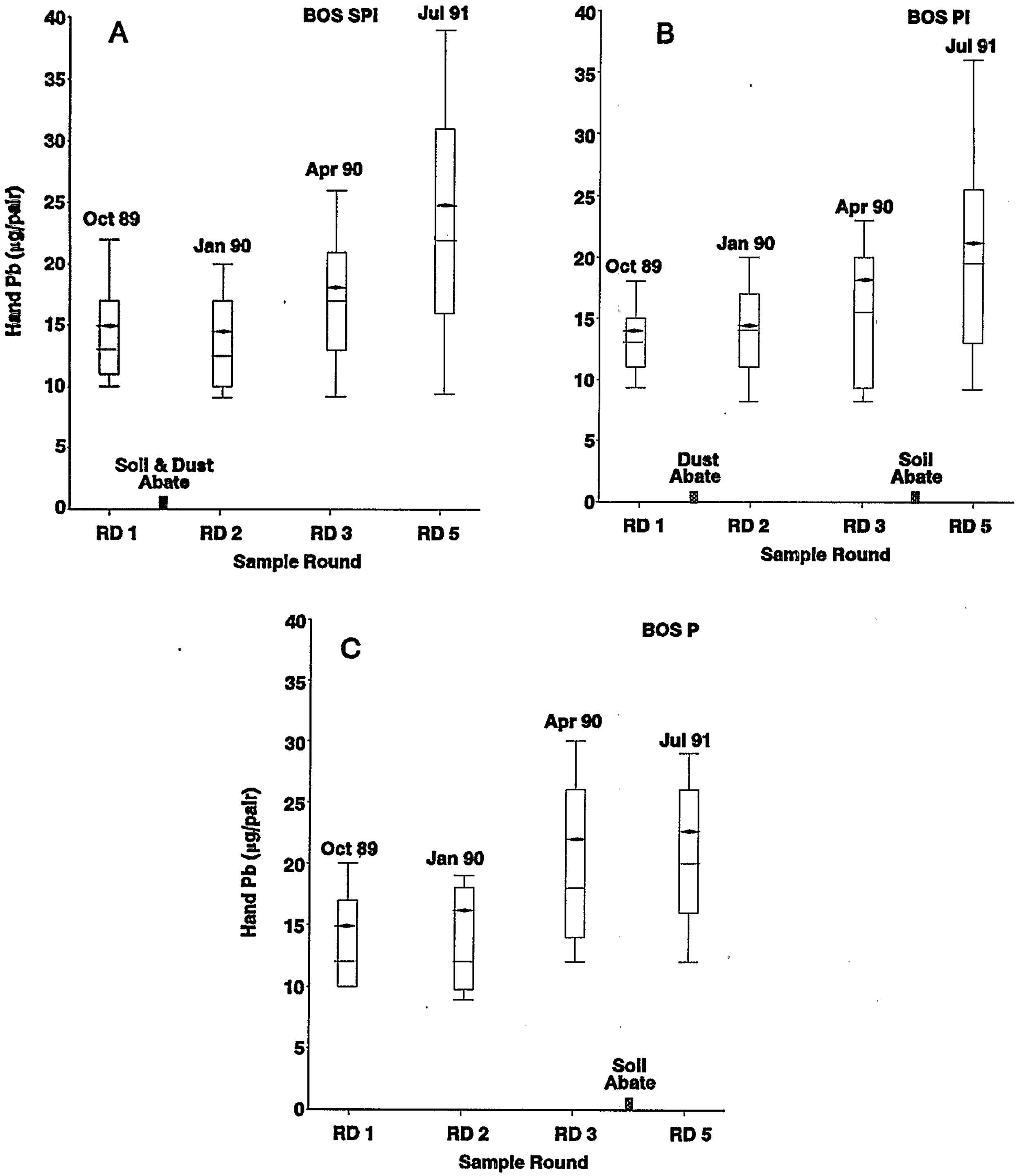

Figure 5-27. Boston hand lead load. The Boston hand lead load increased in all three groups, in contrast to the blood lead concentrations shown in Figure 5-31.

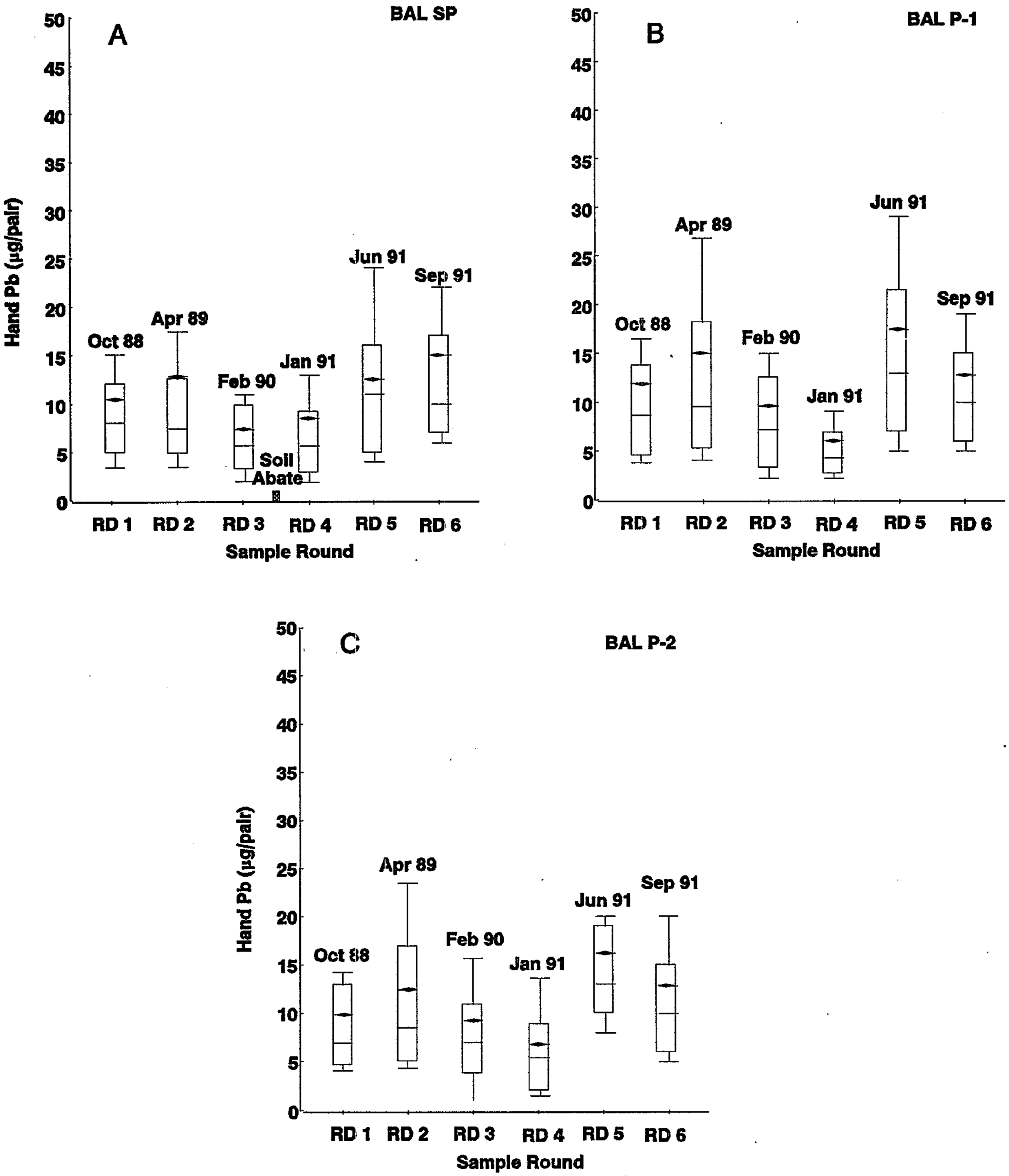

Figure 5-28. Baltimore hand lead load. There were no sequential measurements of Baltimore house dust to compare with the hand lead load.

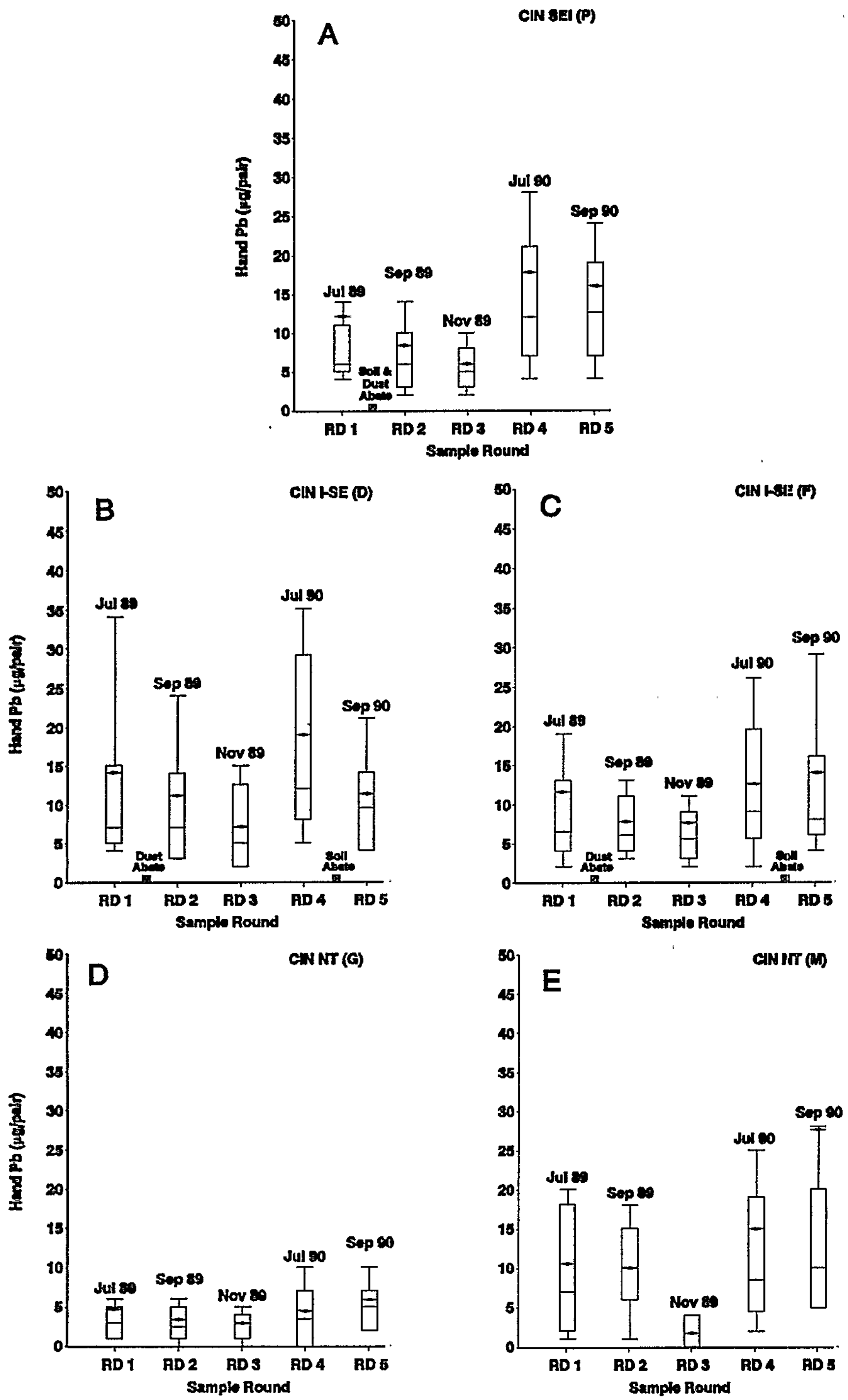

Figure 5-29. Cincinnati hand lead load. The pattern of hand lead load change, both increases and decreases, appears to follow the pattern of floor dust lead load in Figure 5-20.

5.2.4 Changes in Hand Dust Loadings

Because hand-to-mouth activity is one route by which lead may be ingested, the amount of lead on the child's hand is an indicator of exposure. Only lead loading information is available because it was necessary to take the sample with wet wipes and there is no measure of the amount of dust removed. The units of measurement are micrograms per pair of hands rather than micrograms per square meter.

In Boston, there was a general increase in hand lead throughout the study (Figure 5-27). Although there is no explanation for this increase, there appears to be less of an effect for the groups that received soil and dust intervention, and this reduction is greatest for the group that received soil, dust, and paint intervention.

Baltimore hand lead values did not follow a discernable pattern (Figure 5-28) and there appear to be no systematic differences among the groups.

In Cincinnati, the hand dust lead load (Figure 5-29) appears to follow the pattern of change observed in the floor dust lead load (Figure 5-20). This is an important link in the exposure pathway that measures actual external contact with the child's dust environment. Hand lead loadings were expected to respond more quickly to environmental changes than blood lead concentrations. The hand lead data were informative and showed a number of similar patterns across the three studies. The discussion below of the relationship of hand lead to blood lead will shed further light on this critical pathway.

5.2.5 Changes in Blood Lead Concentrations

5.2.5.1 Baltimore Study Blood Lead Data

The blood lead concentrations for the three Baltimore groups are shown in Figure 5-30. The data are for all children participating in the round. They show that the groups were similar prior to soil abatement between Rounds 3 and 4. Following abatement, the groups responded according to treatment, but the difference was not significant 10 months after abatement. The lack of postabatement measurements of soil and house dust limits the ability to interpret these data by more than a simple analysis of variance.

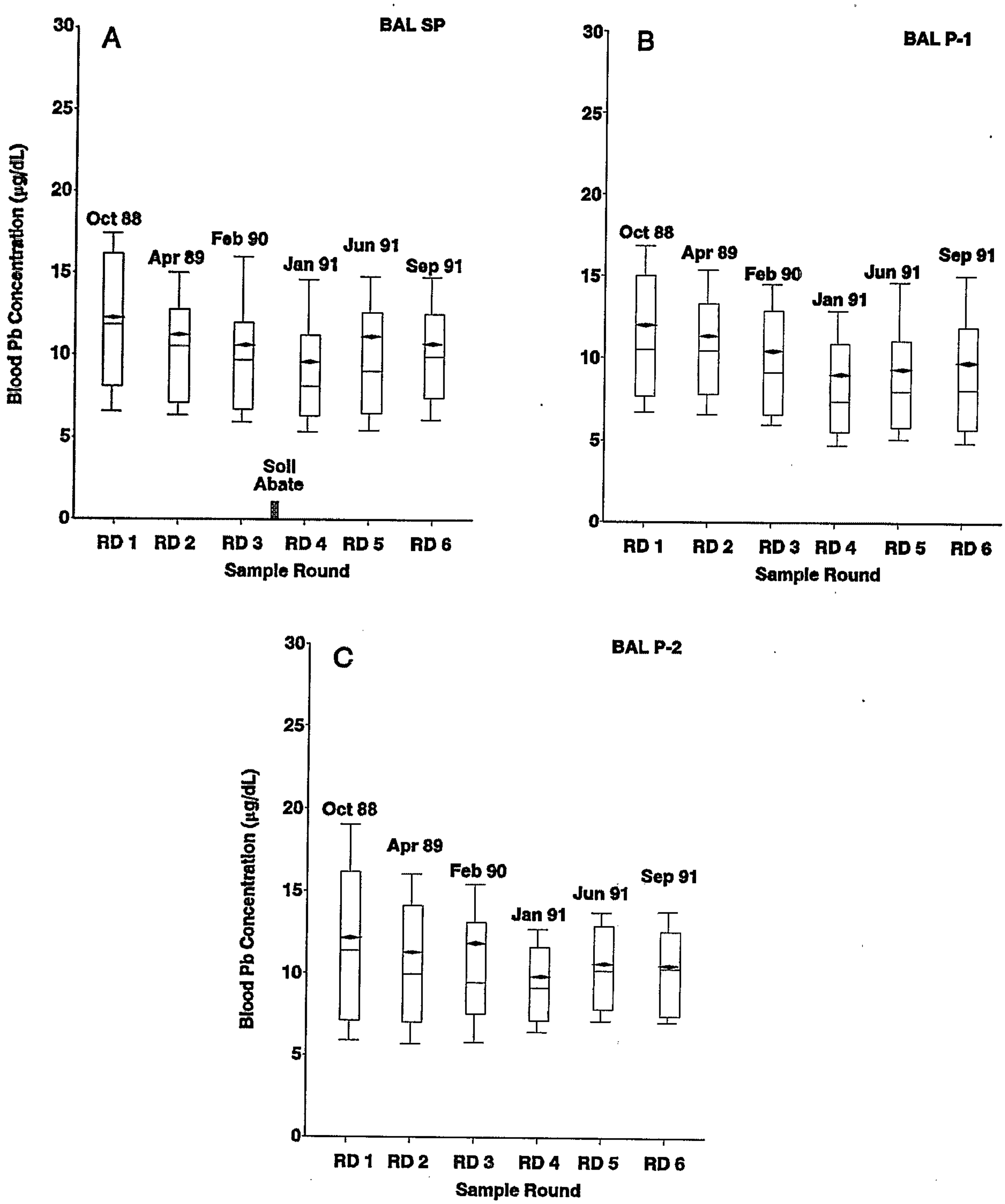

Figure 5-30. Baltimore blood lead concentrations. There appears to be little difference between study groups.

5.2.5.2 Boston Study Blood Lead Data

The blood lead concentrations for the Boston study are shown in Figure 5-31, where they graphically illustrate the conclusions of the Boston report, that intervention probably accounted for a decrease of 0.8 to 1.5 μg/dL in the blood lead. The observation that all three Boston study groups experienced an increase in blood lead concentrations between Round 3 (April 1990) and Round 4 (September 1990) is consistent with similar observations in the hand dust lead load and, to a lesser degree, the window dust lead load. The apparent absence of a comparable increase in floor dust lead load runs counter to the expected pattern of the floor dust lead load being the primary route for dust exposure in children.

5.2.5.3 Cincinnati Study Blood Lead Data

The wealth of information from the more detailed measurements of household dust in the Cincinnati study presents a proportionally greater challenge to the modeling of dust exposure pathways. The blood lead concentrations shown in Figure 5-32 correspond roughly to the changes observed in the hand dust lead loads of Figure 5-29. And there are several points where the blood lead concentrations are consistent with the observed changes in the various forms of house dust. The floor and window dust lead loads are especially indicative of the exposure route, and the mat dust lead load seems to account for the increase in blood lead concentrations after November 1990. The group that received soil abatement in the first year, CIN SEI, continued to show increasing blood lead concentrations through the following year, and the CIN I-SE(B) and CIN I-SE(D), and CIN I-SE(F) groups continued to decrease following soil and exterior dust abatement in the second year.

5.3 PRE- AND POSTABATEMENT DIFFERENCES IN INDIVIDUALS

5.3.1 Individual Changes in Blood Lead and Soil Lead

Section 5.2 provides a visual presentation of longitudinal changes in population means for specific parameters over the course of the study. This section presents information on an individual child basis through the use of a series of double difference plots where the difference between pre- and postabatement blood lead concentrations are plotted against the

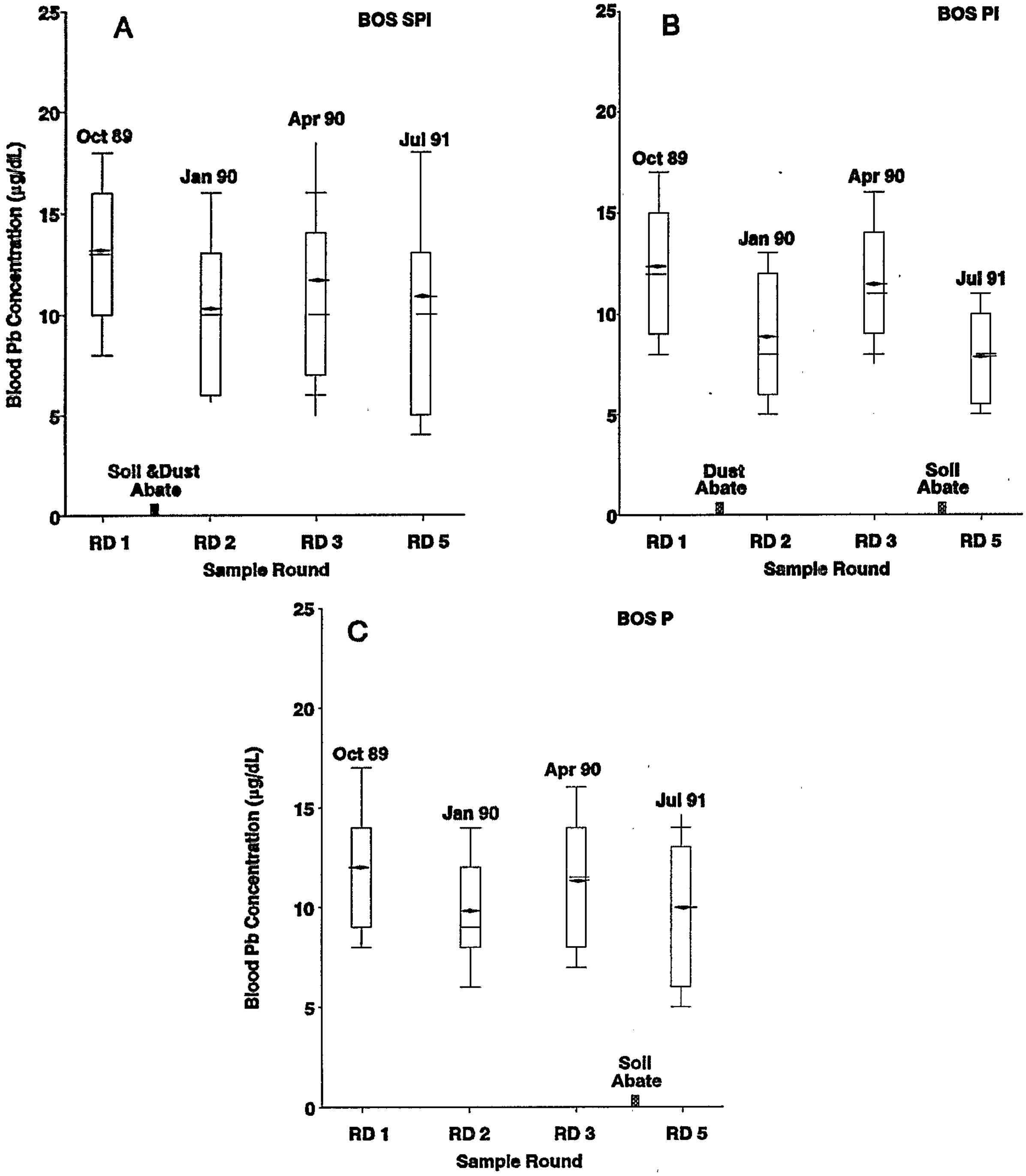

Figure 5-31. Boston blood lead concentrations.

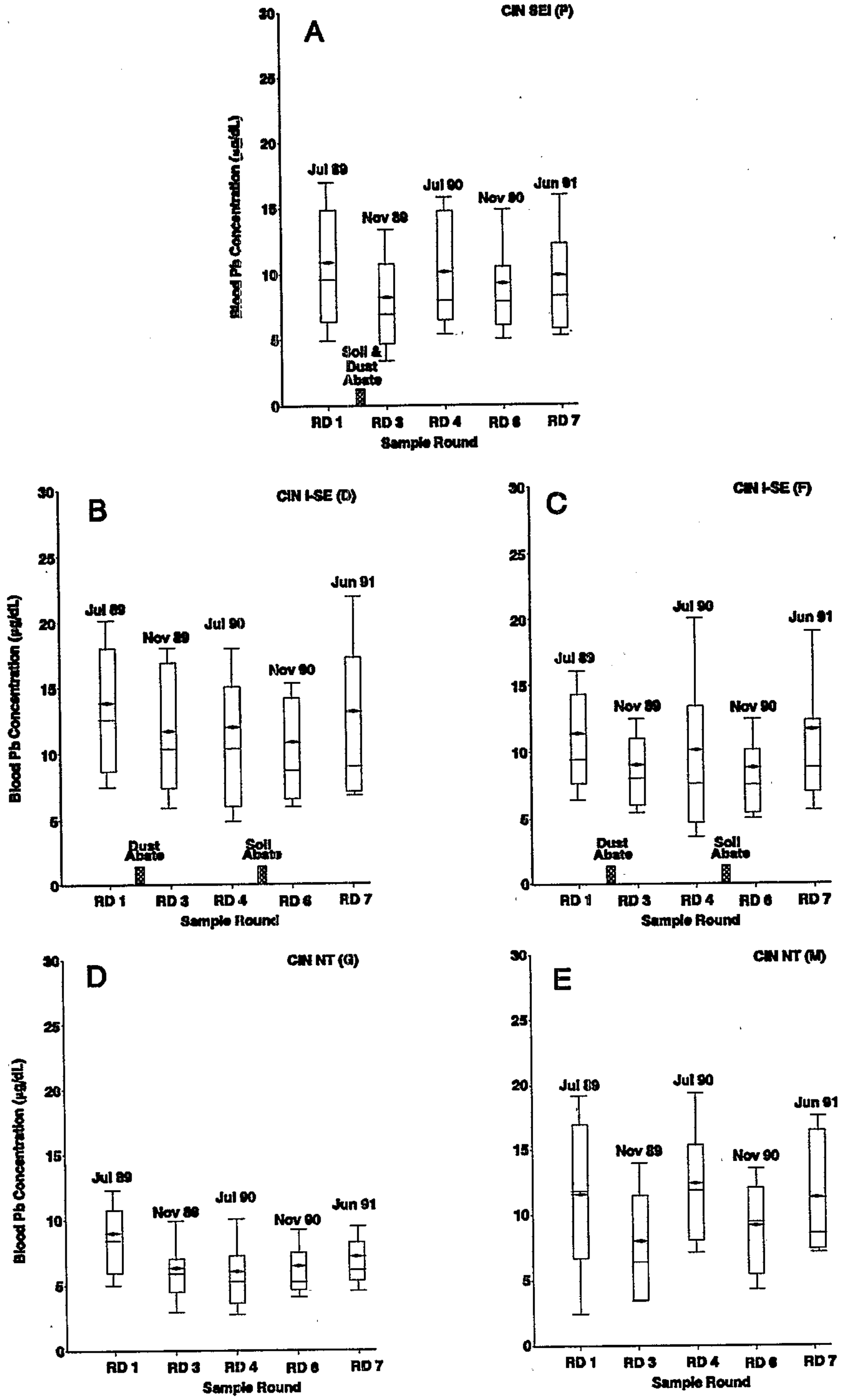

Figure 5-32. Cincinnati blood lead concentrations. Compare to hand lead load patterns in Figure 5-29.

difference in pre- and postabatement soil lead concentrations, dust lead concentrations or dust lead loadings.

