September 27, 1996

RISK ASSESSMENT FOR THE SECTION 403 RULEMAKING

Draft Report

VOLUME I
CHAPTERS 1 TO 8

DO NOT CITE OR QUOTE

Prepared

by

Battelle
505 King Avenue
Columbus, Ohio 43201

for

Chemical Management Division
Office of Pollution Prevention and Toxics
Office of Prevention, Pesticides, and Toxic Substances
U.S. Environmental Protection Agency
Washington, D.C. 20460

DISCLAIMER

The material in this document has not been subject to Agency technical and policy review. Views expressed by the authors are their own and do not necessarily reflect those of the U.S. Environmental Protection Agency. Mention of trade names, products, or services does not convey, and should not be interpreted as conveying, official EPA approval, endorsement, or recommendation. Do not quote or cite this document.

This report is copied on recycled paper.

TABLE OF CONTENTS

TABLE OF CONTENTS
(Continued)

TABLE OF CONTENTS
(Continued)

TABLE OF CONTENTS
(Continued)

LIST OF TABLES

TABLE OF CONTENTS
(Continued)

Page

TABLE OF CONTENTS
(Continued)

Page

TABLE OF CONTENTS
(Continued)

TABLE OF CONTENTS
(Continued)

Page

LIST OF FIGURES

TABLE OF CONTENTS
(Continued)

Page

1.0 INTRODUCTION, BACKGROUND, AND OVERVIEW

CHAPTER 1 SUMMARY

This introductory chapter provides the background, purpose, and objectives of this report. §403 of Title IV, as amended in Title X, requires EPA to define standards for lead-based paint hazards, lead-contaminated dust, and lead-contaminated soil. This report

- *documents the scientific basis for §403.*
- *characterizes the health risks to young children from exposures to lead*
- *estimates the reductions in childhood health risks and blood-lead concentrations expected to results from various options for §403, and*
- *estimates the numbers of children and housing units affected by various options for §403.*

This information is provided to help the risk managers evaluate and compare various regulatory options for §403.

Title X of the Housing and Community Development Act, known as the Residential Lead-Based Paint Hazard Reduction Act of 1992, contains legislation designed to evaluate and reduce exposures to lead in paint, dust, and soil in the nation's housing. This act provides the framework for developing a national strategy for reducing and preventing lead exposures to children. Consistent implementation of this strategy by federal, state, local and private agencies requires a uniform definition of lead hazards. Title X includes legislation that requires the U.S. Environmental Protection Agency (EPA) to define standards for lead in paint, dust, and soil. More specifically, §403 of Title IV of the Toxic Substances Control Act (TSCA), as amended in Title X, requires EPA to "promulgate regulations which shall identify, for purposes of this title and the Residential Lead-Based Paint Hazard Reduction Act of 1992, lead-based paint hazards, lead-contaminated dust, and lead-contaminated soil."

§403 will set standards (condition and location of lead-based paint, levels of lead in dust and soil) against which to compare a residential environment when evaluating the presence and magnitude of lead-based paint hazards. Federal, state, local, and private agencies will use these standards to determine which homes require actions be taken to reduce or prevent the threat of

childhood lead poisoning. Following the conduct of these actions health risks associated with childhood lead poisoning will be reduced for children currently residing in the residence as well as those that may later live in the residence. Proper selection of the standards requires both an understanding of the health risks associated residential exposures to lead, the amount by which these risks can be reduced through interventions and the numbers of homes and children affected by the standards.

This report was prepared to support the §403 rulemaking, and to help EPA to document the scientific basis for §403. In addition, the report provides information to the risk managers on the relative efficacy of various options for the §403 standards for reducing the health risks associated with lead exposures. First, the report summarizes EPA's assessment of the health risks to young children from exposures to lead-based paint hazards, lead-contaminated dust, and lead-contaminated soil in the nation's housing. Second, the report documents the approach developed by EPA to estimate the reductions in these risks following implementation of §403, and applies this methodology to evaluate several regulatory options. The benefits of a regulatory option for §403 are expressed in terms of the reduction in health risks attained from the passage of an option for the §403 standards. Finally, the report provides estimates of the numbers of homes and children that will be affected by various options for the standards.

Information presented in this Risk Assessment will ultimately be used as input to the Regulatory Impacts Analysis (RIA) for the proposed rule, as well as any interim cost-benefit analyses. While the Risk Assessment quantifies the impact of §403 in terms of health risks and blood-lead levels and documents the scientific basis for the rule, the RIA expresses the impact of §403 in terms of costs: monetary costs of implementing the regulation, monetary benefits associated with reductions in health risks and blood lead concentrations for various options for the regulation, and the estimated economic impacts of the regulations. The RIA also examines the likelihood of interventions actually taking place. Finally, the RIA summarizes other regulatory actions designed to reduce risks from lead, and presents environmental equity analyses for adults and children.

This report documents the critical decisions on risk-assessment-related tools and data that are being relied upon in the RIA analyses, which relate environmental levels of lead to children's blood-lead levels and, ultimately, potential health effects. This report includes a description of

the data used, an assessment of the strengths and weaknesses of that data, and discussions of any additional uncertainties which result from using these particular data sets and tools to create estimates of risk reduction on a national basis. Section 1.1 discusses the statutory requirements of §403 and EPA's response to the statutory requirements. The objectives of the risk assessment are presented in Section 1.2. Summaries of each component of the risk assessment are given in Section 1.3. Section 1.4 provides an overview of the methodology utilized to conduct the integrated risk assessment. Finally, Section 1.5 describes the major data sources and analysis tools employed in each component of the risk assessment along with a discussion of their limitations.

1.1 REQUIREMENTS OF §403

1.1.1 Statutory Requirements

On October 29, 1992, the United States Congress enacted the Residential Lead-Based Paint Hazard Reduction Act (Title X of HR 5334). This includes an amendment to the Toxic Substances Control Act (Title IV: Lead Exposure Reduction) that requires the EPA Administrator to identify lead-based paint hazards. Specifically, §403 of TSCA Title IV states:

> "The Administrator shall promulgate regulations which shall identify, for purposes of this title and the Residential Lead-Based Paint Hazard Reduction Act of 1992, lead-based paint hazards, lead-contaminated dust, and lead-contaminated soil."

This statute requires EPA to define standards for lead in dust and soil and to define what constitutes a lead-based paint hazard. The statute defines lead-based paint to be dried paint film with a lead content exceeding 1.0 mg/cm^2 or 0.5 percent (5,000 parts per million (ppm)) by weight. The statute requires EPA to identify lead-based paint hazards, i.e., the condition, location and amount of lead-based paint that causes exposures to lead in paint, lead-contaminated dust and lead-contaminated soil that would result in adverse health risks.

The Title X statute provides definitions for lead-based paint, lead-based paint hazard, lead-contaminated soil, lead-contaminated dust and other relevant terms. Definitions given in the statute and utilized in the risk assessment are provided in the glossary in Appendix A.

1.1.2 EPA's Response to Statutory Requirements

The Agency's approach to the §403 requirements is to establish quantitative standards for levels of lead in residential soil and dust. For soil, the standard will be defined in units of mass of lead per mass of soil (μg Pb/g soil). A tiered standard was utilized for the soil standard specified in the Interim 403 Guidance (EPA, 1994) and the Agency has decided to continue to use a two tiered standard for soil. The lower level recognizes levels of lead in soil that may be of a health concern and thereby warrant interim controls to reduce this concern. Because of the limited data available on the efficacy of interim controls for soil, the Agency felt more extreme actions may be required for high levels of lead in soil. The higher level for the soil standard identifies soil levels that present a larger health concern and require more intensive action to eliminate this concern, such as soil abatement.

There are two methods for measuring the amount of lead in dust. Dust-lead loading measures the mass lead collected per surface area sampled and is usually expressed in terms of micrograms of lead collected per square foot sampled (μg Pb/ft^2). Dust-lead concentration measures the mass of lead collected per mass of dust collected and is usually stated in terms of micrograms of lead collected per gram of dust collected (μg Pb/g dust). Both are useful measures for evaluating exposures to lead in dust. Dust-lead loading measures the amount of lead available to the child and dust-lead concentration measures the source strength of lead in the dust. A high dust-lead loading might represent a surface containing a large amount of dust at a low lead concentration or a surface containing a small amount of dust at a high lead concentration. Both measures have been used to predict blood-lead concentrations and, there is currently no consensus on which may be the better predictor.

There are two approaches for collecting samples of dust from a surface: wipe and vacuum sampling. Although dust-lead loadings can be measured using both wipe or vacuum sampling, dust-lead concentrations can be measured only via vacuum sampling. The interim standards provided for dust lead in the Interim 403 Guidance Document (EPA, 1994) were defined in terms of lead loading. The decision there was based, in part, on the wider availability, familiarity, and lower cost of wipe sampling compared to vacuum sampling. The agency made a policy decision to define the §403 dust standards in terms of lead loading.

Dust can accumulate on multiple surfaces in the home: floors, furniture, window sills, and window troughs. Levels of lead accumulated in dust on the window sill may not represent the same level of health concern as lead in floor dust. The Interim 403 Guidance Document (EPA, 1994) provided standards for lead in dust on floors, window sills, and window troughs. Technical analyses conducted to support the Risk Assessment for §403 indicated that there is mixed evidence on whether levels of lead in window trough dust provide additional information on the impact of lead on childhood blood lead beyond the information contained in floor and window sill dust. Window troughs are also more difficult to sample than window sills. EPA's approach for the §403 dust standard is to provide two standards, one for lead in floor dust and one for lead in window sill dust.

With respect to paint, the Agency recognizes that a principal pathway of childhood exposure to lead is through the contamination of dust and soil. Activities designed to reduce exposure to lead in dust and soil will therefore mitigate the contribution of lead-based paint to childhood health effects and blood lead by the elimination or reduction of the pathway from paint to dust and soil. By statutory definition, intact lead-based paint is not considered a hazard unless it is present on accessible, friction, or impact surfaces. However, in cases where painted surfaces are deteriorated, there is an increased potential for both direct ingestion of paint chips containing lead and for contamination of the residential dust and soil. As a result, the standard for the lead-based paint hazard is defined in terms of the condition, location and amount of painted surfaces that contain lead-based paint.

Although the standards defined by this rule will not specifically require the conduct of any lead exposure reduction activities, EPA recognizes that they will be used by federal, state, local, and private entities in their efforts to manage the hazards of lead in paint, dust, and soil. Therefore, EPA's approach to the Risk Assessment is to document the scientific basis for placing standards on lead-based paint hazards, lead-contaminated dust and lead-contaminated soil, to estimate the number of interventions performed to meet the standards defined by this rule and to characterize the reductions in childhood health risks and blood-lead concentrations achieved from conducting these interventions.

1.2 OBJECTIVES OF RISK ASSESSMENT

The overall objectives of this report are to

1. Document the scientific basis for §403.

   This report assesses the risks of childhood exposure to lead-based paint hazards, lead-contaminated dust, and lead-contaminated soil. Each component of the risk assessment is documented in this report: hazard identification, exposure assessment, and dose-response assessment.

   These individual characterizations are integrated to assess the risks of lead exposures to young children and the reductions in these risks expected to take place as a result of §403. The objectives and activities of each component of the risk assessment are described further in Section 1.3.

2. Characterize the health risks to young children from specific residential exposures to lead.

   This document estimates risks to young children from specific residential sources of lead. These sources are: (1) interior and exterior lead-based paint; (2) lead-contaminated dust, which may contain lead derived from deteriorated interior paint and tracked- or blown-in exterior soil; and (3) lead-contaminated soil, which may contain lead from deteriorated exterior paint, from past leaded-gasoline vehicle emissions, or from other sources.

   This risk assessment focuses on risks to children aged 1-2 years old. Other populations also certainly face risks from lead exposure including children of other ages, pregnant women, and the general adult population. Characterization of risks and risk reduction for 1-2 year old children was chosen as representative of total risk and risk reduction in the interest of keeping a manageable scope and time frame for the risk assessment. Also, as discussed in Sections 2.3 and 2.4 this may be the subgroup of children most appropriate for estimation of health effects.

3. Characterize the reduction in risk expected to result from implementation of the §403 rule for a variety of options for the §403 standards.

   This report characterizes the incremental risk reductions expected to result from specific environmental interventions in specific types of housing units. Because the §403 rule does not mandate specific action, it was not possible to analyze the risk reductions associated with specific interventions required by the regulation. Instead, the Agency's approach in this risk assessment is to characterize the risk reduction consequences that might occur if broadly defined interventions are undertaken to reduce exposures to lead in dust, soil, and paint. Intervention activities considered in

this report are: cleaning of house dust, maintenance of interior or exterior paint, encapsulation/abatement of interior or exterior paint, soil cover, and soil removal.

4. Estimate the numbers of children and housing units affected by various options for the §403 standards.

   This report estimates numbers of children and numbers of homes in the nation's housing stock that would be affected by the rulemaking. The time frame of the risk assessment is 1997, with the assumption that all actions resulting from the §403 rule occur within that time frame.

Please note that the objectives of this report do not include the actual selection of the §403 standards. Actual selection of the standards is a policy decision to be made by the risk managers. The purpose of this report is to provide relevant information on the scientific basis for setting the standards and the comparative risk reductions that are expected to result for various options for the §403 standards.

1.3 ORGANIZATION OF REPORT

The report has seven chapters, including this introduction, background, and summary. This section summarizes the remaining chapters.

1.3.1 Chapter One – Introduction, Background, and Summary

The questions addressed in Chapter One are: What health risks are addressed in this report? What is the basis and requirements for addressing these risks? What is EPA's response to addressing these requirements? What are the objectives of the risk assessment? What are the data sources and tools used to assess the risks? What are the limitations of the data sources and tools?

The health risks addressed in this report are introduced in Section 1: health risks to young children from exposures to lead-based paint hazards, lead-contaminated dust, and lead-contaminated soil. The regulatory basis for addressing these risks, the regulatory requirements and EPA's response to the requirements are described in Section 1.1. The overall objectives of the risk assessment are presented in Section 1.2, and the objectives of each component of the risk assessment are discussed in Section 1.3. Section 1.5 describes the data sources and analysis tools employed in this risk assessment and discusses their limitations. Section 1.4 provides an

overview of the methodology and approach utilized to assess the risks of childhood exposures to lead-based paint hazards, lead-contaminated dust, and lead-contaminated soil.

1.3.2 Chapter Two - Hazard Identification

The goal of the hazard identification was to answer the following questions: What measure of body burden to lead should be used in this risk assessment? What are the biologic effects of exposures to lead? What are the adverse health effects linked to body burdens of lead? What health endpoints should be quantified in the risk assessment? For what age groups of children should the health endpoint be evaluated?

Blood-lead concentration, as discussed in Section 2.1, is the most commonly used measure of lead body burden for linking exposures to lead toxicity and is utilized throughout this risk assessment. A summary on the mechanisms of lead toxicity is presented in Section 2.2 For a more comprehensive assessment, the reader is referred to the evaluations conducted by EPA (EPA, 1986) and ATSDR (ATSDR, 1993) and other literature. Several health effects are linked to childhood lead body burdens in Section 2.3, focusing on the adverse neurotoxic effects of decreased intelligence, developmental delays, behavioral problems, seizures and even coma. These effects are of particular significance because of the developing nervous system of the young child and because the central nervous system is the primary target organ for lead toxicity in children. (ATSDR, 1988). The health risks associated with childhood lead poisoning encompasses a wide range of exposure levels. IQ point deficit and elevated blood-lead concentration are selected in Section 2.4 to represent the spectrum of health effects of lead exposure. This report assess the health risks and blood-lead concentrations of children ages 1-2. The reasons for selecting this age group of children are discussed in Section 2.4

1.3.3 Chapter Three - Exposure Assessment

Chapter Three examines the following questions: What measures of exposure should be assessed? Are lead-based paint hazards, lead-contaminated dust and lead-contaminated soil sources and environmental pathways of lead exposures? Is there evidence of a relationship between exposures to lead in the environment and blood-lead concentrations? What is the

distribution of lead-based paint hazards, lead contaminated dust, and lead-contaminated soil in the nation's housing? What is the distribution of children's blood-lead concentrations?

Both environmental levels of lead and childhood blood lead are used in this report to characterize exposures to lead. Pathways and sources of environmental lead exposure, including lead-based paint, lead contaminated dust, and lead-contaminated soil, are summarized in Section 3.1. The rather extensive evidence on the relationship between childhood blood-lead concentrations and environmental-lead levels is summarized in Section 3.2. The distribution of lead in residential dust, soil, and paint in the nation's housing is estimated in Section 3.3. Tables of results contained in that section show that residential levels of lead can be very high for some homes and that older homes (built before 1940) tend to have higher levels of residential lead compared to newer homes (homes built after 1960). Estimating the national distribution of lead exposures required estimating the number of homes in the nation's housing stock and the number of children in 1997. The methods employed to determine these two quantities are discussed in Appendix C. Finally, the national distribution of blood-lead concentrations for children of ages 1-2 is presented in Section 3.4

1.3.4 Chapter Four - Dose-Response Assessment

The primary questions explored in Chapter Four are: What is the dose-response relationship between exposures to environmental lead and the health effects evaluated in this risk assessment? What are reasonable ranges of options for the standards to be further examined in the risk assessment?

The linkage between IQ deficits and lead exposures is usually provided in terms of blood-lead concentrations rather than environmental-lead levels. Therefore, the solution to the first question prompted further inspection of two additional questions: What is the dose-response relationship between environmental lead and childhood blood-lead concentration? What is the dose-response relationship between childhood blood-lead concentrations and health effects? Two dose-response models are used in this report to estimate the relationship between environmental lead and childhood blood-lead concentration. First, a mechanistic model developed by EPA, EPA's Integrated Exposure, Uptake and Biokinetic Model for Lead (IEUBK Model), is discussed in Section 4.11. Second, a regression model constructed using the data

from the Rochester Lead-in Dust Study (EPI model) is described in Section 4.12. The IEUBK model does not account for the contribution of lead-based paint on childhood blood-lead concentrations. The approach implemented for estimating the effect of pica for paint on childhood blood lead is presented in Section 4.3. Because EPA's approach is to define the dust-lead standard in terms of a wipe dust-lead loading and because dust samples in the HUD National Survey were collected via vacuum sampling, an additional question was examined: Can dust-lead loadings collected using a vacuum sampler be converted to a wipe equivalent dust-lead loading? Section 4.2 presents equations for converting a dust-lead loading collected via a vacuum sampler to a wipe equivalent dust-lead loading. Finally, the methods for computing the childhood blood lead and health risks evaluated in this report from blood-lead concentrations and from environmental lead exposures are detailed in Section 4.4.

Two approaches were employed to estimate ranges of options for the §403 standards: regression models and sensitivity/specificity analyses. Table 4.8 in Section 4.5 presents the results of these analyses.

1.3.5 Chapter Five - Integrated Risk Assessment

This chapter integrates the characterizations from the hazard identification, exposure assessment, and the dose-response assessment to answer the following questions: What are the blood-lead concentrations and health risks to children ages 1-2 resulting from residential exposures to lead? What interventions or abatements might be performed to comply with the §403 standards? What are the expected reductions in these endpoints resulting from the rulemaking for various options for the §403 standards. How many children and housing units will be affected by various options for the §403 standards? What assumptions and data inputs are likely to have the largest impact on the risk assessment? How sensitive are the results of the risk assessment to the assumptions and data inputs?

An overview of the methodology employed in the integrated risk assessment is provided in Section 1.4. Blood-lead concentrations and health risks to children ages 1-2 estimated to exist in 1997 prior to the passage of §403 (pre-§403) are shown in Table 5-1 in Section 5.1. Intervention options and environmental levels of residential lead expected after conduct of these interventions are presented in Tables 5.2 and 5.3, respectively, in Section 5-2. Tables 5.7 to 5.10

in Section 5.3 display the numbers of homes affected by various options for the §403 standards and characterize the childhood blood-lead concentrations and health risks expected to occur in the post-§403 environment for these options. Figures 5-3 to 5-6 in Section 5.3 graphically illustrate the impact of changing the levels of lead in the §403 standards on both the number of homes requiring an intervention and the post-§403 childhood blood-lead concentrations and health risks.

Table 5-11 in Section 5-4 lists the assumptions and inputs considered to have the greatest impact on the risk assessment and Tables 5-12 to 5-15 in Section 5-4 present the outcomes of the sensitivity analyses.

1.3.6 Chapter Six - Validation Studies for the IEUBK Model

This chapter considered one question: Is the IEUBK model an appropriate tool for predicting a national distribution of blood-lead concentrations based on a national distribution of environmental-lead levels?

The analyses summarized in Chapter 6 did not find any evidence to conclude that the IEUBK model is not an appropriate tool for predicting a national distribution of blood lead levels based on a national distribution of environmental lead levels.

1.3.7 Chapter Seven - Risk Summary

On an overall basis, after each of the activities described in the previous chapters have been completed, this chapter summarizes the conclusions of the risk assessment.

- The health risks to young children from exposure to lead-based paint hazards, lead-contaminated dust, and lead-contaminated soil are unacceptably high.
- The health risks to our nation's children can be reduced.
- The standards defined by §403 will help reduce the health risks to our nation's children.

1.4 OVERVIEW OF METHODOLOGY

This section presents the conceptual approach taken by the Agency in the integrated risk assessment, including the basis for choosing the specific risk assessment tools and data that were used, as well as the major assumptions that were made in implementing those tools.

1.4.1 General Approach

The general approach taken in this risk assessment is to estimate the health effects benefits derived from intervention activities expected to be performed in response to candidate standards, which can then be compared to the costs of those interventions. As with most risk assessments addressing the health impacts attributable to reduced environmental exposures to lead, the level of lead in blood is used as the index of exposure. The predicted decrease in children's blood lead concentrations is then related to the decrease in expected adverse health effects.