Most children in each neighborhood experienced some change in blood lead, either an increase or decrease, during the course of the study. Some of this change is due to changes brought about by intervention. Another part may be due to seasonal effects, age (see Figure 2-6), or changes in exposure not related to intervention.

A child exposed to decreasing soil lead concentrations is expected to experience a decrease in blood lead concentration. In Figure 5-33, this child would be represented in the lower left quadrant (III). Conversely, a child exposed to increasing lead concentrations should experience an increase in blood lead concentrations. This child would be represented in the upper right quadrant (I). If there were no other factors involved, all children should be in the upper right or lower left quadrants, or centered around the origin if there were little or no change. If the relationship between blood lead and soil lead were strictly linear, and if blood lead concentrations increased by the same mechanism as they decrease, all points would lie on a straight line passing through the origin.

In these studies, there does not appear to be a linear response for any of the double difference plots, and there are many cases where data lie in one of the excluded quadrants II and IV, indicating blood lead increased when environmental lead decreased, or vice versa (Figures 5-33 to 5-41).

This type of plot is especially helpful to the reader in understanding the variability of the measurements and the possible significance of patterns or clusters. They are designed to show the interaction of only two variables at a time, not the multiple interactions of several variables. In Section 5.4, statistical techniques such as repeated measures analysis and structural equation modeling are used to extract information from the systematic variability using more appropriate methods for comparison than observed on these double difference plots but in the context of several variables interacting at the same time.

There are a few observations worth noting in the double difference plots. In Boston and Baltimore, the more intense interventions (BAL SP and BOS SPI) placed a greater number of points in quadrant III. Even though soil seemed to have a greater impact than floor dust (Figures 5-34 through 5-36), later analyses in Sections 5.4 and 5.5 suggest otherwise. Entry way dust lead concentrations and loadings in Cincinnati do not seem to

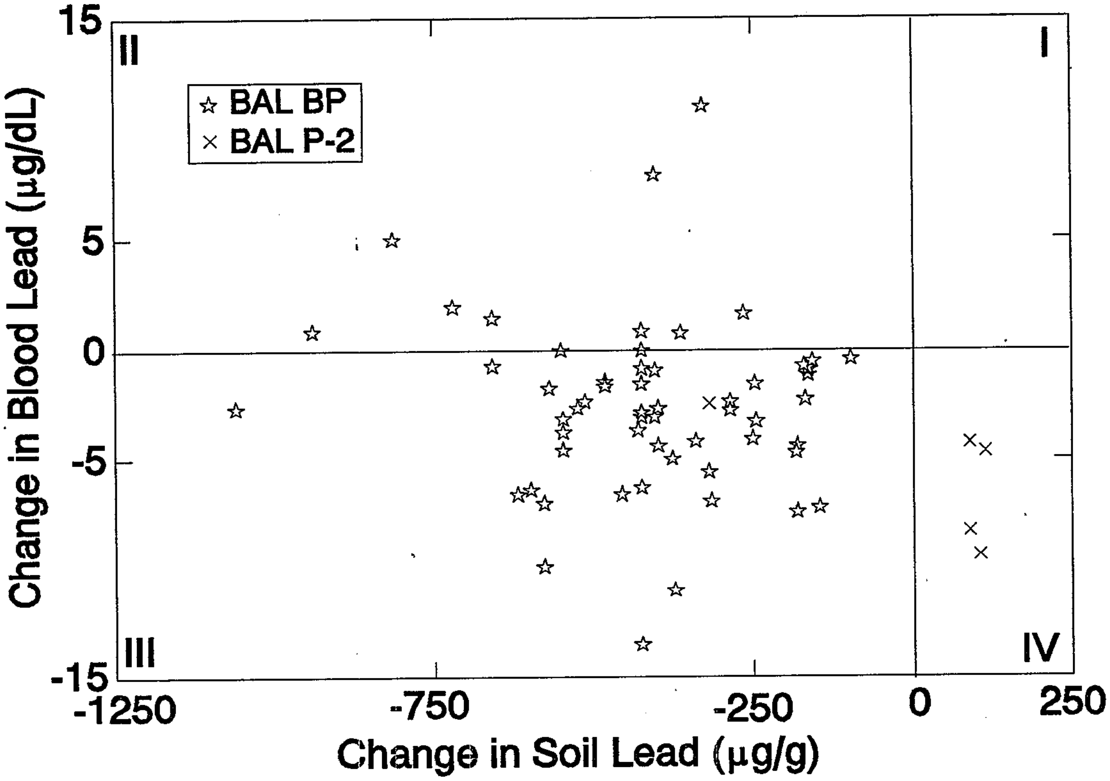

Figure 5-33. Double-difference plot of the change in soil lead versus the change in blood for the Baltimore study. Except for a few measurements in BAL P-2, postabatement soil measurements were taken in BAL SP only.

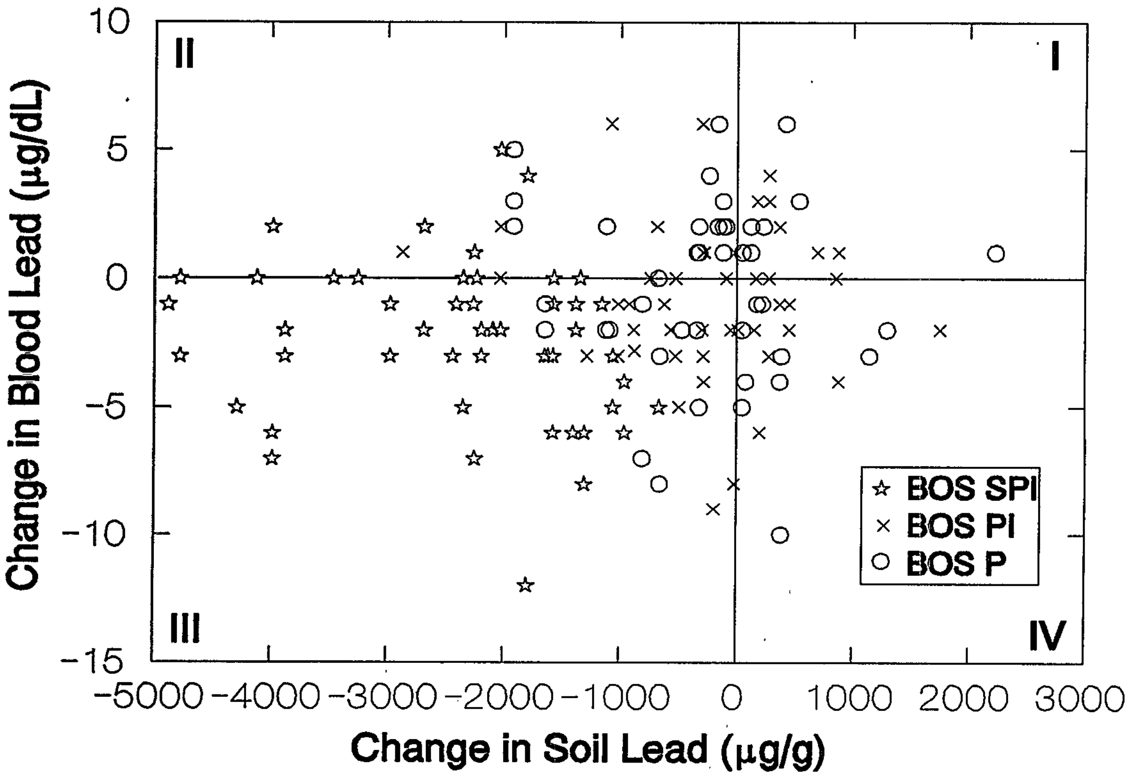

Figure 5-34. Double-difference plot for Boston soil and blood lead data.

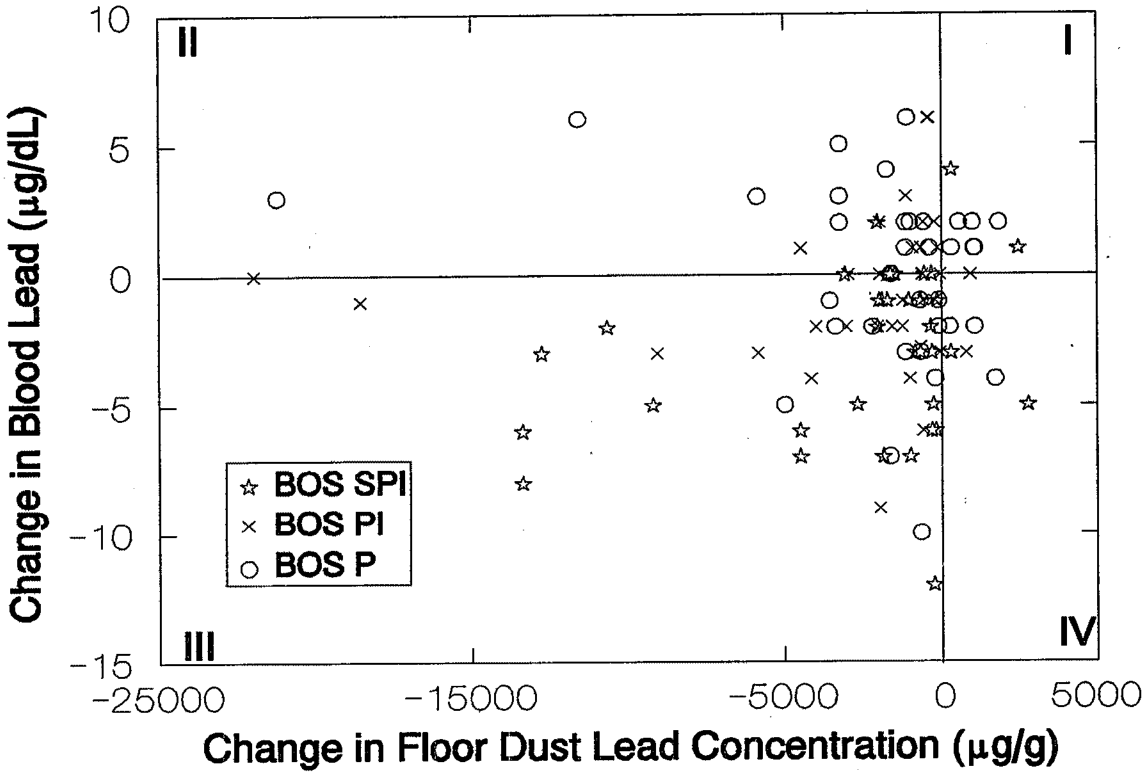

Figure 5-35. Double-difference plot for Boston floor dust lead concentrations and blood lead concentrations.

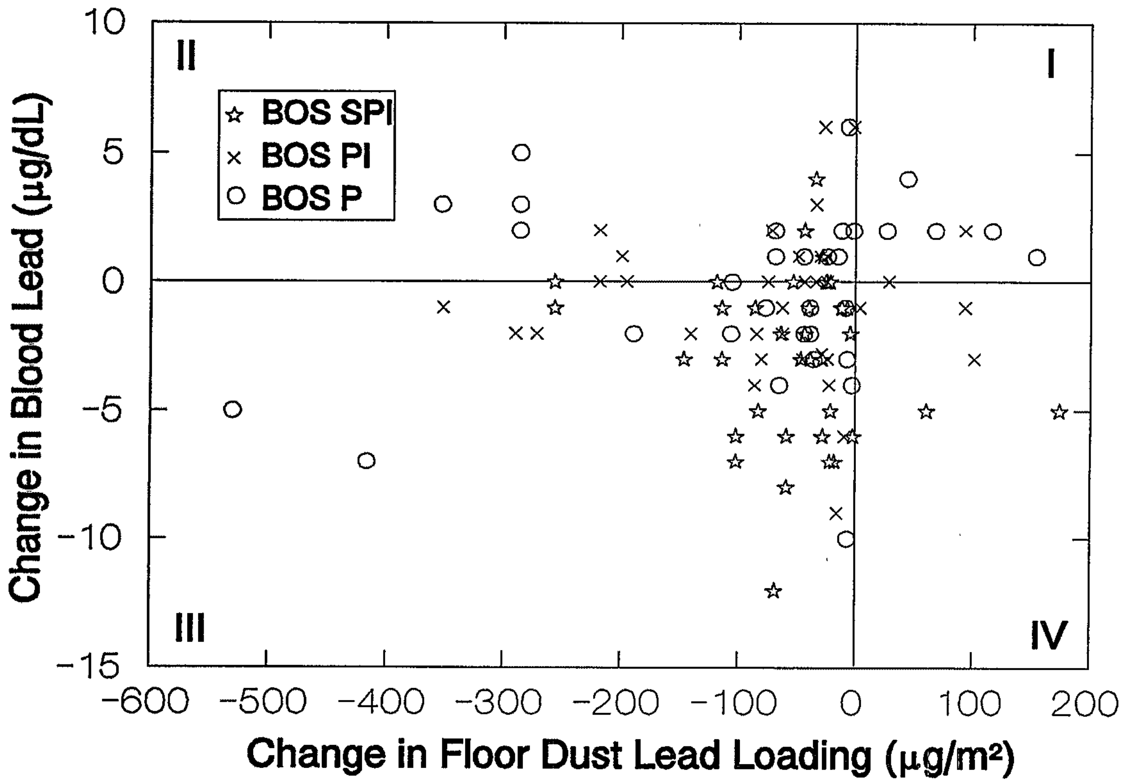

Figure 5-36. Double-difference plot for Boston floor dust lead loading and blood lead concentrations.

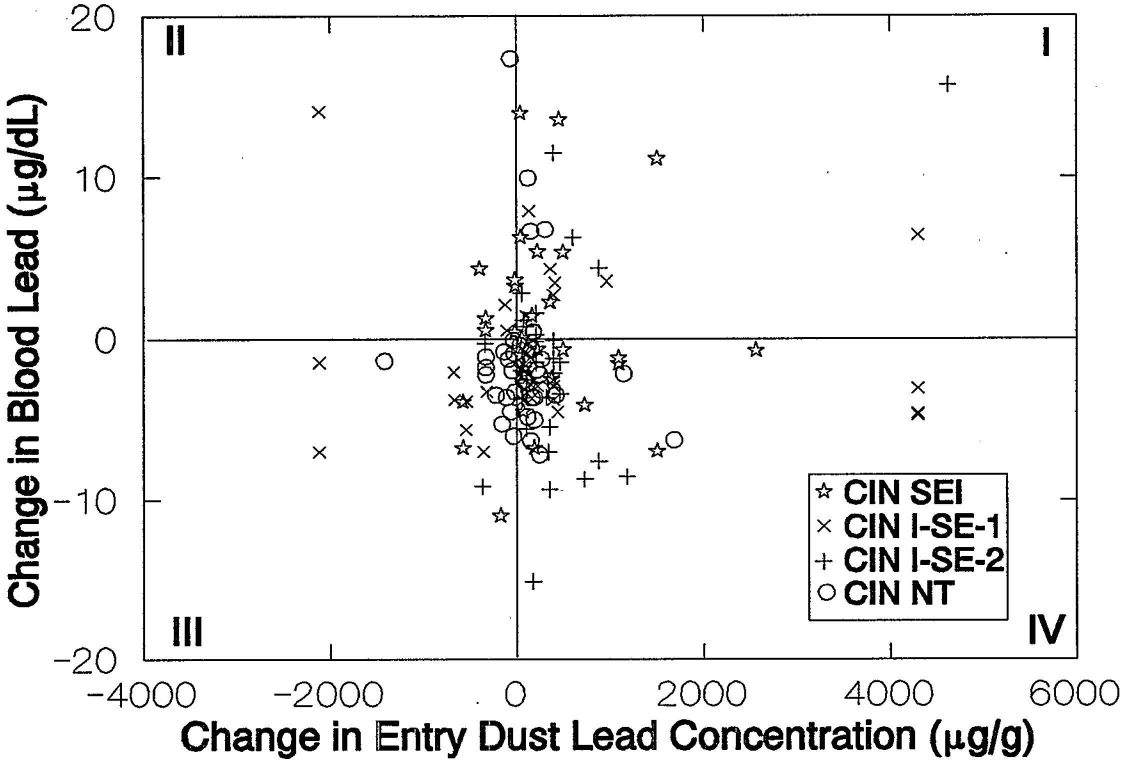

Figure 5-37. Double-difference plot for Cincinnati entry dust lead concentrations and blood lead concentrations.

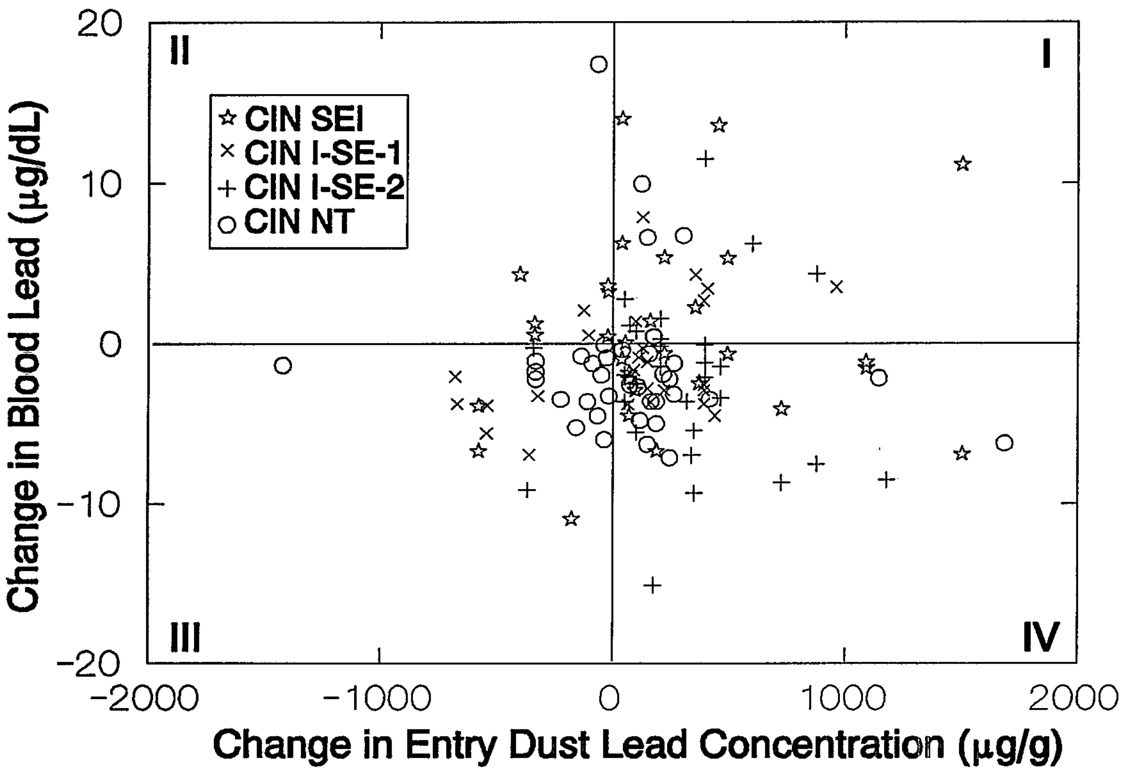

Figure 5-38. Double-difference plot for Cincinnati entry dust lead concentrations and blood lead concentrations. Entry dust lead concentrations are truncated at 2,000 µg/g to enhance resolution near the origin.

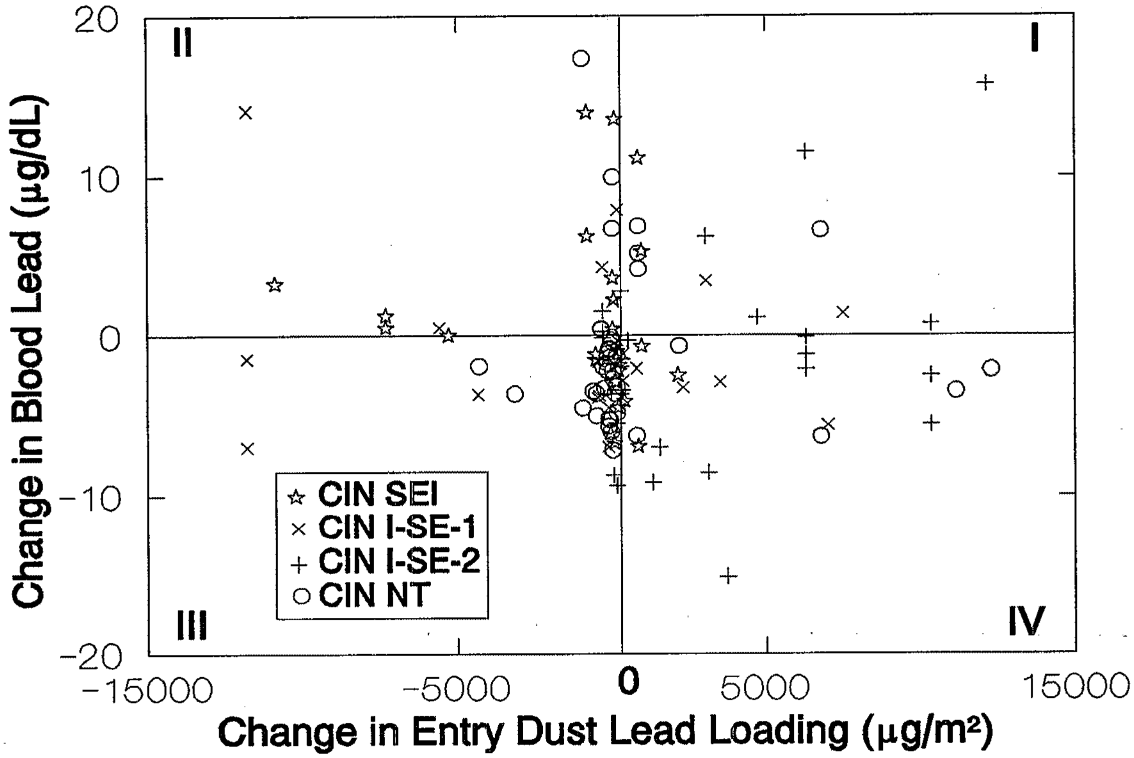

Figure 5-39. Double-difference plot for Cincinnati entry dust lead loading and blood lead concentrations.

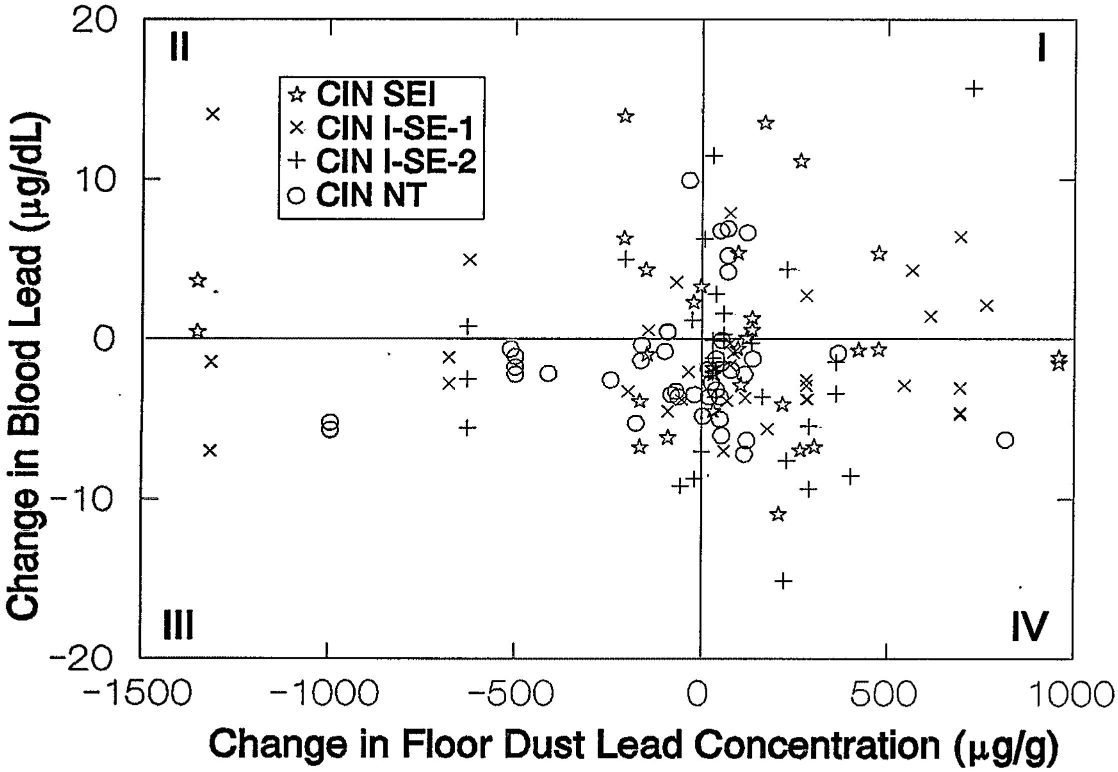

Figure 5-40. Double-difference plot for Cincinnati floor dust lead concentrations and blood lead concentrations.

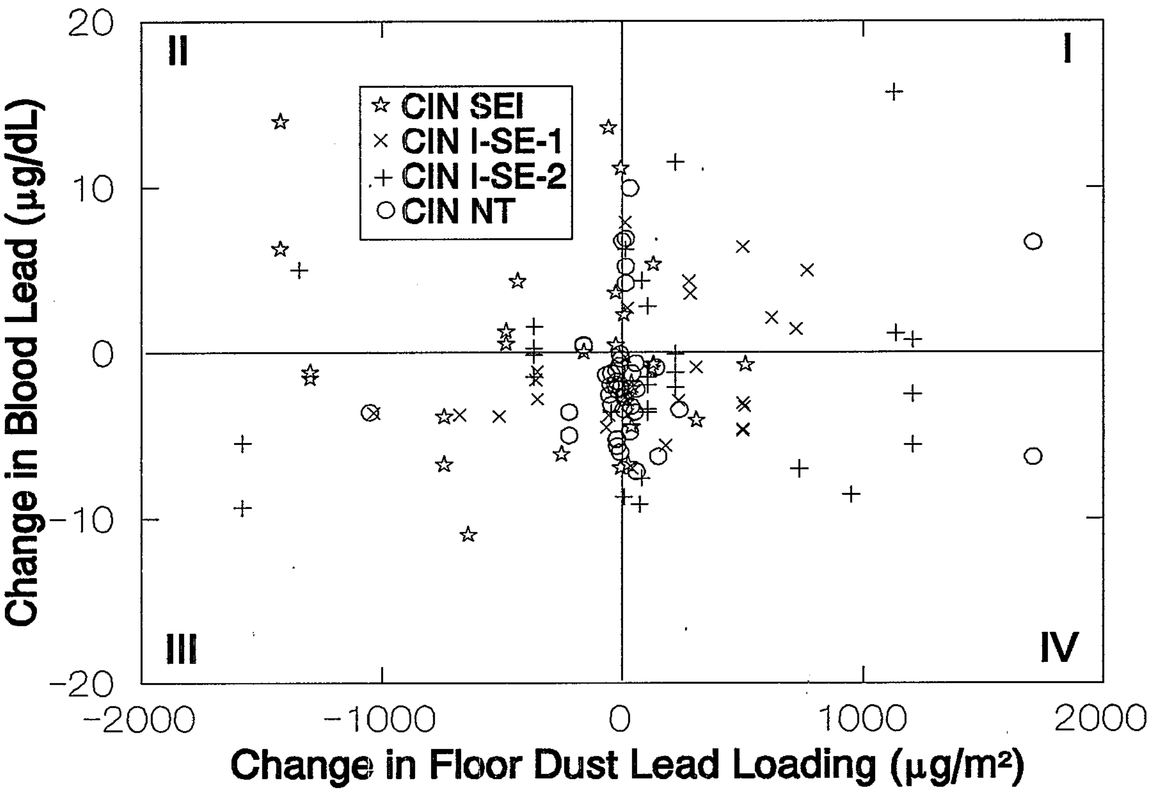

Figure 5-41. Double-difference plot for Cincinnati floor dust lead loading and blood lead concentrations.

have a significant impact on blood lead concentrations, although later analyses show this variable is important in estimating the influence of exterior dust or soil on house dust. Floor dust lead concentrations and lead loadings in Cincinnati (Figures 5-40 and 5-41) show a large number of points in quadrant IV. The increase in exterior dust that eventually impacted interior dusts may not have yet caused an increase in blood lead concentrations.

5.4 COMPARISON BY REPEATED MEASURES ANALYSIS

5.4.1 Baltimore Study

The Baltimore study results for blood lead are shown in Figure 5-42, and for hand lead in Figure 5-43. For each of the three groups, the central points show the geometric mean and the ends of the bars around the points show the uncertainty of the geometric mean as measured by the geometric standard error, where the upper bar is the geometric mean multiplied by one geometric standard error and the lower bar is the geometric mean divided by one geometric standard error. The geometric standard error is a factor equal to exponent (SEL), where SEL is the standard error of the mean logarithm of hand lead or blood lead. The ends of the bars also define a 68% confidence interval for the geometric mean of natural log. The intervals are based on an assumed normal distribution for the natural logarithm of the geometric mean, and so are not quite symmetric around the geometric mean. Each measurement made before abatement must be paired with a measurement made after abatement in order to calculate the effect of the abatement, so that the statistical uncertainty of the intervention differences cannot be calculated from the separate standard errors shown in these figures. Preabatement is Round 3, and postabatement is one year later, Round 6.

The geometric mean blood lead profiles for the BAL P-1 and BAL P-2 control groups in Figure 5-42 are almost parallel and horizontal, similar to the example in Figure 5-6. There is a slight decrease in the BAL SP blood lead levels between Rounds 3 and 6, resembling Figure 5-6. This suggests that there was a slight decrease in blood lead levels in Baltimore soil abatement children relative to either control group. However, hypothesis tests in Table 5-1 showed no significant differences in blood lead rates of change related to soil abatement.

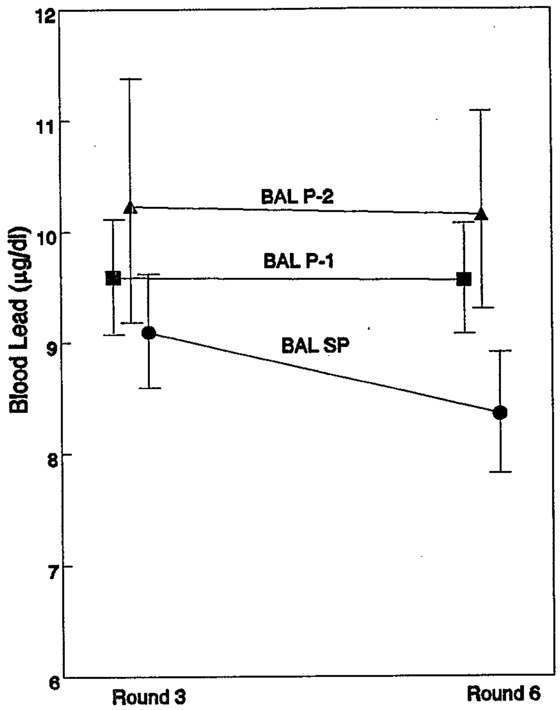

Figure 5-42. Change in preabatement geometric mean blood lead levels in Baltimore study 1 year after abatement.

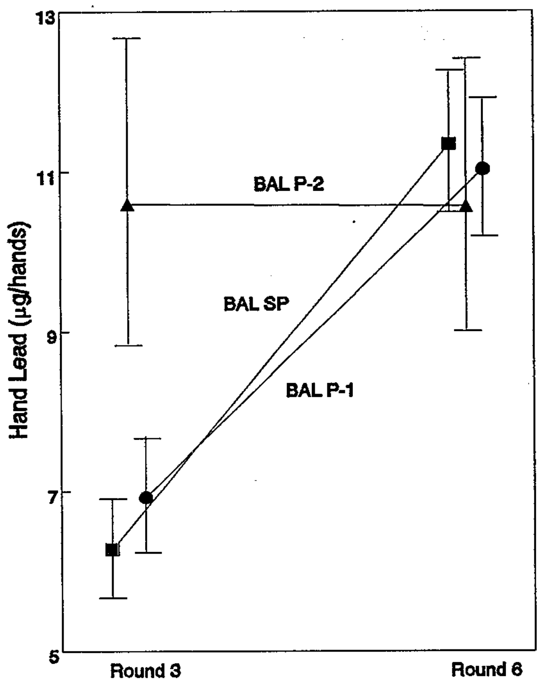

Figure 5-43. Change in preabatement geometric mean hand lead levels in Baltimore study 1 year after abatement.

TABLE 5-1. STATISTICAL SIGNIFICANCE OF BALTIMORE REPEATED MEASURES ANALYSES FOR BLOOD LEAD, ROUNDS 3 AND 6 (PRE- AND POSTABATEMENT), AFTER COVARIATE ADJUSTMENT

		Significance of Effect		
Covariate	N	Time * Group	Time * Covariate	Time * Group * Covariate
None	149	0.4247	------	-------
Log of Soil Lead	149	0.3780	0.0361*	0.3217
Log of Dust Lead Loading	145	0.2012	0.0125*	0.2106
Log of AA Dust Con.	149	0.6212	0.6304	0.7895
Log of XRF Dust Conc.	149	0.3144	0.6560	0.4741
Log of Interior Paint + 1	143	0.1783	0.1564	0.0988^{+}
Log of Exterior Paint + 1	136	0.8418	0.3878	0.7342
Age in years (categorical)	149	0.4043	0.5761	0.8224

[1]In this chapter, the convention for indication signficance by ranges of p-values is:
$^{+}p = 0.05$ to 0.10
$^{*}p = 0.01$ to 0.05
$^{**}p = 0.005$ to 0.01
$^{***}p = 0.001$ to 0.005
$^{****}p < 0.001$

The geometric mean hand lead profiles for the BAL P-1 and BAL SP groups in Figure 5-43 are almost identical and increase during the study, whereas the profile for BAL P-2 is nearly horizontal. The interpretation of Figure 5-43 is that hand lead levels in the soil abatement group rose at a faster rate than in the control groups. However, when adjusted for initial floor dust lead concentration before abatement, the rate of increase of hand lead levels was significantly less than in the Baltimore P-1 control group. When adjusted for initial floor dust lead concentration before abatement, the rate of increase of hand lead levels in the low-soil adjacent control group BAL P-2 was significantly greater than in the Baltimore P-1 control group. Without adjusting for the preabatement dust lead concentration, then as shown in Figure 5-43, the rate of increase of hand lead levels in the low-soil adjacent control group BAL P-2 appears to be significantly less than in the Baltimore P-1 control group.

The statistical significance of the covariate-adjusted repeated measures analyses is shown in Table 5-1. Comparisons of changes in blood lead concentrations showed no effect of treatment group, with or without adjustments, with all P values > 0.178. Similar lack of significant treatment group effect was shown when the covariates were tested one at a time, except for a marginal effect of interior lead paint (P = 0.10). Because interior lead paint was not abated, there may have been some mitigation of any beneficial soil lead abatement effect that might have occurred by the nonremediated interior lead-based paint. There was, however, a significant positive statistical relationship of blood lead reduction to the preabatement soil lead concentration and dust lead loading. Remediating households with higher soil lead had more benefit than remediating those with lower soil lead, but the higher nonremediated dust lead and interior lead paint loadings offset any beneficial effects of soil lead remediation that might have occurred.

Hand lead loadings show many statistically significant relationships to the study group in Table 5-2. There are also significant interactions of study group with covariates, but these effects show little relation to soil abatement. Detailed examination of these relationships (not shown here) finds that the increase in hand lead is different between the two control groups, BAL P-1 and BAL P-2, and that there is little difference between the control group and the soil abatement group, BAL SP, in Area 1. It is of some interest that there is usually a larger difference in the average change in lead between the two neighborhood control groups than between the control group and soil abatement group in the same neighborhood in Baltimore.

TABLE 5-2. STATISTICAL SIGNIFICANCE OF BALTIMORE REPEATED MEASURES ANALYSES FOR THE LOGARITHM OF HAND LEAD, ROUNDS 3 AND 6 (PRE- AND POSTABATEMENT), COVARIATE ADJUSTMENT

		Significance of Effect		
Covariate	N	Time * Group	Time * Covariate	Time * Group * Covariate
None	288	0.0015^{**}	-----	-----
Log of Soil Lead	288	0.0448^{*}	0.1324	0.0186^{*}
Log of Dust Lead Loading	274	0.0366^{*}	0.7750	0.0011^{**}
Log of AAS Dust Conc.	288	0.1869	0.4023	0.0071^{**}
Log of XRF Dust Conc.	288	0.6598	0.7519	0.0419^{*}
Age in years (categorical)	288	0.0032^{**}	0.4465	0.5888

Age plays a significant role in hand lead loading, but not in blood lead differences among study groups. The child's age appears to be a useful variance-reducing covariate that can explain some of the differences among children, but is not useful as a significant modifier of the soil abatement effect in the Baltimore study.

5.4.2 Boston Study

The Boston study results for blood lead are shown in Figure 5-44, for hand lead in Figure 5-45, for floor dust lead concentration in Figure 5-46, and for floor dust lead loadingin Figure 5-47. For each of the three groups, the central points show the geometric mean and the ends of the bars around the points show the uncertainty of the geometric mean, calculated for one geometric standard error, as in Section 5.4.1.

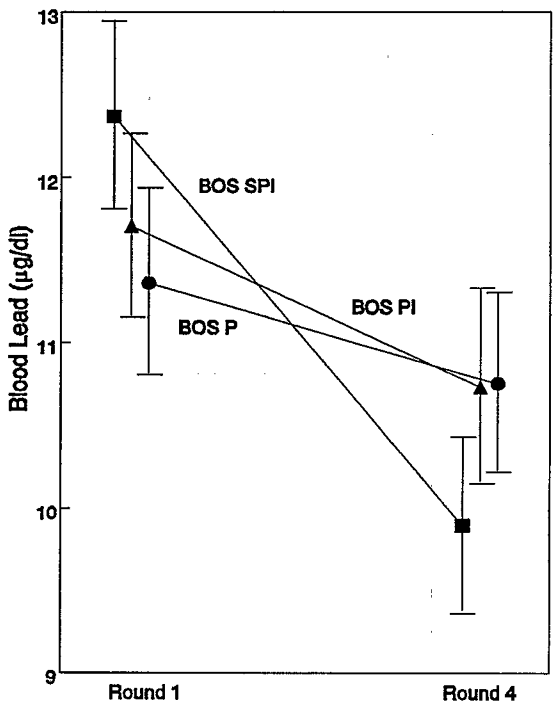

Figure 5-44. Change in preabatement geometric mean blood lead levels in Boston study 1 year after abatement.

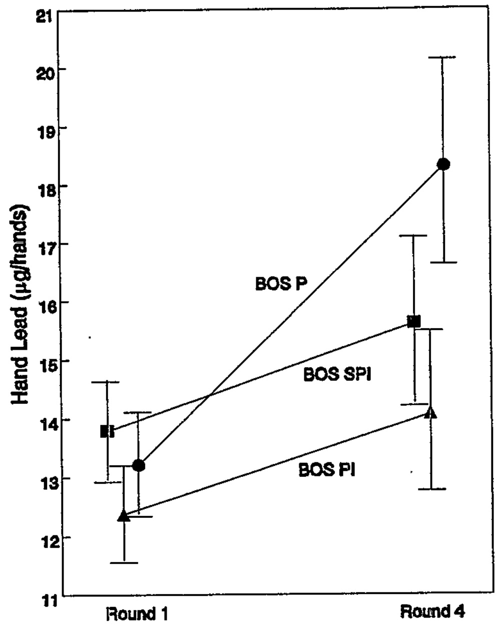

Figure 5-45. Change in preabatement geometric mean hand lead levels in Boston study 1 year after abatement.

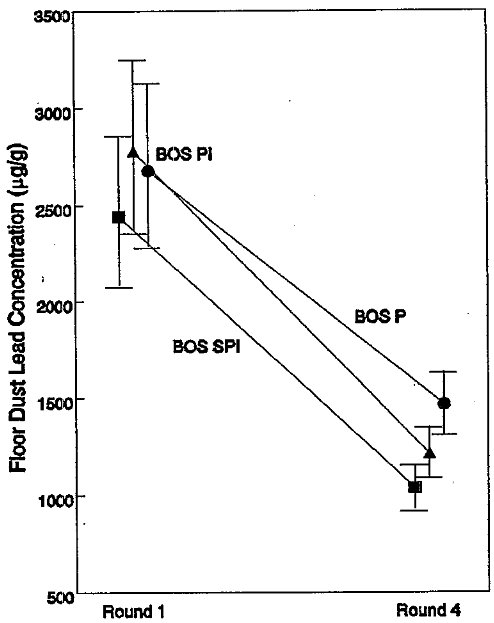

Figure 5-46. Change in preabatement geometric mean floor dust lead concentration in Boston study 1 year after abatement.

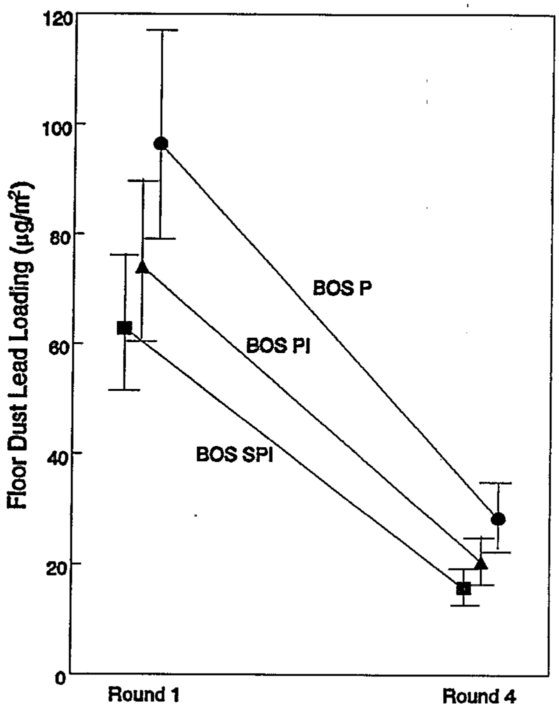

Figure 5-47. Change in preabatement geometric mean floor dust lead loading in Boston study 1 year after abatement.

The geometric mean blood lead profiles for the BOS PI and BOS P control groups after one year, shown in Figure 5-44, are almost identical and intersecting, as in Figure 5-6 for example. There is a much greater decrease in the BOS SPI blood lead levels between Rounds 1 and 4, even more greatly resembling Figure 5-6. This suggests that there was a slightly greater decrease in blood lead levels in the Boston interior dust abatement children in BOS PI than in the negative control group BOS P. Boston children in the soil abatement group BOS SPI showed a much greater decrease in blood lead relative to either control groups BOS PI and BOS P, demonstrating a beneficial soil abatement effect that was statistically significant.

The geometric mean hand lead profiles for the BOS SPI and BOS PI groups in Figure 5-45 are almost parallel and increase during the study, whereas the profile for BOS P increased much more rapidly. The interpretation of Figure 5-45 is that hand lead levels in the control group BOS P rose at a faster rate than in the soil or dust abatement groups.

However, none of these differences were statistically significant even when adjusted for initial lead exposures.

The geometric mean floor dust lead concentration profiles for the BOS SPI and BOS PI groups in Figure 5-46 are almost parallel and decreased rapidly during the study, whereas the concentration profile for BOS P decreased more slowly. The interpretation of Figure 5-46 is that the soil and dust abatements both had a beneficial effect in reducing floor dust lead levels more than in the control group BOS P. However, none of these differences were statistically significant even when adjusted for initial soil and paint lead exposures.

The geometric mean floor dust lead loading profiles for the BOS SPI and BOS PI groups in Figure 5-47 are almost parallel and decreased rapidly during the study, whereas the loading profile for BOS P decreased more rapidly. The interpretation of Figure 5-47 is that the soil and dust abatements had little effect in reducing floor dust lead loadings. However, none of these differences were statistically significant even when adjusted for initial soil and paint lead exposures.

The Boston study showed clear and statistically significant differences in the decrease of blood lead between Rounds 1 and 3, as shown in Table 5-3. When the relationship was adjusted for initial soil lead, dust lead, or paint lead, the differences among treatment groups became nonsignificant. This suggests that the quantitative characterization of abatement by change in soil lead or dust lead is sufficiently strong in the Boston study that remediation group effect is largely subsumed by the changes in environmental lead concentrations. The environmental changes in the Boston study are twofold: large and persistent reductions in soil lead and dust lead in the soil abatement group, and small changes in the other two groups. The corresponding effects are moderately large reductions in blood lead the first year after abatement in the soil abatement group. Blood lead continues to decrease in the second postabatement year in those households where recontamination did not occur, as expected from the biokinetics of lead storage in bone.

Unlike the Baltimore study, hand lead loadings in Boston showed little relation to soil or dust abatement, as seen in Table 5-4. Reasons for this difference are not obvious.

The Boston study also found that child age was an important and highly significant covariate for changes in blood lead. As in the Baltimore study, there was no strong evidence that age modified the effect of soil abatement versus other treatments.

TABLE 5-3. STATISTICAL SIGNIFICANCE OF BOSTON REPEATED MEASURES ANALYSES FOR BLOOD LEAD, ROUNDS 1 AND 3 (PRE- AND POSTABATEMENT), AFTER COVARIATE ADJUSTMENT

		Significance of Effect		
Covariate	N	Time * Group	Time * Covariate	Time * Group * Covariate
None	147	0.0074**	-----	-----
Log of Soil Lead	147	0.4589	0.8844	0.5644
Log of Dust Lead Loading	147	0.4046	0.2138	0.4516
Log of Dust Lead Conc.	133	0.9932	0.3890	0.8453
Log of 1 + Chipped Paint	132	0.0774+	0.4375	0.4937
Log of 1 + Interior XRF	141	0.7993	0.7961	0.8645
Age in years (categorical)	147	0.0004***	0.2800	0.1695
Sex	147	0.0107*	0.6425	0.6497

TABLE 5-4. STATISTICAL SIGNIFICANCE OF BOSTON REPEATED MEASURES ANALYSES FOR NATURAL LOGARITHM OF HAND LEAD, ROUNDS 1 AND 3 (PRE- AND POSTABATEMENT), AFTER COVARIATE ADJUSTMENT

		Significance of Effect		
Covariate	N	Time * Group	Time * Covariate	Time * Group * Covariate
None	150	0.0781+	-----	-----
Log of Soil Lead	150	0.3102	0.5085	0.3873
Log of Dust Lead Loading	150	0.7893	0.6812	0.6643
Log of Dust Lead Conc.	136	0.6985	0.6148	0.7412
Log of 1 + Chipped Paint	134	0.3190	0.3909	0.7912
Log of 1 + Interior XRF				
Age in years (categorical)	150	0.8924	0.4400	0.4007
Sex	150	0.0840+	0.6808	0.9521

5.4.3 Cincinnati Study

The results on significant neighborhood treatment group effects for the Cincinnati study are shown in Tables 5-5 and 5-6. There was a significant difference in blood lead changes among the Cincinnati neighborhoods, which also became nonsignificant when adjusted for differences in dust lead concentrations or loadings in the residence unit interior entry or floor. This suggests that preabatement environmental dust lead characterizes changes in the child's blood lead at least as well as does the remediation group for the neighborhood. Even

TABLE 5-5. STATISTICAL SIGNIFICANCE OF CINCINNATI REPEATED MEASURES ANALYSES FOR BLOOD LEAD, ROUNDS 1 AND 4 (12 MONTHS), AFTER COVARIATE ADJUSTMENT

		Significance of Effect		
Covariate	N	Time * Group	Time * Covariate	Time * Group * Covariate
None	156	0.0477*	-----	------
Log Dust Conc. Floor	146	0.9990	0.9753	0.9912
Log Dust Conc. Entry	139	0.8364	0.3386	0.9050
Log Lead Load Floor	146	0.9883	0.2217	0.8812
Log Lead Load Entry	143	0.3106	0.5823	0.7317
Log XRF Interior Trim	153	0.5036	0.6762	0.1190
Log XRF Interior Wall	154	0.0280*	0.9342	0.4964
Log XRF Exterior Trim	154	0.0161*	0.2827	0.0026*
Log XRF Exterior Wall	132	0.1237	0.7934	0.4410
Age (years)	156	0.0521+	0.0001****	0.0438*

TABLE 5-6. STATISTICAL SIGNIFICANCE OF CINCINNATI REPEATED MEASURES ANALYSIS FOR HAND LEAD, ROUNDS 1 AND 4 (12 MONTHS), AFTER COVARIATE ADJUSTMENT

		Signficance Effect		
Covariate	N	Time * Group	Time * Covariate	Time * Group * Covariate
None	---	----	---	---
Log Dust Conc. Floor	111	0.8142	0.6746	0.7780
Log Dust Entry Floor	106	0.4226	0.7115	0.3937
Log Lead Load Floor	111	0.9513	0.9860	0.9530
Log Lead Load Entry	110	0.9172	0.3734	0.9077
Age (years)	120	0.2119	0.0406*	0.9179

though the Cincinnati study was largely restricted to gut-rehab housing, interior lead-based paint on walls, and exterior lead-based paint on trim were significantly related to blood lead changes in different neighborhoods. Finally, there were significant age-related effects on blood lead changes during the study that were also related to the neighborhood or equivalent treatment group.

Hand lead loadings showed no significant relationship to remediation group, neighborhood, environmental covariates, but did show an age effect, as shown in Table 5-6.

The Cincinnati study results for blood lead are shown in Figure 5-48, for hand lead in Figure 5-49, for floor dust lead concentration in Figure 5-50, and for floor dust lead loading in Figure 5-51. For each of the four groups, the central points show the geometric mean and the ends of the bars around the points show the uncertainty of the geometric mean, calculated for one geometric standard error as described in Section 5.4.1.