This assessment focuses on two routes of childhood lead exposure: ingestion of dust and soil through normal hand-to-mouth activity, and ingestion of paint chips. These are believed to be the major exposure routes that will be affected by the Title X Lead-Based Paint Program. (The sources and pathways of lead exposure are summarized in Section 3.1.)

In initially formulating the details of its approach, the Agency decided to rely for the most part upon existing data on the prevalence of lead in residential environments and on the relationship between environmental lead and adverse health impacts to children.

The Agency has also used measured empirical data whenever such data existed and was deemed to be of sufficient quality to provide reasonable estimates of the phenomena of interest (*e.g.*, the national blood-lead distribution in children, paint conditions and lead levels in residential dust and soil, *etc.*). Best technical judgement and predictive models were used to estimate changes in environmental-lead levels due to intervention activities and consequent changes in the distribution of blood-lead concentrations in children. Where assumptions are believed to be tenuous or thought to be critical to any of the conclusions, analyses have been conducted to demonstrate the sensitivity of the results to those assumptions.

The key concern with predictive models used to relate environmental-lead levels to children's blood lead is whether the models provide plausible predictions of blood-leads over the

range of environmental-lead levels of interest. Two different types of predictive models are utilized for determining the relationship between environmental lead and blood lead: the IEUBK model and an EPI model. To help ensure that the IEUBK and EPI models provide plausible predictions of blood-lead concentrations, EPA developed an approach to calibrate reductions in blood-lead concentrations predicted from these models to the baseline blood-lead concentrations developed from empirical data. This approach is summarized as follows:

1. NHANES III was employed to characterize the baseline (pre-§403) distribution of blood-lead concentration for children aged 1-2 years.

2. Use the environmental-lead levels for HUD National Survey units as input to either the IEUBK or EPI model to predict the decline in the distribution of blood-lead concentration for children aged 1-2 expected to result from the §403 rulemaking.

3. Use the baseline distribution of blood-lead concentration and the estimated decline in the distribution of blood-lead to derive a final post-intervention blood-lead distribution (post-§403).

Additionally, the models are used only to estimate the "primary prevention" benefits of the standards (*i.e.*, benefits to children who are born into units where interventions have already occurred and, therefore, who have not been previously exposed to higher levels of environmental lead). The Agency believes that use of the models in these limited circumstances avoids or minimizes many of the confounding factors that are often encountered when attempting to predict blood-lead responses related to a wide range of environmental conditions, exposure situations, and intervention methods.

The process of assessing the risk reductions associated with the §403 standards consists of a number of discrete steps, as depicted in Figure 1-1. The Agency's approach to each of these steps is summarized below, and described in detail in the chapters that follow.

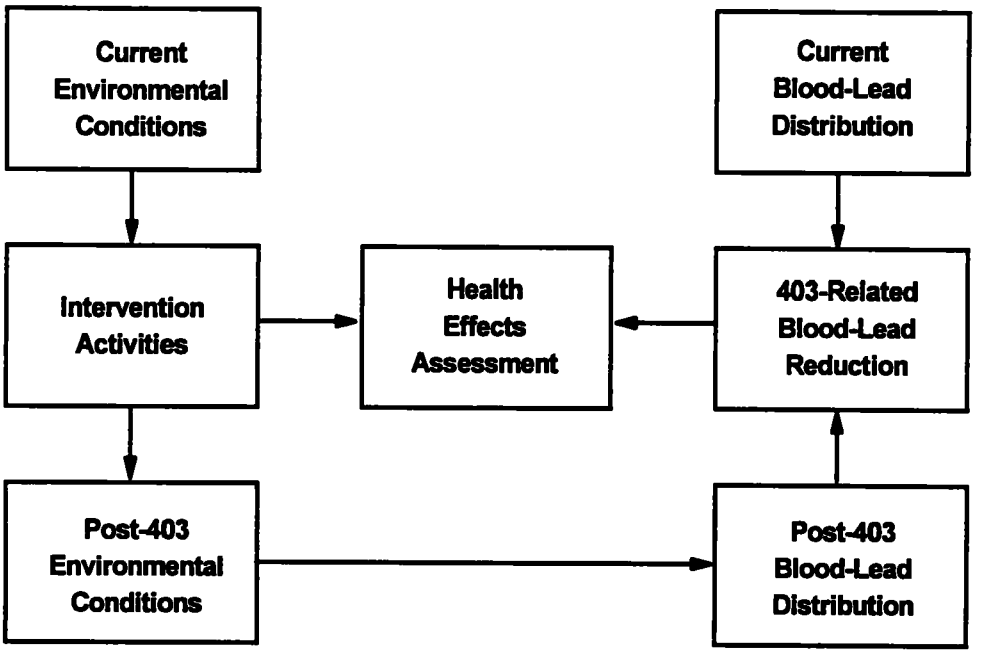


Figure 1-1. §403 Risk Assessment Steps

1.4.2 Current Blood-Lead Distribution

The current "pre-§403" distribution of children's blood lead has been assumed to be that described by the third National Health and Nutrition Examination Survey (NHANES III). The Agency is assuming 1997 as the "pre-§403" time frame since the rule is expected to be promulgated in that year. NHANES III reported blood-lead results for children aged 1-2 years and 3-5 years. Although the results reported were for the years 1988-1989, the Agency is using these results to represent the current blood-lead distribution. The Agency recognizes that children's blood leads may be lower today (levels reported in previous NHANES surveys have shown significant declines over time), but has no information upon which to project additional declines from the 1988-1989 time frame to the present. The Agency notes that much of the reduction in children's blood-lead concentrations that occurred from the 1960s through the 1970s was due to the phase-out of leaded gasoline and activities to halt the use of leaded solder in food cans. It is likely that the benefits from these types of activities have already been realized and that the sharp declines in blood-lead concentrations seen in the 1970s and 1980s would not continue in the period from NHANES III to the present. The results of the NHANES survey are discussed in Section 3.4.

1.4.3 Baseline Environmental Lead Conditions

An estimated baseline distribution of current environmental lead conditions is necessary in order to predict the post-§403 conditions. The Agency's estimate of current conditions is based upon the Department of Housing and Urban Development's national survey, which was conducted in 1989-1990 (HUD National Survey). The HUD National Survey contains data on age of housing, condition and location of deteriorated lead-based paint, and levels of lead in interior dust and exterior soil. More recent data from the 1993 American Housing Survey (AHS) were used to update the information on numbers of housing units obtained from the HUDH Survey. This survey, conducted every two years by HUD, collects data on the nation's housing characteristics such as age of home, number of rooms, number and ages of residences, and income levels of residences. The AHS survey does not collect information on environmental-lead levels.

Additional methods were used to project the numbers of units in the current housing stock from the 1993 AHS. Together, this information allowed the Agency to estimate the current distribution of environmental lead conditions in the nation's housing according to housing age (pre-1940, 1940-1959, 1960-1979, and post 1979). This categorization of the housing stock allows the Agency to account for differences in numbers, types, and costs of intervention activities that may be associated with the various categories. The details of the Agency's approach are described in Section 3.3 and Appendix C.

1.4.4 Post-§403 Environmental Lead Conditions

In order to project the distribution of environmental lead conditions that would result from the promulgation of the §403 standards, the Agency identified a number of specific intervention activities that were assumed to occur at housing units that would be identified as posing a hazard under the standards. These intervention activities include both interim controls and more permanent, abatement measures. For each of the intervention activities, post-intervention environmental lead conditions were assumed. In general, these intervention activities are assumed to be media-specific. For example, if a unit was identified as a hazard based only upon the presence of deteriorated exterior paint, the intervention activity would address only the exterior paint. An exception is that both interventions dealing with either

interior paint or activities involving soil excavation. These two interventions are assumed to be followed by cleaning of interior dust to HUD clearance levels. This is due to the expectation that these activities would create high levels of interior leaded dust that would warrant special cleaning.

Finally, the expected duration of the reduction in environmental-lead levels that result from each intervention was also estimated. The assumed effectiveness and duration of interventions are presented in Section 5.2.

1.4.5 Post-§403 Blood-Lead Distribution

For projecting the estimated blood-lead distributions expected to result from the post-§403 environmental conditions, two modeling approaches have been used. In one, the Agency's IEUBK model was used, with dust and soil input values taken from the post-§403 environmental levels described above. The remaining environmental input parameter values were selected to represent national average levels of exposure to other media. The IEUBK-based approach does not account for exposures due to the direct ingestion of chips of lead-based paint through the model itself. Rather, for homes with damaged lead-based paint (where paint chips were assumed to be available for ingestion), a set percentage of children were assumed to have a specified increase in blood-lead concentration due to this route of exposure. Where paint stabilization or removal was the intervention expected to occur in these units, the avoidance of the increase in blood lead concentration from ingestion of paint chips was assumed.

In the other approach, an empirical model (EPI model) was developed using the data from the Rochester Lead-in-Dust Study. This model directly accounts for ingestion of paint chips as well as dust and soil via hand-to-mouth activity.

In combination, these models are used to estimate a range of predicted blood-lead concentrations that are expected in children exposed to the post-§403 conditions. The details of the Agency's approach are described in Section 4.1.

The estimated post-§403 blood-lead distribution is used to predict the reductions in health effects and blood-lead concentrations that are expected to result from implementation of §403. The predicted post-§403 blood-lead distributions are based on distributions of environmental-lead levels developed from the HUD National Survey data, predicted declines in the distributions

of environmental lead that are expected to result from intervention activities performed under §403, and either the IEUBK or EPI model. The predicted post-§403 blood-lead distributions are used herein for the purposes of comparing various regulatory options. The predicted post-§403 blood-lead distributions and associated health effects are NOT MEANT to be an accurate reflection of what childhood blood-lead concentrations and health effects will be in future years after §403.

1.4.6 Projected Population of Children

The number of children estimated to occupy the housing categories was calculated based upon published literature predicting the numbers of children expected to reside in the United States in 1997 by age group. Numbers of children of ages less than one year, 1 to 2 years, and 3 to 5 years were calculated for each of the four housing group age categories. As discussed in Section 2.4, the risk assessment is evaluating the health risks and blood-lead concentrations for children aged 1-2. In addition, the statute defines target housings to be housing constructed before 1978 that has the possibility of housing a child less than six years of age. Analyses are conducted in the sensitivity analyses to determine the impact of basing the risk assessments on children ages 1-2.

The numbers of children occupying housing categories is necessary to estimate the numbers of these children who will be affected by interventions that are expected to occur at each housing type. The details of the Agency's approach are described in Section 3.3.2 and Appendix C.

1.4.7 Reductions in Childhood Blood Lead and Health Effects

Benefits from the estimated blood-lead reductions were quantified for three health outcomes: decrease in IQ scores, incidence of IQ scores less than 70, and incidence of blood-lead concentrations greater than 25 μg/dl. Low IQ scores are associated with lower levels of educational attainment and lower lifetime earnings. IQ scores less than 70 are indicative of costs ranging from special education to life-long institutional care. Blood-lead concentrations greater than 25 μg/dL represent levels at which medical intervention may be necessary. The details of the Agency's approach are described in Section 4.4.

1.5 DATA SOURCES, ANALYSIS TOOLS AND LIMITATIONS

All data sources and research studies referred to in this report are listed in the references at the end of the report. Data sources and analysis tools are described in more detail in the individual sections of the report where they are applied. This section highlights the major data sources, and analysis tools implemented in the risk assessment and points out possible limitations and data gaps in the risk assessment.

1.5.1 Hazard Identification

There is a wealth of information on adverse biological responses to lead. An exhaustive compilation or review of all available data on the effects of lead is not within the scope of this report. For a comprehensive review, the reader is referred to EPA's report, *Air Quality Criteria for Lead* (EPA, 1986), and the two ATSDR reports, *Toxicological Profile for Lead* (ATSDR,1993) and *The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress* (ATSDR, 1988). The documented evidence on the adverse biological responses to lead is one of the major strengths of this risk assessment.

Quantifying health risks to children from exposures to lead requires the selection of specific endpoints. The neurotoxic and blood lead endpoints selected for this risk assessment have been used to support previous regulatory decisions. It is possible that if other endpoints were selected, the baseline risks to lead exposures would be larger and the potential risk reduction associated with various options might be larger.

1.5.2 Exposure Assessment

The primary data set used in this study to estimate a national distribution of exposures to lead-based paint hazards, lead-contaminated dust, and lead-contaminated soil is the HUD National Survey. This study was designed to be a nationally representative study of environmental-lead in the nation's housing built prior to 1980. Possible limitations of this dataset are: 1) environmental levels may have declined since the study was conducted in 1988-1991, 2) blood samples were not taken in the study, 3) at most three floor dust samples were collected in each dwelling unit, 4) dust samples may not have been collected from areas frequented by children, 5) considerable measurement error may be present in some of the

measured dust-lead concentrations due to the small amounts of dust collected for some samples, and 6) the sample size of 284 homes is a small number for characterizing environmental-lead levels in our nation's housing stock. A laboratory study was conducted, methodology developed, and dust-lead concentrations adjusted (revised downwards) to correct for the small weights of dust collected for some samples. These results are presented in Appendix Z. For comparison purposes, exposures to lead in paint, dust, and soil from two epidemiologic studies, Baltimore Repair and Maintenance (R&M) study and the Rochester Lead-in-Dust (Rochester) Study, are also presented in Section 3.

The HUD National Survey characterized residential environmental-lead levels only in homes built prior to 1980. Therefore, an additional limitation of the exposure assessment is that environmental-lead levels in houses built after 1980 had to be inferred.

The primary data set used in the risk assessment to estimate a national distribution of blood-lead concentration is NHANES III. This is an ongoing survey with a statistical sampling plan to insure representativeness. Some possible limitations of NHANES III are 1) blood-lead concentrations may have declined since the study was conducted in 1988-1991, and 2) seasonal rhythms in blood-lead concentrations are not accounted for in the database. If blood-lead concentrations have declined since the conduct of NHANES III due to the activities of federal, state, local, and private agencies, then the baseline risks due to childhood lead exposures will be overestimated and the reductions in those risks resulting from §403 will also be overestimated. Because of the serious implication of this data limitation, the sensitivity analysis examines the impact of a 1% annual decline in childhood blood-lead concentrations on the estimated baseline risks and the reduction in risks resulting from §403.

The evidence in the literature supporting the existence of a positive relationship between environmental lead and blood-lead concentration is one of the strengths of this risk assessment. Eight epidemiological studies were selected to help demonstrate that relationship. However, quantification of this relationship, as discussed below in Section 1.5.3, is more problematic

The 1993 American Housing Survey (AHS) is the principal data source used for estimating the nations's housing stock. A possible limitation with the 1993 AHS survey is that it characterizes the nation's housing in 1993. Other datasets and assumptions, as described in Section 3.4, were required to extrapolate this information to 1997. Estimates of the number of

children who reside in each home were based on information in the 1993 AHS on the average number of residents per home and Census projections of the number of children per resident (Day, 1993). A possible limitation in the analyses is the assumption that the average number of residents per home is the same for all types of homes.

1.5.3 Dose-Response Assessment

Conversions factors were developed to convert dust-lead loadings based on the vacuum samplers used in the HUD National Survey and the R&M study to wipe equivalent dust-lead loadings. However, only limited data were available for constructing these conversion factors and there is considerable uncertainty associated with predictions generated from these equations. Therefore, the sensitivity analysis examined the impact of the uncertainty in the conversion equations on the wipe equivalent dust-lead loadings and the predicted blood-lead concentrations based on these equations.

Currently, the data from just one study, the Rochester Study, was used for development of the EPI model. Limitations of the EPI model derived from the Rochester Study data concern the use of data from a single city, with over 84% of the sampled homes built prior to 1940 and approximately 40% of the sampled children African American. The IEUBK model is a biological simulation model for predicting a plausible distribution of blood-lead concentrations based on available information on children's exposure to lead. Analyses conducted in Section 5 are based on the default values for the intake and uptake parameters recommended in the guidance manual (EPAa, 1994) and the soil- and dust- lead concentrations are being used.

A major limitation of this Risk Assessment is whether or not the model predicted blood-lead concentrations based on the soil and dust lead concentrations measured in the HUD National Survey are representative of blood-lead concentrations in exposed children in the nation's housing. Because of possible limitation in both the EPI model and the IEUBK model for predicting blood-lead concentrations from environmental-lead levels, results from both models are presented in this report. Furthermore, analyses are conducted in Section 6 to assess whether there is any evidence to show that utilizing the IEUBK model in the Risk Assessment is not appropriate.

Several papers were available in the literature for estimating the relationship between blood lead and IQ decrements. In addition, there were at least three papers that reviewed and evaluated the data from multiple studies to develop a model based on more than one study (meta analysis). The relationship between IQ decrement and blood-lead concentration employed in this Risk Assessment is based on the results of the meta-analysis in (Schwartz, 1994). Heath risks associated with childhood lead exposures are very sensitive to the estimated IQ points lost per one μg/dL change in blood lead. The sensitivity analysis examines the impact of other alternative estimates of this relationship on the Risk Assessment.

1.5.4 Integrated Risk Assessment

The integrated risks assessment employs the datasets mentioned in Section 1.5.2 for exposure assessment and the dose-response models referred to in Section 1.4.3. In addition, in order to estimate the impact of §403, generic interventions that might be implemented by federal, state, local and private agencies to comply with the §403 standards were developed. These interventions were not meant to simulate every possible nuance of post-§403 activities, but rather serve as categories of interventions to capture generic activities conducted to meet the standards for lead-based paint hazards, lead-contaminated dust and lead-contaminated soil. However, evaluation of the impact of §403 required specification of lead levels in paint, dust and soil after each one of the interventions. Only limited data were available for estimating the post-intervention levels of lead. This constitutes one of the major data gaps and limitations for this risk assessment. The sensitivity analyses examines the impact of changes in the post-intervention environmental-lead levels on the risk reductions expected to occur as a results of §403. In addition, the sensitivity analysis examines an alternative approach that does not require specification of post-intervention environmental-lead levels.

Tables 5.7 to 5.11 in the Integrated Risk Assessment Chapter (Section 5.3) present estimates of the health and blood-lead effects predicted to exist after implementation of the §403 rule for a variety options for the §403 standards. There is considerable uncertainty in these numbers due to uncertainty in the

1. estimated distribution of environmental-lead levels in the nation's housing,

2. the measures of lead in dust, soil, and paint considered in this risk assessment,

3. the estimated number of homes that will take action in order to comply with the §403 standards,

4. assumed efficacy of interventions conducted to comply with the §403 standards,

5. dose-response models used to predict average (geometric mean) blood-lead concentrations from environmental-lead levels,

6. dose-response models used to blood-lead concentrations to relate IQ related health effects,

7. methodology employed to predict a national distribution of post-§403 blood-lead concentrations and associated health effects.

The intent of the risk characterization is to estimate risks associated with residential environmental lead exposure and reductions in risk that will be associated with different sets of §403 standards. It is well known that there is much uncertainty associated with characterizing specific health risks associated with environmental lead exposure. There are numerous sources and pathways of lead exposure, particularly for children. There are other significant factors affecting children's blood-lead concentration and health risks that are not captured in the risk assessment methodology nor affected by the §403 rule. Such factors include home and personal cleaning habits, diet and nutritional status, bio-availability of the lead found in residential environmental media, non-residential exposures, parent's occupation, water, hobbies, and children's behavioral patterns. Characterization of environmental levels of lead in paint, dust, and soil are subject to many sources of uncertainty which include chemical analysis, sampling, spatial, and temporal variability. Use of available data introduces additional uncertainty, introduced by conversion factors and locality differences. If one accepts a 1.6 GSD as reflective of variability in blood-lead concentrations in a population of similarly exposed individuals, then a perfect model to predict a national distribution of blood-lead concentrations would account for the additional variability (2.05 GSD) observed in the NHANES national distribution. However, even in this ideal case, the model would only account for approximately 57% of the variance in

the national distribution. (Calculated as: $[(\log 2.05)^2 - (\log 1.6)^2] / (\log 2.05)^2$.) Modeling is particularly difficult for children with moderately high blood-lead concentrations (e.g. 10 μg/dL to 20 μg/dL) and exposures to multiple media. This uncertainty in characterizing the relationship between environmental levels of lead and health effects was a major factor in EPA's decision to approach this rule from a risk management perspective. From this perspective, the results of this risk assessment should not be taken as precise estimates of total health risks associated with a particular set of standards. Rather the risk assessment should be taken as providing a reasonable analysis of:

1. The degree to which implementation of a §403 rule that results in environmental interventions may be expected to reduce health risks associated with lead exposure; and

2. The relative change in risk reduction when different options for the §403 standards are chosen.

The fact that all predictions in the modeling process are in terms of geometric means and associated distributions lends credibility to the analyses. Nevertheless, the substantial uncertainty in the modeling effort to relate multi-media lead exposure to a national distribution of health effects is recognized. The sensitivity analyses help characterize the uncertainties in the analysis.

2.0 HAZARD IDENTIFICATION

CHAPTER 2 SUMMARY

This chapter presents information on the toxicity of lead, through a discussion of how body-lead burden is measured, how lead works in the body, and the resulting adverse health effects. Two health effects, elevated blood-lead concentration and IQ point deficits, are identified to represent the spectrum of adverse health effects resulting from lead exposure. These representative effects are used in the integrated risk analysis to assess the potential benefits of the proposed §403 rule.