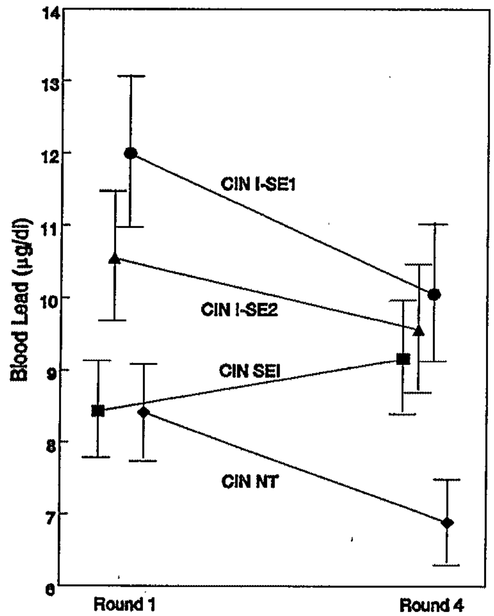

Figure 5-48. Change in preabatement geometric mean blood lead levels in Cincinnati study 1 year after abatement.

The geometric mean blood lead profiles for the CIN I-SE-1, CIN I-SE-2, and CIN NT control groups in Figure 5-48 are almost parallel and nonintersecting, as in Figure 5-6 for example. There is an increase in the CIN SEI blood lead levels between Rounds 1 and 5, somewhat resembling Figure 5-6. This suggests that there was a moderate increase in blood lead levels in the Cincinnati soil abatement children in CIN SEI than in the positive or negative control groups. The unexpected direction of the soil abatement effect was

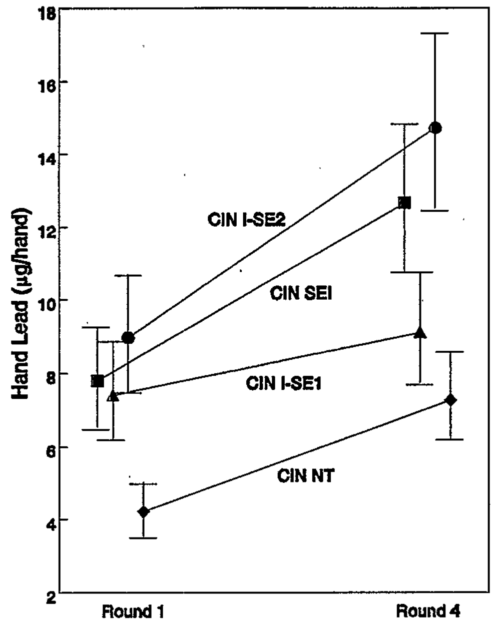

Figure 5-49. **Change in preabatement geometric mean hand lead levels in Cincinnati study 1 year after abatement.**

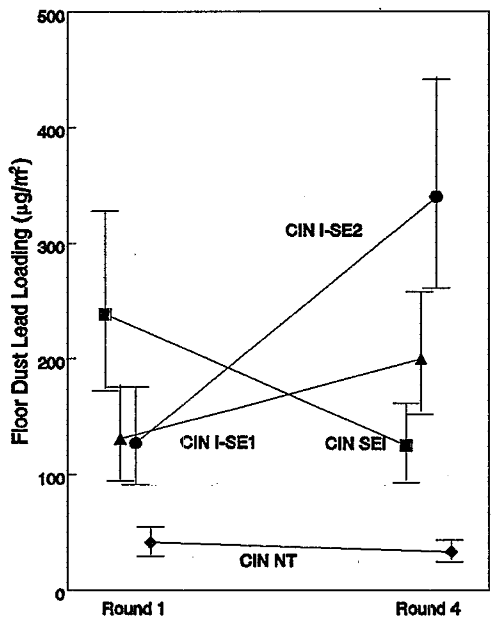

Figure 5-50. **Change in preabatement geometric mean floor dust lead concentrations in Cincinnati study 1 year after abatement.**

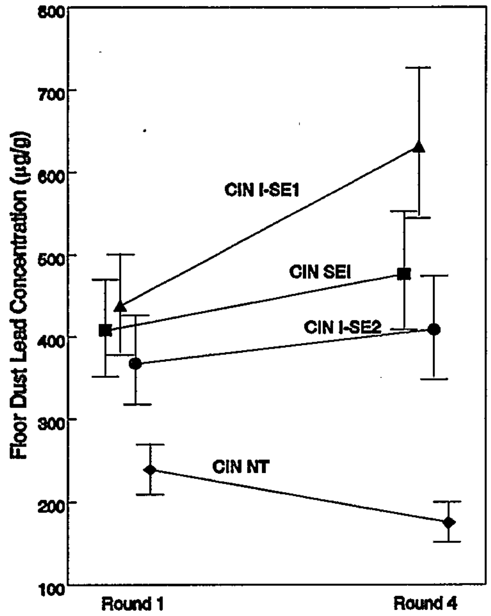

Figure 5-51. Change in preabatement geometric mean floor dust lead loading in Cincinnati study 1 year after abatement.

statistically significant relative to the no-treatment group CIN NT, but not relative to the interior dust abatement groups.

The geometric mean hand lead profiles for the CIN SEI and CIN I-SE-2 groups in Figure 5-49 are almost parallel and increase during the study, whereas the profiles for CIN I-SE-1 and CIN NT have a flatter slope. The interpretation of Figure 5-49 is that hand lead levels in the control groups CIN NT and CIN I-SE-1 rose at a slower rate than in the soil or dust abatement groups CIN SEI and CIN I-SE-2. The only differences that were statistically significant were between CIN I-SE-1 and CIN I-SE-2, and only when adjusted for initial floor dust and entrance dust lead concentrations.

The geometric mean floor dust lead concentration profiles for the CIN SEI, CIN I-SE-1 and CIN I-SE-2 groups in Figure 5-50 are almost parallel and increased during the study, whereas the concentration profile for CIN NT decreased. The interpretation of Figure 5-50 is that the soil and dust abatements apparently had no effect in reducing floor dust lead concentrations more than in the control group CIN NT. However, none of these differences were statistically significant except for CIN SEI versus CIN NT.

The geometric mean floor dust lead loading profiles for the CIN I-SE-1 and CIN I-SE-2 groups in Figure 5-51 increased rapidly with different slopes during the study, whereas the lead loading profile for CIN SEI and CIN NT decreased. The interpretation of Figure 5-51 is that the soil and dust abatements had little effect in reducing floor dust lead loadings in CIN I-SE-1 and CIN I-SE-2. The differences between CIN I-SE-1 and CIN I-SE-2 were statistically significant after adjusting for entrance dust lead loading. The differences between CIN SEI and CIN NT were statistically significant even when adjusted for initial entrance dust lead loadings, with the rate of decrease proportionally larger in CIN NT. The pattern of changes in dust lead loadings is not easy to interpret without invoking additional sources of lead in these neighborhoods where there were no exterior soil or dust interventions.

5.4.4 Repeated Measures Analyses Adjusted for Environmental Analysis and Demographics

5.4.4.1 Results from Boston Study

The results of repeated measures analyses for a variety of models are shown in Table 5-7. Eleven models to be tested have been specified in advance, so that any model for which there is a soil abatement effect with P value less than about 0.05 / 11 = 0.0045 can be regarded as showing a significant effect 8 to 10 months after soil and interior dust abatement, with a group-wise significance level less than 0.05. The eleven models can be described as follows:

- Soil abatement group versus Other two groups combined;
- Soil abatement group versus Other two groups combined, adjusted for change in floor dust lead concentration from pre- to postabatement;
- Comparison of all three groups, not adjusted for covariates;
- Comparison of all three groups, adjusted for covariates one at a time:
  - Change in soil lead concentration from pre- to postabatement
  - Change in floor dust lead concentration
  - Change in floor dust load
  - Change in floor dust lead loading
  - Age at beginning of study
  - Ethnicity/race category
  - SES
  - Sex.

TABLE 5-7. REPEATED MEASURES ANALYSES OF BLOOD LEAD IN BOSTON STUDY FOR FIRST YEAR AFTER ABATEMENT, ADJUSTED FOR DIFFERENCES IN ENVIRONMENTAL INDICES AND DEMOGRAPHICS

		Statistical Significance					
Comparison Groups	Covariates	Comparison * Time		Covariate * Time		Comp * Cov * Time	
		df[1]	p	df	p	df	p
Soil Abatement vs. others	None	1,148 Soil↓	0.0394	----	----	----	----
Soil Abatement vs. others	Dust Pb Conc, Floor	1,101 Soil↓↓	0.0077	1,101	0.2537	1,101	0.24330
Soil, Dust, Control	None	2,147 Soil↓	0.1035	----	-----	-----	-----
Soil, Dust, Control	Soil Pb	2,143 Soil↓	0.2209	1,143	0.5121	2,143	0.8778
Soil, Dust, Control	Dust Pb Conc.	2,101 Soil↓	0.0084	1,101	0.3389	2,101	0.5134
Soil, Dust, Control	Dust Load	2,144 Soil↓	0.0641	1,144	0.8027	2,144	0.6634
Soil, Dust, Control	Dust Pb Load	2,144 Soil↓	0.1070	1,144	0.9894	2,144	0.9651
Soil, Dust, Control	Age	2,144 Soil↓	0.7599	1,144	0.4159	2,144	0.9996
Soil, Dust, Control	Ethnicity Category	2,82 Soil↓ Dust↓	0.0020	3,82 Black↑↑	0.0074	6,82	0.0006
Soil, Dust, Control	SES	2,140 Soil↓	0.0720	1,140	0.6222	2,140	0.2355
Soil, Dust, Control	Sex	2,144	0.4248	1,144	0.9487	2,144	0.7257

[1]df = degrees of freedom, expressed here as two numbers: a, b. The value *a* is the number of degrees of freedom of the effect being tested; the value *b* is the number of degrees of freedom of the residual error term used as the basis for the hypothesis tests.

Soil abatement showed a test-wise significant reduction in blood lead for four of the models:

- Soil abatement group versus other two groups combined, P = 0.0394
- Soil abatement group versus other two groups combined, adjusted for change in floor dust lead concentration, P = 0.0077
- Comparison of all three groups, adjusted for covariates one at a time:
  - Change in floor dust lead concentration, P = 0.0084;
  - Ethnicity/race category, P = 0.0020.

Soil abatement also showed some marginally significant effects wiith other covariate adjustments:

- Comparison of all three groups, not adjusted for covariates, P = 0.1035
- Comparison of all three groups, adjusted for covariates one at a time:
  - Change in floor dust loading from pre- to postabatment, P = 0.0641
  - Change in floor dust lead loading, P = 0.1070
  - SES, P = 0.0720.

There was only one significant interaction term between treatment group and covariate in the nine models that had covariate adjustments, but the interaction between abatement group and ethnicity was the most significant effect among all of the treatment group and covariate effects that were tested. The interaction between ethnicity/race category and treatment effect had P = 0.0006. Although the soil abatement group had a significantly greater reduction in blood lead than the other groups in the tests described above, the dust abatement group also had a smaller but statistically significant reduction in blood lead compared with the control group when race/ethnicity was taken into account. It is clear that sociodemographic factors may affect the response of child blood lead to soil remediation. In the Boston study, it is possible that race or ethnicity was a surrogate for type or quality of housing or some other characteristic of the household that affects the response of the children in a household to soil or dust abatements.

The soil abatement group effect ranged from about 1.3 to 1.9 μg/dL, whereas the dust abatement group effect ranged from about 0.3 to 0.6 μg/dL. Covariate effects were not statistically significant modifiers of treatment group effect, except for ethnicity/race. However, including the covariates and interactions in the models greatly reduced the uncertainty about the treatment effect size. It appears that the treatment effect for soil abatement may be partially subsumed by changes in environmental variables, particularly by changes in the floor dust lead concentration. Floor dust loading may also play a role, but it is not clear from these analyses whether the role of dust loading is as a modifier of floor dust lead concentration or as a sociodemographic surrogate variable.

5.4.4.2 Results of Baltimore Study

The results of the repeated measures analyses of a variety of models for the Baltimore study are shown in Table 5-8. In contrast to the Boston study, the treatment group effect was never statistically significant, but the covariate effects of age and of changes in dust lead loading were statistically significant. There was a broad range of ages in the Baltimore

TABLE 5-8. REPEATED MEASURES ANALYSES OF BLOOD LEAD IN BALTIMORE STUDY FOR FIRST YEAR AFTER ABATEMENT, ADJUSTED FOR DIFFERENCES IN ENVIRONMENTAL INDICES AND DEMOGRAPHICS

		Statistical Significance					
		Comparison * Time		Covariate * Time		Comp * Cov * Time	
Comparison Groups	Covariates	df	p	df	p	df	p
Soil Abatement Ctrls 1 and 2	None	2,176	0.3357	----	----	----	----
Soil Abatement, Ctrl 1, 2	Dust Pb Conc AAS	2,105	0.6546	1,105	0.4995	2,105	0.4680
Soil, Abatement, Ctrls 1, 2	Dust Pb Conc XRF	2,102	0.2008	1,102	0.9409	2,102	0.1670
Soil, Abatement, Ctrls 1, 2	Dust Load	2,111	0.6306	1,111	0.9928	2,111	0.4703
Soil, Abatement, Ctrls 1, 2	Dust Pb Load	2,105	0.9530	1,105	0.0727	2,105	0.0910
Soil, Abatement, Ctrls 1, 2 Soil Pb > 500	None	2,61	0.3633	----	----	----	----
Soil, Abatement, Ctrls 1, 2, Soil Pb ≤ 500	None	2,112	0.6287	----	----	----	----
Soil, Abatement Ctrls 1, 2	Age, year category	2,169	0.6450	7,169	0.0021	----	----
Soil, Abatement Ctrls 1, 2	Age, year Dust Pb Load	2,98	0.9610	7,98 1,98	0.0139 0.00206	2,98	0.0714

study, so age was treated as a categorical variable with seven categories: age 0 years (0 to 11 months), age 1 year (12 to 23 months), and so on. Blood lead increased greatly by Round 6 for children less than twelve months of age at Round 3, increased slightly for children who were 12 to 35 months of age at Round 3, and decreased modestly for children whose age at Round 3 was greater than 35 months. Children whose households had greater reductions in dust lead loading had significantly smaller increases in blood lead than children whose households showed no such reduction. However, on average, blood lead increased in all three groups, with insignificantly greater increases in the soil abatement group than in Control Groups 1 or 2.

5.4.4.3 Results of the Cincinnati Study

The results of the Cincinnati study are shown in Tables 5-9 and 5-10. Comparison of soil abatement or dust abatement groups with combined control groups was much less informative than comparison with separate control neighborhoods. Based on discussions with the Cincinnati investigators, it appears that, in spite of the small number of subjects, the Mohawk neighborhood is a more appropriate control group for the soil abatement neighborhood of Pendleton than was the much more remote neighborhood of Glencoe. Mohawk and Pendleton had more similar housing than Glencoe, and were located in the Ohio River Valley rather than on the surrounding hills.

Table 5-9 shows that there are substantial differences in changes in blood lead among the six Cincinnati neighborhoods during the first postabatment year. Differences in treatment group are test-wise statistically significant when adjusted for changes in dust load at the interior entry ($P = 0.018$) or for changes in lead loading at the entry ($P = 0.037$). When adjusted for age as well, the differences among neighborhoods were more pronounced when adjusted for changes in floor dust lead concentration ($P = 0.029$), floor dust lead loading ($P = 0.034$), entry dust lead loading ($P = 0.002$), and entry dust load ($P < 0.001$), and nearly significant when adjusted for changes in floor dust load ($P = 0.055$). This is even more impressive because of the small sample size for Mohawk ($N = 6$ including floor dust measurements, $N = 8$ for entry dust) and for Pendleton ($N = 32$ to 35).

In general, the three neighborhoods that received only dust abatement during the first year (Back Street, Dandridge, and Findlay) were not significantly different and showed the largest decreases in blood lead. Glencoe children also showed a large decrease in blood lead, which differed significantly from children in Mohawk who showed a large increase in blood lead. The children in the Pendleton neighborhood where soil abatement was carried out showed a very small increase in blood lead, significantly larger than the distant neighborhood of Glencoe, but smaller than the children in the nearby Mohawk neighborhood.

Table 5-10 shows results of testing a variety of models in which the soil abatement neighborhood of Pendleton is compared with the proximate control neighborhood of Mohawk. The differences in goodness of fit among the models in Table 5-10 is small, with residual standard deviations ranging from 2.95 to 3.16 μg/dL. The overall treatment group effect is not statistically significant in any of these models, but the interaction of treatment

TABLE 5-9. REPEATED MEASURES ANALYSES OF BLOOD LEAD IN CINCINNATI STUDY FOR FIRST YEAR AFTER ABATEMENT, ADJUSTED FOR DIFFERENCES IN ENVIRONMENTAL INDICES

		Statistical Significance					
		Comparison * Time		Covariate * Time		Comp * Cov * Time	
Comparison Groups	Covariates	df	p	df	p	df	p
Pendleton vs. Other	None	1,154 Pend ↑	0.022 Other ↓	----	----	----	----
Pendleton vs. Controls	None	1,85 Pend ↑	0.077 Controls ↓	----	----	----	----
Pendleton vs. Glencoe, Mohawk	None	2,84 Pend ↑ Glen ↓	0.018 Moha ↑↑	----	----	----	----
Controls: Glencoe vs. Mohawk	None	1,42 Glen ↓	0.016 Moha ↑↑	----	----	----	----
Dust Abate: Back, Find, Dand	None	2,66 All ↓	0.549	----	----	----	----
Nbhds	None	5,150	0.048	----	----	----	----
Nbhds	Age	5,144	0.001	1,144	0.000	5,144	0.015
Nbhds	Age	5,114	0.000	1,114	0.000	5,114	0.009
	Dust Pb Conc. Entry			1,114	0.148	5,114	0.066
Nbhds	Dust Pb Conc. Entry	5,120	0.011	1,120	0.835	5,120	0.060
Nbhds	Dust Pb Conc Floor	5,125	0.247	1,125	0.571	5,125	0.958
Nbhds	Dust Load Entry	5,120	0.018	1,120	0.394	5,120	0.814
Nbhds	Dust Load Floor	5,125	0.376	1,125	0.920	5,125	0.511
Nbhds	Dust Pb Load, Entry	5,127	0.037	1,127	0.302	5,127	0.719
Nbhds	Dust Pb Load, Floor	5,125	0.407	1,125	0.916	5,125	0.966
Nbhds	Age	5,119	0.029	1,119	0.000	5,119	0.102
	Dust Pb Conc, Floor			1,119	0.872	5,119	0.819

TABLE 5-9 (cont'd). REPEATED MEASURES ANALYSES OF BLOOD LEAD IN CINCINNATI STUDY FOR FIRST YEAR AFTER ABATEMENT, ADJUSTED FOR DIFFERENCES IN ENVIRONMENTAL INDICES

		Statistical Significance					
		Comparison * Time		Covariate * Time		Comp * Cov * Time	
Comparison Groups	Covariates	df	p	df	p	df	p
Nbhds	Age	5,114	0.000	1,114	0.000	5,114	0.022
	Dust Load, Entry			1,114	0.646	5,114	0.949
Nbhds	Age	5,119	0.055	1,119	0.000	5,119	0.217
	Dust Load, Floor			1,119	0.931	5,119	0.815
Nbhds	Age	5,121	0.002	1,121	0.000	5,121	0.051
	Dust Pb Load, Entry			1,121	0.781	5,121	0.951
Nbhds	Age	5,119	0.034	1,119	0.000	5,119	0.144
	Dust Pb Load, Floor			1,119	0.976	5,119	0.772

group and covariate is slightly significant after adjustment for changes in dust lead loading at the entry (P = 0.037) or dust loading on the floor (P = 0.0432). Dust lead loading on the floor is not significant by itself, but becomes marginally significant (P = 0.097) when dust loading is included in the model (P = 0.0207). Age category is highly significant, with children whose age at the beginning of the study in Round 1 was less than 12 months, and modest decreases in blood lead for children of age 2 years or older.

Although the evidence for a soil abatement effect is suggestive, it is hardly conclusive in the Cincinnati study. Some children in both the Mohawk and Pendleton neighborhoods had large increases in blood lead during the first post-abatement year, possibly associated with increases in dust lead loading and dust loading. This suggests that additional sources of dust exposure may have been occurring that were not under control by the study. Although some recontamination from other non-abated urban sources was expected, the magnitude of these effects was larger than expected. This may be one of the major challenges in doing urban soil lead remediation.

TABLE 5-10. REPEATED MEASURES ANALYSES OF BLOOD LEAD IN CINCINNATI STUDY FOR FIRST YEAR AFTER ABATEMENT, ADJUSTED FOR DIFFERENCES IN ENVIRONMENTAL INDICES: MOHAWK VERSUS PENDLETON

		Statistical Significance					
		Comparison * Time		Covariate * Time		Comp * Cov * Time	
Residual S.D. µg/dl	Covariates	df	p	df	p	df	p
2.97	Age GRP	1,24	0.6613	5,24	0.001180	----	----
	Dust Pb Conc, Entry	----	----	1,24	0.4590	----	----
	Dust Load, Entry	----	----	1,24	0.4888	----	----
	Dust Pb Load, Entry	----	----	1,24	0.3908	----	----
	Dust Pb Conc, Floor	----	----	1,24	0.8165	----	----
	Dust Load, Floor	----	----	1,24	0.0463	----	----
	Dust Pb Load Floor	----	----	1,24	0.0902	----	----
3.09	Age GRP	1,27	0.4957	5,27	0.000068	----	----
	Dust Pb Conc, Entry			1,27	0.3155	1,27	0.1667
	Dust Load, Entry	----	----	1,27	0.4074	1,27	0.4041
	Dust Pb Load, Entry	----	----	1,27	0.1340	1,27	0.1256
2.98	Age GRP	1,25	0.3454	5,25	0.000164	----	----
	Dust Pb Conc, Floor	----	----	1,25	0.1676	1,25	0.1615
	Dust Load, Floor	----	----	1,25	0.3579	1,25	0.2241
	Dust Pb Load, Floor	----	----	1,25	0.2046	1,25	0.1592
3.03	Age GRP	1,29	0.5527	5,29	0.000031	----	----
	Dust Pb Conc, Entry	----	----	1,29	0.5271	1,29	0.2384
	Dust, Pb Load, Entry	----	----	1,29	0.0445	1,29	0.0370

TABLE 5-10 (cont'd). REPEATED MEASURES ANALYSES OF BLOOD LEAD IN CINCINNATI STUDY FOR FIRST YEAR AFTER ABATEMENT, ADJUSTED FOR DIFFERENCES IN ENVIRONMENTAL INDICES: MOHAWK VERSUS PENDLETON

		Statistical Significance					
		Comparison * Time		Covariate * Time		Comp * Cov * Time	
Residual S.D. µg/dl	Covariates	df	p	df	p	df	p
3.06	Age GRP	1,27	0.3838	5,27	0.000191	----	----
	Dust Pb Conc, Floor	----	----	1,27	0.0551	1,27	0.0607
	Dust Load, Floor	----	----	1,27	0.0868	1,27	0.0426
3.13	Age GRP	1,31	0.4635	5,31	0.000051	----	----
	Dust Load, Entry	----	----	1,31	0.0780	1,31	0.0776
3.08	Age GRP	1,32	0.2644	5,32	0.000030	----	----
	Dust Pb Load, Entry	----	----	1,32	0.0369	1,32	0.0345
3.03	Age GRP	1,29	0.2643	5,29	0.000265	----	----
	Dust Load, Floor	----	----	1,29	0.0432	1,29	0.2582
3.16	Age GRP	1,29	0.8991	5,29	0.000397	----	----
	Dust Pb Load, Floor	----	----	1,29	0.4564	1,29	0.2460
3.04	Age GRP	1,30	0.5001	5,29	0.000336	----	----
	Dust Load, Floor	----	----	1,29	0.0868	----	----
2.95	Age GRP	1,29	0.5830	5,29	0.000128	----	----
	Dust Load, Floor	----	----	1,29	0.0207	----	----
	Dust Pb Load, Floor	----	----	1,29	0.0970	----	----

5.5 COMPARISONS USING STRUCTURAL EQUATIONS MODELS

The effectiveness of environmental lead intervention may be assessed in any of several ways, depending on the purposes of the analyses. One of the most important goals in the analysis of environmental lead data from the USLADP is the identification of the effects of different lead interventions on environmental pathways from lead sources through different media (especially household dust) to which the child may be exposed. A generic structural equation model is shown in Figure 5-52, and is analogous to individual segments of Figure 5-4. This is an *environment-only* model and assumes that the soil and dust lead interventions have no effects apart from those that can be identified by differences in lead concentrations in soil and dust, dust lead loadings, and total lead loadings, and long-term reductions in treatment group blood lead concentrations. Although these relationships are expressed by a series of interconnected algebraic equations, they may be more easily understood from the environmental pathway diagrams shown in Figure 5-52. The assumptions of the model are as follows:

1. Preabatement dust loadings depend on sociodemographic variables that affect household dustiness, such as the age of the house, and on environmental dust sources such as chipping and peeling interior paint;

2. Pre-abatement soil lead concentrations are independent or exogenous variables that may depend on exterior lead-based paint and on historic deposition of airborne lead particles from stationary sources (e.g., lead smelters or nonferrous metal processing operations) and from mobile sources (combustion of leaded gasoline);

3. Dust lead concentrations both pre- and postabatement are related to current soil lead concentrations at the time of measurement and to other sources such as deteriorating interior lead-based paint;

4. Dust lead loadings are the product of dust loading per unit area and the concentration of lead in house dust, an exact mathematical relationship denoted "X" in the figures;

5. Blood lead concentrations are related to lead in soil and to lead loading or concentration in house dust at or shortly before blood leads are measured, to prior or historic lead exposures that have accumulated a (primarily skeletal) body burden of lead that contributes to current blood lead concentrations, and on the child's age as well as many other individual behavioral or demographic factors;

6. Soil lead concentrations change very slowly over time, in the absence of interventions;

7. Blood lead concentrations from stored body burdens decrease relatively slowly over time, and in children such as those in the Boston USLADP who have had several years of exposure to high concentrations of environmental lead with consequently large skeletal lead pools, stored body burdens may account for 1-year postabatement blood lead concentrations that may be as high as 66% of the

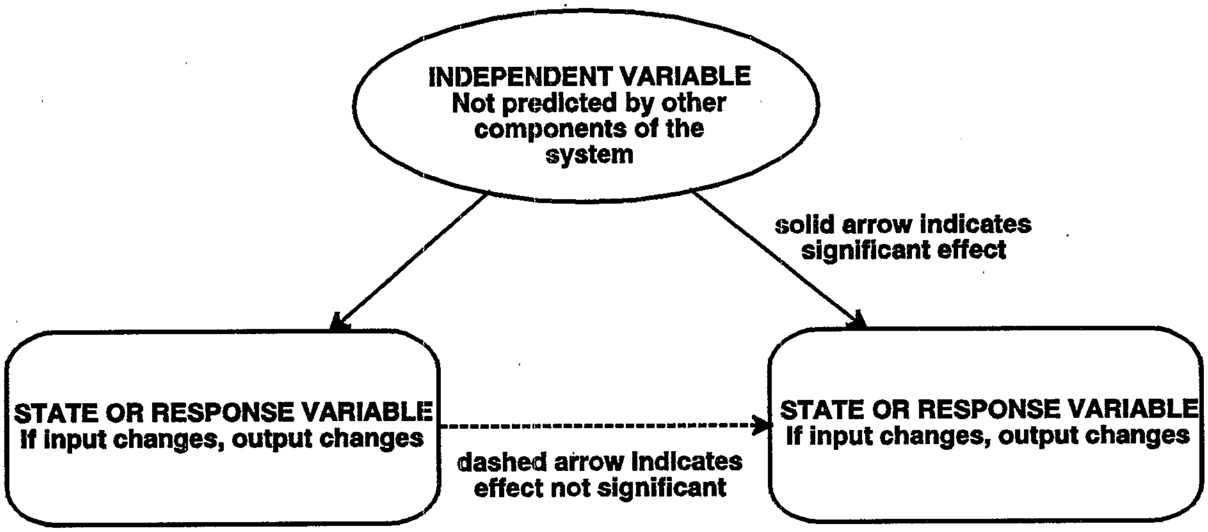

Figure 5-52. Explanation of the terms and features of the structural equation model diagram in Figures 5-53, 5-54, and 5-55.

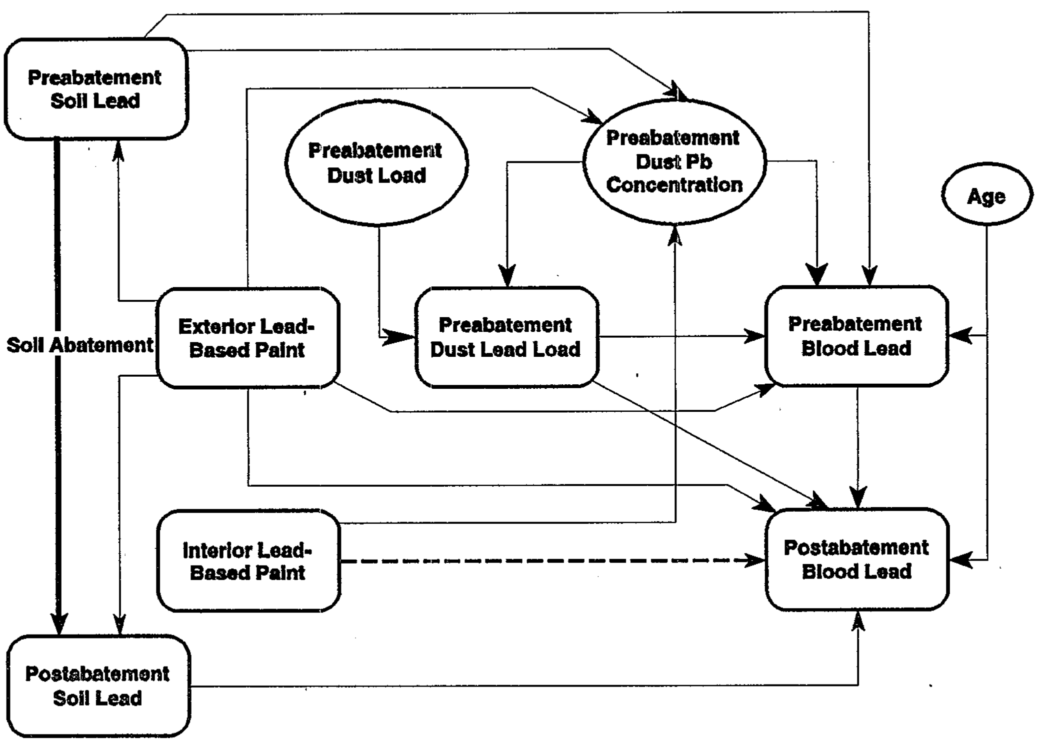

Figure 5-53. Structural equation model for childhood exposure in Baltimore.

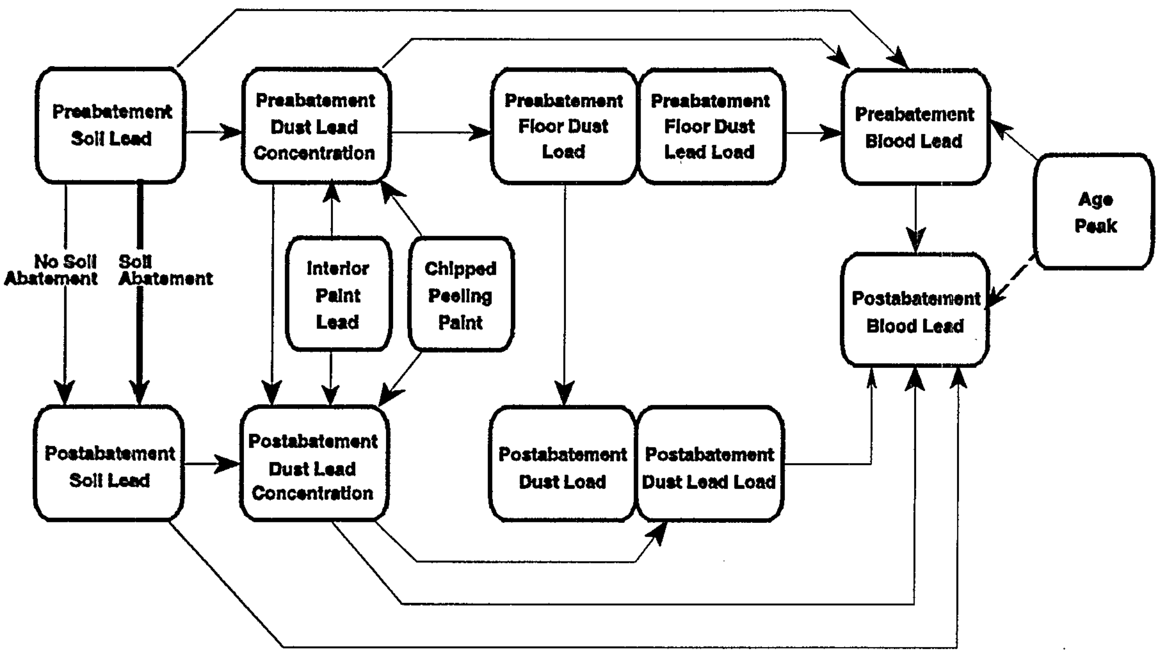

Figure 5-54. Structural equation model for Boston.

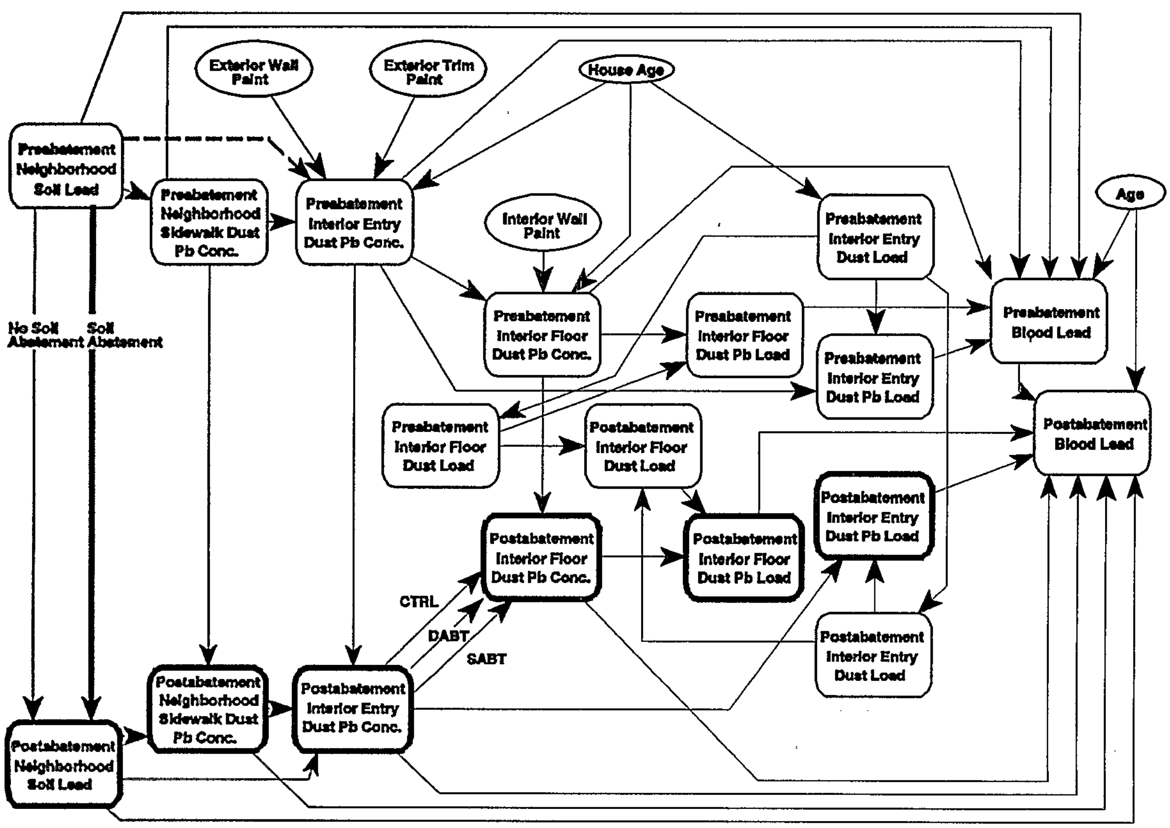

Figure 5-55. Structural equation model for Cincinnati.

preabatement concentrations, which may severely limit the potential effectiveness of any environmental lead intervention in treating currently lead-burdened children, and suggests that lead intervention may be far more effective in preventing lead poisoning in children who have never been exposed to elevated environmental lead.

5.5.1 General Issues in Structural Equation Modeling

The purpose of structural equation modeling is to elucidate pathways for environmental lead exposure from source to child. From this perspective, the development and testing of pathway models for urban lead is an exploratory model-building activity that does not readily lend itself to hypothesis testing. It is well known that "specification searches" such as stepwise regression have complicated inferential properties (Leamer, 1978), and the true P level for an estimated regression coefficient may be quite different from the nominal P value. An up-and-down search procedure was employed that started with a plausible pathway diagram, and dropped nonsignificant blocks of parameters if all estimates of the same or analogous parameters in different groups were zero or nonsignificant. New parameters were added for each new pathway in the model, based on prior beliefs and on sample correlation coefficients.

Structural equation models are useful in evaluating hypothetical causal pathways among multiple variables. This is particularly useful in assessing intervention studies in which changes in one part of a system can have both direct and indirect effects on other components of the system. The general framework for all of the models is shown in Figure 5-52. Independent variables (covariates, predictors) are those measured components of a system that are not predicted from other components. The independent variables are functionally independent of each other, but may be correlated with each other. It is not necessary to model an explicit causal pathway among the independent variables. Independent variables are shown by elliptical figures.

In Figure 5-52, dependent variables are shown as rectangular figures. The dependent variables of the system are assumed to have some predictive relationship to the independent variables and to each other. Although it is not necessary to dwell on the concept that there is a "causal" implication for any proposed predictive relationship, it should be noted that in a longitudinal lead study, most of the lead in yard soil at the earlier measurement will still be there at a later measurement unless the yard soil is removed; some of the lead in house dust will be left for later collection; and some of the lead in the child's body (even in blood and soft tissues) will be circulating in blood at a later measurement. Thus, estimates of lead concentrations in earlier samples are expected to be predictive of measurements from later samples, which are estimated of the same quantity, in part. The models do not depend on causal interpretations, however, but do assume a temporal direction in which the dependent variables depend on values of other variables measured at the same time, or measured previously, but not on values measured in the future. The direction of statistical dependence is shown by a line with an arrow. The line is solid if the relationship is statistically significant in the study, otherwise the line is dotted.

5.5.2 Results of Structural Equation Model Analyses

5.5.2.1 Baltimore Study

The structural equation model (denoted SEM) developed for the Baltimore study is shown in Figure 5-53. The model has three dependent variables with estimated parameters:

(1) Pre-abatement floor dust lead concentration measured by AAS, denoted DCFAR1.

(2) Pre-abatement blood lead concentration measured at Round 3, denoted BCR3.

(3) Post-abatement blood lead concentration measured at Round 6, denoted BCR6.

The preabatement floor dust lead loading, denoted LLFAR1, is calculated from the preabatement floor dust lead concentration DCFAR1 and from the preabatement total floor dust loading denoted DLFR1, which does not involve unknown parameters:

$$LLFAR1 = DLFR1 * DCFAR1 / 1,000$$

where the factor of 1,000 converts dust loading in mg/cm^2 and dust lead concentration in $\mu g/g$ into dust lead loading in $\mu g\ Pb/cm^2$.

The model also has a number of independent variables:

- SCR1 = soil lead concentration, preabatement
- SCR4 = soil lead concentration, postabatement (soil abatement group only; otherwise, SCR4 = SCR1 if no soil abatement)

- EP = exterior paint lead P-XRF
- IP = interior paint lead P-XRF
- DLR1 = total dust loading, preabatement
- AGE2 = (age in months at Round 3 - 36 months), squared.

A linear model was fitted in logarithmic form in order to stabilize variances. The parameters in the model are denoted D_, B_, and A_, with affixes:

log(DCFAR1) = log(DC0 + DS * SCR1 + DE * EP + DI * IP)

log(BCR3) = log(B0 + BC * DCFAR1 + BL * LLFAR1 + BE * EP + BI * IP + BS * SCR1 + BA2 * AGE2)

log(BCR6) = log(A0 + AB * BCR3 + AC * DCFAR1 + AL * LLFAR1 + AE * EP + AI * IP + AS * SCR4 + AA2 * AGE2).

The following equation defined dust lead loading, but had no parameters to estimate:

log(LLFAR1) = LC * log(DCFAR1) + LD * log(DLR1) - log(1,000),

where LC = 1 and LD = 1. The estimated parameters for two such models are shown in Table 5-11 and 5-12. All other parameters that were determined to be nonsignificant were set to 0 in the analysis reported here.

In Table 5-11, interior and exterior lead paint, and lead in soil make marginally significant contributions to floor dust lead concentrations in these Baltimore residences. However, preabatement blood lead shows little relationship to dust lead loading or exterior lead paint in this model. On the other hand, postabatement blood lead is highly correlated with dust lead loading, but only weakly associated with lead paint once the influence of starting blood lead (parameter AB) is taken into account. Interior lead and dust lead loading are somewhat confounded, because including dust lead loading tends to reduce the interior paint lead contribution to pre- and postabatement blood lead to nonsignificant levels.

The primary contribution of interior paint for these children appears to be as an indirect source of house dust. In Table 5-12, the contributions of soil lead, interior and exterior paint to house dust lead concentration are all statistically significant. The contribution of interior paint to blood lead pre- and postabatement is statistically significant, but interior paint does

TABLE 5-11. BALTIMORE STRUCTURAL EQUATION MODEL FULL INFORMATION MAXIMUM LIKELIHOOD METHOD

Dependent Variable	R^2 for Log Model	Predictor or Independent Variable	Coefficient ± S.E.	Units
Dust Lead Conc., AAS-PRE	0.0828	If Control Group 1: Intercept	1328 ± 1519	μg/g
		If Control Group 2: Intercept	504 ± 573	μg/g
		If Soil Abate: Intercept	−131 ± 395	μg/g
		Soil Lead Conc. - PRE	1.728 ± 1.257	μg/g per μg/g
		Interior Paint Lead XRF	203 ± 132	μg/g per mg/cm^2
		Exterior Paint Lead XRF (All groups)	86.0 ± 56.6	μg/g per mg/cm^2
Blood Lead - PRE	-0.0043	Intercept	9.76 ± 1.05	μg/dL
		Age-Squared	−0.00066 ± 0.00070	μg/dL per $month^2$
		Dust Lead Loading - PRE	0.79 ± 2.91	μg/dL per 1,000 $μg/m^2$
		Exterior Paint Lead XRF	0.118 ± 0.112	μg/dL per mg/cm^2
Blood Lead - POST	0.5459	Intercept	3.91 ± 0.33	μg/dL
		Age-Squared	−0.00095 ± 0.00011	μg/dL per $month^2$
		Dust Lead Loading - PRE	14.61 ± 2.18	μg/dL per 1,000 $μg/m^2$
		Interior Paint Lead XRF	0.036 ± 0.056	μg/dL per mg/cm^2
		Exterior Paint Lead XRF	0.012 ± 0.022	μg/dL per mg/cm^2
		Blood Lead - PRE	0.5629 ± 0.0274	μg/dL per μg/dL

TABLE 5-12. BALTIMORE STRUCTURAL EQUATION MODEL FULL INFORMATION MAXIMUM LIKELIHOOD METHOD

Dependent Variable	R^2 for Log Model	Predictor or Independent Variable	Coefficient ± S.E.	Units
Dust Lead Conc., AAS-PRE	0.0699	If Control Group 1: Intercept	1111 ± 1204	μg/g
		If Control Group 2: Intercept	326 ± 394	μg/g
		If Soil Abate: Intercept	−182 ± 288	μg/g
		Soil Lead Conc. - PRE	1.656 ± 0.790	μg/g per μg/g
		Interior Paint Lead XRF	241 ± 113	μg/g per mg/cm^2
		Exterior Paint Lead XRF (All groups)	87.4 ± 38.5	μg/g per mg/cm^2
Blood Lead - PRE	0.0211	Intercept	8.96 ± 0.83	μg/dL
		Age-Squared	−0.00094 ± 0.00071	μg/dL per $month^2$
		Dust Lead Loading - PRE	0.135 ± 1.866	μg/dL per 1,000 $\mu g/m^2$
		Interior Paint Lead	0.697 ± 0.287	μg/dL per 1,000 $\mu g/m^2$
		Exterior Paint Lead XRF	0.108 ± 0.081	μg/dL per mg/cm^2
Blood Lead - POST	0.5414	Intercept	3.41 ± 0.70	μg/dL
		Age-Squared	-0.00061 ± 0.00021	μg/dL per $month^2$
		Interior Paint Lead XRF	0.648 ± 0.197	μg/dL per mg/cm^2
		Exterior Paint Lead XRF	0.025 ± 0.049	μg/dL per mg/cm^2
		Blood Lead - PRE	0.5533 ± 0.0694	μg/dL per μg/dL

not make a significant contribution to blood lead when dust lead loading is included as a predictor of postabatement blood lead.

The models presented here do not include postabatement dust lead data because there were a substantial number of missing values, 25/80 in the soil abatement group, 4/21 in the Area 1 nonabatement group, and 40/76 in the Area 2 control group. Additional analyses using non-missing postabatement dust lead data may be useful.

5.5.2.2 Boston Study

The structural equation model (denoted SEM) developed for the Boston study is shown in Figure 5-54. The preabatement blood lead model had no statistically significant parameters other than the intercept, so that all preabatement lead variables are taken as independent variables. The model has four dependent variables with estimated parameters:

- Postabatement floor dust lead concentration at Round 4, denoted DCFR4
- Postabatement soil lead concentration at Round 3, denoted SCR3
- Postabatement floor dust loading at Round 4, denoted DLFR4
- Postabatement blood lead concentration measured at Round 3, denoted BCR3.

The pre- and postabatement floor dust lead loadings, denoted LLFR1 and LLFR4 respectively, are calculated from the preabatement floor dust lead concentrations DCFR1 and DCFR4, and from the pre- and postabatement total floor dust loadings denoted DLFR1 and DLFR4, which do not involve unknown parameters:

$$LLFR1 = DLFR1 * DCFR1 / 1{,}000$$

$$LLFR4 = DLFR4 * DCFR4 / 1{,}000$$

where the factor of 1000 converts dust loading in mg/cm^2 and dust lead concentration in $\mu g/g$ into dust lead loading in μg Pb/cm^2.

The model also has a number of independent variables:

- SCR1 = soil lead concentration, preabatement
- DCFR1 = floor dust lead concentration, preabatement
- BCR1 = blood lead concentration, preabatement
- IP = interior paint lead XRF
- CPTO = total area of chipped and peeling paint
- DLR1 = total dust loading, preabatement

- AGE2 = (age in months at Round 3 - 36 months), squared
- PRAY = property age (0 if post-1940, 1 if pre-1940).

A linear model was fitted in logarithmic form in order to stabilize variances. The parameters in the model are denoted D_, B_, and A_, with affixes:

log(SCR3) = log(SCNO * SCR1) if no soil abatement

log(SCR3) = log (SS) if soil abatement

log(DCFR4) = log(DC0 + DS * SCR1 + DI * IP + DCP * CPTO + DCC * DCFR1)

log(DLFR4) = log(DL0 + DLD * DLFR1 + DLP * CPTO)

log(BCR3) = log(A0 + AB * BCR1 + AC * DCFR4 + AL * LLFR4 + AI * IP + AS * SCR2 + AA2 * AGE2).

The following equations defined dust lead loading, but had no parameters to estimate:

log(LLFR1) = LC * log(DCFR1) + LD * log(DLR1) - log(1,000),

log(LLFR4) = LC * log(DCFR4) + LD * log(DLR4) - log(1,000),

where LC = 1 and LD = 1. The estimated parameters for two such models are shown in Table 5-13 and 5-14. All other parameters that were determined to be nonsignificant were set to 0 in the analysis reported here. The interior paint variables were not significant and were omitted from Figure 5-54.

In Tables 5-13 and 5-14, lead in soil makes a significant contributions to postabatement floor dust lead concentrations in these Boston residences. However, preabatement dust lead shows little relationship to interior lead paint or paint condition in this model. Dust loading is significantly correlated with dust lead loading the preceding year (parameter DLD) nonsignificant levels.

On the other hand, postabatement blood lead in Table 5-13 is highly correlated with dust lead loading, but only weakly associated with lead paint once the influence of starting blood lead (parameter AB) is taken into account. As shown in Table 5-14, the correlation of postabatement blood lead with dust lead concentration is weaker than the association with dust lead loading. Soil lead was not a significant direct predictor of blood lead.

The primary contribution of soil lead for these children appears to be as an indirect source of house dust. In Tables 5-13 and 5-14, the contribution of soil lead to house dust

TABLE 5-13. BOSTON STRUCTURAL EQUATION MODEL BLOOD LEAD VERSUS DUST LEAD LOADING FULL INFORMATION MAXIMUM LIKELIHOOD METHOD

Dependent Variable	R^2 for Log Model	Predictor or Independent Variable	Coefficient ± S.E.	Units
Soil Lead - POST	0.8453	If Soil Abate: Intercept	129 ± 15	μg/g
		If No Soil Abate: Soil Pb - PRE	0.832 ± 0.104	μg/g per μg/g
Dust Lead Conc. - POST	0.0845	Intercept	892 ± 149	μg/g
		Dust Pb Conc. - PRE	0.0111 ± 0.0208	μg/g per μg/g
		Soil Pb Conc. - POST	0.1697 ± 0.0775	μg/g per μg/g
Dust Load - POST	0.1357	Intercept	10.43 ± 2.94	mg/m^2
		Dust Load - PRE	0.2736 ± 0.0834	mg/m^2 per mg/m^2
Blood Lead - POST	0.3908	Intercept	2.38 ± 0.48	μg/dL
		Age-Squared (Peak at 36 Months)	0.00021 ± 0.00100	μg/dL per $month^2$
		Dust Lead Loading - POST	7.99 ± 4.01	μg/dL per 1,000 $\mu g/m^2$
		Blood Lead - PRE	0.5961 ± 0.0409	μg/dL per μg/dL

lead concentration are statistically significant, as is the contribution of dust lead loading to blood lead. In the Boston study, soil abatement produced a persistent reduction in soil lead, which was associated with a persistent reduction in dust lead that accounted for a persistent reduction in blood lead during the first year after abatement. Recent analyses (Aschengrau et al., 1994) show that additional decreases in blood lead occurred in the second year as well, provided no dust recontamination occurred.