Blood-lead concentration is a commonly used measure of body lead burden. An extensive body of research relates health effects of lead exposure to blood-lead concentration. For example, lead-related reductions in intelligence, impaired hearing acuity, and interference with vitamin D metabolism have been documented in children at blood-lead concentrations as low as 10 to 15 µg/dL, with no apparent threshold. At higher exposure levels, these effects become more pronounced and other adverse health effects are observed in a broader range of body systems. Increased blood pressure, delayed reaction times, anemia, and kidney disease may become apparent at blood-lead concentrations between 20 and 40 µg/dL. Symptoms of very severe lead poisoning, such as kidney failure, abdominal pain, nausea and vomiting, and pronounced mental retardation, can occur at blood-lead levels as low as 60 µg/dL. At even higher levels, convulsions, coma, and death may result.

Adverse health effects (e.g., IQ deficits, neurological dysfunction) in children have long been associated with elevated lead exposure. The concentration of lead in whole blood, usually expressed in micrograms of lead per deciliter of whole blood (µg/dL), is the most common measure of a person's internal exposure to lead. Blood lead can be measured easily and accurately as compared to alternative physiological measures such as lead in bone or hair, and is more directly relevant to the assessment of exposure than are environmental measures (CDC, 1991; EPA, 1986). Centers for Disease Control guidelines on childhood lead poisoning prevention have traditionally been and currently are defined in terms of blood-lead concentration (CDC, 1991). While lead exposure in adulthood is a concern, fetuses, infants, and young children are the population most at risk from exposure to lead (EPA, 1986; ATSDR, 1993). This intensified risk is due to children's increased oral activity (e.g., hand-to-mouth behavior) and ability to absorb lead, coupled with the susceptibility of their rapidly developing central nervous

systems (Bellinger, 1995; Goyer, 1993). The central nervous system is the primary target organ of lead, though lead is stored throughout the body (e.g., bones, tissues, blood) (ATSDR, 1993).

The blood-lead concentration at which health professionals express concern has decreased over time. At high blood-lead concentrations (i.e., lead poisoning), lead exposure can cause coma, convulsions, and death. At lower concentrations, observed adverse effects from lead exposure in young children include reduced intelligence, reading and learning disabilities, impaired hearing, and slowed growth (CDC 1991). The phase-out of leaded gasoline (halting the fallout from leaded gasoline emissions), the restriction on the residential use of lead-based paints, and restrictions against lead solder in cans and water systems have greatly reduced blood-lead concentrations nationwide (EPA, 1986; Brody et al., 1994; Pirkle et al., 1994; Section 3.0). Simultaneously, however, further research suggested that levels previously thought safe were, in fact, hazardous. In the first half of the 20th Century, medical care providers were concerned about childhood blood-lead levels above 80 μg/dL; by the 1960s the level of concern was reduced to 60 μg/dL and above; by the 1970s the level of concern was at 40 μg/dL; and by the 1980s the level was lowered to 25 μg/dL (CDC, 1991). In 1991, the CDC reduced its blood-lead concentration community level of concern to 10 μg/dL (CDC, 1991). This was in response to scientific evidence appearing in the immediately preceding years that adverse health effects occur at blood-lead concentrations at least as low as 10 μg/dL. In fact, no discernable threshold in the relationship between adverse health effects and blood-lead concentrations has been identified (ATSDR, 1993).

Despite the nationwide reductions in blood-lead concentrations over time, one effect of reducing the blood-lead concentration of concern is increasing the number of children expected to have blood-lead concentrations above that level. The National Health and Nutrition Examination Surveys (NHANES) trace the health and nutritional status of the U. S. population. The results of the most recent NHANES (NHANES III, phase 1, 1988–1991) demonstrate that significant declines in childhood blood-lead concentrations have occurred in recent years, but that significant numbers of children's blood-lead concentrations remain above 10 μg/dL. The geometric mean blood-lead concentration, reported in NHANES III – phase 1, for children between the ages of one and five years was 3.6 μg/dL (Pirkle et al., 1994). This is 11.4 μg/dL lower than the estimate reported in NHANES II (conducted between 1976 and 1980) of 15 μg/dL

(Pirkle et al., 1994). Despite the lower blood-lead concentration reported in NHANES III, approximately 1.7 million children between one and five years of age are estimated to have blood-lead concentrations above 10 μg/dL (Brody et al., 1994).

There is an extensive body of literature relating health effects of lead exposure to measures of body-lead burden. This literature is summarized in several government reports, including

- Air Quality Criteria for Lead (EPA, 1986)
- The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress (ATSDR, 1988a)
- Air Quality Criteria for Lead: Supplement to the 1986 Addendum (EPA, 1990)
- Comprehensive and Workable Plan for the Abatement of Lead-Based Paint in Privately Owned Housing (HUD, 1990)
- Preventing Lead Poisoning in Young Children (CDC, 1991)
- Toxicological Profile for Lead (ATSDR, 1993)

These sources were used extensively in the next sections, although the original sources are cited for specific results whenever possible. Section 2.1 discusses commonly used measures of body lead burden. The mechanisms of lead toxicity are described in Section 2.2. The scientific evidence on resulting health effects is presented in Section 2.3. Finally, specific health effects are selected in Section 2.4 for use in this risk assessment.

2.1 MEASURES OF BODY-LEAD BURDEN

For purposes of risk assessment, it would be ideal to precisely relate particular health outcomes, such as decreased learning deficits or increased motor coordination, to environmental lead levels. Unfortunately, most studies of lead in the environment use measures of body-lead burden, such as blood-lead concentration, as biomarkers of lead exposure. Similarly, studies that assess lead hazard interventions tend to use blood-lead concentration to measure intervention effectiveness (EPA, 1995). There is extensive evidence that body-lead burden is associated with

lead levels in environmental media (EPA, 1986; CDC, 1991). In addition, there is an extensive body of literature relating health effects of lead exposure to measures of body-lead burden.

The most common screening and diagnostic measure of body-lead burden is blood-lead concentration. Other measures include lead in bones, teeth, and hair. Approximately 94% of the total body burden of lead in adults (73% in children) is found in the bones (Barry, 1975). Blood-lead concentration has the advantage of being easily and inexpensively measured. A disadvantage, however, is that it reflects a mixture of recent and past exposure. Bone-lead levels are more reflective of cumulative exposure to lead. The half-life of lead in the blood of adults is approximately one month (Griffin, et al., 1975b; Rabinowitz, et al., 1976), whereas the half-life of lead in bone is several decades (ATSDR, 1993). Because lead cycles between the blood and bone, a single blood lead measurement cannot distinguish between low-level chronic exposure and high-level acute exposure (ATSDR, 1993). Because of recycling from the bone, both types of exposure could result in the same blood-lead concentration. Despite these limitations, blood-lead concentration remains the one readily accessible measure that can demonstrate in a relative way the relationship of various effects to increases in lead exposure (ATSDR, 1993).

Of the other measures, bone and tooth lead may be used to measure cumulative exposure to lead, while hair lead is an indicator of more recent exposure. Bone-lead content may be measured by x-ray fluorescence (XRF), although the reliability of this method is questioned by many researchers, especially at levels below 10 ppm (Wedeen, 1988). Since teeth can store lead up to the time of shedding or extraction, levels of lead in shed teeth have been used as an indicator of lead exposure in some studies (Smith, et al., 1983; Pocock, et al., 1989; Bergomi, et al., 1989; Needleman, et al., 1990). Hair lead has been used as an indicator for intermediate exposure (2 months) in children (Wilhelm, et al., 1989). However, artificial hair treatments such as dyeing, bleaching, or permanents, can invalidate metal analysis of hair (Wilhelm, et al., 1989) and external surface contamination problems are such that it is difficult to differentiate between externally and internally deposited lead (EPA, 1986). After consideration of the disadvantages of using bone, tooth, and hair lead as biomarkers of exposure, most researchers in the area of lead exposure conclude that blood lead remains the most efficient and useful way to assess body lead burden.

Physiological changes, such as alterations in heme (the component of blood that contains iron) synthesis, that are known to implicate lead exposure may also be used as biomarkers of exposure. Generally, blood-lead levels are determined concurrently with these physiological biomarkers. Interference with heme synthesis following lead exposure can lead to a reduction of hemoglobin concentration in blood (Bernard and Becker, 1988) and an increase in urinary coproporphyrin (EPA, 1986). A relationship between δ-aminolevulinate-dehydratase (ALAD) activity measured in erythrocytes and blood-lead levels of 5 to 95 μg/dL has been observed (Hernberg, et al., 1970). Although δ-aminolevulinate (ALA), a potential neurotoxin that accumulates from decreased ALAD activity, can be detected in urine when blood-lead levels are above 35 μg/dL in adults (25 to 75 μg/dL) (Roels and Lauwerys, 1987), ALA in urine is not considered as sensitive a measure of current lead exposure as ALAD activity (Hernberg, et al., 1970). The concentration of erythrocyte protoporphyrin (EP) rises above background at blood-lead levels of 25 to 30 μg/dL and there is an association between blood-lead levels and EP (CDC, 1985; Hernberg, et al., 1970). Determination of EP in blood is used as an indicator of past chronic exposure, since elevated EP reflects average blood-lead levels for about 4 months following the exposure (Janin, et al., 1985). In the case of each of these physiological measures, other conditions may produce similar effects, leading to false positive outcomes when these measures are used alone as biomarkers for body lead burden.

2.2 MECHANISMS OF LEAD TOXICITY

Lead is a very dynamic compound with a wide spectrum of effects in humans. Its effects are seen at the subcellular level as well as at the level of general function that encompasses all systems in the body. The subcellular mechanisms of action, followed by a discussion of the neurotoxic effects and the heme effects of lead poisoning, are included in this chapter. Wherever possible, mechanisms included in the subcellular mechanisms section are related to the specific effects of lead on the nervous system and the blood. There remain many gaps, however, in the information needed to explain the varied mechanisms of lead in the body in different organs.

Lead has been recognized as a naturally occurring element since the beginning of civilization. Today, the major environmental sources of lead are paint, auto exhaust, food, dust, soil, and water. "Inorganic lead" includes the metallic form of lead, its salts and oxides.

Inorganic lead primarily enters the body through inhalation (breathing in air) and ingestion (eating or drinking). Inorganic lead is absorbed, distributed throughout the body, and removed from the body (excreted). It is not broken down to simpler compounds (metabolized) in the body. "Organic lead," which is formed by lead in combination with an alkyl group (carbon and hydrogen), is found primarily in gasoline as tetraethyl lead. Organic lead enters the body through inhalation and can pass through the skin because of its properties. It is broken down (metabolized) in the liver and then removed from the body.

The rate at which lead is absorbed into the body depends on the chemical and physical properties of the form of lead and on the physiological characteristics of the exposed person (nutritional status, age, etc). When lead is inhaled it becomes deposited in the lower respiratory tract and is completely absorbed. The amount of lead absorbed from the gastrointestinal tract of adults is 10-15% of the amount ingested. In pregnant women and children, the amount absorbed can increase to 50%. The amount absorbed greatly increases during compromised nutritional status of the individual or during periods of iron or calcium deficiency. Once lead is absorbed it enters the bloodstream and is dispersed throughout the body where it is distributed between the blood, the mineralizing tissue (bone and teeth), and soft tissue (kidney, bone marrow, liver, and brain).

The lead in the mineralizing tissues accumulates in two different areas of the bone: 1) an area where lead can quickly be exchanged in the blood and 2) a more stable area where lead is stored long-term. This stable area for storing lead can pose a special risk because, when the body is under stress such as during pregnancy, lactation, or chronic disease, this lead may be 'mobilized,' thereby increasing the blood lead. Because of this more stable compartment of lead in bone, significant declines in blood lead can require months or years to occur after exposure has been stopped.

Of the lead found in the blood, 99% is associated with the red blood cells (erythrocytes). The remaining 1% is in the plasma, where it can be released to tissues. The blood lead not retained is either excreted by the kidneys, or through bile, enters the gastrointestinal tract. In exposures to a single dose of lead, one-half of the lead from the original exposure remains in the blood about 25 days after exposure, in soft tissues about 40 days, and in stable bone more than 25 years. Consequently, after a single exposure a person's blood-lead concentration may begin to

return to normal, but the total body burden (amount of lead in the body) may still be elevated. Lead exposure does not need to be acute for lead poisoning to occur. It is the total body burden, accumulated over a lifetime, that is related to the risk of adverse health effects.

2.2.1 Physiological Mechanisms

Lead affects the cellular organelle structures and processes as well as the general functioning of the body, which results in neurotoxicity, hematological effects, possible hypertension, kidney damage, reproductive dysfunction, developmental abnormalities, etc. These effects are described in Section 2.3.

The biological basis of lead toxicity is its ability to bind (attach) to substances crucial to various physiological functions. In this way, lead may interfere with cell functions by competing with native (substances normally found in the body), essential metals for binding sites, inhibiting enzyme activity, and inhibiting or otherwise altering essential ion transport. These effects are modulated by the stability of the binding site, how the lead is distributed in the body, and the differences in biochemical organization of different cells and organs. As a result, there is no single, well-defined mechanism that explains how lead works in all tissues in humans.

Studies on the mechanism of lead toxicity at the cellular level appear to implicate the mitochondria (energy powerhouse in the cell) and membranes (both cellular and intracellular) as primary targets for lead (EPA, 1986).

Lead effects mitochondria in numerous ways. These include structural changes and marked disturbances in mitochondrial function within the cell, especially energy metabolism and ion transport. These effects are associated with the accumulation of lead within the mitochondria. Structural changes include the swelling of mitochondria, and the distortion and loss of the small inner folds, called cristae, which carry many enzymes. The uncoupling of energy metabolism, inhibition of cellular respiration, and altered activities of intracellular calcium in mitochondria due to lead have been demonstrated in many studies. These investigations have particularly been concerned with the effects of lead on the brain, heme synthesis, and erythropoiesis (formation and production of red blood cells).

Lead also effects cellular and intracellular membranes and the mitochondria by altering ion transport, particularly calcium. This leads to the inhibition of enzymes and interferes with

normal transport systems. The overall impact of these effects is to disturb the development and functioning of many of the major organ systems in the body, particularly the central nervous system, resulting in significant adverse health effects.

2.2.2 Neurotoxic Effects of Lead

The data assessing the neurotoxicological mechanisms of lead provide limited information about how lead affects the nervous system. For over a decade the hippocampus was thought to be the principal target of lead in the brain. The hippocampus was selected because: 1) the hippocampus contains relatively high concentrations of zinc, and zinc-dependent functions may be sensitive to lead, 2) the hippocampus contains a dense plexus of cholinergic fibers that are affected by lead exposure, and 3) the hippocampus is functionally related to behaviors involving memory and learning (Petit, 1983). More recent investigations have shown that other areas, particularly the mesolimbic system (Moresco, 1988, Lasley, 1988) where low levels of lead have been found, cannot be excluded. Continuing research may help to determine which areas of the brain have an affinity for lead.

Lead is an ion and the hypothesis for its molecular mechanism has been based on its interaction with other physiologically important ions like calcium (Pounds, 1984) and zinc (Fowler, 1989). In addition, the activity of protein kinase C (Markovac, 1988) is effected by lead due to its zinc binding sites. Calcium and zinc are found in a multitude of sites and reactions throughout the body. It is thought, however, that there must be other, more specific events that determine how lead acts at a defined site. How this occurs is unknown at this time.

A number of scientists working on the neurotoxicity of lead discuss the proposed mechanisms of how lead affects the nervous system. Among these scientists, Silbergeld (1992) and Bellinger (1995) both discuss possible mechanisms for lead neurotoxicity in the context of neurodevelopmental (effects occurring during development of the nervous system), and neuropharmacological (interaction of lead with cells of the brain) effects.

Neurodevelopmental Effects: During development, the central nervous system (the brain and spinal cord) goes through a number of programmed changes involving the overall growth in cell numbers, size in the organ, and proliferation and outgrowth of cells that establish connections between cells. Many factors regulate these processes, including growth factors,

neurotransmitters functioning as trophic agents, and glycoprotein cell adhesion molecules (Jacobson, 1990).

One of the potential mechanisms for lead's effect on the developing brain has been investigated by Goldstein (1990), who suggests that the immature endothelial cells forming the capillaries of the developing brain are less resistant to the effects of lead than are capillaries from mature brains. As a result, blood carrying lead to the brain may easily pass into the newly forming compartments of the brain and effect many parts of this developing organ. In comparison, the capillaries of adults are developed and help to prevent the passage of ions like lead across the blood-brain barrier. It has been suggested that lead may affect the differentiation of capillary endothelial cells from the fetal brain in a similar way to its effects on neurons undergoing development (Bressler and Goldstein, 1991). This provides a neurotoxicological basis for the observed increased risk to pregnant women, infants, and young children of exposure to lead.

Silbergeld (1990) found that exposure of fetal animals to lead affects both regional growth and neuron-specific differentiation/synaptogenesis (development of synapses) in the central nervous system. Of these, synaptogenesis appears to be the more sensitive (Silbergeld, 1991; Regan, 1989), and lead is thought to interfere with the normal development of synapses in the brain. A synapse is a junction where the axon of one neuronal cell (or neuron) terminates with the dendrite of another neuron. Nerve impulses move from one nerve cell to another by traveling through the synapse. The normally functioning brain seems to exhibit a deletion of synapses that are unused. Those synapses which are frequently used are kept and strengthened. Goldstein (1990, 1992) suggests that lead may disrupt, or delay, this normal synaptic developmental process and that perhaps the resulting connections in the brain are "poorly chosen," leading to functional impairment in the brain. Although this hypothesis is speculative, lead's ability to facilitate the unstimulated release or prevent the stimulated release of neurotransmitters, which are important for the morphological organization of neurons, may be related to how neurons are chosen to survive (Audesirk, 1985). This may result in a nervous system that appears normal but in which cell to cell connections are not normal. These abnormalities then may be translated into the kind of neurobehavioral deficits which result in cognitive and behavioral deficits.

At the age of two the synaptic density and number of synapses per neuron peaks in the frontal cortex in humans. It is suggested that this area of the brain is an important target for lead (Huttenlocher, 1979). The age of two is also important from the standpoint of scientists evaluating lead toxicity outcome. Many studies have been conducted at this age because blood-lead concentrations tend to peak at age two, children are more cooperative for assessment at this age, hand to mouth activity is greatest, and level of cognitive ability is sufficiently developed.

Neuropharmacological Effects of Lead: Lead may also act as a neuropharmacological toxicant in the brain (Silbergeld, 1992; Bellinger, 1995). Silbergeld (1992) discusses how lead interferes with the synaptic mechanisms of the release of neurotransmitters and signal transduction. These effects are due to the presence of lead in the synapse. Theoretically, these effects are reversible if lead is removed. However, exposure to lead for a long time may result in permanent alteration in cellular responsiveness at pre- and post-synaptic levels. These pharmacologic effects may include the effects of lead to facilitate transmitter release, modulate ion conductance and, as a result, alter the electrophysiological output of the neuron.

Disruption of ion transport at membranes may be the mechanism by which lead produces its pharmacologic effects in the nervous system. Lead can also substitute for calcium and zinc in ion transport events at the synapse. These events include sodium channels, calcium channels, calcium-binding modulators like calmodulin, messengers like adenyl cyclase and protein kinase C (Bressler, 1991). Lead may affect ion channels by occupying zinc-binding sites and preventing ion movements (Alkodon, 1990).

At the neuron, lead seems to be more disruptive inside than outside. For example, inside the neuron, mitochondrial release of calcium is quite sensitive to lead (Silbergeld, 1975). Protein kinase C, which is very sensitive to lead, modulates receptor currents affecting long-term potentiation and other forms of synaptic response that may underlie learning and memory (Markovac, 1988). Dopamine sensitive adenyl cyclase, Na,K-ATPase, is also relatively sensitive to lead (Ewers, 1980, Fox, 1991).

Outside the neuron, neurotransmitter release or transmitter-gated ion channels are sensitive to lead at higher concentrations (Alkondon, 1990; Minnema, 1986; Kostial, 1957; Silbergeld, 1974l; Audesirk, 1985). If lead is kept out of the neuron, relatively high levels of lead are required to affect function. However, lead can enter certain neurons under the right

conditions (Sulzer, 1987; Silbergeld, 1977). Under these conditions mitochondria, protein kinase C, and other intracellular sites become accessible. The differential ability to prevent lead entry may be an important protective mechanism to prevent neurotoxicity. There has been speculation of a lead-binding protein in humans (DuVal, 1989) which may serve to concentrate and transport lead to certain parts of the brain.

Peripheral Neuropathy: Lead induces degeneration of the protective Schwann cells in the motor neurons of the peripheral nervous system, which causes segmental loss of the myelin covering of the neuron and possible neuron degeneration (Fullerton, 1966). Dyck (1980) and Windebank (1980) suggest that lead induces a breakdown in the blood-nerve barrier, allowing lead and fluid to enter the endoneurium, and disruption of myelin membranes. The degeneration of sciatic and tibial nerve roots is also possible. Sensory nerves are less sensitive to lead than motor nerves. Peripheral neuropathy is usually present only after prolonged high exposure to lead. Peripheral neuropathy may be reversible or permanent depending on the severity of exposure. Motor nerve dysfunction has been assessed clinically by the electrophysiologic measurement of nerve conduction velocities and shown to occur at blood-lead levels as low as 40 μg/dL.

To summarize, the mechanisms for lead neurotoxicity are not well understood. Several mechanisms have been proposed which seek to explain why children are more sensitive than adults to lead and how lead acts molecularly to effect the nervous system. Because most of the molecular events in the nervous system are also found throughout the rest of the body, it is difficult to explain why the nervous system is the most sensitive system in the body to lead.