TABLE 5-14. BOSTON STRUCTURAL EQUATION MODEL BLOOD LEAD VERSUS DUST LEAD CONCENTRATION FULL INFORMATION MAXIMUM LIKELIHOOD METHOD

Dependent Variable	R^2 for Log Model	Predictor or Independent Variable	Coefficient ± S.E.	Units
Soil Lead - POST	0.8485	If Soil Abate: Intercept	132 ± 19	μg/g
		If No Soil Abate: Soil Pb - PRE	0.867 ± 0.125	μg/g per μg/g
Dust Lead Conc. - POST	0.0611	Intercept	940 ± 158	μg/g
		Dust Pb Conc. - PRE	0.0105 ± 0.0220	μg/g per μg/g
		Soil Pb Conc. - POST	0.1821 ± 0.0768	μg/g per μg/g
Dust Load - POST	0.1374	Intercept	10.42 ± 2.79	mg/m^2
		Dust Load - PRE	0.2705 ± 0.0849	mg/m^2 per mg/m^2
Blood Lead - POST	0.4155	Intercept	3.39 ± 0.48	μg/dL
		Age-Squared (Peak at 36 Months)	−0.00193 ± 0.00111	μg/dL per $month^2$
		Dust Lead Conc. - POST	0.225 ± 0.194	μg/dL per 1,000 μg/g
		Blood Lead - PRE	0.5834 ± 0.0440	μg/dL per μg/dL

5.5.2.3 Cincinnati Study

The structural equation model developed for the Cincinnati study is shown in Figure 5-55. Because the study collected a larger number of interior and exterior environmental indices than did the Baltimore or Boston studies, it was possible to develop a more detailed environmental pathway model than in the other studies. The Cincinnati model has twelve dependent variables with estimated parameters:

- Preabatement neighborhood sidewalk lead concentration at Round 1, denoted DCWR1

- Preabatement interior entry dust lead concentration at Round 1, denoted DCER1
- Preabatement floor dust lead concentration at Round 1, denoted DCFR1
- Preabatement floor dust loading at Round 1, denoted DLFR1
- Preabatement blood lead concentration measured at Round 1, denoted BCR1
- Postabatement neighborhood soil lead concentration at Round 4, denoted SCR4
- Postabatement neighborhood sidewalk lead concentration at Round 4, denoted DCWR4
- Postabatement interior entry dust lead concentration at Round 4, denoted DCER4
- Postabatement floor dust lead concentration at Round 4, denoted DCFR4
- Postabatement interior entry dust loading at Round 4, denoted DLER4
- Postabatement floor dust loading at Round 4, denoted DLFR4
- Postabatement blood lead concentration measured at Round 4, denoted BCR4.

The pre- and postabatement floor dust lead loadings, denoted LLFR1 and LLFR4 respectively, and the interior entry dust lead loadings, denoted LLER1 and LLER4 respectively, are calculated from the preabatement floor and interior entry dust lead concentrations DCFR1, DCFR4, DCER1, and DCER4, and from the pre- and postabatement total floor dust loadings denoted DLFR1, DLFR4, DLER1, and DLER4, which do not involve unknown parameters:

$$LLFR1 = DLFR1 * DCFR1 / 1,000$$

$$LLFR4 = DLFR4 * DCFR4 / 1,000$$

$$LLER1 = DLER1 * DCER1 / 1,000$$

$$LLER4 = DLER4 * DCER4 / 1,000$$

where the factor of 1,000 converts dust loading in mg/cm^2 and dust lead concentration in μg/g into dust lead loading in μg Pb/cm^2.

The model also has a number of independent variables:

- SCR1 = neighborhood soil lead concentration, preabatement
- DLER1 = interior entry dust loading, preabatement
- XMET = exterior trim paint lead, mean XRF

- XMEW = exterior wall paint lead, mean XRF
- XMIT = interior trim paint lead, mean XRF
- XMIW = interior wall paint lead, mean XRF
- AGE2 = (age in months at Round 3 - 36 months), squared
- SIB = number of preschool children in household
- PRAY = property age (0 if post-1940, 1 if pre-1940).

A linear model was fitted in logarithmic form in order to stabilize variances. The parameters in the model are denoted S_, F_, E_, L_, D_, B_, and A_, with affixes:

log(SCR4) = log(SCNO * SCR1) if no soil abatement
log(SCR4) = log (SS) if soil abatement

log(DCWR1) = log(DW1 + DWS1 * SCR1)
log(DCWR4) = log(DW4 + DWS4 * SCR4)

log(DCER1) = log(DE1 + DEW1 * DCWR1 + DET1 * XMET + DEW1 * XMEW + DEY1 * PRAY)

log(DCER4) = log(DEC4 + DEW4 * DCWR4) if CONTROL group;
log(DCER4) = log(DED4 + DEW4 * DCWR4) if DUST ABATE group;
log(DCER4) = log(DES4 + DEW4 * DCWR4) if SOIL ABATE group;

log(DCFR1) = log(DF1 + DFE1 * DCER1 + DFIW1 * XMIW + DEY1 * PRAY)

log(DCFR4) = log(DFC4 + DFEC4 * DCER4) if CONTROL group;
log(DCFR4) = log(DFD4 + DFED4 * DCER4) if DUST ABATE group;
log(DCFR4) = log(DFS4 + DFES4 * DCER4) if SOIL ABATE group;

log(DLER4) = log(DLEC4 + DLE4 * DLER1) if CONTROL group;
log(DLER4) = log(DLED4 + DLE4 * DLER1) if DUST ABATE group;
log(DLER4) = log(DLES4 + DLE4 * DLER1) if SOIL ABATE group;

log(DLFR1) = log(DLF1 + DLFE1 * DLER1 + DLFY1 * PRAY)

log(DLFR4) = log(DLFC4 + DLF4 * DLFR1) if CONTROL group;
log(DLFR4) = log(DLFD4 + DLF4 * DLFR1) if DUST ABATE group;
log(DLFR4) = log(DLFS4 + DLF4 * DLFR1) if SOIL ABATE group;

log(BCR1) = log(B0 + AK * SIB + ACW1 * DCWR1 + AL1 * LLFR1 + AA2 * AGE2).
log(BCR4) = log(A0 + AB * BCR1 + ACW4 * DCWR4 + AL4 * LLFR4 + AA2 * AGE2).

The following equations defined dust lead loading, but had no parameters to estimate:

$$\log(LLFR1) = LC * \log(DCFR1) + LD * \log(DLFR1) - \log(1,000),$$

$$\log(LLFR4) = LC * \log(DCFR4) + LD * \log(DLFR4) - \log(1,000),$$

$$\log(LLER1) = LC * \log(DCER1) + LD * \log(DLER1) - \log(1,000),$$

$$\log(LLER4) = LC * \log(DCER4) + LD * \log(DLER4) - \log(1,000),$$

where LC = 1 and LD = 1. The estimated parameters for one such model is shown in Table 5-15. All other parameters that were determined to be nonsignificant were set to 0 in the analysis reported here. The interior paint trim variable was not significant and was omitted from Figure 5-55.

In Table 5-15, lead in soil makes a significant contribution to pre- and postabatement sidewalk dust lead concentrations in these Cincinnati neighborhoods. Both pre- and postabatement interior entry dust lead shows a statistically significant relationship to neighborhood sidewalk dust lead concentrations. Exterior lead paint on walls and trim contributes significantly to preabatement interior entry dust lead concentrations in this model, even though these are "gut rehab" housing units. The dust lead pathway can be traced further by statistically significant relationships between preabatement entry dust lead and floor dust lead concentrations, and by a marginal statistically significant relationship between postabatement entry dust and floor dust lead concentration in the dust abatement neighborhoods. Dust loading is not significantly correlated with dust loading at the interior entry or floor a year earlier, but preabatement floor dust loading is significantly correlated with interior entry dust loading in the same residence. This suggests a consistent but complex pattern of movement of particles from the soil and other sources to the sidewalk and surface areas outside these urban residential properties, then into the individual dwelling units within the property.

Preabatement blood lead shows a significant relationship to dust lead loading at the interior entry, but not to dust lead loading on the unit floor or sidewalk lead concentration. On the other hand, postabatement blood lead in Table 5-15 is more highly correlated with sidewalk dust lead concentration than with interior entry or floor dust lead concentration or loading, once the influence of starting blood lead (parameter AB) is taken into account. Soil lead was not a significant direct predictor of blood lead, but its effect as an indirect source can be traced along the soil-to-sidewalk-to-entry-to-floor dust pathway.

TABLE 5-15. CINCINNATI STRUCTURAL EQUATION MODEL BLOOD LEAD VERSUS SIDEWALK DUST LEAD CONCENTRATION ITERATED TWO-STAGE LEAST SQUARES METHOD

Dependent Variable	R^2 for Log Model	Predictor or Independent Variable	Coefficient ± S.E.	Units
Soil Lead - POST	0.8999	If Soil Abate: Intercept	129 ± 5	µg/g
		If No Soil Abate: Soil Lead - PRE	0.898 ± 0.025	µg/g per µg/g
Dust Lead Conc. - PRE, Sidewalk	0.3989	Intercept	202 ± 301	µg/g
		Soil Lead - PRE	5.84 ± 0.89	µg/g per µg/g
Dust Lead Conc. - POST, Sidewalk	0.1032	Intercept	1587 ± 683	µg/g
		Soil Lead - POST	6.00 ± 2.75	µg/g per µg/g
Dust Lead Conc. - PRE, Int. Entry	0.2902	Intercept	90 ± 48	µg/g
		Property Age (0 = new, 1 = old)	111 ± 168	µg/g
		Dust Lead Conc. - PRE, Sidewalk	0.033 ± 0.013	µg/g per µg/g
		Exterior Trim Paint Lead XRF	48.3 ± 27.4	µg/g per mg/cm^2
		Exterior Wall Paint Lead XRF	78.4 ± 47.4	µg/g per mg/cm^2
Dust Lead Conc. - POST, Int. Entry	0.0893	If Control: Intercept	275 ± 113	µg/g
		If Dust Abate: Intercept	−190 ± 434	µg/g
		If Soil Abate: Intercept	263 ± 190	µg/g
		Dust Lead Conc. - POST, Sidewalk (All Groups)	0.139 ± 0.079	µg/g per µg/g

TABLE 5-15 (cont'd). CINCINNATI STRUCTURAL EQUATION MODEL BLOOD LEAD VERSUS SIDEWALK DUST LEAD CONCENTRATION ITERATED TWO-STAGE LEAST SQUARES METHOD

Dependent Variable	R^2 for Log Model	Predictor or Independent Variable	Coefficient ± S.E.	Units
Dust Loading - POST, Int. Entry	0.1929	If Control: Intercept	474 ± 243	mg/m^2
		If Dust Abate: Intercept	3753 ± 1251	mg/m^2
		If Soil Abate: Intercept	−2394 ± 1346	mg/m^2
		Dust Loading - PRE, Int. Entry	0.0027 ± 0.0077	mg/m^2 per mg/m^2
Dust Lead Conc. - PRE, Floor	0.2022	Intercept	22 ± 61	µg/g
		Property Age	−96.6 ± 133.9	µg/g
		Dust Lead Conc. - PRE, Int. Entry	0.976 ± 0.239	µg/g per µg/g
		Interior Wall Paint Lead XRF	48.1 ± 42.0	mg/g per mg/cm^2
Dust Lead Conc. - POST, Floor	0.3250	If Control: Intercept	191 ± 45	µg/g
		Dust Lead Conc. - POST, Int. Entry	0 (constr.)	µg/g per µg/g
		If Dust Abate: Intercept	340 ± 127	µg/g
		Dust Lead Conc. - POST, Int. Entry	0.315 ± 0.175	µg/g per µg/g
		If Soil Abate: Intercept	141 ± 253	µg/g
		Dust Lead Conc. - POST, Int. Entry	0.124 ± 0.522	µg/g per µg/g
Dust Loading - PRE, Floor	0.5675	Intercept	129 ± 25	mg/m^2
		Dust Loading - PRE, Int. Entry	0.125 ± 0.035	mg/m^2 per mg/m^2
		Property Age	−104 ± 101	mg/m^2

TABLE 5-15 (cont'd). CINCINNATI STRUCTURAL EQUATION MODEL BLOOD LEAD VERSUS SIDEWALK DUST LEAD CONCENTRATION ITERATED TWO-STAGE LEAST SQUARES METHOD

Dependent Variable	R^2 for Log Model	Predictor or Independent Variable	Coefficient ± S.E.	Units
Dust Loading - POST, Floor	0.0789	If Control: Intercept	202 ± 76	mg/m^2
		If Dust Abate: Intercept	278 ± 71	mg/m^2
		If Soil Abate: Intercept	73 ± 117	mg/m^2
		Dust Loading - PRE, Floor (All Groups)	0.0477 ± 0.0328	mg/m^2 per mg/m^2
Blood Lead - PRE	0.2879	Intercept	10.09 ± 1.45	μg/dL
		Age for Peak Blood Lead	47.2 ± 19.1	months
		Age-Squared	−0.0026 ± 0.0028	μg/dL per month
		Number of Preschool Children	0.33 ± 0.67	μg/dL per child
		Dust Lead Loading - PRE, Floor	0.191 ± 0.124	μg/dL per 1,000 $\mu g/m^2$
		Dust Lead Conc. - PRE, Sidewalk	0.078 ± 0.252	μg/dL per 1,000 μg/g
Blood Lead - POST	0.3430	Intercept	0.86 ± 6.04	μg/dL
		Age-Squared	0.0019 ± 0.0042	μg/dL per month
		Dust Lead Conc. - POST, Sidewalk	0.454 ± 0.488	μg/dL per 1,000 μg/g
		Blood Lead - PRE	0.5501 ± 0.4468	μg/dL per μg/dL

The primary contribution of soil lead for these children appears to be as an indirect source of lead in house dust. In the Cincinnati study, soil abatement did not produce a persistent reduction in dust lead or blood lead during the first year after abatement.

5.6 SUMMARY OF STATISTICAL ANALYSES

5.6.1 General Observations

This integrated assessment of the USLADP includes a reevaluation of the results of the analyses carried out by the original investigators and of the conclusions reached by the investigators based on their analyses. While we have largely confirmed the numerical results of the analyses, other interpretations of the results are also consistent with these numerical findings and, in some cases, may be more plausible than the conclusions published by the investigators. We have also extended the results of the original investigations by carrying out additional analyses, using a consistent set of powerful analytical techniques not available when the original reports were published.

5.6.1.1 Combining Studies

There were substantial differences in the design of the three studies that precluded completely identical analyses of the data. It was technically possible to create a combined data set, given that all three studies included data on blood lead and hand lead before and after abatement, as well as carefully coordinated measures of family demographic characteristics, soil and dust lead at the child's residence. However, there were substantial differences in study design, such as the characterization of the "control" groups, pre-abatement paint stabilization, age distribution at the time of abatement, ethnic and racial characteristics of the populations, and pre-abatement soil lead exposure. Mathematically similar measures of effect in each study would therefore have very different interpretations, and would not be clearly generalizable to other study designs, much less to soil lead abatement in other communities. However, some parameters are the same, such as the persistence parameter for blood lead used in structural equation models.

5.6.1.2 Measurement Error

Statistical characteristics of these studies must be interpreted in the light of so-called "measurement error". QA/QC procedures were instituted to minimize analytical errors in the measurement of blood lead, soil lead, and dust lead concentrations. However, a larger part of the possible difficulty in reproducing lead measurements is likely to be found in the necessity of sampling highly variable phenomena. Blood lead concentrations are known to change over time as a function of changes in behavior (e.g., ingestion of soil or hand washing), diet (intake of calcium, iron, lactate, vitamins, fiber, etc.), and metabolism (thyroid function, etc.). Soil lead concentrations may change only slowly over time, but there are obviously serious difficulties in sampling precisely the same location at different times. This raises serious questions about the appropriate method for monitoring changes in soil lead over time, or characterizing soil lead for potential child exposure in a yard or some portion of a parcel of land. Finally, there are even more serious questions about defining a dust lead concentration or dust lead loading for child exposure. Dust lead exposure depends on the sections of bare or carpeted floor sampled, on the selection of rooms and sampling areas, and on variable factors such as season, frequency of opening of doors and windows, house cleaning, and other variable factors. In spite of these difficulties, there are statistically strong correlations among lead in soil and dust, on child's hands and in child's blood that are found in almost all recent studies.

5.6.2 Summary of Results

The data presented in this section lead to the following conclusions:

(1) Soil abatement in each study effectively reduced the concentration of lead in the soil in the areas where soil abatement was performed.

(2) In the Boston and Cincinnati studies, the effectiveness of soil abatement was persistent through the end of the study. There were no followup measurements of soil in Baltimore to demonstrate persistency.

(3) Exterior dust abatement, performed only in Cincinnati, was not persistent, indicating a source of lead other than soil at the neighborhood level.

(4) Interior dust with soil abatement, as performed in Cincinnati and Boston, appeared to respond to subsequent changes in exterior dust and soil lead in Cincinnati. Entry way measurements of lead concentration and lead load may be a good indicator of the movement of environmental lead into the living unit.

(5) Hand lead measurements often reflected general trends in blood lead measurements and may be a reasonable estimate of recent exposure. Hand lead, as measured in these studies, can be a useful complement to blood lead measurements.

(6) Paint stabilization as performed on all homes with lead-based paint in Boston (interior) and Baltimore (exterior), was intended to reduce the potential confounding effects from contamination of soil and dust, but in retrospect, paint stabilization itself may be a form of intervention in this study.

5.6.3 Limitations of the Statistical Methods

The statistical methods used here were reasonable and appropriate, and could be used by other investigators with access to standard statistical software packages. However, the methods have certain limitations that should be understood. The repeated measures analyses assume only that the response variables are correlated with each other, with no implication of temporal causality. The goodness of fit of the models was significantly improved by use of covariate analyses. Some repeated measures analyses require that the covariates have no time dependence. In most applications in this chapter, only two time points (before and after abatement) were used and the pre-post difference in environmental covariates was used.

A problem arises if the response variable must be transformed, say by a logarithmic transformation for blood lead or for hand lead, in order to reduce skewness and to stabilize variances across treatment groups. The implied model for the original untransformed variable is then *multiplicative* in treatment effects and random variation. This is probably acceptable for the analysis of variance, but is likely to produce a physically or biologically meaningless specification for the covariate model when the covariates are indicators of distinct and additive sources of lead, such as soil lead and interior lead-based paint. The logarithmic model does not reproduce the *additive* nature of the separate exposure pathways.

Extension of repeated measures analyses to covariates such as environmental lead levels that change with time can be done using a single technique, structural equation modeling. These methods provide more powerful interpretive tools. The availability of environmental data to characterize time-varying lead exposures in the Boston and Cincinnati studies suggests that more powerful statistical methods, such as structural equation models, could be more appropriate.

5.6.4 Comparison Across the Three Studies

The effectiveness of soil lead abatement in reducing blood lead varied greatly among the three cities. The variability in abatement effects is probably due to substantial differences in lead sources and pathways among the neighborhoods in these studies. These differences for each study are discussed below.

The Baltimore study had two neighborhoods, Upper Park Heights and Walbrook Junction. The area to which abatement was assigned (Park Heights) had enrolled families whose residences did not have soil lead levels that were high enough to justify abatement. The soil lead levels in the nonabatement premises in Park Heights that were measured in the preabatement phase were not significantly smaller than those of the control premises in Walbrook Junction. Therefore, the nonabatement houses in Park Heights were used as an additional control group. Unlike the other two studies, the soil abatement in Baltimore was not accompanied by interior dust abatement. There was essentially no significant effect of soil abatement in the abated houses, compared to the control group. Statistical covariate adjustment in both repeated measures analyses showed that the differences in blood lead levels both before and after abatement were significantly dose-related to interior lead-based paint and (nonabated) interior dust. It is likely that interior paint contributed to child lead exposure, either directly by ingestion of paint chips, or indirectly by the hand-to-mouth exposure pathway, as follows:

$$\text{interior paint} \Rightarrow \text{interior dust} \Rightarrow \text{hands} \Rightarrow \text{blood}.$$

Cross-sectional and longitudinal structural equation analyses could be used to explore this hypothesis. However, because there were no repeated measurements of household dust lead, it will be very difficult to assess changes in exposure over time except by use of hand lead data. Concerning the Baltimore study, we conclude that:

> *It is likely that soil lead abatement had little effect on the primary factors responsible for elevated child blood lead levels in these two neighborhoods, which appear to be interior lead-based paint and interior dust lead.*

The Boston study was conducted with blood and hand leads measured at one preabatement round and at about 8 months after abatement. Soil and dust lead measurements were available for pre- and postabatement at about the same time. These data allowed a very complete analysis of blood lead responses to changes in dust and soil lead over time. Relative to the no treatment group, the results showed clearly that there was a persistent reduction in blood lead levels (1.5 μg/dL) in the soil lead abatement children, and that, on average, the postabatement blood leads were lowest in premises that had the lowest postabatement soil lead and dust lead loadings. Interior and exterior lead paint were not significant predictors of blood lead for Boston children. Concerning the Boston study, we conclude:

When soil and dust lead levels show a persistent decrease as a result of effective abatement, blood lead levels also show a persistent decline.

Because the Cincinnati study had collected blood lead and environmental samples in six Cincinnati neighborhoods, analyses comparable to those reported for the Baltimore and Boston studies can be made. After some analyses using models similar to those for Baltimore and Boston, it became evident that the neighborhoods within each of their treatment group were not comparable in every way. Although there was a strong dependence of blood lead on environmental lead, particularly on hand lead and on current floor or entry dust lead there was no clear pattern of change or response of interior dust lead levels after abatement.

We are inclined to accept the conclusion of the Cincinnati investigators that blood and dust lead levels were affected differently at different times and places by other events not under their control. However, the dose-dependence exhibited in the models suggests that reducing interior dust lead levels did reduce blood lead levels, at least for a while. The problem is that the abatements did not always persistently reduce dust lead levels. We therefore conclude that:

There were additional sources of environmental lead exposure that had different effects on the neighborhoods during the course of the Cincinnati study and were not related to the abatement methods used in the study. It will be necessary to use other analysis methods, such as structural equations modeling, in order to assign changes in Cincinnati child blood lead levels to changes in lead exposure.

6. INTEGRATED SUMMARY AND CONCLUSIONS

6.1 PROJECT OVERVIEW

This project focuses on the exposure environment of the individual child, looking at three indicators of exposure: blood lead, hand lead, and house dust lead. From the perspective of the child's environment, changes in the soil concentration are expected to bring about changes in the house dust concentration, the hand dust loading, and the blood lead concentration.

In the past 25 years, concern for children with lead poisoning has steadily increased with mounting evidence for the subtle but serious metabolic and developmental effects of lead exposure levels previously thought to be safe. Childhood lead poisoning was formerly considered a severe medical problem usually traced to swallowed chips of peeling lead-based paint. Scientific evidence has systematically revealed deleterious effects of lead at lower levels of exposure. Agencies such as the U.S. Environmental Protection Agency and the Centers for Disease Control and Prevention (CDC) have repeatedly lowered the level of concern for children's lead burden that recommends environmental or clinical intervention from a blood lead level of 30 μg/dL established in 1978 by CDC to 25 μg/dL in 1985, just prior to the start of the project, then to the present level of 10 μg/dL, which was defined in October 1991 by CDC as a blood lead level that should trigger community-wide prevention activities if observed in many children.

The purpose of Urban Soil Lead Abatement Demonstration Project (USLADP) was to determine to what extent intervention in the form of soil abatement in residential neighborhoods would be effective as a means to reduce childhood lead exposure. Each of the three studies in the project is a longitudinal study of the impact of an altered environment on the lead exposure of children. The studies focused on evaluation of the exposure environment of the children living mainly in inner city neighborhoods. Measurements of lead in key external environmental media (e.g., soil, exterior and interior dust, and paint) were obtained prior to soil abatement, along with more direct indices of personal exposure in terms of hand wipes and blood lead levels. Abatement of soil lead generally involved removal of contaminated soil and replacement with "clean" soil. Postabatement lead levels in the above media and children's blood lead were remeasured at varying intervals to determine the effect of soil abatement, alone or in combination with paint stabilization or dust abatement, on blood lead concentrations. There are few other longitudinal studies of this type, and none of this scope or duration. Because the three studies were conducted using mutually agreed upon protocols, with few exceptions, a common ground exists for understanding an array of information available from the three individual studies that broadens the base of information beyond the limits of a single study or location.

Although the three studies were conducted independently, an effort was made to coordinate the critical scientific aspects of each study in order to provide comparable data at their completion. This effort included seventeen workshops where the study designs, sampling procedures, analytical protocols, and QA/QC requirements of each study were discussed with a goal toward reaching a common agreement. In most cases, a consensus was reached on the resolution of specific issues, but the individual studies were not bound to conform to that consensus or to adhere to it throughout the study. This procedure produced similar studies with some differences in study design and experimental procedures.

The individual results for each of the three cities were originally presented at an EPA-sponsored symposium in August 1992. These presentations included the data analysis and conclusions for each of the three individual city studies. Following this open discussion with the scientific community, the three research teams submitted their respective reports to the designated EPA regional offices (Boston, Region I; Baltimore, Region III; and Cincinnati, Region V). These reports and their associated data sets were then provided to EPA's Office of Research and Development (ORD) and Office of Solid Waste and Emergency Response (OSWER) for further analysis and preparation of this Integrated Report.

The EPA review of the study designs, chemical analytical procedures and data quality measures has found no major flaws that would cast doubt on the findings of the individual reports. The data sets submitted to EPA were systemically scrutinized for errors and inconsistencies, and were reviewed and revised by the principal investigators for each of the three cities prior to the completion of the analyses reported here. The few data corrections found to be necessary were minor and would not have altered the conclusions of the individual city reports.

This draft integrated report has reached its present form after an extensive review process. First, the reports of the individual studies were peer reviewed by non-EPA experts, revised, and presented to EPA in their final form, along with the data sets that were used as the basis for the individual reports. These data sets were then reanalyzed by EPA using rigorous statistical techniques to extract information not easily accessible from any individual study. An earlier draft of integrated report was next written based on those initial analyses. Following internal review and revision, the integrated report was released in draft form for public comment and external review at an expert workshop. Further statistical analyses (based in part on peer review comment recommendations) have since been carried out, and this draft of the integrated report incorporates changes reflecting the new analyses and earlier comments from the external experts. Another round of review and revision of the draft report is now being carried out prior to its final release.

Electronic copies of the underlying three cities data sets will be made available to members of the scientific community for continued review and analysis along with the release of the final version of this report. This continuing reanalysis means that new perspectives on the USLADP data may emerge. Although it is unlikely that major findings have been overlooked during these extensive review phases, it is not at all unreasonable that still further information will be retrieved and reported by the extended investigations to be made possible by this open policy for data release.

6.2 SUMMARY OF FINDINGS

6.2.1 EPA Integrated Report Results

This integrated assessment looks at the three individual studies collectively to determine if a broad overview can be taken of the project results when each study is placed in its correct perspective.

The key findings of this integrated assessment with regard to the Boston study are as follows:

1. The median preabatement concentration of lead in soil was relatively high in Boston, averaging about 2,400 μg/g with few samples below 1,000 μg/g.

2. Abatement of the soil effectively reduced the median concentration of lead in the soil to about 150 μg/g (an average decrease of about 2,300 μg/g).

3. Soil was clearly a part of the exposure pathway to the child, contributing significantly to house dust lead.

4. Other sources of lead, such as interior lead-based paint were minimized by stabilization.

5. The reductions of lead in both soil and house dust persisted for at least two years.

6. Blood lead levels were reduced by approximately 1.6 μg/dL at 10 mo after soil lead abatement.

7. Additional reductions in blood lead of about 1.0 μg/dL (relative to non-abated) were observed at 22 mo postabatement for children in houses where the soil lead was abated and the interior house dust lead was consequently reduced and remained low.

Thus, in the Boston study, the abatement of soil resulted in a measureable, statistically significant decline in blood lead concentrations in children, and this decline continued for at least two years. It appears that the following conditions were present, and perhaps necessary for this effect: (a) a notably elevated starting soil lead concentration (e.g., in excess of 1,000 to 2,000 μg/g); (b) a marked reduction of more than 1,000 μg/g in soil lead consequent to soil abatement accompanied by (c) a parallel marked and persisting decrease in house dust lead.

These conclusions are consistent with those reported by the Boston research team. This integrated assessment found no basis for modifying their conclusions, although we choose not to express these findings as a broadly generalizeable linear relationship between soil and blood, such as change in micrograms of lead per deciliter of blood per change in micrograms of lead per gram of soil, because we believe that such a linear expression of abatement effects is highly site specific for the soil-to-blood relationship. We found evidence that the dust-to-blood relationship is more significant and, perhaps, more linear than the soil-to-blood relationship.

With regard to the Baltimore analyses conducted for this integrated assessment, the participants in the abatement neighborhood that did not receive abatement were treated as a separate control group, rather than combined with the nonabatement neighborhood (as the Baltimore research team did). The reason for this was to establish a control group not influenced by differences between neighborhoods. This alternative approach used in this integrated assessment had little impact on the statistical significance of soil abatement effects as reported by the Baltimore research team.

The key findings of this integrated assessment for Baltimore are:

1. The preabatement concentrations of lead in soil were notably lower (i.e., averaging around 500 to 700 μg/g, with few over 1,000 μg/g) than in Boston.

2. The actual reduction of lead in soil by abatement was small (a change of about 400 μg/g), compared to the Boston study (a change of about 2,300 μg/g).

3. Measurements of blood lead were made for only ten months following abatement; and no significant decreases in blood lead consequent to soil abatement were observed compared to non-abatement control group children.

4. Except for exterior lead-based paint, there was no control of other sources of lead, such as the stabilization of interior lead-based paint (as done in Boston) or abatement of house dust (as done in Boston and Cincinnati).

5. Follow-up measurements of soil (except immediately postabatement) were not made to establish the persistency of soil abatement, and its possible effects on house dust.

Thus, in Baltimore, where starting soil lead concentrations were much lower than in Boston and soil abatement resulted in much smaller decreases in soil lead levels and no interior paint stabilization or dust abatement was performed, no detectable effects of soil lead abatement on blood lead levels were found.

These conclusions are consistent with those reported by the Baltimore research group, and are not inconsistent with those above for the Boston study. At soil concentratons much lower than the Boston study, the Baltimore group would have likely been able to see only a very modest change in blood lead concentrations (perhaps less than 0.2 μg/dL) assuming similarity between the study groups in Boston and Baltimore and the same linear relationship between change in soil concentration and change in blood lead. Furthermore, the interior paint stabilization and house dust abatement performed in Boston perhaps enhanced and reinforced the impact of soil abatement on childhood blood lead, whereas in Baltimore, any possible small impact of soil abatement would have likely been swamped by the large reservoir of lead in the interior paint and the large unabated amounts of lead in interior house dust.

As for the Cincinnati study, because of differences in the neighborhoods, we found that combining neighborhoods into treatment groups often obscures important effects, and chose to analyze each of the six Cincinnati neighborhoods as separate treatment groups. One neighborhood, Back Street, had an insufficient number of participants and was dropped from some analyses. The Back Street group started with nine families, but by Round 5 there was only one participating family in the study. We also found that the two control neighborhoods, Glencoe and Mohawk, were substantially different, and that the three remaining treatment groups, Pendleton, Dandridge, and Findlay, were more comparable, both demographically and in geographic proximity, to Mohawk than to Glencoe.

On this basis, we concluded that, in most cases, the effect of soil abatement could not be clearly determined, and offer the following explanation for this conclusion:

1. Most of the soil parcels in each neighborhood were not adjacent to the living units, and this soil was therefore not the primary source of lead in house dust. Evidence for this statement includes the observation that street dust lead concentrations are much higher than soil concentrations, indicating there is a large source of lead contributing to street dust in addition to soil lead.

2. The preabatement median soil lead concentrations in the three treatment groups were about 300 μg/g in Pendleton, 700 μg/g in Findlay, and 800 μg/g in Dandridge, and the postabatement soil concentrations were less than 100 μg/g, so that the reduction of lead in soil was small, as in Baltimore.

Evidence for the impact of dust abatement or dust and soil abatement consists of a statistically significant difference between changes in blood lead between Rounds 1 and 4, approximately one year apart. Some Cincinnati neighborhoods showed decreased blood lead concentrations in response to dust abatement or dust and soil abatement. The two neighborhoods that received only interior dust abatement in the first year, Dandridge and Findlay, showed a small decrease in blood lead concentrations, compared to large increases in the nearest control group, Mohawk. The treatment group that received soil, exterior dust and interior dust abatement, Pendleton, showed a smaller effect than did the Dandridge and Findlay neighborhoods. After consultation with the Cincinnati research team, we suspect that there was recontamination of street dust in Pendleton during the study, probably caused by demolition of nearby buildings in the neighborhood.

The consistent theme across the outcomes for all three studies is that soil abatement must be both effective and persistent in markedly reducing soil lead concentrations accompanied by a corresponding reduction in house dust lead in order to result in any detectable reduction of blood lead. The location of the soil relative to the exposure environment of the child is important. In this project, the movement of lead from soil or street dust into the home seems to be a key factor in determining blood lead concentrations. Although these USLADP results provide substantial evidence for the link between soil or street dust and house dust lead, there is insufficient information by which to clearly quantify this relationship in terms of the lowest level of soil or street dust lead reduction that will yield a measurable decrease of lead in blood.

6.2.2 Application of Findings to Conceptual Framework of Soil Lead Exposure Pathway

This integrated assessment attempts to answer the following question: If residential soil is abated will blood lead concentrations decline? To confirm or reject this soil lead/blood lead hypothesis, this report builds a framework of logical arguments described below. Each step of the pathway from soil to blood must be scrutinized closely and related data examined in detail. This means that if dust lead derived from soil is not ingested, either directly or after passing through other sources, then blood lead concentrations cannot respond to changes in soil lead concentrations.

1. There is a substantial amount of lead in soil.

   Lead was measured in soil in the range of less than 50 μg/g to more than 18,000 μg/g. If a parcel of 100 m^2 had an average of 500 μg Pb/g soil, then the upper 2 cm of soil on this parcel (about 4,000,000 g) would contain 2 billion μg or two kilograms of lead. Before abatement, there was an estimated 25,000 kilograms of soil lead on the participating properties of this project.

   A 2-cm soil core was deemed better than a 15-cm core commonly used in previous studies. When there is a decreasing gradient between the top and bottom of the 15-cm core, the effect is to dilute the concentration, giving a distorted picture of what is available at the surface. In this project, some measurements were made of the soil concentration in the bottom 2-cm of the 15-cm core in order to determine the depth of excavation. The Boston study reported there was not a large gradient between the top and bottom of the 15-cm core, as had been expected.

   Finally, there is little information on the types of surfaces that a child plays on. If these surfaces are mostly soil, as opposed to asphalt or concrete, then the soil measurement may be a good estimate of exposure. However, exterior dust is probably a better estimate of exposure from hard play surfaces (item 5 below).

Exterior dust represents lead from several sources, including soil, and may also be a better estimate of the lead transferred to household dust.

2. Lead in soil can move to other compartments of the child's environment, such as exterior dust.

   Limited evidence for this statement was shown in the Cincinnati study. In the Cincinnati study, the relationship between soil and exterior dust was found to be very weak, giving rise to the next statement.

3. There are sources of lead other than soil that contribute to exterior dust.

   Because the changes in lead in soil do not account for all of the changes in exterior dust, it is reasonable to conclude from the Cincinnati study that there are other sources for lead in exterior dust. In Cincinnati, the soil parcels were not on the individual properties of the participating families, as was the case in Boston and Baltimore. There are no measurements of exterior dust in the Boston or Baltimore studies.

4. Lead in exterior dust can also move into other components of the child's environment, such as interior dust.

   In the Cincinnati study, when exterior dust lead concentrations changed, interior dust lead concentrations also changed. This was especially obvious when the exterior dust sample closest to the residence was compared to the interior floor dust sample taken just inside the entryway door.

   A living unit with 130 m^2 of floor space (1,400 ft^2) and 1,000 μg Pb/m^2 (a relatively high value from tables in Section 3.3) would have 130,000 μg of lead, or less than 1% of the lead available from soil in paragraph 1 above (see Figure 6-1). Additional lead would be in rugs and upholstered furniture.

5. There are sources of lead other than exterior dust that contribute to interior dust.

   Taken individually, none of the studies decisively demonstrated this effect. The most obvious source of lead inside the home is lead-based paint, which was common in the Boston and Baltimore studies, but less important in the Cincinnati study. Because neither Boston nor Baltimore measured exterior dust, measurements of interior dust in these studies cannot easily be broken down into contributions from lead-based paint and from exterior dust. However, structural equation analyses on the Boston study showed a strong influence of both interior and exterior lead-based paint on interior dust.

6. Lead in soil can move directly onto the child's hand.

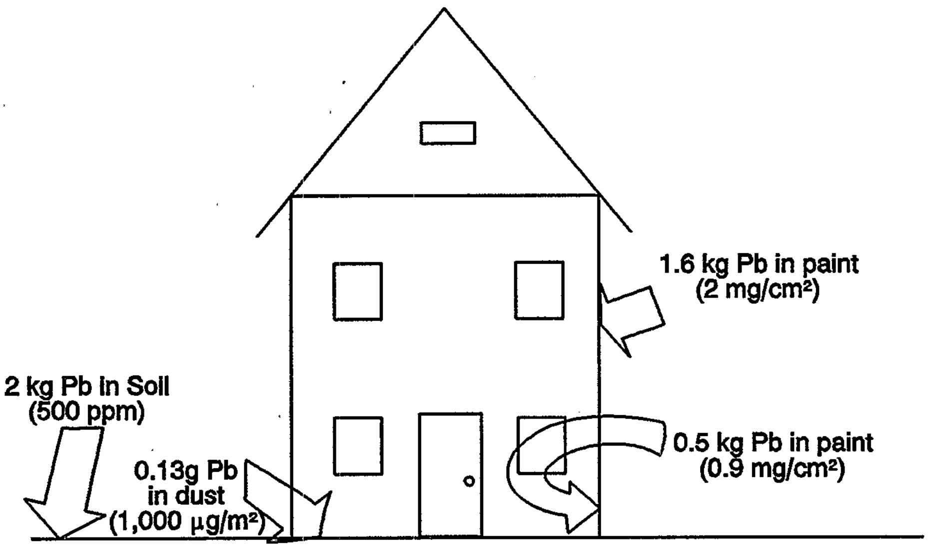

Figure 6-1. Total amounts of lead in various compartments of a child's environment, using the assumptions for concentration (soil, top 2 cm) or lead loading (dust and paint) in parentheses. Although house dust is only a small fraction of the total lead in the child's environment, it is the most accessible component. The concentrations and loadings are illustrative, not typical.

Conceptually, the transfer of lead from soil to the child's hand is difficult to measure. A child playing outside usually gets soil on his/her hands, but it is not certain whether this soil is adequately represented by a composite of 2 cm soil cores.

7. Lead in exterior dust can move directly onto the child's hand.

There is no portion of these studies that directly measures this effect. Baltimore reported that the lead loading on hands increased during the summer months, by inference due to the increased playtime outside. During the interviews with the family, questions were asked in all three studies about the activity patterns of the children, including the amount of time spent outside, but none of the studies attempted to assess the play activities immediately before the hand wipe sample was taken.

8. Lead in interior dust can move directly onto the child's hand.

In most cases, when interior dust changed, hand dust changed. Because hand dust lead is only a measure of the amount of lead on the hand, not the concentration nor the amount of dust, it is difficult to make a quantitative estimate of this pathway. It is not likely that the amount of dust on the hand is strictly a function of the amount of dust on the playing surface, as there is probably an equilibrium effect where some dust falls off after time. There is no aspect of these studies that could measure this interesting problem.

9. Lead in interior dust can also move into other components of the child's environment, such as food.

   This pathway was not investigated by any of the three studies. Measurements of lead in food before and after kitchen preparation would be required. Conceptually, this lead and other routes such as the direct mouthing activities on toys, furniture, and window sills is included in the measurement of interior dust when the assumption is made that a child ingests about 100 mg dust/day by all routes and through all activity patterns.

10. There are sources of lead other than dust that contribute to the child's lead exposure.

    In this project, lead was measured in drinking water once or twice during each study. Low ambient levels (ca. 0.1 $\mu g/m^3$) of lead in air (typical of U.S. metropolitan areas in 1990) were assumed, as were national averages of lead in food. Ethnic food preferences and individual use of cosmetics or other lead containing products were not investigated.

6.3 INTEGRATED PROJECT CONCLUSIONS

The main conclusions of this Integrated Report report are two-fold:

(1) *When soil is a significant source of lead in the child's environment, the abatement of that soil will result in a reduction in exposure that will, under certain conditions, cause a reduction in childhood blood lead concentrations.*

(2) *Although these conditions for a reduction in blood are not fully understood, it is likely that four factors are important: (1) the past history of exposure of the child to lead, as reflected in the preabatement blood lead; (2) the magnitude of the reduction in soil lead concentrations; (3) the magnitude of other sources of lead exposure, relative to soil; and (4) a direct exposure pathway between soil and the child.*

The basis for the first conclusion is: in Boston, where the soil lead concentrations were high and the contribution from lead-based paint was reduced by paint stabilization, there was a measurable reduction of blood lead concentrations. This reduction continued to increase for two years following abatement in Boston.

Conversely, in Baltimore and Cincinnati, where soil was not a significant source of lead relative to other sources, there was no measurable reduction of blood lead except in cases where those sources were also removed or abated. In Baltimore, these sources may have been interior lead-based paint that was not stabilized, or house dust that was not abated. In Cincinnati, the principle source of lead seemed to be neighborhood dust that may have been contaminated with lead-based paint.

The basis for the second conclusion is: in those cases where all important elements of the exposure pathway were available for assessment, the structural equation model analyses showed that preabatement blood lead concentration was a major predictor of postabatement blood lead, suggesting that the remobilization of bone lead is a major component of the measured blood lead.

All other factors being equal, the measurable reduction in blood lead was observed only at higher concentrations of soil lead. In the absence of information about other sources of lead, no clear statement can be made about the possibility of smaller reductions in blood lead at lower soil lead concentrations.

In spite of the recent successes in reducing exposure to lead by removing lead from gasoline and canned food, lead exposure remains a complex issue. This integrated assessment attempts to assess exposure to lead in soil and house dust. Lead in soil and lead-based paint are closely linked in the child's environment. If there is exterior lead-based paint, then soil lead is likely to be elevated with a consequent elevation in house dust lead. If there is interior lead-based paint, then efforts to reduce the impact of soil lead on house dust will be only partially effective. The maximum reduction in lead exposure will not be achieved unless both paint and soil abatement are implemented.

There is evidence from all three studies that lead moves through the child's environment. This means that lead in soil contributes to lead in street or playground dust, lead in exterior paint contributes to lead in soil, and lead in street dust contributes to lead in house dust. A more detailed analysis of the data may show the relative contribution from two or more sources, but the present analyses imply that this transfer takes place.

The analysis of the data from the three studies showed evidence that blood lead responds to changes in house dust lead. There is also evidence for the continued impact of other, independent sources following abatement of one source. This means that abatement of soil or exterior paint does not necessarily reduce the contribution of lead from other sources such as interior lead-based paint.

The conclusions of this report suggest that soil abatement alone will have little or no effect on reducing exposure to lead unless there is a substantial amount of lead in soil and unless this soil lead is the primary source of lead in house dust. At a minimum, when implemented, both soil abatement and interior dust removal should both be performed to be fully effective. Conversely, soil abatement should be considered in conjunction with paint abatement when it is likely that soil will otherwise continue to contaminate house dust after a paint abatement is completed.

From one perspective, decisions about soil abatement should be made on an individual home basis. For an individual home, the owner or renter needs to know that the property is safe for children. This report shows that, on an individual house basis, soil abatement may reduce the movement of lead into the home and its incorporation into house dust. The magnitude of this reduction depends on the concentration of lead in the soil, the amount of soil-derived dust that moves into the home, the frequency of cleaning in the home and the cleanability of the home. The number and ages of children and the presence of indoor/outdoor pets are factors known to increase this rate of dust movement, whereas frequent cleaning with an effective vacuum cleaner, use of entry dust mats, and removing shoes at the door serve to reduce the impact of soil lead on house dust.

From another perspective, soil abatement at the neighborhood level poses problems not pertinent to individual homes. Playground, vacant lot, and other plots of soil may pose an immediate problem if they are accessible to children and there is a direct pathway for dust generated by this soil to enter the home. Likewise, sources of lead other than soil may contribute more to exterior dust than soil itself. The evidence in this report suggests that the key to reducing lead exposure at the neighborhood level is to abate significant sources of lead contributing to exterior dust, in addition to the soil and paint abatement that would be performed on an individual property.

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APPENDIX A:

GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

The data in Table A-1 were derived using the PROC UNIVARIATE feature of SAS 6.10 (SAS, 1994). The treatment groups are as described in Chapter 5, using data identical to that plotted in Figures 5-8 through 5-32. Data for blood lead concentration and hand lead are calculated with one value for each child; for floor and window dust, one value for each living unit; and for soil, one value for each property or soil parcel. The group assignments and numbers of individuals are different from the individual study reports and different also from the summaries of these reports in Chapter 4. In particular, the data are different from Tables 4-2 through 4-4.

TABLE A-1. GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Boston	Soil Pb Conc.	BOS SPI	1	35	1485	1678	2413	3367	4020	2625
	(μg/g)		2	26	83	98	125	160	190	139
			3	35	50	70	113	192	380	234
			5	21	83	100	174	284	297	206
		BOS PI	1	36	1469	1813	2477	3300	4400	2831
			3	35	1460	1480	2148	3286	3833	2502
			5	22	88	161	278	505	570	429
		BOS P	1	30	1355	1611	2268	3890	4064	2728
			3	30	1493	1572	2115	3880	4240	2679
			5	17	50	110	204	240	350	307
	Floor Dust Pb Conc.	BOS SPI	1	40	943	1000	2100	4045	8480	5797
	(μg/g)		2	30	580	700	1040	1900	4770	2111
			3	38	530	570	845	1620	2700	2803
			4	31	480	600	760	1200	2090	1120
			5	28	550	619	726	1182	1568	1239
		BOS PI	1	39	1090	1240	2240	3800	5000	3712
			2	32	670	775	1105	1810	2470	1560
			3	33	580	750	1150	1360	1700	1301
			4	31	610	750	1030	1400	1680	1189
			5	27	400	517	806	1450	2500	1192
		BOS P	1	33	920	1250	2200	3900	7460	4627
			3	32	600	700	950	1370	1900	1280
			4	29	730	990	1300	1750	2380	1646
			5	21	550	644	862	1250	1485	1041
	Floor Dust Load	BOS SPI	1	40	9.09	11.24	23.56	69.86	81.01	51.03
	(mg/m^2)		2	30	10.75	14.88	35.55	58.69	76.88	52.42
			3	38	11.16	13.33	22.78	62.00	94.59	53.14
			4	34	6.89	8.15	15.20	29.76	45.88	25.63

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Boston	Floor Dust Load		5	28	17.36	24.15	31.29	60.35	88.87	50.90
	(mg/m²)	BOS PI	1	40	9.87	10.80	24.39	41.85	66.96	38.86
			2	32	7.44	9.45	19.43	39.27	45.47	31.23
			3	33	7.75	14.57	26.29	47.12	54.56	38.31
			4	31	5.24	8.50	16.86	35.03	45.35	26.42
			5	27	16.12	17.01	31.00	50.10	54.56	37.22
		BOS P	1	33	9.92	15.25	39.68	70.68	87.63	46.97
			3	32	9.28	11.68	28.32	52.70	94.24	46.47
			4	29	7.09	9.92	19.34	38.44	50.84	29.88
			5	21	9.92	13.33	36.85	76.88	86.30	55.92
	Floor Dust Pb Load	BOS SPI	1	40	22.92	30.31	51.90	107.80	157.40	303.19
	(μg/m²)		2	30	14.38	19.44	39.85	61.92	91.35	127.43
			3	38	6.80	11.07	22.98	63.61	167.40	212.02
			4	31	3.91	6.60	15.85	26.31	71.25	33.77
			5	28	12.87	16.03	24.03	58.59	117.99	55.17
		BOS PI	1	39	16.74	31.25	58.95	179.60	208.32	102.19
			2	32	6.48	10.05	23.57	66.96	87.42	43.93
			3	33	8.63	11.78	26.66	55.39	67.65	44.54
			4	31	5.49	7.79	17.61	32.19	74.77	33.25
			5	27	13.14	15.71	28.21	56.07	67.72	39.58
		BOS P	1	33	22.59	32.92	75.04	180.54	381.42	263.23
			3	32	6.75	8.90	26.28	77.50	109.45	70.69
			4	29	5.21	11.61	21.27	66.09	107.41	59.31
			5	21	8.68	13.90	37.08	65.97	73.78	55.25
	Window Dust Pb Conc.	BOS SPI	1	41	5067	7575	13340	24733	38333	19326
	(μg/g)		2	35	2667	5260	9667	16167	21833	10911

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Boston	Window Dust Pb Conc		3	41	4040	4800	10500	19742	21667	13229
	(μg/g)		4	38	3317	4300	18896	38633	46367	23183
			5	24	2983	4587	8780	16035	21950	14336
		BOS PI	1	41	3067	7200	19670	31250	41020	22178
			2	36	900	1167	2400	4917	8367	5706
			3	37	2450	4867	10000	26100	29667	13813
			4	37	2133	5100	15650	38000	45800	25137
			5	24	2023	3322	6870	10475	19267	8844
		BOS P	1	35	2100	3867	17400	52500	60717	26452
			3	37	2350	3940	15500	24240	43667	19552
			4	37	1250	1320	12667	40000	45000	22807
			5	19	2947	4457	12350	24050	24647	14060
	Window Dust Load	BOS SPI	1	41	70	133	295	630	796	450
	(mg/m^2)		2	35	29	43	111	209	445	401
			3	41	122	249	440	707	913	592
			4	38	157	226	391	780	932	662
			5	24	228	385	919	1404	2579	1326
		BOS PI	1	41	106	159	303	522	757	624
			2	36	17	22	31	158	177	114
			3	37	126	227	380	712	1174	583
			4	37	161	262	570	1095	1516	785
			5	24	92	155	500	766	993	556
		BOS P	1	35	74	142	239	444	629	494
			3	37	83	135	239	595	949	762
			4	37	91	239	504	990	1957	834
			5	19	169	185	797	976	1279	829

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
	Window Dust Pb Load	BOS SPI	1	41	1250	1694	6656	14228	17446	9668
	($\mu g/m^2$)		2	35	176	270	1374	3479	7854	6084
			3	41	910	1100	4535	8151	11601	6253
			4	38	1641	2554	5626	11102	16950	11097
			5	24	1252	1777	5402	20982	26748	14425
Boston	Window Dust Pb Load	BOS PI	1	41	907	1578	7196	14746	21577	23227
	($\mu g/m^2$)		2	36	14	36	88	691	1313	881
			3	37	483	1438	4624	11549	15319	8091
			4	37	1205	2664	5697	13404	36373	15279
			5	24	701	1089	2553	6092	9175	5654
		BOS P	1	35	244	1123	4179	17878	24890	31055
			3	37	445	1130	4441	12220	13986	10552
			4	37	162	2521	5559	16338	32017	11635
			5	19	1569	1638	6018	28169	30796	12671
	Hand Pb Load	BOS SPI	1	54	9.4	11.00	13.00	17.00	17	14.97
	(μg/pair)		2	54	8.2	10.00	12.50	17.00	20	14.52
			3	53	8.8	13.00	17.00	21.00	23	18.06
			5	33	11.0	16.00	22.00	31.00	29	24.82
		BOS PI	1	51	10	11.00	13.00	15.00	20	13.97
			2	49	9	11.00	14.00	17.00	19	14.44
			3	46	12	9.30	15.50	20.00	29	18.10
			5	32	15	13.00	19.50	25.50	29	21.20
		BOS P	1	47	10	10.00	12.00	17.00	22	14.88
			2	46	9.1	9.80	12.00	18.00	20	16.18
			3	46	9.7	14.00	18.00	26.00	24	21.99
			5	26	9.7	16.00	20.00	26.00	37	22.64