2.2.3 Hematologic Effects of Lead

Red blood cells, which carry oxygen to body tissues, develop in the bone marrow of the body. Lead has adverse affects on heme synthesis (the formation of hemoglobin), which can result in anemia, and red blood cell formation, which can result in decreased life span of these cells.

Hemoglobin constitutes 90% of red blood cells. Hemoglobin consists of globin protein and heme, which is a metal complex consisting of an iron atom in the center of a porphyrin structure and provides the red color to hemoglobin.

Effect of Lead on Heme Synthesis: When an individual is exposed, lead quickly reaches the blood, circulates in the body, and enters different tissues including the bone marrow, where it can have an impact on various parts of the formation of heme. The process of heme biosynthesis starts with glycine and succinyl-coenzyme A, proceeds through the formation of a protein entity called protoporphyrin IX, and culminates with the insertion of iron into the porphyrin ring to form heme. In addition to being a constituent of hemoglobin, heme is found in many hemoproteins, such as myoglobin, the P-450 component of the mixed-function oxygenase system, and the cytochromes of cellular energetics. Therefore, disturbing heme biosynthesis by exposure to lead poses the potential for multiple-organ toxicity.

Lead's effects on the heme synthesis pathway are: 1) stimulation of mitochondrial δ-aminolevulenic acid synthetase (ALA-S), which mediates formation of δ-aminolevulinic acid (ALA), 2) direct inhibition of the cytosolic enzyme, δ-aminolevulenic acid dehydrase (ALA-D) which catalyzes formation of porphobilinogen from ALA, and 3) inhibition of insertion of iron into protoporphyrin IX to form heme, a process mediated by ferrochelatase.

Lead's mechanism of action seems to be due to its effect on cellular mitochondria. Lead enters the mitochondria of the cell where it impairs mitochondrial function and exercises many of its effects on the production of heme. In the mitochondria, lead increases the activity of the enzyme ALA-S, which increases the amount of ALA that is formed. Lead, in the cytosol of the cell, also decreases the activity of ALA-D, an enzyme which catalyzes reactions of ALA to form other molecules in heme biosynthesis. The result is an increase in the level of ALA.

Ferrochelatase, an enzyme also found in the mitochondria, catalyzes the incorporation of iron into protoporphyrin IX to form heme. Lead tends to inhibit ferrochelatase from incorporating the iron into the protoporphyrin ring, thereby preventing the formation of heme. Instead, there is an increase in erythrocyte protoporphyrin in the red blood cells. Erythrocyte protoporphyrin (EP) can be measured in blood as zinc protoporphyrin (ZPP) or free erythrocyte protoporphyrin (FEP).

Effect of Lead on Hemoglobin Production and Red Cell Formation: As described above, heme production is decreased by lead. Heme production regulates globin production so that globin production is also decreased, resulting in the decreased production of hemoglobin. These effects can lead to anemia (reduction in circulating red blood cell mass). Lead can induce

two types of anemia. Acute high-level lead poisoning has been associated with hemolytic (excessive red blood cell destruction) anemia. In chronic lead poisoning, lead induces a hypochromic (light colored red cells) normocytic (normal size red cells) anemia by both interfering with normal hemoglobin production and by diminishing red cell survival (cells are incapable of functioning in a normal manner and the life span is shortened).

The molecular mechanism for the diminished red cell life span is thought to be due to lead's inhibition of the enzymes (Na+, K+)-ATPase and pyrimidine-5-nucleotidase (Py-5-N). With enzyme inhibition there is irreversible loss of potassium ion from the cell with undisturbed input of sodium into the cell, resulting in a relative increase in sodium. Because the cells tend to shrink, there is an increase in sodium concentration which results in increased mechanical fragility and cell lysis (destruction of cells through rupture of cell membrane) in lead-induced anemia. It has also been suggested that enzymes (like Py-5-N and glucose-6-phosphate dehydrogenase) that help stabilize the red cell membrane may be effected by lead.

2.3 HEALTH EFFECTS OF LEAD EXPOSURE

Lead is a powerful toxicant with no known beneficial purpose in the human body (ATSDR, 1988a). The toxic effects of lead are seen primarily in the central nervous system, but virtually all parts of the body can be damaged at high exposure levels. Acute lead poisoning, associated with blood-lead levels above 70 μg/dL, causes abdominal pains, vomiting, and diarrhea. Without proper treatment, lead poisoning can result in convulsions, coma, and even death. At lower exposure levels, subtle neurological effects are of most concern.

Although occupational exposure to lead is dangerous and the subsequent health effects are well-documented, infants and young children are more at risk from lead exposure than are adults, as their neurological systems are developing and are more vulnerable to damage. At the same time, their frequent hand-to-mouth activities bring them into greater contact with lead in the environment and their bodies absorb a larger percentage of ingested lead than do those of adults. The increased risk of lead exposure appears to be most evident at age 2 (Clark, 1985; Goyer, 1993), and studies have shown a strong association between blood-lead concentration measured at age 2 and IQ scores later in life (Bellinger, 1992; Schwartz, 1994; Pocock, 1994). Because lead is readily transferred across the placenta, a developing fetus is at risk for lead

exposure and toxicity. For this reason, women of childbearing age are also a population of concern.

Over time, the lead exposure levels at which adverse health effects are reported have declined dramatically. Although cases of severe lead poisoning still occur, recent research has focused on the effects of chronic, low-level lead exposure. At blood-lead levels as low as 10 to 15 μg/dL, researchers have documented slower reaction times, reductions in intelligence and short-term memory, other neurobehavioral deficits, adverse effects on heme (iron in blood) biosynthesis, and on vitamin D and calcium metabolism. In addition, longitudinal studies have shown reductions in gestational age and birth weight associated with prenatal blood-lead levels of 10 to 15 μg/dL. At or above 40 μg/dL, children may experience reduced hemoglobin, the accumulation of a potential neurotoxicant known as ALA, and mild anemia. Very severe lead poisoning involving symptoms such as kidney failure, gastrointestinal problems, coma, convulsions, seizures, encephalopathy, and pronounced mental retardation, can occur at blood-lead levels of 60 μg/dL or higher. Specific health effects of lead exposure, the blood-lead levels at which these effects have been observed, and the scientific literature in which the effects were reported are summarized in Table B-1 in Appendix B. This table is reproduced from the *Toxicological Profile for Lead* (ATSDR, 1993).

2.3.1 Neurological Effects of Lead

The most severe neurological effect of lead in adults and children is lead encephalopathy, a general term used to describe various diseases that affect brain function. Early symptoms include dullness, irritability, poor attention span, headache, muscular tremor, loss of memory, and hallucinations. The condition may worsen, sometimes abruptly, to delirium, convulsions, paralysis, coma, and death (Kumar, et al., 1987). While physical symptoms of lead poisoning can be treated, the effects on the central nervous system may be irreversible. Long-lasting impacts on intelligence, motor control, hearing, and emotional development of children have been documented at levels of lead in the body that are not associated with obvious symptoms and were once thought to be safe.

Effects on Adults: Occupational exposure to lead has often been associated with subjective signs of neurotoxicity. The literature contains numerous case reports and small cohort

studies that describe symptoms such as malaise, forgetfulness, irritability, lethargy, headache, fatigue, and dizziness at blood-lead levels that range from 40 to 120 μg/dL, following acute, intermediate, and chronic-duration occupational exposure to lead (Awad, et al., 1986; Baker, et al., 1979; Haenninen, et al., 1979; Holness and Nethercott, 1988; Marino, et al., 1989; Matte, et al., 1989; Pagliuca, et al., 1990; Pasternak, et al., 1989; Pollock and Ibels, 1986; Schneitzer, et al., 1990).

Neurobehavioral testing has revealed effects of lead in adults at blood-lead levels between 40 and 80 μg/dL, well below the levels that cause encephalopathy (120 μg/dL – EPA, 1986). Disturbances in oculomotor function (saccadic eye movements) have been observed in lead workers with mean blood-lead levels of 57 to 61 μg/dL (Baloh, et al., 1979; Spivey, et al., 1980; Glickman, et al., 1984). Disturbances in reaction time, visual motor performance, hand dexterity, IQ test and cognitive performance, nervousness, mood, or coping ability were observed in workers with blood-lead levels of 50 to 80 μg/dL (Arnvig, et al., 1980; Haenninen, et al., 1978; Hogstedt, et al., 1983; Mantere, et al., 1982; Valciukas, et al., 1978). However, there is some evidence to the contrary. No neurobehavioral effects were observed in a study of 288 randomly selected lead workers with mean blood lead of 40.1 μg/dL, compared to 181 demographically similar controls with mean blood lead of 7.2 μg/dL (Ryan, et al., 1987).

Numerous studies measure the conduction velocity of electrically stimulated nerves in the arm or leg of occupationally exposed workers. Nerve conduction velocity (NCV) is used to measure slowed reaction times associated with lead exposure and is considered a sensitive indicator of lead toxicity. Studies indicate that NCV effects occur in adults at blood-lead levels below 70 μg/dL, possibly as low as 30 μg/dL. Decreased NCV has been observed in both prospective and cross-sectional studies (Seppalainen, et al., 1983; Rosen, et al., 1983; Treibig, et al., 1984; Araki, et al., 1980). There is some evidence indicating that changes in NCV associated with lead exposure may be transient (Araki, et al., 1980; Muijser, et al., 1987).

Effects on Children: High-level lead exposure produces encephalopathy in children, starting at approximately 80 to 100 μg/dL (NAS, 1972; Bradley and Baumgartner, 1958; Bradley, et al., 1956; Gant, 1938; Rummo, et al., 1979; Smith, et al., 1983; EPA, 1986). However, low-level exposure also may result in long-lasting impacts on intelligence, motor control, hearing, and neurobehavioral development of children.

Results are available from four large-scale, longitudal studies conducted in Boston, Cincinnati, Cleveland, and Port Pirie, Australia. These studies indicate that disturbances in early neurobehavioral development occur at exposure levels that until recently were considered safe, or even normal. In the Boston study, 4-8 point differences in performance on the Bayley Mental Development Index (MDI) were reported at 6, 12, 18, and 24 months, after adjusting for other covariates, between low (prenatal mean of 1.8 μg/dL) and high (prenatal mean of 14.6 μg/dL) exposure infants (Bellinger, et al., 1985a, 1985b, 1986a, 1986b, 1987a). These findings were confirmed in more recent studies (Bellinger, et al., 1989a, 1989b). Additional follow-up showed that deficits in McCarthy General Cognitive Index scores at age 5 were significantly correlated with blood-lead levels at age 24 months, although not with prenatal blood lead measures. Similar results were reported in the Cincinnati study (Dietrich, et al., 1986, 1987a, 1987b). In addition, study results suggest that the effect of prenatal lead exposure on the MDI was mediated in part through its effects on birth weight and reduced gestational age, which were each significantly associated with MDI scores (Dietrich, et al., 1987a). Results reported for the Cleveland study were mixed, but while the authors tended to conclude that there was not strong evidence of developmental effects of lead (Ernhart, et al., 1985, 1986, 1987, 1988; Wolf, et al., 1985; Ernhart and Green, 1990), other reviewers suggest that such effects may be inferred from the reported results (Davis and Svendsgaard, 1987; EPA, 1986; ATSDR, 1993). In the Port Pirie study, reduced MDI scores at 24 months were associated with postnatal blood-lead levels measured at age 6 months, but not with prenatal exposure measured through cord and maternal blood-lead levels (Baghurst, et al., 1987; Vimpani, et al., 1985, 1989; Wigg, et al., 1988). Results of a follow-up neurobehavioral assessment conducted at age 3 to 4 years, using the McCarthy Scales of Children's Abilities, indicated significant associations between postnatal blood-lead levels (geometric means of 14 μg/dL at 6 months and approximately 21 μg/dL at 15 and 24 months) and ability test scores (McMichael, et al., 1988).

In addition, all four studies report lower IQ scores at school-age for children who had earlier exhibited elevated blood-lead levels. In Boston, slightly elevated blood-lead levels at age 24 months (mean of 6.5 μg/dL) were associated with intellectual and academic performance deficits at age 10 years (Bellinger, 1992). In Cincinnati, postnatal blood-lead levels measured through age 3 years were inversely associated with IQ scores measured at age 5, although the

effect was not statistically significant when adjusted for covariates (Deitrich, et al., 1993). In Cleveland, a significant association was reported between blood-lead concentration at age 2 (mean of 16.7 μg/dL) and IQ measured at 5 years (Ernhart, et al., 1989). In Port Pirie, statistically significant associations were reported between IQ measured at age 7 and blood-lead levels from birth through age 7, with the strongest associations for blood-lead levels measured at 15 months to 4 years (Baghurst, et al., 1992).

Taken together, these studies provide strong evidence that low-level prenatal or early postnatal exposure to lead results in neurobehavioral developmental delays through age 5. Strong relationships between blood-lead concentration in early childhood, age 15 months to 4 years, and IQ scores were also reported, even when only slight elevations in blood-lead levels were present.

Additional evidence of IQ point loss associated with elevated blood-lead levels in school-age children is reported in cross-sectional studies throughout the world. A study of Danish children related tooth-lead concentration to performance on several psychometric tests (Hansen, et al., 1989). Children with elevated tooth-lead levels (above 18.7 μg/g) were matched by sex and socioeconomic status with children with lower levels (below 5 μg/g). High lead children scored lower on the Wechsler Intelligence Scales for Children (WISC) IQ test than children with lower lead levels, although no difference in scores was observed for the Performance IQ and several experimental tests. Impaired neuropsychological functioning due to lead exposure was observed through differences in performance on the Bender Visual Motor Gestalt Test and on a behavioral rating scale. A study of school children in Edinburgh, Scotland (Fulton, et al., 1987) found that elevated blood-lead levels (mean of 11.5 μg/dL) were associated with lower scores on IQ tests and on mathematical and reading attainment tests, after adjusting for covariates. No threshold in the relationship, below which lead does not have an effect on intelligence and attainment, was observed even for blood-lead concentrations below 10 μg/dL. A study of Chinese children (Wang, et al., 1989) also reported a significant dose-response relationship between blood-lead concentration (above 10 μg/dL) and IQ scores, after adjusting for covariates.

A significant effect of lead on IQ is not uniformly reported, however. Children randomly selected from birth records in Birmingham, United Kingdom, were assessed using a variety of cognitive, performance, neuropsychological, and behavioral endpoints (Harvey, et al., 1988).

The effect of lead (mean of 13.5 μg/dL) was not significant for most endpoints, and for none of the three IQ measures. Both tooth lead and blood lead were examined as predictors of intelligence in a study of 6 year old children in London (Smith, et al., 1983; Pocock, et al., 1989). Neither measure of lead exposure was a significant predictor, once social factors were controlled. No evidence of an association between blood-lead levels (mean of 12.75 μg/dL) and intelligence was reported in another study of London children that included more middle class families (Lansdown, et al., 1986).

A possible explanation for these seemingly contradictory results is that the effect of lead on IQ may be overshadowed by the effects of home and societal factors, such as birth order, parental IQ and level of education, and socioeconomic status. For example, a study of 104 children under age 7 and of lower socioeconomic status indicated that MDI and IQ scores were significantly associated with blood-lead levels ranging from 6 to 59 μg/dL, after controlling for socioeconomic and other factors (Schroeder, et al., 1985). In a five-year follow-up of 50 of these children, IQ was inversely correlated with initial and concurrent blood-lead levels, but the effect of lead was not significant when socioeconomic status and other covariates were included in the analysis (Schroeder and Hawk, 1987). However, in a replication of the study among children of uniformly low socioeconomic status, the effect of lead was evident at both the initial and five-year follow-up (Hawk, et al., 1986; Schroeder and Hawk, 1987). These results suggest that the effects of lead may be more easily detected in groups with similar home and societal backgrounds.

Both current and long-term indicators of lead-exposure were studied to establish which indicator was best correlated with psychometric test scores (Bergomi, et al., 1989). Total and verbal IQ scores were negatively correlated with tooth-lead levels and ALAD activity. Tooth-lead levels were also negatively correlated with Toulouse Pieron test results, which evaluate ability figure identification, discrimination, and attention. The most predictive measure of lead exposure was tooth lead, which is indicative of cumulative lead exposure. Blood lead, which is indicative of a mix of current and past exposure, and hair lead, which is indicative of short-term exposure, had little predictive value in this study.

The effect of lead on IQ and other developmental indicators is well-established for children with markedly elevated blood-lead concentrations. For example, five point IQ

decrements, fine motor dysfunction, and altered behavioral profiles were reported among preschool children exhibiting pica for paint and plaster, whose blood-lead levels were greater than 40 μg/dL (mean of 58 μg/dL), when compared with matched controls who did not eat paint and plaster (de la Burde and Choate, 1972). At age 7 to 8, three point IQ decrements and impairments in learning and behavior were reported for these children, even though blood-lead levels had declined (de la Burde and Choate, 1975). Blood-lead concentrations for control children were not reported, but, given the timing of the study, children in the control population may have had what would now be considered elevated blood-lead levels. A study that included children who had previously had encephalopathy indicated that these children had increased incidence of hyperactivity and IQ decrements of approximately 16 points resulting form lead exposure (Rummo, et al., 1979). In the same study, asymptomatic children with long-term exposure (means of 51-56 μg/dL) had IQ decrements of 5 μg/dL on average, compared to control children (mean of 21 μg/dL).

A study of the long-term effects of low-level lead exposure found that children with higher dentin lead levels were more likely to drop out of high school and have a reading disability (Needleman, et al., 1990). Higher lead levels were also associated with lower ranking in high school class and increased absenteeism. Lower scores on vocabulary and grammatical-reasoning tests were reported, along with poor hand-eye coordination, delayed reaction times, and slowed finger tapping, compared to children with lower lead exposure. Earlier results indicated that children with high dentin lead levels had deficits in IQ scores, speech and language processing, attention, and classroom performance in first and second grades (Needleman, et al., 1979). IQ deficits continued through the fifth grade. In addition, children with higher lead levels needed more special academic services, and had a higher failure rate in school (Bellinger, et al., 1986c).

A lead-related decrease in hearing acuity has been reported in young children, with hearing thresholds at 2000 Hz increasing with blood-lead levels in the range of 6 to 59 μg/dL (Robinson, et al., 1985). Analysis of NHANES II data indicated that the probability of increased hearing thresholds at 500, 1000, 2000, and 4000 Hz was associated with increased blood-lead levels from below 4 μg/dL to over 50 μg/dL. In addition, this study reported increased

probability a child was hyperactive and delays in developmental milestones (age at which child first sat up, walked, and talked) associated with elevated blood lead (Schwartz and Otto, 1987).

2.3.2 Hematological Effects of Lead

The effects of lead on the blood's biochemical functions are interrelated and have variable biological impact. Heme (the component of blood that contains iron) is critical to the basic function of many organ systems, including the blood-forming tissue, liver, brain, and kidneys. As noted earlier, lead can disturb the formation of hemoglobin (red blood cells), which may cause anemia at high exposure levels. The heme-mediated generation of an important hormonal metabolite of vitamin D (1,25-dihydroxyvitamin D) may be disturbed by lead. This hormone serves a number of functions in humans, including the regulation of calcium metabolism. In addition to the direct effects of lead on heme biosynthesis, there are potentially significant indirect impacts on the central nervous system, caused by the accumulation of the potential neurotoxicant, ALA. Lead also inhibits coproporphyrin utilization and the conversion of zinc erythrocyte protoporphyrin (ZPP) into heme. The effects of lead on heme biosynthesis are described in detail in Section 2.2.3 and in the *Air Quality Criteria for Lead* (EPA, 1986).

The threshold blood-lead level for a decrease in hemoglobin is approximately 50 μg/dL in occupationally exposed adults and 40 μg/dL in children (EPA, 1986). However, adverse effects on hematocrit may occur at even lower blood-lead levels in children. In a cross-sectional study of 579 children ages 1 to 5 years, a strong association between blood-lead level and the probability of anemia was observed between 20 and 100 μg/dL, with the strongest effect in the youngest children. In this study, anemia (defined as hematocrit below 35%) was not observed at lead levels below 20 μg/dL (Schwartz, et al., 1990).

Anemia is not usually an early manifestation of lead poisoning and is evident only when the blood-lead level is significantly elevated for prolonged periods. Some of the hematologic signs of lead poisoning resemble other diseases or conditions. Two rare diseases, acute intermittent porphyria (a group of diseases with unusual and characteristic manifestations, which have in common the excretion of porphyrins) and coproporphyria (high excretion of coproporphyria), also result in heme abnormalities similar to those of lead poisoning. Nutritional deficiencies may increase the development of anemia since lack of proper vitamins and minerals

may result in iron deficiency anemia. Iron deficiency makes lead induced anemia worse in children and vice versa. Lead also exacerbates hemolytic anemia associated with vitamin E deficiency by enhancing mechanical fragility of cells.

Lead-induced disturbances in red blood cell formation and maturation also occur by way of alterations in pyrimidine metabolism, as described in Section 2.2.3. Erythrocite Py-5^1-N activity is inhibited in lead workers, with the greatest inhibition and marked accumulations of pyrimidine mucleotides apparent in workers with overt intoxication, including anemia (Paglia, et al., 1975, 1977). Erythrocyte Py-5^1-N activity is inhibited in children at very low blood-lead levels, with no threshold apparent (Angle and McIntire, 1978; Angle, et al., 1982). The adverse effects of decreased Py-5^1-N activity at low blood-lead levels, in the absence of detectable effects on hemoglobin levels and erythrocyte function or survival, are not known.

Lead can inhibit ALA-D activity and stimulate ALA-S activity, which results in accumulation of ALA in the body and excretion. ALA may be neurotoxic at higher levels. General population studies indicate that ALA-D activity is inhibited at very low blood-lead levels, with no threshold apparent, in adults (Hernberg and Nikkanen, 1970; Roels, et al., 1976), children (Chisolm, et al., 1985; Roels and Lauwerys, 1987), and newborns (cord blood) and their mothers at delivery (Lauwerys, et al., 1978). The adverse effects of the decreased ALA-D activity observed at low blood-lead levels are not known.

2.3.3 Other Effects of Lead

Death: It is well known that severe lead poisoning can lead to encephalopathy and death. There is some evidence, too, of higher death rates due to cerebrovascular disease among lead workers (Fanning, 1988; Malcolm and Barnett, 1982; Michaels, et al., 1991). In infants, high levels of lead have been suggested as a causative agent in Sudden Infant Death Syndrome (SIDS) (Drasch, et al., 1988).

Hypertension: There may be a relationship between lead exposure and hypertension. Increased heart rate and hypertension have been noted in occupationally exposed workers after exposure to high levels of lead following exposure durations of as short as four weeks (Marino, et al., 1989). Hypertension has also been associated with lead exposure in the general populations (Khera, et al., 1980b; Pirkle, et al., 1985), although the evidence is mixed (Pocock,

et al., 1984, 1985, 1988; Gartside, 1988; Coate and Fowles, 1989). Cardiovascular effects other than blood pressure changes have been observed in individuals occupationally exposed to lead (electrocardiographic (ECG) abnormalities – Kosmider and Petelenz, 1962; ischemic ECG changes – Kirkby and Gyntelberg, 1985).

Gastrointestinal Effects: Colic is a consistent early symptom of lead poisoning in occupationally exposed cases, in individuals acutely exposed to high levels of lead, such as occurs during the removal of lead-based paint. Colic is characterized by a combination of the following symptoms: abdominal pain, constipation, cramps, nausea, vomiting, anorexia, and weight loss. Although gastrointestinal symptoms typically occur at blood-lead levels of 100 to 200 μg/dL, they have sometimes been noted in workers whose blood-lead levels were as low as 40 to 60 μg/dL (Studies listed in Table B-1). EPA has established a lowest observed adverse effect limit (LOAEL) of 60 to 100 μg/dL for colic in children (EPA, 1986).

Renal Effects: The characteristics of early, or acute, lead-induced nephropathy (kidney disease) include nuclear inclusion bodies, mitochondrial changes, and cytomegaly of the proximal tubular epithelial cells; disfunction of the proximal tubules (Fanconi's syndrome) manifested as aminoaciduria, glucosuria, and phosphaturia with hypophosphatemia; and increased sodium and decreased uric acid excretion. These effects appear to be reversible. Characteristics of chronic lead nephropathy include progressive interstitial fibrosis, dilation of tubules and atrophy or hyperplasia of the tubular epithelial cells, and few or no nuclear inclusion bodies, reduction in glomerular filtration rate, and azotemia. These effects are irreversible. The acute form is reported in lead-intoxicated children and sometimes in lead workers. The chronic form is reported mainly in lead workers. A summary of studies reporting acute or chronic nephropathy may be found in ATSDR, 1993. Additional detail is reported in EPA, 1986.

Vitamin D Metabolism: Lead appears to interfere with the conversion of vitamin D to its hormonal form, 1,25-dihydroxyvitamin D. Evidence for this effect comes primarily from studies of children with high lead exposure (Rosen, et al., 1980; Mahaffey, et al., 1982). However, the effect of lead on vitamin D metabolism may only be apparent in children with chronic nutritional deficiency and chronically elevated blood-lead levels (Koo, et al., 1991).

Thyroid: Limited evidence from occupationally exposed workers suggests that lead may adversely affect thyroid function (Tuppurainen, et al., 1988). However, no effects of lead on thyroid function have been found in children (Siegel, et al., 1989).

Growth: A number of epidemiological studies have reported an association between blood-lead levels and growth in children (Nye, 1929; Johnson and Tenuta, 1979; Lauwers, et al., 1986; Schwartz, et al., 1986; Lyngbye, et al., 1987; Angle and Kuntzelman, 1989). However, a study of lead-poisoned subjects and nonexposed sibling controls failed to establish an association between blood-lead levels and growth or the genetic predisposition for adult height (Sachs and Moel, 1989). Moreover, a recent longitudinal study in Cleveland found no statistically significant effect of blood-lead levels on growth (height, weight, and head circumference) from birth through age 4 years and 10 months (Greene and Ernhart, 1991). Growth rates, measured as covariate-adjusted increases in stature from 3 and 15 months of age, were inversely correlated with corresponding increases in blood-lead levels in a longitudinal study of 260 infants in Cincinnati (Shukla, et al., 1987, 1989).

Development: Lead-related effects on children's development, such as reduced birth weight, reduced gestational age, and neurobehavioral developmental deficits, have been reported. The evidence on birth weight and gestational age is mixed, with some studies reporting reductions associated with lead exposure (Moore, et al., 1982; McMichael, et al., 1986), while others report no differences (Needleman, et al., 1984; Factor-Litvak, et al., 1991; Green and Ernhart, 1991). The evidence on neurobehavioral development is more consistent, with most studies reporting an association between lead exposure and developmental deficits (Baghurst, et al., 1987; Vimpani, et al., 1985, 1989; Wigg, et al., 1988; Bellinger, et al., 1985a, 1985b, 1986a, 1986b, 1987a, 1989a, 1989b; Dietrich, et al., 1986, 1987a, 1987b). A short summary of these results is included in Section 2.3.1. There is some evidence that early developmental deficits may not persist until age 4-5 (Bellinger, et al., 1991). Finally, one study demonstrated an association between cord blood-lead levels and minor congenital anomalies (Needleman, et al., 1984), although lead was not associated with increased incidence of major congenital anomalies.

Immune System: The data on immunological effects of lead in occupationally exposed adults are inconsistent, but indicate that while lead may have an effect on the cellular component of the immune system, the humoral component is relatively unaffected (ATSDR, 1993). The

data on immunological effects of lead on children are very limited, but no effects have been detected (ATSDR, 1993; Reigart and Graber, 1976).

Reproduction: A large body of literature clearly indicates that high levels of lead cause adverse effects on both male and female reproductive functions. Women who are exposed to high levels of lead during pregnancy have experienced an increased rate of miscarriages and stillbirths (Nordstrom, et al., 1979; Baghurst, et al., 1987; McMichael, et al., 1986; Wibberly, et al., 1977). In addition, women who were significantly exposed during childhood may be at risk of spontaneous abortion and stillbirth and their children more likely to experience learning disabilities (Hu, 1991). Lead-induced effects on male reproductive functions, including reduced sperm production, have been reported in studies of occupationally exposed males (Chowdhury, et al., 1986; Assennato, et al., 1987; Lancrajan, et al., 1975; Wildt, et al., 1983). Reproductive effects of chronic low-level exposure are less known. A recent prospective study found no effect on the rate of spontaneous abortions among women who resided near a lead smelter (mid-pregnancy mean blood lead concentration of 15.9 μg/dL) (Murphey, et al., 1990).

Genotoxic Effects: Results of assays made following *in vivo* exposure from occupational sources are contradictory, but do suggest that lead may have an effect on chromosomes. While increased frequencies of chromosomal aberrations have been observed in occupationally-exposed workers,(Huang, et al., 1988b; Nordenson, et al., 1978), most of the available data show no such increase (Bauchinger, et al., 1977; Maki-Paakkanen, et al., 1981; O'Riordan and Evans, 1974; Schmid, et al., 1972). Sister chromatid exchanges may (Huang, et al., 1988b; Grandjean, et al., 1983; Leal-Garza, et al., 1986), or may not (Maki-Paakkanen, et al., 1981; Dalpra, et al., 1983) be increased as a result of lead exposure.

Cancer: The information available that has examined the association of occupational exposure to lead with increased cancer risk is generally limited in its usefulness because the actual compound(s) of lead, the route(s) of exposure, and level(s) of lead to which the workers were exposed were not reported. Furthermore, the potential for exposure to other chemicals, including arsenic, exists, particularly in lead smelters. Nonetheless, a statistically significant increase in total malignant neoplasms, largely due to small, statistically nonsignificant increases in digestive, respiratory, and urinary tract tumors has been observed among lead production workers (Cooper, 1976; Cooper and Gaffey, 1975; Kang, et al, 1980). In addition, a statistically

significant increase in rectal cancer was found in workers exposed to tetraethyl lead (Fayerweather, et al, 1991).

2.4 REPRESENTATIVE HEALTH EFFECTS

The childhood lead poisoning problem encompasses a wide range of exposure levels, with varying health effects at different levels of exposure. As described in the previous section, even low-level exposure to lead can result in adverse health effects. At low levels, the health effects may not be severe or obvious, but a large number of children are affected. As the exposure level increases, the severity of the health effects increases, but the number of children affected decreases.

Both individuals and society as a whole are damaged by adverse health effects associated with lead exposure. In this section, two representative effects, elevated blood-lead concentration and IQ point deficit, are identified to represent the spectrum of health effects of lead exposure. Each representative end point may be used both to estimate the number of children who will benefit under the proposed rule and also the economic benefit to society. The representative health effects and blood-lead concentrations are used in the integrated risk analysis in Chapter 5 to estimate the numbers of children who may benefit under the proposed §403 rule. The estimation of economic benefits was considered in selecting the health endpoints, as economic benefits resulting from the rule are estimated in the accompanying §403 RIA. Estimation of the benefits of reducing lead exposure requires selection of an age group for characterizing the health risks of lead exposure. The selection of the age group in this risk assessment was based on the most appropriate age of child for the estimation of health effects. The effects of lead exposure are thought to be strongest for fetuses, infants, and young children, because of their rapidly developing central nervous system and because the brain is a primary target organ of lead. Furthermore, the increased risk of lead exposure appears to be most evident at age 2 (Clark, 1985; Goyer, 1993) and studies have shown a strong association between blood-lead concentration measured at age 2 and IQ scores later in life (Bellinger, 1992; Schwartz, 1994; Pocock, 1994). Therefore, the health benefits of reducing childhood lead exposure are estimated at age 2 in this risk assessment.

2.4.1 Elevated Blood-Lead Concentration

Although an elevated blood-lead concentration is not a health effect in and of itself, the relationship between blood-lead concentration and adverse health effects is well-established. In addition, CDC guidelines on childhood lead poisoning prevention traditionally have been and currently are defined in terms of blood-lead concentrations. Table 2-1 summarizes CDC's recommended actions for children with elevated blood-lead concentrations (CDC, 1991). Based on these guidelines, two levels of elevated blood-lead concentration are used to estimate benefits under the proposed rule:

Incidence of blood-lead levels greater than 10 μg/dL: This level is the lowest blood-lead level at which a child is considered lead poisoned by CDC. While extensive interventions are not always recommended by CDC, children with blood-lead concentrations at or above 10 μg/dL require more frequent rescreening at minimum, and may require environmental or more extensive medical interventions.

Incidence of blood-lead levels greater than 25 μg/dL: This level is the blood-lead level at which extensive medical intervention may be necessary. Current CDC guidelines (CDC, 1991) recommend a provocative chelation test for children with blood-lead concentrations between 25 μg/dL and 44 μg/dL. It is further recommended that children with positive provocative chelation tests, and all children with blood-lead concentrations at or above 45 μg/dL, should receive one or more courses of chelation therapy. For these children, the medical intervention accompanies an environmental assessment, remediation of sources of lead, and parental education on ways to reduce lead exposure.

2.4.2 IQ Point Deficits

In this section, two IQ based endpoints are identified to represent the spectrum of neurotoxicological effects of lead. While tests that focus on a specific neurological effect might be more sensitive to the effects of lead than IQ tests, the selection of a representative effect is difficult. Differences in the level, timing, and route of exposure for individuals may result in differing effects of lead. For example, early exposure to lead (before age 2) may affect language skills, while later exposure is more likely to affect spatial-symbolic skills (Shaheen, 1984). In the absence of details of the exposure scenario, which are rarely available, exposure-related

Table 2-1. Interpretation of Blood-Lead Concentrations and Follow-Up Actions Recommended by CDC

Class	Blood-Lead Concentration (μg/dL)	Recommended Action
I	≤ 9	A child in Class I is not considered to be lead-poisoned. No action is recommended.
IIA	10 - 14	Many children (or a large proportion of children) with blood-lead levels in the range should trigger communitywide childhood lead poisoning prevention activities. Children in this range may need to be rescreened more frequently.
IIB	15 - 19	A child in Class IIB should receive nutritional and educational interventions and more frequent screening. If the blood-lead level persists in this range, environmental investigation and intervention should be done.
III	20 - 44	A child in Class III should receive environmental evaluation and remediation and a medical examination. Such a child may need pharmacologic treatment of lead poisoning. A provocative chelation test is recommended for children with blood-lead levels between 25 and 44 μg/dL.
IV	45 - 69	A child in Class IV will need both medical and environmental interventions, including chelation therapy.
V	≥ 70	A child with Class V lead poisoning is a medical emergency. Medical and environmental management must begin immediately.

differences will be most apparent on tests, such as IQ tests, that measure performance over a range of neurological functions (Bellinger, 1995). The relationship between blood-lead concentration and IQ scores has been reported consistently in the literature and is quantified by meta-analysis (Needleman and Gatsonis, 1990; Schwartz, 1993; Schwartz, 1994; Pocock, et al., 1994; Section 4.4; Appendix D). The following IQ-based health endpoints are used in the risk assessment to represent the neurotoxicological effects of lead exposure:

IQ Points Lost: This health effect is used to represent the neurological loss due to low level lead exposure. Lower IQ scores are associated with a lower level of educational attainment and lower life-time earnings.

Increased Incidence of IQ scores less than 70: This health effect is selected to represent the increased likelihood of mental retardation resulting from lead exposure. An IQ of 70 is two standard deviations below the population mean and is used as an indicator of mental retardation. Children who are mildly mentally retarded require special education classes in school. Children who are severely mentally retarded may require life-long institutional care.

3.0 EXPOSURE ASSESSMENT

CHAPTER 3 SUMMARY

The goal of this exposure assessment is to document the important sources of lead in the environment, to document the major pathways by which lead is exposed to children, to characterize the current distribution of environmental-lead levels in the nation's housing stock, and to characterize the current distribution of average blood-lead concentration among the nation's children. Information from the exposure assessment is used with the findings of hazard identification (Chapter 2) and dose response assessment (Chapter 4) to provide input to the risk characterization (Chapter 5).

The 1997 national housing stock is predicted to contain 99,272,000 occupied housing units, containing nearly eight million children aged 1 to 2 years. Of these units, approximately 62% are forecast to contain lead-based paint, and 14% to contain more than 5 ft^2 of deteriorated lead-based paint. In this risk assessment, the HUD National Survey is used to characterize the distribution of environmental-lead levels in these units. Data from this survey and other epidemiological studies suggest that lead levels in dust and soil tend to decrease with the age of the unit. Data from the Baltimore Repair and Maintenance Study provide strong evidence that environmental-lead levels are high in the presence of deteriorated lead-based paint. Data from the Rochester Lead-in-Dust Study indicate that lead levels can be high within urban environments and in older units.

Data from Phase I of the Third National Health and Nutrition Examination Survey (NHANES III) were used to characterize a pre-intervention distribution of children's blood-lead levels in the nation's housing stock. The geometric mean blood-lead concentration for children aged 1-2 years is 4.05 µg/dL, with a geometric standard deviation of 2.06. Slightly over 10% of these children are estimated to have blood-lead concentrations greater than 10 µg/dL. Blood-lead concentration data from the Baltimore Repair and Maintenance Study and the Rochester Lead-in-Dust Study indicate that blood-lead concentrations are higher in the presence of lead-based paint and high environmental-lead levels.

The goal of this exposure assessment is to document the important sources of lead in the environment, to document the major pathways by which lead is exposed to children, to characterize the current distribution of environmental-lead levels in the nation's housing stock, and to characterize the current distribution of average blood-lead concentration among the nation's children. Information from the exposure assessment is used with the findings of hazard identification (Chapter 2) and dose response assessment (Chapter 4) to provide input to the integrated risk analysis (Chapter 5).

Section 3.1 provides documented sources and pathways of lead exposure in the nation's residential environment. A number of epidemiological studies have investigated the extent to which lead is present in certain residential environments and how this lead exposure affects blood-lead concentration in children. These studies are introduced and summarized in Section 3.2.

It is of interest to assess lead exposure in that portion of the national housing stock in which children reside or can potentially reside (hereafter referred to simply as the "national housing stock"). Section 3.3 characterizes the extent to which lead exposure is present in the national housing and Section 3.4 characterizes the distribution of childhood blood-lead concentrations. These characterizations are provided for 1997, the year in which regulations developed in response to §403 are expected to be promulgated.

This chapter provides several sources of data on housing stock characteristics, population estimates, and environmental-lead levels in housing units. In the exposure assessment, these data have been used to make inferences on residential lead exposure to children in the United States. The extent to which any exposure assessment accurately portrays the exposure scenario of interest depends on the relevance and representativeness of the data used in the analyses and in the methods applied to these data to meet the objectives of the exposure assessment. Therefore, an effort has been made in this chapter to present the methods used, to identify assumptions and approximations made in the analysis and when they were made, and to identify uncertainties and limitations in the data. Supporting information and detailed results to accompany the information in this chapter are presented in Appendix C.

3.1 SOURCES AND PATHWAYS OF LEAD

Lead is a heavy, stable element occurring naturally in the earth's crust. Through natural activity such as crustal weathering and human activity such as mining, this metal has been distributed throughout the human environment. Lead's historic use as an ingredient in various manufactured and refined products has increased its introduction into the environment. As a result, lead has been detected in water, soil, air, plants, animals, and humans. As lead does not naturally biodegrade, its exposure potential tends to accumulate over time as more and more lead is deposited in the environment.

Research has identified a variety of environmental sources and reservoirs of lead which can contribute to overall lead exposure in a child. Figure 3-1 illustrates the major sources and reservoirs of lead, how lead is introduced into the human environment, and various pathways of human exposure. According to this figure, both natural sources (e.g., crustal weathering) and manufactured sources (e.g., auto and industrial emissions, paint and industrial dusts, solder, lead glazes) have contributed lead to various components of the human environment. These components act as lead reservoirs. Lead is exchanged among these reservoirs by various environmental pathways (e.g., ambient air to soil to dust). Lead in such media as inhaled air, dusts, food, or drinking water contributes to human lead exposure via direct pathways between these reservoirs and man. As data supporting the dangers of lead exposure have been identified, a combination of state and Federal action has curtailed the impact of certain sources and reservoirs of lead in the environment, resulting in a change in the predominance of historically significant sources.

In the scientific literature (e.g., Bornschein et al., 1986), quantitative exposure models, or *pathways models*, have been applied to data from environmental-lead studies to identify the most significant pathways by which residential, childhood environmental-lead exposure occurs and to provide quantitative estimates of the relative contributions of the numerous hypothesized exposure paths. These pathways models support Figure 3-1 by identifying direct and indirect effects of lead levels in the residential environment on lead concentrations in children's blood. Examples of direct effects on a child's blood-lead concentration include the effect of refinishing painted surfaces within a housing unit, and the effect of a child's pica habits or mouthing behavior. An indirect effect occurs when lead in one residential environmental medium (e.g., soil) contaminates another medium (e.g., interior dust), which in turn contributes directly to elevated blood-lead concentration.

The information that follows provides the current status of the sources of lead included in Figure 3-1 that have historically been recognized in the scientific literature as most associated with elevated blood-lead concentrations in children. Most of the information comes from detailed investigations on sources of lead documented in EPA (1986), CDC (1991), and ATSDR (1993).