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Boston	Blood Pb Conc	BOS SPI	1	54	8	10.00	13.00	16.00	17	13.19
	(μg/dL)		2	54	5	6.00	10.00	13.00	13	10.31
			3	54	8	7.00	10.00	14.00	16	11.70
			5	33	5	5.00	10.00	13.00	11	10.88
		BOS PI	1	51	8	9.00	12.00	15.00	17	12.37
			2	48	6	6.00	8.00	12.00	14	8.85
			3	49	7	9.00	11.00	14.00	15	11.49
			5	32	5	5.50	8.00	10.00	13	7.89
		BOS P	1	47	8	9.00	12.00	14.00	18	12.02
			2	46	6	8.00	9.00	12.00	16	9.83
			3	46	6	8.00	11.50	14.00	16	11.35
			5	26	4	6.00	10.00	13.00	17	9.96
Cincinnati	Soil Pb Conc.	CIN SEI (P)	1	112	52	90	314	1368	2172	1139
	(μg/g)		2	104	0	0	0	102	216	190
			3	104	19	21	32	74	159	152
			4	100	24	28	48	90	179	161
			5	100	26	29	44	129	215	188
			6	101	26	28	48	140	343	227
			7	103	26	29	43	134	223	192
		CIN I-SE (B)	1	26	29	49	102	123	141	141
			2	26	52	57	100	132	167	135
			3	26	61	67	107	151	166	176
			4	26	70	72	114	145	151	318
			5	26	46	57	74	107	109	77
			6	26	38	47	60	90	104	68
			7	26	40	42	64	88	106	79
		CIN I-SE (D)	1	-	-	-	-	-	-	-
			2	92	181	252	871	1794	2548	1312
			3	88	178	262	767	1610	2055	1111

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Soil Pb Conc		4	86	178	353	883	1637	2217	1247
	(μg/g)		5	84	17	20	34	178	899	404
			6	82	22	26	47	180	946	384
			7	88	23	28	78	534	1152	584
		CIN I-SE (F)	1	46	64	120	699	1633	2338	1201
			2	48	72	143	874	2001	2463	1444
			3	49	87	124	338	1333	2064	939
			4	48	85	106	436	1276	1670	936
			5	48	18	24	42	696	1347	588
			6	48	20	22	81	1616	2940	1068
			7	47	30	37	58	820	1415	960
		CIN NT (G)	1	118	0	9	79	254	411	203
			2	120	0	20	132	308	473	232
			3	120	36	52	142	354	598	659
			4	119	31	47	112	207	263	215
			5	120	39	50	114	248	399	221
			6	119	37	52	125	232	344	206
			7	121	30	44	128	227	360	195
		CIN NT (M)	1	44	60	115	401	1356	1986	930
			2	55	80	160	732	1582	1728	1165
			3	49	82	126	388	914	1288	752
			4	49	90	125	318	585	1124	603
			5	48	104	126	402	774	1122	705
			6	47	98	118	417	975	1082	577
			7	48	72	151	478	989	1182	609
	Floor Dust Pb Conc.	CIN SEI (P)	1	30	196	245	364	606	799	561
	(μg/g)		2	30	222	237	359	581	675	459
			3	30	193	208	325	516	622	459
			4	25	220	284	474	604	906	612

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Floor Dust Pb Conc		5	24	0	0	0	0	0	0
	(μg/g)		6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (B)	1	10	117	172	308	506	544	592
			2	9	250	348	400	495	1650	645
			3	8	302	322	477	633	633	465
			4	3	398	398	402	523	523	441
			5	1	0	0	0	0	0	0
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (D)	1	23	234	331	411	529	613	438
			2	22	243	315	415	746	820	493
			3	23	191	248	411	570	618	444
			4	25	290	381	494	741	850	576
			5	21	0	0	0	0	0	0
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (F)	1	23	245	271	466	827	1224	890
			2	23	242	333	450	1282	1735	902
			3	22	265	288	389	635	751	522
			4	29	255	317	389	950	1182	1115
			5	22	0	0	0	0	0	0
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (G)	1	31	92	124	186	330	472	267
			2	28	125	150	207	272	329	250
			3	29	124	144	183	257	300	214
			4	41	109	121	179	247	350	222
			5	35	0	0	0	0	0	0
			6	-	-	-	-	-	-	-

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Floor Dust Pb Conc		7	-	-	-	-	-	-	-
	($\mu g/g$)	CIN NT (M)	1	9	272	314	389	459	760	587
			2	9	211	338	558	694	719	589
			3	6	183	209	368	481	548	360
			4	14	217	253	430	655	766	775
			5	15	0	0	0	0	0	0
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
	Floor Dust Load	CIN SEI (P)	1	30	88	106	380	2248	4833	1714
	(mg/m^2)		2	30	36	53	136	397	1428	942
			3	30	83	93	135	307	358	285
			4	25	62	84	197	796	2123	784
			5	-	-	-	-	-	-	-
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (B)	1	10	45	57	127	1274	1433	1869
			2	9	11	22	31	53	103	45
			3	8	21	39	79	96	106	86
			4	3	85	85	137	266	266	163
			5	-	-	-	-	-	-	-
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (D)	1	23	72	80	231	573	1269	791
			2	22	22	24	36	80	178	296
			3	23	47	86	119	263	331	205
			4	25	203	304	775	1745	3752	3289
			5	-	-	-	-	-	-	-
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Floor Dust Load	CIN I-SE (F)	1	23	87	100	207	1140	3837	2839
	(mg/m²)		2	23	21	31	52	103	122	116
			3	22	68	77	195	257	301	177
			4	29	154	293	420	623	986	704
			5	-	-	-	-	-	-	-
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (G)	1	31	45	60	138	532	911	501
			2	28	49	79	138	209	275	327
			3	29	88	108	152	223	309	202
			4	41	67	102	196	345	646	499
			5	-	-	-	-	-	-	-
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (M)	1	9	86	105	207	319	332	258
			2	9	56	89	115	139	143	151
			3	6	14	98	258	333	438	233
			4	14	111	186	319	1161	1887	1102
			5	-	-	-	-	-	-	-
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
	Floor Dust Pb Load	CIN SEI (P)	1	30	20	32	167	1164	1633	861
	(μg/m²)		2	31	9	14	77	213	317	629
			3	30	25	27	54	100	128	179
			4	25	13	33	130	519	916	669
			5	24	9	34	76	263	576	320
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Floor Dust Pb Load	CIN I-SE (B)	1	10	12	12	40	414	652	4957
	($\mu g/m^2$)		2	9	4	5	9	26	170	46
			3	8	9	10	40	58	61	37
			4	3	34	34	55	139	139	76
			5	1	19	19	19	19	19	19
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (D)	1	23	28	30	122	206	521	280
			2	22	6	8	18	36	53	37
			3	23	20	27	69	107	115	93
			4	25	115	144	296	1264	1568	1599
			5	21	25	48	148	540	744	431
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (F)	1	23	21	43	123	788	3000	5711
			2	23	9	12	28	64	179	101
			3	22	22	36	74	140	156	94
			4	29	60	111	175	542	938	657
			5	23	10	16	86	206	624	202
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (G)	1	31	8	15	35	80	195	139
			2	28	12	14	29	64	77	77
			3	29	15	18	31	59	75	42
			4	41	10	15	22	85	231	118
			5	30	21	26	69	132	219	140
			6	-	-	-	-	-	-	-

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Floor Dust Pb Load		7	-	-	-	-	-	-	-
	($\mu g/m^2$)	CIN NT (M)	1	9	27	48	88	128	140	245
			2	9	20	25	59	80	80	142
			3	7	3	3	53	160	160	89
			4	14	79	80	127	509	1000	512
			5	14	47	52	140	435	594	282
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
	Window Dust Pb Conc.	CIN SEI (P)	1	30	503	724	1541	2652	3600	2755
	($\mu g/g$)		2	28	457	653	1299	1763	3121	2990
			3	28	536	670	922	1556	1905	1201
			4	25	790	980	1920	3348	6476	4134
			5	24	209	244	502	696	758	752
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (B)	1	10	721	2353	2782	3868	6527	4085
			2	8	515	598	800	1212	1321	2776
			3	8	469	574	1159	2728	3713	1682
			4	3	807	807	1069	3320	3320	1732
			5	1	1111	1111	1111	1111	1111	1111
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (D)	1	23	879	1083	1822	2515	3190	2643
			2	18	695	1098	1954	2491	3077	1965
			3	23	628	1019	1308	2209	2213	1508
			4	25	1208	1367	1958	2741	2930	7127
			5	21	408	534	752	1295	1306	1304
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Window Dust Pb Conc	CIN I-SE (F)	1	21	507	1193	2000	2733	15737	7355
	($\mu g/g$)		2	23	765	1156	2102	8224	14565	7433
			3	22	534	630	1432	2676	4688	2541
			4	29	1193	1345	2379	3299	7291	4277
			5	23	241	270	491	1039	1136	851
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (G)	1	31	311	506	907	2206	3235	2136
			2	29	321	481	731	1435	2463	1337
			3	28	151	208	484	1208	1886	864
			4	41	411	539	1084	2081	2517	1484
			5	29	169	187	274	424	503	332
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (M)	1	9	378	913	1355	1889	4200	3611
			2	8	310	637	1456	4932	8253	5825
			3	7	290	290	638	1703	1703	1503
			4	15	1978	2043	3824	14505	17177	7361
			5	15	423	426	679	982	1118	1259
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
	Window Dust Load	CIN SEI (P)	1	30	52	137	729	3479	9217	10396
	($\mu g/m^2$)		2	28	96	112	443	1083	1563	3396
			3	28	80	110	254	507	675	531
			4	25	192	663	4524	21259	34180	20554
			5	24	553	613	966	1389	1699	1092
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Window Dust Load	CIN I-SE (B)	1	10	177	330	517	6659	8967	4436
	(mg/m^2)		2	8	61	62	222	830	1132	636
			3	8	68	100	179	630	702	355
			4	3	225	225	1514	34180	34180	11972
			5	1	164	164	164	164	164	164
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (D)	1	23	316	544	1831	6146	14201	6147
			2	18	56	113	327	1230	9390	2719
			3	23	73	115	257	790	1200	798
			4	25	898	3574	7623	17658	34994	18089
			5	21	266	399	697	979	1189	1334
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (F)	1	21	113	178	1139	4381	13300	14711
			2	23	120	205	397	3748	5530	4223
			3	22	89	111	239	472	1203	1071
			4	29	935	4231	9632	17374	29250	15903
			5	23	248	329	649	977	1216	720
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (G)	1	31	209	340	2621	5292	9615	4473
			2	29	89	316	2733	11895	20524	16777
			3	28	189	212	311	1040	1909	809
			4	41	1544	2767	8200	16956	31488	19333
			5	28	404	483	711	1198	1540	897
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Window Dust Load	CIN NT (M)	1	9	349	409	1151	1412	2170	1660
	(mg/m^2)		2	8	66	80	474	4270	4298	1894
			3	7	200	200	405	681	681	418
			4	15	971	2164	3863	13530	19250	8526
			5	15	329	451	704	1139	1176	783
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
	Window Dust Pb Load	CIN SEI (P)	1	30	39	100	1075	9583	14599	34966
	(μg/m^2)		2	31	48	80	484	1606	5074	67217
			3	28	83	148	242	377	1156	605
			4	25	285	383	15385	45405	86113	53241
			5	24	221	264	397	442	543	456
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (B)	1	10	341	488	1198	25000	25758	44274
			2	9	32	50	189	360	438	5335
			3	8	36	62	146	1646	2067	973
			4	3	747	747	1222	36538	36538	12836
			5	1	182	182	182	182	182	182
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (D)	1	23	731	869	3034	10578	19431	51617
			2	22	37	60	194	3250	3600	5003
			3	23	67	130	350	1033	1802	1398
			4	25	1959	4115	14306	41818	109286	251438
			5	21	261	288	410	722	893	1112
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (F)	1	21	87	309	2600	13871	54217	368809

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Window Dust Pb Load		2	23	182	354	1129	12471	25465	13976
	($\mu g/m^2$)		3	22	128	173	352	1065	2029	2810
			4	29	1588	9773	29078	80000	101136	77220
			5	23	213	241	329	398	458	353
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (G)	1	31	69	262	2065	8929	22083	11940
			2	29	121	215	1200	12000	47188	27281
			3	29	49	63	125	804	1095	1009
			4	41	818	2128	13676	29215	41616	26389
			5	29	132	140	215	248	307	239
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (M)	1	9	228	435	1718	1981	7308	3172
			2	9	34	64	357	6929	8647	6026
			3	7	58	58	358	689	689	673
			4	15	2743	4457	30256	83482	121053	61825
			5	15	242	274	378	716	746	504
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
	Entry Dust Pb Conc.	CIN SEI (P)	1	29	132	0	222	382	862	348
	($\mu g/g$)		2	31	380	429	661	950	1230	1110
			3	30	204	256	433	810	892	556
			4	24	118	263	491	867	1404	755
			5	24	25	44	211	1115	5357	1567
			6	22	201	207	382	584	764	440
			7	17	148	262	488	824	863	819
		CIN I-SE (B)	1	10	185	185	296	421	755	329

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Entry Dust Pb Conc		2	9	247	410	467	504	2370	849
	(μg/g)		3	8	335	363	442	560	569	508
			4	3	45	45	425	576	576	349
			5	1	56	56	56	56	56	56
			6	2	478	478	698	918	918	698
			7	1	277	277	277	277	277	277
		CIN I-SE (D)	1	23	270	331	494	752	1104	622
			2	22	353	381	631	1006	1140	690
			3	23	309	351	685	1034	1513	869
			4	25	472	586	825	1003	1998	1244
			5	21	72	94	183	656	1083	1355
			6	21	308	327	652	1199	1360	905
			7	18	290	392	703	892	975	730
		CIN I-SE (F)	1	22	239	161	351	800	1176	669
			2	22	364	462	626	1000	1452	1150
			3	22	229	268	407	749	966	1948
			4	29	222	301	387	808	1123	739
			5	23	19	21	67	117	1625	1232
			6	24	220	268	424	1164	1444	844
			7	18	215	381	475	773	850	550
		CIN NT (G)	1	30	86	117	217	311	444	284
			2	27	181	189	303	455	546	446
			3	29	192	220	307	359	474	329
			4	39	150	180	293	486	577	414
			5	29	18	28	56	278	450	602
			6	35	138	192	307	466	526	401
			7	31	203	221	268	439	466	335
		CIN NT (M)	1	9	332	332	419	597	1471	1740

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Entry Dust Pb Conc		2	9	389	404	573	805	1375	1046
	(μg/g)		3	7	167	167	434	791	791	534
			4	15	326	452	877	3873	13000	3938
			5	15	68	100	352	945	3130	7820
			6	12	257	326	575	1413	2063	976
			7	9	283	478	637	1698	4855	1714
	Entry Dust Load	CIN SEI (P)	1	21	108	119	154	723	98101	2386
	(mg/m^2)		2	31	35	48	114	2601	8344	6479
			3	30	112	145	230	837	1425	4920
			4	24	259	407	590	4060	26992	8088
			5	24	42	3118	12671	63462	92160	40218
			6	22	62	64	97	344	426	314
			7	17	105	143	301	1183	5020	2250
		CIN I-SE (B)	1	9	49	118	272	278	1543	396
			2	9	29	36	49	79	323	1009
			3	8	55	80	284	357	371	258
			4	3	249	249	1156	42535	42535	14647
			5	1	48214	48214	48214	48214	48214	48214
			6	2	115	115	139	163	163	139
			7	1	260	260	260	260	260	260
		CIN I-SE (D)	1	22	56	88	375	863	1024	745
			2	22	31	39	59	125	144	159
			3	23	62	69	192	362	570	723
			4	25	377	419	2591	6266	14322	8989
			5	21	2493	3512	12796	27000	37500	21517
			6	21	83	93	179	339	622	1081

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Entry Dust Load		7	18	214	261	534	1628	4160	1741
	(mg/m^2)	CIN I-SE (F)	1	19	81	99	150	522	5537	1205
			2	22	38	63	116	203	285	169
			3	22	48	68	107	291	622	574
			4	29	130	244	913	4371	7605	9648
			5	23	359	11066	40299	128571	142105	62379
			6	24	59	80	182	926	2191	891
			7	18	109	199	632	3059	4856	3335
		CIN NT (G)	1	27	60	93	267	963	2462	647
			2	27	141	193	244	604	723	2002
			3	29	159	2112	296	447	500	341
			4	39	165	236	435	3007	8824	34584
			5	29	3541	7521	34364	93103	150000	64155
			6	35	53	75	165	369	1789	3306
			7	31	190	367	952	1931	3388	26981
		CIN NT (M)	1	8	54	235	495	1296	3642	965
			2	9	51	56	223	379	415	506
			3	7	65	65	223	299	299	233
			4	15	105	197	1341	7889	11616	4899
			5	15	424	660	4265	13745	27000	14109
			6	12	35	61	102	440	2989	662
			7	9	523	1020	1616	5417	14591	5298
	Entry Dust Pb Load	CIN SEI (P)	1	30	8	20	116	6958	31000	17480
	($\mu g/m^2$)		2	31	19	24	112	1375	14167	8629
			3	30	30	42	167	272	1163	2944
			4	25	38	139	250	2267	8434	11323

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Entry Dust Pb Load		5	24	324	956	2502	2700	2700	1930
	($\mu g/m^2$)		6	22	13	21	57	122	217	151
			7	17	27	34	150	900	2450	3619
		CIN I-SE (B)	1	10	9	40	82	489	520	885853
			2	9	12	12	18	96	163	2263
			3	8	41	52	91	200	211	116
			4	3	52	52	106	24500	24500	8219
			5	1	2700	2700	2700	2700	2700	2700
			6	2	78	78	92	106	106	20
			7	1	72	72	72	72	72	72
		CIN I-SE (D)	1	22	24	29	222	636	859	512
			2	22	19	20	40	55	74	154
			3	23	30	56	176	245	270	542
			4	25	346	356	2000	5000	8401	17034
			5	21	1908	2304	2700	2700	2700	2314
			6	21	43	61	117	317	350	705
			7	18	78	189	376	572	2750	2310
		CIN I-SE (F)	1	23	16	30	145	2358	4688	12803
			2	23	19	28	87	216	248	252
			3	22	16	19	44	203	1600	509
			4	29	76	101	306	2388	7222	10163
			5	23	584	1512	2700	2700	2700	2086
			6	24	29	44	125	534	900	664
			7	18	30	56	214	1850	4271	2482
		CIN NT (G)	1	31	13	16	71	303	478	1140
			2	28	30	42	82	217	510	509

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Entry Dust Pb Load		3	29	40	72	88	109	233	116
	($\mu g/m^2$)		4	40	33	49	154	952	3000	29473
			5	35	1512	1512	2700	2700	2700	2089
			6	35	13	16	46	209	495	1202
			7	31	41	117	240	636	1500	15971
		CIN NT (M)	1	9	12	248	384	1209	7000	25158
			2	9	36	36	73	176	1848	731
			3	7	44	44	80	176	176	99
			4	15	41	57	4444	12556	13433	26391
			5	15	290	450	1512	1875	2304	1404
			6	12	21	48	83	208	1300	436
			7	9	127	545	1533	2744	86000	21575
	Street Dust Pb Conc.	CIN SEI (P)	1	105	521	661	1286	2764	4127	2319
	($\mu g/g$)		2	85	515	757	1182	2024	2839	1900
			3	75	326	458	647	988	1526	1097
			4	66	453	684	994	2900	3603	1836
			5	89	601	749	1294	3171	3756	2386
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (B)	1	47	554	728	1407	1878	2275	1452
			2	47	334	643	1001	1656	1790	1172
			3	35	387	535	978	1331	1688	1927
			4	37	509	893	1298	1709	3191	1933
			5	42	758	955	1499	1966	2184	1836
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Street Dust Pb Conc	CIN I-SE (D)	1	20	635	680	1262	5384	5768	2782
	(μg/g)		2	22	566	696	1457	2056	2164	1488
			3	18	331	405	1011	1616	1956	1178
			4	19	439	461	1207	1766	1982	1491
			5	21	671	708	1024	4973	5245	2982
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (F)	1	33	1147	1283	1681	2553	4085	2371
			2	42	662	835	1274	2123	3631	2948
			3	34	525	715	1273	2406	5207	2239
			4	41	809	876	1520	3491	4832	2741
			5	42	1058	1197	2055	4606	6088	3793
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (G)	1	15	75	118	212	343	348	238
			2	27	43	131	263	389	452	276
			3	14	117	132	229	325	352	261
			4	17	165	192	283	337	397	288
			5	14	84	86	162	357	367	234
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (M)	1	33	793	964	1217	1903	2138	1480
			2	35	520	512	584	785	1930	670
			3	35	473	512	584	785	865	670
			4	35	542	615	1157	1433	1801	1258
			5	35	562	655	884	1671	1908	1494

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Street Dust Pb Conc		6	-	-	-	-	-	-	-
	(μg/g)		7	-	-	-	-	-	-	-
	Sidewalk Dust Pb Conc.	CIN SEI (P)	1	84	788	1007	1809	4862	7565	3999
	(μg/g)		2	60	923	1240	2004	4622	8408	3748
			3	49	464	575	1478	2730	5307	3519
			4	48	773	930	1910	5779	7581	4150
			5	74	885	1078	2139	6310	8493	4534
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (B)	1	61	765	1375	2376	4093	5777	3448
			2	44	745	1055	2330	4820	5983	3599
			3	36	790	1163	2691	4928	6171	3882
			4	37	808	1167	1646	5130	9525	3853
			5	45	887	1066	1899	3677	5232	3261
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (D)	1	19	776	1037	1801	3087	4128	3899
			2	24	748	1228	2060	4577	5758	3577
			3	22	449	484	1294	5050	5325	2961
			4	20	669	794	2090	4738	5720	3413
			5	30	394	516	1696	3929	5890	3155
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (F)	1	38	1471	1760	4456	9915	11342	6318
			2	45	1065	1420	3941	6215	7611	4727
			3	33	961	1284	3103	5587	9846	4839

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Sidewalk Dust Pb Conc		4	35	1177	1466	3365	9892	12393	6442
	(µg/g)		5	42	1307	1777	3125	5371	7334	4505
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (G)	1	27	114	152	304	486	823	399
			2	41	101	158	297	541	631	390
			3	27	165	227	304	482	535	343
			4	25	146	178	315	369	490	314
			5	23	101	121	233	425	511	272
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (M)	1	37	495	938	1377	2539	3636	2310
			2	41	693	856	1410	4441	5683	2947
			3	36	533	686	1203	1999	2634	1886
			4	31	631	761	1101	4924	7706	3219
			5	34	477	564	1199	2714	2967	2448
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
	Hand Wipe Pb Load	CIN SEI (P)	1	51	4	5.00	6.00	11.00	14	12.14
	(µg/pair)		2	51	2	3.00	6.00	10.00	14	8.37
			3	35	2	3.00	5.00	8.00	10	5.94
			4	37	4	7.00	12.00	21.00	28	17.73
			5	30	4	7.00	12.50	19.00	24	16.03
		CIN I-SE (B)	1	24	4	5.00	17.50	33.00	43	24.17
			2	16	4	7.50	12.00	38.00	45	21.81
			3	11	4	4.00	7.00	10.00	11	6.45

TABLE A-1 (cont'd). GROUP MEAN PARAMETERS FOR EACH STUDY BY SAMPLE TYPE, TREATMENT GROUP, AND ROUND

	Sample Type	Treatment Group	Round	N	16 PCTL	Q1	Median (Q2)	Q3	84 PCTL	Arithmetic Mean
Cincinnati	Hand Wipe Pb Load		4	5	2	2.00	2.00	5.00	6	3.40
	(μg/pair)		5	4	3	3.00	3.00	3.00	3	3.00
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (D)	1	43	4	5.00	7.00	15.00	34	14.09
			2	43	3	3.00	7.00	14.00	24	11.19
			3	32	2	2.00	5.00	12.50	15	7.13
			4	41	5	8.00	12.00	29.00	35	18.93
			5	34	4	4.00	9.50	14.00	21	11.29
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN I-SE (F)	1	30	2	4.00	6.50	13.00	19	11.57
			2	33	3	4.00	6.00	11.00	13	7.79
			3	30	2	3.00	5.50	9.00	11	7.67
			4	48	2	5.50	9.00	19.50	26	12.54
			5	34	4	6.00	8.00	16.00	29	13.97
			6	-	-	-	-	-	-	-
			7	-	-	-	-	-	-	-
		CIN NT (G)	1	46	1	1.00	3.00	5.00	6	4.74
			2	48	0	1.00	2.50	5.00	6	3.40
			3	34	0	1.00	3.00	4.00	5	2.88
			4	58	-1	0	3.50	7.00	10	4.52
			5	46	2	2.00	5.00	7.00	10	5.91
			6	-	-	-	-	-	1	-
			7	-	-	-	-	-	1	-
		CIN NT (M)	1	10	1	2.00	7.00	18.00	20	10.60