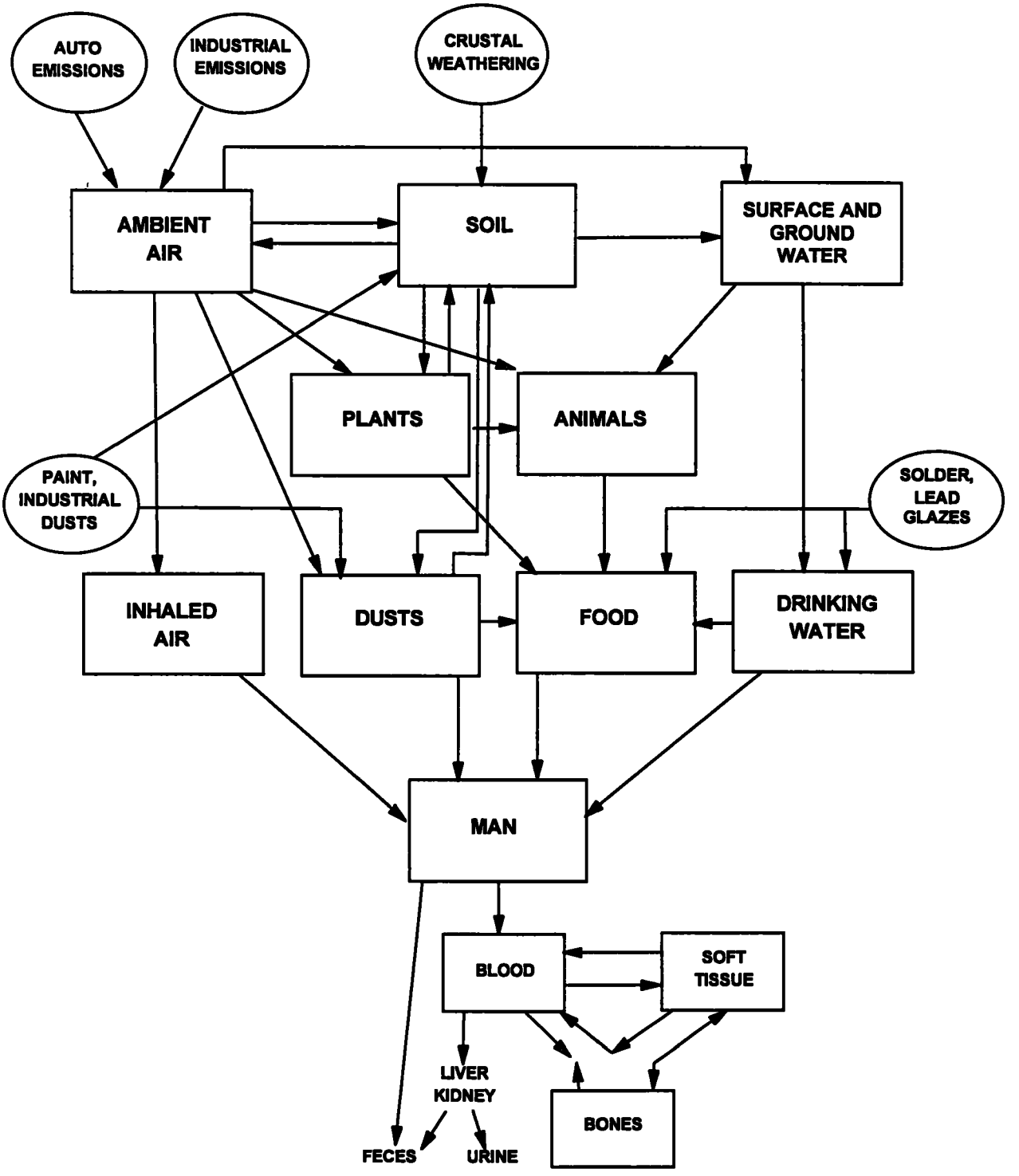


Figure 3-1. Pathways of Lead from the Environment to Humans, Main Organs of Absorbsion and Retention, and Main Routes of Excretion

(Sources: EPA, 1986; EPA, 1996)

Airborne Lead

Historically, emissions from lead smelters, battery manufacturing plants, solid waste incinerators, and automobiles have made major contributions to airborne lead levels. Fallout of atmospheric lead contributes to lead levels in soil, household dust, and street dust. Lead is deposited on soil, plants, and animals, which thereby is incorporated into the food chain.

Until recently, leaded gasoline emissions was one of the primary sources of lead exposure in the United States. Under Title II of the Clean Air Act (FR 1973 December 6), EPA specified lead as a pollutant compound of concern, and instituted a controlled phase-out of leaded gasoline by December 31, 1995. As a result, there was a 73% reduction in lead consumed in gasoline from 1975 to 1984 (EPA, 1986), and a 64% reduction in national lead emissions from 1985 to 1989 (ATSDR, 1993). This reduction has corresponded to a similarly dramatic decrease in average lead concentration in children's blood (CDC, 1991; Annest, 1983). The phase-out of leaded gasoline has contributed to airborne lead becoming only a minor lead-exposure pathway for children not exposed to specific point-emitting lead sources (CDC, 1991).

Indoor air may be considered an important indirect lead-exposure pathway when lead-based paint or lead-contaminated dust or soil is disturbed during renovation and remodeling activities. Inadequate dust control or use of paint stripping techniques that vaporize lead in paint are ways that lead is introduced into the air during renovation and remodeling activities (EPA, 1994d).

EPA has set a National Ambient Air Quality Standard of 1.5 μg of lead per cubic meter of air averaged over three months (ATSDR, 1993; 40 CFR 50.12).

Drinking and Cooking Water

Detectable levels of lead are rare in surface and ground water that serve as sources of drinking water in this country. Typically, lead contamination of drinking water occurs after the water leaves the treatment plant (CDC, 1991). By traveling within service lines and household plumbing, drinking water can become contaminated upon encounter with lead pipes, connectors, and solder. At a residence, water can also become contaminated within lead-containing water fountains, coolers, faucets, and other fixtures. Through the authority of the 1986 Safe Drinking Water Act and its amendments, EPA banned the use of lead materials and solders in new

plumbing and plumbing repairs, required that public water suppliers notify the public about lead presence in drinking water, and encouraged local government measures to test and remediate lead-contaminated drinking water in schools and day-care centers. As a result, lead in drinking and cooking water is generally not a predominant source of lead exposure among lead-poisoned children (CDC, 1991).

Analysis of environmental-lead data from several studies, including the Baltimore R&M Study and the Rochester Study (Section 3.2), concluded that lead levels in drinking water generally do not have a statistically significant effect on blood-lead concentrations. However, lead in drinking water is still considered an important exposure source when present due to the high absorption rate of lead in water (CDC, 1991).

The Safe Drinking Water Act set an action level for lead in drinking water of 15 ppb. Those systems that exceed the action level must inform the public, while taking measures to reduce lead levels and continue monitoring procedures. In 1991, EPA promulgated maximum contaminant level goals (MCLGs) and national primary drinking water regulations (NPDWRs) for lead and copper (56 FR 26460, June 7, 1991). This rule set the MCLG for lead within drinking water at the tap to be 0 ppb (ATSDR, 1993; 40 CFR 141,142).

Food

Many studies have shown that children's dietary intake of lead has receded over recent years. For example, data from the U.S. Food and Drug Administration (FDA) indicate that dietary lead intake in two-year-old children has declined from an approximate average of 30 μg/day in 1982 to 5 μg/day in the period 1986-1988 (CDC, 1991). U.S. FDA intervention and outreach activities, along with reduced lead entering the food chain due to the phase-out of leaded gasoline, have contributed to this decline. The phase-out of lead-soldered food cans (1.4% of the U.S.-produced food and soft drink cans in 1989, compared to 47% of such cans produced in 1980), along with public education on proper food storage and cooking techniques, have made large contributions to reducing the amount of lead ingested with food (CDC, 1991). Education is especially important in those areas of the country with traditions of using lead-containing pottery in cooking and preparing folk remedies containing lead.

While production of lead-soldered food and soft drink cans have been virtually eliminated in the U.S., such cans may still be used by other countries who import food to the U.S. In addition, lead can be introduced to food grown in lead-contaminated soil. Improper handling of food in the home (e.g., storing food in containers such as lead-soldered cans and lead-glazed pottery) can cause food to be a source of lead exposure. Thus, while lead exposures through food ingestion have declined considerably in recent years, these exposures can still occur if proper precautions are not addressed.

Lead-Based Paint

Lead-based paint is currently considered the most significant high-dose source of lead exposure in pre-school children (CDC, 1991). From the turn of the century through the 1940's, paint manufacturers used lead as a primary ingredient in many oil-based interior and exterior house paints. Usage gradually decreased through the 1950s and 1960s, as largely lead-free latex paints and exterior paint with lower lead concentrations were manufactured. Although the Consumer Product Safety Commission (CPSC) banned lead-based paints from residential use in 1978 (currently, paints may not have greater than 0.06% lead by weight), the presence of lead-based paint in the nation's housing stock remains high. An estimated 64 million (or 83% of) privately-owned, occupied housing units built prior to 1980 contain some components covered with lead-based paint (EPA, 1995a). Approximately 12 million of these units contain at least one child under the age of seven years. The estimated percentage of public housing units built prior to 1980 and containing lead-based paint is even higher: 86% (EPA, 1995a).

The exposure to lead from lead-based paint is considerably higher when the paint is in a deteriorated state or is found on accessible, chewable, impact, or friction surfaces (EPA, 1986; CDC, 1991). Thus, young children are especially susceptible to lead poisoning from lead-based paint, as they may ingest lead-based paint chips or come into contact with dust or soil that has been contaminated by deteriorated lead-based paint (see below). Both adults and children can be exposed to hazardous levels of lead by inhaling the fine dust or by ingesting paint-dust during hand-to-mouth activities. The U.S. Department of Housing and Urban Development (HUD) has prepared guidelines on controlling lead-based paint hazards, as improper control procedures can actually increase the threat of lead-based paint exposure by dispersing fine lead dust particles in

the air and over accessible household surfaces (HUD, 1995b; Farfel and Chisolm, 1990). The potential for lead-based paint to contaminate a variety of environmental media within a household makes lead-based paint the greatest source of public health concern regarding lead exposure (CDC, 1991).

Contaminated Dust and Soil

While enforcement of national air quality standards continues to reduce the threat of lead exposure via air from point sources, the fallout of atmospheric lead over time has resulted in a continued exposure route through soil (CDC, 1991). In addition, soil can become contaminated by deteriorated lead-based paint or by the improper removal of lead-based paint from a housing unit. The same soil, once tracked indoors, can become a component of household dust causing yet another source of lead exposure. Children are exposed to lead from soil or dust in their homes during typical hand-to-mouth activities.

Lead-contaminated soil and dust are thought to be the major pathway by which young children are exposed to lead from lead-based paint hazards (EPA, 1986). Exterior house paint can flake off or leach into the soil around the outside of a home, contaminating children's playing areas. Indoors, normal wear of lead-based paint (especially around windows and doors) and contaminated soil tracked into the house can contaminate interior dust. When lead takes the form of small particles, as it typically does when found within household dust (Que Hee et al., 1985), it is more easily absorbed into the body (Mahaffey, 1977).

A number of studies have assessed the effect of dust- and soil-lead levels on childhood blood-lead concentrations. A few studies have concluded that the effect of residential lead-based paint on blood-lead levels occurs via the pathway of dust- and soil-lead to blood. For example, analysis of data from the Cincinnati Longitudinal Study (Section 3.2.5) identified a significant lead pathway from exterior dust to interior dust to hands to blood, with lead in paint and soil contributing to lead in exterior dust (Bornschein et al., 1986). Analysis of data from the Brigham and Women's Hospital Longitudinal Study (Section 3.2.6) concluded a significant pathway from soil to window sill-dust to floor-dust to blood (Menton et al., 1995). It is likely that exposure of young children to lead in dust and soil is primarily due to their propensity to mouth fingers, toys, and other nonfood items that contain contaminated dust. In unpublished, EPA-supported

pathways analyses of data from the Baltimore R&M Study (Section 3.2.1) and the Rochester Study (Section 3.2.2), mouthing tendencies were found to be an important contributor to childhood blood-lead concentrations.

3.2 SUPPORTING EVIDENCE IN EPIDEMIOLOGIC STUDIES

Extensive evidence of the relationship between childhood blood-lead concentrations and environmental-lead levels is offered in the scientific literature. Evidence from two types of studies is available. *Epidemiological studies* investigate the association between elevated blood-lead concentrations and elevated levels of lead in a child's residential environment. *Intervention studies* investigate the impact on children's blood-lead concentrations of reducing childhood lead exposure via a range of intervention strategies. Epidemiological studies have demonstrated that elevated blood-lead concentrations are associated with elevated lead levels in the dust, paint, and soil of the surrounding environment. Causation, however, is better demonstrated by intervention studies. If children receiving an intervention strategy that targets a particular lead exposure source (e.g., paint, dust, or soil) exhibit greater reductions in blood-lead concentrations than those reported for a suitable control population, then the targeted source may be at least partially responsible for the prior exposure.

A review of intervention studies (EPA, 1995b) concluded that reductions in blood-lead concentrations have occurred following interventions of lead in paint, dust, and soil. While such studies suggest causation, their results are not necessarily indicative of the magnitude of the association between the levels of lead in targeted environmental media and blood-lead concentrations. This is because intervention studies typically examine children already exposed to environmental lead. Exposed children retain a store of lead in their tissues that routinely mobilizes into the blood. In fact, this mobilization is heightened following an intervention (Schroeder and Tipton, 1968; Rabinowitz, 1991) as the change in exposure caused by the intervention disrupts the body's equilibrium. Blood-lead concentrations following the intervention, therefore, represent a combination of the now reduced environmental lead exposure and the increased (at least temporarily) mobilized lead stores.

During the past 25 years, studies have been conducted to investigate the sources responsible for lead exposure in children. Many of these studies are limited, small, or not

relevant to the current exposure situation. These studies include investigations of the sources and extent of lead exposure in both urban and smelter communities. The studies listed in Tables 3-1 and 3-2 provide evidence regarding associations between childhood blood-lead concentrations and environmental-lead levels in urban and smelter communities, respectively. The results of these studies are qualitatively similar in that the association between environmental lead and blood lead is consistently positive and, when considered without the confounding from additional variables, usually found to be statistically significant. However, any effort to combine the disparate results from multiple studies into a single set of coefficients that provide one representative, quantitative measure of the relationship between blood-lead concentration and soil- and dust-lead levels is complicated by the qualitative dissimilarity among studies (e.g., differences in sampling and analysis methods, sampling locations, studied populations, and types of communities).

Table 3-1. Childhood Lead Exposure Studies Conducted in Urban Communities That Present Evidence of the Positive Relationship Between Environmental-Lead Levels and Blood-Lead Concentrations

Study/Community	Study Duration	Study Type	Reference(s)
Baltimore (MD) Repair and Maintenance Study	1992-1997	Abatement Efficacy	Farfel and Lim, 1995
Rochester (NY) Lead-in-Dust Study	1993	Health Assessment	Rochester School of Medicine and NCLSH, 1995; Lanphear et al., 1995
Baltimore (MD) Urban Soil Lead Abatement Demonstration Project (USLADP)	1988-1991	Soil Abatement Efficacy	EPA, 1996; Weitzman et al., 1993; Aschengrau et al., 1994
Boston (MA) USLADP	1989-1991		
Cincinnati (OH) USLADP	1989-1991		
Birmingham (UK) Urban Lead Uptake Study	1984-1985	Health Assessment	Davies et al., 1990; Thornton et al., 1990; Davies et al., 1987
Cincinnati (OH) Longitudinal	1980-1987	Health Assessment	Bornschein et al., 1985a; Que Hee et al., 1985; Bornschein et al., 1985b; Bornschein et al., 1986
Brigham and Women's Hospital Longitudinal Study (Boston, MA)	1980-1983	Health Assessment	Bellinger et al., 1986; Rabinowitz et al., 1985a; Rabinowitz et al., 1985b; Rabinowitz et al., 1984a; Rabinowitz et al., 1984b; Rabinowitz et al., 1982
New Haven, CT	1977	Health Assessment	Stark et al., 1982; Stark et al., 1978
Omaha, NE	1970-1977	Health Assessment	Angle and McIntire, 1979; Angle et al., 1974; Angle et al., 1984

Table 3-2. Childhood Lead Exposure Studies Conducted in Smelter Communities That Present Evidence of the Positive Relationship Between Environmental-Lead Levels and Blood-Lead Concentrations

Study/Community	Study Duration	Reference(s)
Granite City (IL) Educational Intervention	1991	Kimbrough et al., 1994
Butte-Silver Bow (MT) Environmental Health	1990	Butte-Silver Bow Dept. of Health, et al., 1991
Clear Creek/Central City (CO) Mine Waste Exposure	1990	ATSDR, 1992
Midvale (UT) Community	1989	Bornschein et al., 1990; Que Hee et al., 1985
Child Lead Exposure Study (Leeds, AL)	1989	ATSDR, 1991a
Philadelphia (PA) Neighborhood Lead	1989	ATSDR, 1991b
Leadville (CO) Metals Exposure	1988	Colorado Dept. Of Health, et al., 1990
Silver Creek Mine Tailings Exposure (Park City, UT)	1987	ATSDR, 1988
Telluride, ID	1986	Bornschein et al., 1989; Que Hee et al., 1985
Kellogg (ID) Revisited	1983	Panhandle District Health Dept. et al., 1986
Helena Valley (MT) Child Lead	1983	Lewis and Clark County Health Dept. et al., 1986
El Paso, TX	1971-1973	Landrigan et al., 1975

Early childhood lead exposure studies emphasized exposure to lead in paint, leaded gasoline emissions, and emissions from industrial sources. These studies, therefore, measured lead levels in these media and sought to relate them directly to resident children's blood-lead concentrations. Due to the assessment by many researchers in childhood lead exposure that ingestion of dust and soil via hand-to-mouth behavior represents the principal mechanism of lead exposure in young children today (CDC, 1991), more recent studies have focused principally on lead exposure from residential soil and dust. As indicated in Figure 3-1, residential soil and dust are assumed to have been contaminated by these same original sources: lead-based paint, industrial emissions or tailings, and leaded gasoline emissions.

Due to the reduction in lead sources such as gasoline emissions over time, the most recent epidemiologic studies provide a more accurate picture of the relationship between child blood-lead concentrations and lead-based paint hazards. Consequently, the §403 risk assessment has relied primarily on information from recent studies conducted in urban areas in the absence of specific point emission sources. The remainder of this section summarizes key objectives and conclusions on the effects of childhood lead exposure for eight urban studies in Table 3-1 that were conducted in the 1980s and 1990s. Environmental-lead data and blood-lead concentration data from two of these studies, the Baltimore Repair and Maintenance Study and the Rochester Lead-in-Dust Study, are summarized in Sections 3.3 and 3.4. These data were also used to develop statistical models to relate blood-lead concentration to environmental-lead levels. Necessary information on data from the three USLADP studies was not made available in time to allow the data to be included in this risk assessment. Data for the three other studies (Birmingham Urban Lead Uptake Study, Cincinnati Longitudinal Study, and the Brigham and Women's Hospital Longitudinal Study) were either not available to the risk assessment effort or were not considered for specific reasons.

3.2.1 Baltimore Repair and Maintenance (R&M) Study

The objectives of the Baltimore R&M Study were to characterize the efficacy of comprehensive lead-paint abatement up to six years post-abatement and to characterize the efficacy and costs of three levels (low, medium and high) of less costly Repair and Maintenance interventions. While published analyses of the R&M data are not yet available, these data were available for use in the §403 risk assessment.

In 1992, three types of housing units were recruited for this study. In the first group, 15 previously-abated dwellings were chosen from 90 low-income housing units that were abated between May, 1988, and April, 1992, by Baltimore City and Kennedy Krieger Institute Pilot Abatement Projects. The second group, slated to receive R&M interventions in this study, consisted of 75 older (mostly pre-1940), low-income dwellings in Baltimore City. Finally, 15 modern urban dwellings free of lead-based paint were chosen to represent control units. These units were chosen from an urban renewal area and included units that were fully gut-rehabilitated

since 1980. All units in the study had to include at least one eligible child aged 6 to 48 months who spent most of his/her time at the unit.

Prior to any intervention in this study, blood-lead concentrations were measured for 115 children that lived in 87 of the housing units. Although environmental and blood samples were collected both before and after interventions, only the results and data of the pre-intervention samples are considered in this report. The BRM vacuum method, consisting of a modified HVS3 cyclone collector, was the primary dust sampling method used in the R&M Study (EPA, 1995c). Within each housing unit, rooms were divided into three groups: rooms with windows on the first floor, rooms with windows on the second floor, and rooms with no windows. A composite sample of floor dust was collected from each of the three groups. Two additional composite samples, one of window sill dust and one of window well dust, were collected from the first two groups of rooms.

Lead levels in paint were determined through *in situ* x-ray fluorescence (XRF) measurement. Only those components suspected of being covered with lead-contaminated paint were measured, in order to identify whether a unit contained lead-based paint. As a result, paint-lead measurements in this study were high and do not represent a random sampling of painted surfaces in a housing unit.

Soil samples were taken from (½ inch) soil cores collected at the foundation and property boundaries. However, few units had available soil at these locations to sample. Dust and soil samples were analyzed for lead using inductively coupled plasma-atomic emission spectrometry or graphite furnace atomic absorption spectroscopy. Two-hour stagnation drinking water samples were also collected. A structured questionnaire collected information on study children and the households.

Summaries of pre-intervention environmental-lead levels observed in the Baltimore R&M Study are presented in Section 3.3 and observed blood-lead concentrations are summarized in Section 3.4.

3.2.2 Rochester Study

The Rochester study, conducted in 1993, was a cross-sectional design study whose primary objective was to obtain information on the association between lead levels in house dust

and blood-lead concentrations of resident children (HUD, 1995a) Children between the ages of 12 and 31 months (considered by the study group as the age of greatest risk for lead exposure) and living in the city of Rochester, NY, were eligible for this study, provided they did not satisfy any of the following:

- they or their environment had underwent recent interventions that were likely to alter blood or dust lead (e.g., major renovation, recent ingestion of prescribed iron products, or any medical or environmental intervention for an elevated blood-lead level),
- they spent more than 20 hours per week away from home, or
- they lived with an adult exposed to lead from an occupational or recreational activity.

Random sampling techniques were used to recruit children born from March 1, 1991, to September 30, 1992, at either Rochester General Hospital, Strong Memorial Hospital, or St. Mary's Hospital. Data for 205 families and children were included in the analysis.

During visits to the home of each study participant, an environmental health team obtained a venipuncture blood sample from the eligible child, completed a behavioral questionnaire for the household regarding lead exposure, collected environmental samples (interior dust, exterior soil, water), and took *in situ* measurements of lead in paint. The dust samples were collected from floors, window sills, and window wells within rooms in which the child was frequently present. When considering dust-lead loadings, the §403 risk assessment effort considered only those dust results collected using wipe techniques ("Little Ones" baby wipes). However, because a secondary objective of the Rochester study was to evaluate various dust sampling methods relative to predicting children's blood lead levels, dust samples were also collected using the University of Cincinnati Dust Vacuum Method (DVM) and the BRM vacuum. Lead concentrations of dust samples collected using the BRM vacuum are also summarized. Side-by-side dust samples were collected at specific locations, with each sample corresponding to a particular collection method and the wipe sample being the first to be collected. Dust samples were analyzed using either flame or graphite furnace atomic absorption

spectroscopy. Soil samples, taken at the play area and dripline, were analyzed using flame atomic absorption spectroscopy.

Children enrolled in the Rochester study were not specifically recruited because of elevated blood-lead concentrations. However, a disproportionate percentage of these children exhibited two risk factors associated with elevated blood-lead concentrations: residing in older housing (84% of the homes were built prior to 1940) and belonging to low-income families (55% of households had incomes below $15,500).

The geometric mean blood-lead concentration for the 205 children in the Rochester study was 6.38 μg/dL (geometric standard deviation, 1.85). Twenty-three percent of the children had blood-lead concentrations above 10 μg/dL, 8% above 15 μg/dL, and 3% above 20 μg/dL. Further summaries of blood-lead concentrations in this study are presented in Section 3.4.

A multiple regression approach using backward selection techniques (Neter and Wasserman, 1974) was used to determine those environmental variables and questionnaire variables most important to predicting blood-lead concentration in children. In addition to wipe dust-lead loading, the following factors were significantly associated with increased blood-lead concentrations among children: African-American race, children engaging in soil pica, single parent household, and having a high ferritin level. Adjusting for these factors, wipe dust-lead loading accounted for 10.1% of the variation in blood-lead concentrations (HUD, 1995a).

The Rochester Study also investigated the relationship between soil-lead concentration and children's blood-lead concentration. One composite soil sample was obtained from a maximum of 12 core samples (3 per side of house) taken two feet away from the foundation, and a second composite sample was obtained from 8-10 samples taken where the child frequently played. A coring device was used to take samples at a depth of ½ inches only where bare soil was present. Twenty-seven percent of the children in this study exhibited soil pica. Soil-lead concentration was a significant (positive) predictor of blood-lead concentration, even when adjusting for dust-lead loading.

The Rochester Study concluded that lead-contaminated dust affects children's blood-lead levels, even when those levels are in the low to moderate range (< 25 μg/dL). This relationship differs according to the dust sampling method and the type of surface sampled. At the relatively low levels of dust-lead in this study, dust-lead loadings were found to be a better predictor of

blood-lead concentration than were dust-lead concentrations. The study suggests that of the three dust collection methods considered, dust-lead results from samples collected using either wipe or BRM-vacuum methods should be used when making inferences on children's blood-lead concentrations.

Summaries of environmental-lead levels observed in the Rochester Study are presented in Section 3.3. Data from the Rochester study were employed to develop the epidemiologic model used in this risk assessment (Section 4.3.2).

3.2.3 Urban Soil Lead Abatement Demonstration Project (USLADP)

The USLADP, authorized in 1986 under the Superfund Amendments and Reauthorization Act, was conducted to determine whether reducing lead levels in soil accessible to children decreases their blood-lead concentration. While other observational studies of childhood lead exposure such as the Rochester Study have shown that differences in soil lead exposure are associated with differences in blood-lead concentration, this project specifically addressed whether controlled reductions in external soil lead exposure were associated with reductions in blood-lead concentrations. The USLADP consisted of three studies conducted in Baltimore, MD, Boston, MA, and Cincinnati, OH. This project considered soil abatements in urban areas and focused on inner-city children.

In Baltimore, data were analyzed for 185 children aged 6 to 72 months. These children resided in either the study area (expectation of moderate risk of lead poisoning) or a control area. The Boston study included 149 children aged 6 to 48 months, considered to be at risk for lead exposure and residing in one of the study areas (history of high incidence of lead poisoning). Only children with blood-lead concentrations ranging from 7 to 24 μg/dL were included in the Boston study. In Cincinnati, families with children under five years of age and residing in one of the study areas (selected as having similar socioeconomic, housing type, etc. characteristics) were enrolled in the study. Data for 206 children were analyzed from the Cincinnati study.

Within each city, a series of neighborhoods were considered in the study from which the participating households were selected. Selected units within certain neighborhoods were to have interventions performed, while units in other neighborhoods were selected as control units. For purposes of data summary and analysis, study units were grouped according to intervention

strategy. Environmental media sampled included dust, soil, drinking water, and paint. Household interviews were also conducted to obtain information on such factors as household behavior and socioeconomic status.

The following two main conclusions were drawn from the USLADP (EPA, 1996):

1. *"When soil is a significant source of lead in the child's environment, under certain conditions, the abatement of that soil will result in a reduction in exposure that will 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 five factors are important in determining the magnitude of any possible reduction: (1) the past history of exposure of the child to lead, as reflected in the pre-abatement blood lead; (2) the initial soil lead concentration and the magnitude of the reduction in soil lead concentrations; (3) the initial interior house dust lead loading and the magnitude of reduction in house dust lead loading; (4) the magnitude of other sources of lead exposure, relative to soil; and (5) the strength of the exposure pathway between soil and the child relative to other lead exposure pathways in the child's environment."*

In taking soil samples, the entire soil region surrounding the residence was partitioned into distinct areas (e.g., front, back), and samples were taken from each partition. At each core sample, the top 2" and bottom 2" of the sample core were retained. A single core sample was taken when less than two meters in either direction were available for sampling. Larger areas had core samples taken at the foundation and at the boundary.

Dust samples were collected by vacuum methods in all three cities. In Baltimore, the Sirchee-Spittler dust buster vacuum sampler without a frame (a 4' x 4' sample area is demarcated with tape) was used. A minimum of three areas were sampled: the main entrance to the household and two areas often frequented by the child when playing. In Boston, the same sampler and sampling sites were used, but a plastic 25 cm x 25 cm frame was used instead of tape. In Cincinnati, a personal air monitoring vacuum pump was used with a plastic 25 cm x 25 cm frame. Dust was sampled from a floor area adjacent to the main entrance; a composite of dust samples from at least 3 floors areas including the child's bedroom and a high traffic area in the main living area; a composite of dust samples from at least 3 window well and sill areas including from within the child's bedroom and the main living area; dust from an entryway

floormat placed by sample collection personnel. Dustfall and exterior surface dust were also measured.

Data from the USLADP studies became available for analysis and summary in 1996, when this risk assessment was being performed. As a result, these data were not summarized in this report, and the data have not been used in the analysis efforts of the risk assessment.

3.2.4 Birmingham Urban Lead Uptake Study

This study was conducted in Birmingham, England, from 1984-1985, and consisted of 183 randomly-selected children, aged 24 months (± 2 months), born in and still residing in urban Birmingham. A stratified subset of 106 children were selected for the study, of which 97 completed the study. The objective of the study was to simultaneously examine lead uptake via all identified environmental pathways for young children in an urban environment.

Soil samples were collected using a stainless-steel trowel surface scrape (0-5 cm). One composite soil sample was obtained from 25 core samples. A specially adapted vacuum was used to collect dust samples from the child's main play area, the child's bedroom and under the doormat. All exposed floor space was sampled. Samples were also taken from the bag of the vacuum cleaner most often used by the household.

The main conclusion from this study was that childhood blood-lead concentration was found to be significantly associated with a combination of dust-lead loading, the rate of touching objects, water-lead concentration, and smoking habits of the parents. Only an estimated 3% of a child's average total uptake of lead per day was attributed to breathable air; the remainder was attributed to dust, food, and water ingestion.

Because this study was conducted outside of the United States over ten years ago, it was considered less representative of current childhood lead exposure in the United States than more recent studies. Therefore, the data from this study were not used in the §403 risk assessment.

3.2.5 Cincinnati Longitudinal Study

Objectives of the Cincinnati Longitudinal study, conducted from 1980-1987, were to provide a complete picture of a child's lead exposure history and to investigate the factors responsible for excessive lead exposure. Approximately 250 expectant mothers residing within a

prespecified set of census tracts in the Cincinnati (OH) area were enrolled for this study. These census tracts were identified as having a long history of producing children with elevated blood lead levels. The mothers were patients at one of three prenatal clinics. Once these mothers delivered, blood-lead concentrations were measured in the children from birth through 5 years of age.

Soil samples were collected by surface scrapings. Surface scrapings were collected from the child's play area outside, if one existed. Interior dust samples were collected using a personal sized vacuum within 484 cm^2 plastic frame area wherever child frequents. A maximum of five sites were sampled within the home. Each sample entails three sweeps of the vacuum within the frame. Exterior dust samples were collected via scraping exterior surfaces with a stainless steel spatula. Paint-lead levels from a maximum of 15 surfaces were measured using XRF techniques. Dust collection from children's hands was performed via repeated wiping with multiple pre-moistened wipes.

This study observed high levels of lead contamination in the residential environments, with most contamination occurring in areas immediately outside of the unit and within the entranceways. Statistical analyses indicated that the pathway from exterior dust to interior dust to hands to blood was of most significance in this study.

The data for this study were not available to the §403 risk assessment effort.

3.2.6 Brigham and Women's Hospital Longitudinal Study

The objective of this early study was to examine the relationship between children's blood-lead levels and various environmental factors from late pregnancy to two years of age. Children were selected from births occurring between April 1979 and April 1981 at Brigham and Women's Hospital in Boston, MA. Births were categorized into the highest, lowest, and middle deciles of umbilical cord blood lead. The 249 infants selected were nearly equally drawn from three distinct categories of cord blood levels. All families resided within a 12 mile radius of hospital, spoke English as their primary language, and the infants had no serious illness. Umbilical cord blood was collected, as was blood at 6, 12, 18, and 24 months of age. The sample was predominantly white, middle- to upper-middle class families in an urban environment.

Soil samples were collected at a distance of 3 meters from the road. Dust samples were collected at 1, 6, 18, and 24 months using wipe techniques from a living room surface (floor or furniture top) and from a window sill. Samples were collected from within a plastic frame with a 930 cm^2 opening (a 465 cm^2 opening for window sills). Lead levels in paint were measured by a PGT model XE-3 XRF instrument. Air samples were collected from personal air monitors, and drinking water samples were collected from the kitchen tap after a 4-liter flush.

Mean blood-lead concentrations at 24 months was 6.8 μg/dL. At 24 months, blood-lead concentration was found to be significantly associated with soil-lead levels, dust-lead levels, the presence of deteriorated paint, and the occurrence of recent refinishing activities at the residence. Water-lead and airborne-lead levels were not significant factors. While none of the children were classified as having excessive pica tendencies, evidence existed between mouthing tendencies and increased blood-lead concentration. These findings agreed with earlier studies which considered children with higher blood-lead concentrations. In addition, blood-lead concentrations were found to be approximately 44% higher within specimens collected in summer months, indicating a possible seasonality factor associated with blood-lead concentration.

Due to the age of the study and its focus on a specific area of the country, data from this study were not used in the §403 risk assessment.

3.3 LEAD IN DUST, SOIL, AND PAINT IN THE NATION'S HOUSING

This section provides information on the distribution of environmental-lead levels in the nation's housing stock, with a focus on lead in residential dust, soil, and paint. The §403 risk assessment uses data from the HUD National Survey of Lead-Based Paint in Housing to characterize the distribution of environmental-lead levels in the nation's occupied housing stock in 1997. These environmental-lead data are summarized in Section 3.3.1. To provide supporting information on environmental-lead levels in occupied housing, environmental-lead data from the Baltimore R&M Study and the Rochester Study are also summarized in this section. Section 3.3.1 also includes estimated numbers of occupied housing units in the 1997 national housing stock.

To provide a link between childhood and residential environmental lead exposures, Section 3.3.2 presents estimated numbers of children of specific age groups in 1997 residing within housing units of specific ages.

3.3.1 The Distribution of Lead Levels in Household Dust, Soil, and Paint

In this section, environmental-lead levels in residences are summarized for three studies. The first study presented, the HUD National Survey, is used to characterize environmental-lead levels in the nation's occupied housing stock in 1997, prior to §403 interventions. The other two studies, the Baltimore R&M Study and the Rochester Study, provide supporting information on environmental-lead levels for specific housing groups or exposure conditions.

3.3.1.1 HUD National Survey

For the §403 risk assessment effort, the primary source of information on environmental-lead levels in the national housing stock was the National Survey of Lead-Based Paint in Housing (EPA, 1995a). This survey was sponsored by the U.S. Department of HUD, in response to a mandate in the 1987 amendments to the Lead-Based Paint Poisoning Prevention Act to obtain "an estimate of the amount, characteristics and regional distribution of housing in the United States that contains lead-based paint hazards at differing levels of contamination." Conducted in 1989-1990, the privately-owned unit portion of the survey (cited as the "HUD National Survey" in this document) measured lead levels in paint, dust, and soil within 284 privately-owned, occupied housing units. The units were selected via a statistically-based sampling design to represent the national housing stock built prior to 1980. Units built in 1980 or later were not included in the survey, as they were assumed to be free of lead-based paint because of the Consumer Product Safety Commission's 1978 ban on the sale of lead-based paint and its use in residences.

In the HUD National Survey, lead loadings (μg of lead per square-feet of area sampled) and lead concentrations (μg of lead per gram of sample) were measured from dust samples collected on floors, window sills, and window wells. Dust samples were collected using the Blue Nozzle vacuum method. Lead concentrations in the soil at each unit were measured by collecting soil samples along the foundation, the entryway to the unit, and from remote areas in the yard,

using a soil corer with plunger. Lead concentrations in paint (milligrams of lead per square-centimeter of painted surface) were measured using *in situ* XRF techniques in selected rooms as well as on the exterior of the unit.

In the §403 risk assessment effort, data from the 284 privately-owned units in the HUD National Survey were used to characterize environmental-lead levels in the nation's occupied housing. Table C-7 of Appendix C contains the following summary of environmental-lead levels for each of these units:

- two weighted arithmetic averages of dust-lead loading: one for floors and one for window sills (where sample results were weighted according to area of sample)
- two weighted arithmetic averages of dust-lead concentration: one for floors and one for window sills (where sample results were weighted according to mass of sample)
- the weighted arithmetic average soil-lead concentration (where remote sample results were weighted twice that of the entryway and dripline results)
- the maximum observed paint-lead concentration for the interior and the exterior, as measured by XRF techniques.

Note that the last bullet indicates the maximum observed (or measured) paint-lead concentration in a unit. To identify whether a unit was suspected of containing LBP, statistical modeling was performed in the HUD National Survey to obtain a predicted maximum XRF paint-lead concentration for each unit. If the predicted maximum XRF value for a unit was at least 1.0 mg/cm^2, the unit was considered to contain LBP. In the §403 risk assessment effort, the predicted maximum XRF value was used only to identify the presence of LBP for each unit.

In the HUD National Survey, each unit was assigned a sampling weight equal to the number of pre-1980 privately-owned, occupied units in the national housing stock that were represented by the given unit in the survey. The sum of all 284 sampling weights equaled the number of pre-1980 privately-owned, occupied units in the national housing stock at the time of the survey. Sampling weights in the HUD National Survey were determined according to four demographic variables associated with the units:

- Age category of unit
- Number of units in the building
- Census region
- Presence of a child under age 7 years

In order to use the environmental-lead levels from the HUD National Survey to characterize environmental-lead levels in the 1997 national housing stock, it was necessary to revise the sampling weights of the HUD National Survey units to represent the 1997 occupied housing stock, both publicly-owned and privately-owned. The method for revising the sampling weights is documented in Section C.1.1.2 of Appendix C; Table C-7 of Appendix C lists the revised weights for each unit. The revised weights, therefore, indicate the number of units in the 1997 national housing stock that are represented by the given HUD National Survey unit, and therefore, represented by its environmental-lead levels. The estimated numbers of units in the 1997 national housing stock are presented in Table 3-3, within four age categories.

Table 3-3. Estimated Total Number of Occupied Housing Units in the National Housing Stock in 1997 According to Year-Built Category

Year In Which the Unit Was Built	Number of National Survey Units	Estimated Numbers of Units in the 1997 National Housing Stock
Pre-1940	77	19,676,000
1940-1959	87	19,718,000
1960-1979	120	34,985,000
Post-1979	28[1]	24,893,000
	Estimated Total:	99,272,000

[1] Units built from 1960-1979 and containing no lead-based paint were also placed in this category.

The HUD National Survey did not consider units built after 1979, as all such units were assumed to be free of lead-based paint due to the Consumer Product Safety Commission's 1978 ban on the sale of lead-based paint and its use in residences. Therefore, in characterizing the 1997 national housing stock from the HUD National Survey, post-1979 housing was represented by the 28 units built between 1960 and 1979 and containing no lead-based paint (i.e., the

predicted maximum amount of lead in paint within the unit was less than 1.0 mg/cm^2). Therefore, the revised sampling weight for these 28 units was subdivided into two parts: one part representing 1960-1979 units, and the other representing post-1979 units. See Section C.1.1.3 of Appendix C on the rationale for selecting these 28 units to represent the post-1979 housing stock and on the method for obtaining the portion of the sampling weight representing post-1979 units.

Using the environmental-lead levels and the updated 1997 sampling weights for the HUD National Survey units from Appendix C, Tables 3-4 and 3-5 are a predicted summary of lead loadings and concentrations, respectively, in floor-dust samples across units in the 1997 housing stock. Tables 3-6 and 3-7 summarize lead loadings and concentrations, respectively, in window sill-dust samples. Table 3-8 summarizes lead concentrations in soil. Table 3-9 presents summaries of the observed maximum XRF value from the National Survey units, weighted by the updated 1997 sampling weights. The percentages of units in the 1997 housing stock having lead-based paint, as well as the percentages having damaged lead-based paint, are estimated in Table 3-10.

The tables that summarize dust-lead loadings and dust-lead concentrations (Tables 3-4 through 3-7) indicate that the geometric means and the medians decrease with the age of the unit. This finding is consistent with the hypothesis that the potential for dust contamination by lead is higher in older units, due to their propensity to contain lead-based paint and to be located in older neighborhoods with lead-contaminated soil. Window sill dust-lead levels in units built prior to 1940 were considerably higher than those of the other units. These tables also indicate that lead levels in dust tend to be higher on window sills than floors, especially in older units. The same trends were observed in soil-lead concentration (Table 3-8), whose geometric mean and median decreased with the age of the unit, and whose levels were considerably higher in pre-1940 units than in the other units.

The components tested for lead-based paint in the HUD National Survey were selected based on a predetermined sample design that did not target only components suspected of having lead-based paint. Therefore, the summarized values in Table 3-9 represent both lead-contaminated and lead-free painted surfaces. The relationship between lead levels in paint and age of unit is strongest for the median and upper percentiles, indicating that while low paint-lead

measurements are likely to be observed in all housing regardless of age, large paint-lead measurements are more likely to be observed in older units.

This risk assessment predicts that approximately 62% of the 1997 occupied housing stock will contain LBP (Table 3-10), based on information from the HUD National Survey and under the assumption that no units built after 1979 contain LBP. This percentage is less than 83%, the percentage of pre-1980 occupied housing predicted to contain LBP according to the HUD National Survey (EPA, 1995a). The estimate of 62% is relative to all occupied housing, even units built after 1979. The percentages of units with LBP within the three pre-1980 year-built categories match those reported in the National Survey report (EPA, 1995a). Table 3-10 also indicates that approximately 14% of units are predicted to contain more than five square feet of deteriorated LBP, with over half of these units built prior to 1940.

Table 3-4. Summary of the Distribution of Lead Loadings in Floor-Dust Samples Within Housing Units in the HUD National Survey, Weighted to Reflect the Predicted 1997 Housing Stock

Year Unit Was Built	Floor Dust-Lead Loadings ($\mu g/ft^2$)[1]						
	Geometric Mean	Geometric Standard Deviation	5th Percentile	25th Percentile	Median	75th Percentile	95th Percentile
Before 1940	43.6	3.13	7.19	17.9	32.9	127.	220.
1940-1959	22.5	2.93	3.65	11.3	22.1	52.9	147.
1960-1979	13.8	2.52	3.79	7.05	12.9	26.6	57.8
1960-1979 units with no LBP[2]	10.3	2.04	4.17	5.77	9.15	18.1	42.1

[1] Data summarized in this table are area-weighted arithmetic mean dust-lead loadings from floors for the 284 privately-owned, occupied National Survey units (see Appendix C). These loadings are converted to represent loadings from dust samples obtained from wipe collection techniques. In the summaries, each unit is weighted by its 1997 weight, which is presented in Appendix C.

[2] Units with no LBP have a predicted maximum XRF value (interior and exterior) less than 1.0 mg/cm^2. These units represent post-1979 units in the §403 risk assessment effort.

Table 3-5. Summary of the Distribution of Lead Concentrations in Floor-Dust Samples Within Housing Units in the HUD National Survey, Weighted to Reflect the Predicted 1997 Housing Stock

Year Unit Was Built	Floor Dust-Lead Concentrations (μg/g)[1]						
	Geometric Mean	Geometric Standard Deviation	5th Percentile	25th Percentile	Median	75th Percentile	95th Percentile
Before 1940	511.	4.0	96.9	297.	589.	831.	6320.
1940-1959	204.	2.6	44.7	102.	215.	322.	1240.
1960-1979	123.	3.0	20.2	68.2	134.	207.	487.
1960-1979 units with no LBP[2]	93.	2.2	22.2	53.8	90.5	164.	458.

[1] Data summarized in this table are mass-weighted arithmetic mean dust-lead concentrations from floors for the 284 privately-owned, occupied National Survey units (see Appendix C). These concentrations were adjusted to reflect the weight of the entire dust sample, not just the tap weight. In the summaries, each unit is weighted by its 1997 weight, which is presented in Appendix C.

[2] Units with no LBP have a predicted maximum XRF value (interior and exterior) less than 1.0 mg/cm^2. These units represent post-1979 units in the §403 risk assessment effort.

Table 3-6. Summary of the Distribution of Lead Loadings in Window Sill-Dust Samples Within Housing Units in the HUD National Survey, Weighted to Reflect the Predicted 1997 Housing Stock

Year Unit Was Built	Window Sill Dust-Lead Loadings (μg/ft^2)[1]						
	Geometric Mean	Geometric Standard Deviation	5th Percentile	25th Percentile	Median	75th Percentile	95th Percentile
Before 1940	208.	12.2	2.36	50.0	243.	1440.	9640.
1940-1959	33.1	8.33	0.568	9.74	29.6	143.	795.
1960-1979	24.9	11.0	0.474	4.10	26.8	227.	753.
1960-1979 units with no LBP[2]	13.5	8.02	0.315	5.04	12.8	73.9	180.

[1] Data summarized in this table are area-weighted arithmetic mean dust-lead loadings from window sills for the 284 privately-owned, occupied National Survey units (see Appendix C). These loadings are converted to represent loadings from dust samples obtained from wipe collection techniques. In the summaries, each unit is weighted by its 1997 weight, which is presented in Appendix C.

[2] Units with no LBP have a predicted maximum XRF value (interior and exterior) less than 1.0 mg/cm^2. These units represent post-1979 units in the §403 risk assessment effort.

Table 3-7. Summary of the Distribution of Lead Concentrations in Window Sill-Dust Samples Within Housing Units in the HUD National Survey, Weighted to Reflect the Predicted 1997 Housing Stock

Year Unit Was Built	Window Sill Dust-Lead Concentrations (µg/g)[1]						
	Geometric Mean	Geometric Standard Deviation	5th Percentile	25th Percentile	Median	75th Percentile	95th Percentile
Before 1940	1730.	5.2	103.	698.	1740.	6700.	56500.
1940-1959	487.	4.0	48.1	244.	378.	1360.	3230.
1960-1979	388.	4.9	28.7	138.	519.	1540.	1650.
1960-1979 units with no LBP[2]	247.	3.3	26.0	129.	274.	503.	1250.

[1] Data summarized in this table are mass-weighted arithmetic mean dust-lead concentrations from window sills for the 284 privately-owned, occupied National Survey units (see Appendix C). These concentrations were adjusted to reflect the weight of the entire dust sample, not just the tap weight. In the summaries, each unit is weighted by its 1997 weight, which is presented in Appendix C.

[2] Units with no LBP have a predicted maximum XRF value (interior and exterior) less than 1.0 mg/cm^2. These units represent post-1979 units in the §403 risk assessment effort.

Table 3-8. Summary of the Distribution of Soil-Lead Concentrations for Housing Units in the HUD National Survey, Weighted to Reflect the Predicted 1997 Housing Stock

Year Unit Was Built	Soil-Lead Concentrations (µg/g)[1]						
	Geometric Mean	Geometric Standard Deviation	5th Percentile	25th Percentile	Median	75th Percentile	95th Percentile
Before 1940	464.	3.1	39.5	258.	569.	1160.	2020.
1940-1959	91.7	3.2	22.0	44.3	75.8	146.	485.
1960-1979	32.7	2.6	6.11	19.7	28.6	58.3	186.
1960-1979 units with no LBP[2]	22.4	2.3	5.58	13.6	21.2	45.0	82.5

[1] Data summarized in this table are weighted arithmetic mean soil-lead concentrations for the 284 privately-owned, occupied National Survey units (see Appendix C). Within each unit's average, remote sample results were weighted twice that of the entryway and dripline results. In the summaries, each unit was weighted by its 1997 weight, which is presented in Appendix C.

[2] Units with no LBP have a predicted maximum XRF value (interior and exterior) less than 1.0 mg/cm^2. These units represent post-1979 units in the §403 risk assessment effort.

Table 3-9. Summary of the Distribution of Observed Maximum XRF Lead Levels in Paint for Housing Units in the HUD National Survey, Weighted to Reflect the Predicted 1997 Housing Stock

Year Unit Was Built[2]	# National Survey Units[3]	Observed Maximum XRF Paint-Lead Levels (mg/cm²)[1]						
		Geometric Mean	Geometric Standard Deviation	5th Percentile	25th Percentile	Median	75th Percentile	95th Percentile
Interior of Unit								
Before 1940	72	1.86	3.78	0.30	0.60	0.90	6.40	20.2
1940-1959	83	1.02	2.42	0.40	0.60	0.70	1.60	8.00
1960-1979	116	0.71	1.79	0.30	0.60	0.60	0.90	1.60
Exterior of Unit								
Before 1940	60	3.14	3.75	0.30	0.70	4.00	7.10	26.9
1940-1959	76	1.45	3.05	0.20	0.60	1.50	2.60	10.3
1960-1979	103	0.72	2.43	0.00	0.50	0.60	0.90	3.60

[1] Data summarized in this table are observed maximum XRF paint-lead level for National Survey units across both interior and exterior painted surfaces (see Appendix C). Each unit's observed maximum XRF paint-lead level was weighted by the 1997 weight for the unit, which is presented in Appendix C.

[2] No units built after 1979 were included in the HUD National Survey. In the §403 risk assessment effort, these units are assumed to be free of LBP.

[3] Number of privately-owned units in the HUD National Survey in which an observed maximum XRF paint-lead level was available (for either the interior or exterior).

Table 3-10. Predicted Numbers and Percentages of Units Having Lead-Based Paint in the 1997 Occupied Housing Stock, Based on Information from the HUD National Survey[1]

Year Unit Was Built	Number (%) of Units with Lead-Based Paint	Number (%) of Units with More Than 5 ft² of Lead-Based Paint
Before 1940	17,248,000 (87.7%)	7,755,000 (39.4%)
1940-1959	18,047,000 (91.5%)	3,065,000 (15.5%)
1960-1979	26,452,000 (75.6%)	2,651,000 (7.6%)
After 1979	0 (0%)	0 (0%)
All Housing	61,747,000 (62.2%)	13,470,000 (13.6%)

[1] A unit in the HUD National Survey is labeled as containing LBP if its predicted maximum XRF value in either the interior or the exterior is at least 1.0 mg/cm². Results are weighted using the 1997 weights presented in Appendix C.

In preparing summaries of dust concentration data in Tables 3-5 and 3-7, these data were adjusted for the effect of underestimated sample weights. "Tap weight" is the portion of a dust sample that was tapped out of the sample collection filter. In the HUD National Survey, the dust-lead concentration equaled the amount of lead in the entire dust sample, divided by the tap weight. The adjustment to dust-lead concentration data attempted to correct for the weight of the entire dust sample, not just the tap weight. Appendix Z contains details on the adjustment method. Lead concentrations for dust samples with a tap weight of less than 0.7 mg were set to missing.

As discussed in Section C.1.3, missing values for dust-lead loading, dust-lead concentration, or soil-lead concentration for a National Survey unit were replaced by nonmissing values prior to the data summaries in Tables 3-5 through 3-8. For a particular data parameter, the value assigned to a unit having missing values equaled the average value across units within the same category of year built and having the same lead-based paint status (i.e., presence or absence of a maximum XRF value in the interior or exterior at 1.0 mg/cm^2 or above).

The §403 dust lead standard will be defined as a lead loading of a wipe sample. Dust samples in the HUD National Survey were collected using the Blue Nozzle vacuum sampler. The Blue Nozzle vacuum dust-lead loadings were converted to wipe equivalent dust-lead loadings using the conversion equations presented in Section 4.2.

3.3.1.2 The Baltimore Repair and Maintenance (R&M) Study

In the Baltimore R&M Study (Section 3.2.1), the BRM vacuum was used to collect dust samples. Dust-lead loadings were converted to wipe equivalent dust-lead loadings using the conversion equations presented in Section 4.2. Tables 3-11 and 3-12 summarize pre-intervention lead loadings and concentrations, respectively, in floor-dust samples across study units. Tables 3-13 and 3-14 summarize lead loadings and concentrations, respectively, in window sill-dust samples. Table 3-15 summarizes lead concentrations in soil samples taken at the dripline. Table 3-16 presents summaries of the observed maximum XRF value within study units slated for R&M interventions in the study, for the interior only and the exterior only, as well as for the entire unit. XRF measurements were not made in the previously abated and modern urban homes.

Table 3-11. Summary of Average Pre-Intervention Floor Dust-Lead Loading for Housing Units in the Baltimore R&M Study

Unit Category	# Units	Floor Dust-Lead Loading ($\mu g/ft^2$)[1]			
		Geometric Mean	Geometric Standard Deviation	Minimum	Maximum
All Study Units	90	31.89	2.11	3.78	157.48
Previously Abated Units	16	35.62	1.50	17.67	85.13
Units Slated for R&M Intervention	58	44.41	1.54	18.63	157.48
Modern Urban Units	16	8.60	1.54	3.78	15.28

[1] Data summarized in this table are area-weighted arithmetic mean dust-lead loadings from floors for each unit. These loadings have been converted to represent loadings from dust samples obtained from wipe collection techniques.

Table 3-12. Summary of Average Pre-Intervention Floor Dust-Lead Concentrations for Housing Units in the Baltimore R&M Study

Unit Category	# Units	Floor Dust-Lead Concentration ($\mu g/g$)[1]			
		Geometric Mean	Geometric Standard Deviation	Minimum	Maximum
All Study Units	90	1303.22	4.06	48.85	60304.19
Previously Abated Units	16	1214.02	2.55	331.86	7357.76
Units Slated for R&M Intervention	58	2436.50	2.61	425.62	60304.19
Modern Urban Units	16	144.79	2.19	48.85	704.20

[1] Data summarized in this table are area-weighted arithmetic mean dust-lead concentrations from floors for each unit.

Table 3-13. Summary of Average Pre-Intervention Window Sill Dust-Lead Loading for Housing Units in the Baltimore R&M Study

Unit Category	# Units	Window Sill Dust-Lead Loading ($\mu g/ft^2$)[1]			
		Geometric Mean	Geometric Standard Deviation	Minimum	Maximum
All Study Units	90	320.64	3.27	22.80	1869.49
Previously Abated Units	16	153.34	2.21	45.52	840.35
Units Slated for R&M Intervention	58	668.48	1.62	201.82	1869.49
Modern Urban Units	16	46.75	1.59	22.80	80.74

[1] Data summarized in this table are area-weighted arithmetic mean dust-lead loadings from window sills for each unit. These loadings have been converted to represent loadings from dust samples obtained from wipe collection techniques.

Table 3-14. Summary of Average Pre-Intervention Window Sill Dust-Lead Concentrations for Housing Units in the Baltimore R&M Study

Unit Category	# Units	Window Sill Dust-Lead Concentration ($\mu g/g$)[1]			
		Geometric Mean	Geometric Standard Deviation	Minimum	Maximum
All Study Units	90	5601.53	8.54	7.25	141056.96
Previously Abated Units	16	1881.89	4.61	254.63	31497.47
Units Slated for R&M Intervention	58	20148.29	2.42	2809.83	141056.96
Modern Urban Units	16	160.98	2.66	7.25	447.12

[1] Data summarized in this table are area-weighted arithmetic mean dust-lead concentrations from window sills for each unit.

Tables 3-11 through 3-14 indicate that geometric mean levels are highest for units slated for R&M intervention, while modern urban units have geometric mean levels that are an order of magnitude lower than the other two housing groups. Units slated for R&M interventions should not be considered representative of inner city homes. Prior to the interventions, many of them were in poor condition. They might be considered to represent the worst case of residential environmental-lead levels. The floor dust-lead loadings for previously-abated units and units slated for R&M interventions are comparable to those reported for pre-1940 housing in the National Survey, while these units have considerably higher window sill dust-lead levels compared to the National Survey. Units slated for R&M interventions have very high dust-lead concentrations and window sill dust-lead loadings, due to the deteriorated condition of most of these units. Modern urban units have dust-lead levels that are similar to the National Survey units built from 1960-1979 and containing no LBP.

Soil-lead concentrations summarized in Table 3-15 are based on small numbers of units, due to the lack of available soil to sample for many of the study units. Geometric mean soil-lead concentrations presented in Table 3-15 are high compared to those in the HUD National Survey.

The paint-lead measurements summarized in Table 3-16 are extremely high, as the data represent only units slated for R&M interventions. Paint-lead measurements were taken primarily from components suspected of containing LBP, in order to identify and prioritize surfaces requiring LBP intervention. Thus, the data summarized in Table 3-16 reflect a LBP-contaminated environment and are not typical of all painted surfaces in occupied housing.

Table 3-15. Summary of Average Pre-Intervention Dripline Soil-Lead Concentrations for Housing Units in the Baltimore R&M Study

Unit Category	# Units	Soil-Lead Concentration (μg/g)[1]			
		Geometric Mean	Geometric Standard Deviation	Minimum	Maximum
All Study Units	28	444.48	5.06	28.85	3539.16
Previously Abated Units	2	2192.33	1.60	1570.20	3060.94
Units Slated for R&M Intervention	16	1258.08	1.95	334.73	3539.16
Modern Urban Units	10	61.13	1.65	28.85	153.69

[1] Data summarized in this table are area-weighted arithmetic mean dripline soil-lead concentrations for each unit.

Table 3-16. Summary of Observed Maximum XRF Paint-Lead Concentration at Pre-Intervention for Housing Units Slated for R&M Intervention in the Baltimore R&M Study[1]

Location Within a Unit	# Units	Observed Maximum XRF Paint-Lead Concentration (mg/cm^2)			
		Geometric Mean	Geometric Standard Deviation	Minimum	Maximum
Entire Unit	36	38.38	1.68	9.30	98.10
Exterior Only	35	24.75	2.63	0.60	86.30
Interior Only	36	28.22	1.80	7.40	98.10

[1] XRF data were not available for previously-abated units and modern urban units in the study, as they were assumed to be free of LBP.

3.3.1.3 The Rochester Lead-in-Dust Study

Tables 3-17 and 3-18 summarize lead loadings and concentrations, respectively, in floor-dust samples across study units. Tables 3-19 and 3-20 summarize lead loadings and concentrations, respectively, in window sill-dust samples. Table 3-21 summarizes lead concentrations in soil samples taken at the dripline, while Table 3-22 summarizes lead concentrations in soil samples taken at the child's play area. Table 3-23 presents summaries of the observed maximum XRF value within the study units, for the interior only and the exterior only, as well as for the entire unit. All of these tables summarize results across all units, as well as within the four age categories in which the HUD National Survey units were categorized in Section 3.3.1.1.

Note from Table 3-17 that approximately 84% of the study units were built prior to 1940. Therefore, while the Rochester study considers units in an urban environment and does not attempt to target a particular lead exposure environment in recruiting the units, most of the units are older units and contain families with low income levels.

As seen in the HUD National Survey, dust-lead loadings and concentrations are highest among the units built prior to 1940 (Tables 3-17 through 3-20). For units built prior to 1980, the geometric mean floor dust-lead levels were often lower in the Rochester study than in the HUD National Survey. However, the ten units built after 1979 had higher geometric mean dust-lead levels than for the 1940-1959 and 1960-1979 year-built categories. It is unclear why these ten units would have such high dust-lead levels, other than existing within an urban environment.

Geometric mean soil-lead concentrations were higher than those observed in the HUD National Survey. As seen with dust-lead levels, units built after 1979 had a higher geometric mean soil-lead concentration compared to the 1960-1979 housing group. Less than half of the units had soil samples taken from the play area (Table 3-22), where geometric mean concentrations were generally lower than at the dripline for older units.

Table 3-23 indicates that at least 40% of the units within each age category (even houses built after 1979) contained LBP as measured by XRF. Units built prior to 1940 had the highest geometric mean paint-lead levels and the highest percentage of units with LBP in the study. However, of most interest is the frequency to which LBP was detected in the ten units built after 1979. Two of these units contained LBP in the interior, while four contained LBP in the exterior. It is possible that LBP exists within some of these units, as high lead levels were also observed in dust and soil. As a result, one may not wish to exclude post-1979 housing in urban settings when identifying LBP hazards in the nation's housing. However, interpretation of XRF results should take into account imprecisions associated with the measurements. For example, units with a maximum XRF value slightly greater than 1.0 mg/cm^2 are classified as having LBP, but the maximum value may be statistically equivalent to a value less than 1.0 mg/cm^2.

Table 3-17. Summary of Average Pre-Intervention Floor Dust-Lead Loading for Housing Units in the Rochester Study

Year Unit Was Built	# Units	Floor Dust-Lead Loading ($\mu g/ft^2$)[1]			
		Geometric Mean	Geometric Standard Deviation	Minimum	Maximum
All Units	205	17.70	3.20	1.21	8663.53
Before 1940	172	19.79	3.18	1.66	8663.53
1940-1959	19	8.36	2.61	1.21	26.93
1960-1979	4	7.84	2.40	2.13	13.21
After 1979	10	15.00	3.34	3.48	250.30

[1] Data summarized in this table are area-weighted arithmetic mean dust-lead loadings from floors for each unit. Results included in the summaries are only for dust samples collected using wipe techniques.

Table 3-18. Summary of Average Pre-Intervention Floor Dust-Lead Concentrations for Housing Units in the Rochester Study

Year Unit Was Built	# Units	Floor Dust-Lead Concentration ($\mu g/g$)[1]			
		Geometric Mean	Geometric Standard Deviation	Minimum	Maximum
All Units	204	350.84	3.74	8.25	40717.31
Before 1940	172	395.68	3.58	8.25	40717.31
1940-1959	18	209.43	4.61	16.52	7899.91
1960-1979	4	60.80	2.68	16.93	163.88
After 1979	10	226.14	2.99	57.02	1124.86

[1] Data summarized in this table are area-weighted arithmetic mean dust-lead concentrations from floors for each unit. Results included in the summaries are only for dust samples collected using BRM vacuum techniques.

Table 3-19. Summary of Average Pre-Intervention Window Sill Dust-Lead Loading for Housing Units in the Rochester Study

Year Unit Was Built	# Units	Window Sill Dust-Lead Loading ($\mu g/ft^2$)[1]			
		Geometric Mean	Geometric Standard Deviation	Minimum	Maximum
All Units	196	196.03	3.96	2.83	14901.36
Before 1940	164	233.68	3.67	2.85	14901.36
1940-1959	18	72.01	6.16	2.83	4393.03
1960-1979	4	52.31	1.38	36.23	70.75
After 1979	10	113.09	1.95	26.88	320.40

[1] Data summarized in this table are area-weighted arithmetic mean dust-lead loadings from window sills for each unit. Results included in the summaries are only for dust samples collected using wipe techniques.

Table 3-20. Summary of Average Pre-Intervention Window Sill Dust-Lead Concentrations for Housing Units in the Rochester Study

Year Unit Was Built	# Units	Window Sill Dust-Lead Concentration ($\mu g/g$)[1]			
		Geometric Mean	Geometric Standard Deviation	Minimum	Maximum
All Units	199	2787.03	8.44	3.15	368111.11
Before 1940	166	3859.20	7.33	15.85	368111.11
1940-1959	19	497.25	9.90	5.31	15017.51
1960-1979	4	473.44	2.92	159.77	1900.67
After 1979	10	674.30	8.56	3.15	8625.00

[1] Data summarized in this table are area-weighted arithmetic mean dust-lead concentrations from window sills for each unit. Results included in the summaries are only for dust samples collected using BRM vacuum techniques.

Table 3-21. Summary of Average Pre-Intervention Dripline Soil-Lead Concentrations for Housing Units in the Rochester Study

Year Unit Was Built	# Units	Soil-Lead Concentration (μg/g)[1]			
		Geometric Mean	Geometric Standard Deviation	Minimum	Maximum
All Units	186	731.03	3.68	12.31	21049.00
Before 1940	158	937.83	3.17	12.31	21049.00
1940-1959	14	291.14	3.30	29.70	1788.00
1960-1979	4	66.35	1.79	29.00	111.00
After 1979	10	135.26	3.10	26.00	876.00

[1] Data summarized in this table are area-weighted arithmetic mean dripline soil-lead concentrations for each unit.

Table 3-22. Summary of Average Pre-Intervention Soil-Lead Concentrations from Play Areas for Housing Units in the Rochester Study

Year Unit Was Built	# Units	Soil-Lead Concentration (μg/g)[1]			
		Geometric Mean	Geometric Standard Deviation	Minimum	Maximum
All Units	87	266.90	2.78	28.00	7300.00
Before 1940	79	277.54	2.79	28.00	7300.00
1940-1959	6	184.51	3.09	55.40	767.00
1960-1979	1	138.00	.	138.00	138.00
After 1979	1	215.00	.	215.00	215.00

[1] Data summarized in this table are area-weighted arithmetic mean soil-lead concentrations from play areas for each unit.

Table 3-23. Summary of Observed Maximum XRF Paint-Lead Concentration at Pre-Intervention for Housing Units in the Rochester Study

Year the Unit Was Built	# Units	% of Units with LBP[1]	Observed Maximum XRF Paint-Lead Levels (mg/cm^2)			
			Geometric Mean	Geometric Standard Deviation	Minimum	Maximum
Entire Unit (interior and exterior)						
All Units	205	89%	12.80	3.88	0.50	59.77
Before 1940	172	95%	16.63	3.05	0.50	59.77
1940-1959	19	68%	5.50	4.97	0.50	37.57
1960-1979	4	50%	1.04	1.89	0.57	1.93
After 1979	10	40%	1.93	5.92	0.50	39.43
Interior of Unit						
All Units	205	83%	7.58	4.38	0.50	57.57
Before 1940	172	91%	9.90	3.67	0.50	57.57
1940-1959	19	63%	2.86	4.71	0.50	32.87
1960-1979	4	0%	0.61	1.12	0.57	0.73
After 1979	10	20%	1.32	5.72	0.50	39.43
Exterior of Unit						
All Units	204	79%	8.14	4.91	0.50	59.77
Before 1940	171	84%	10.33	4.40	0.50	59.77
1940-1959	19	63%	3.47	5.22	0.50	37.57
1960-1979	4	50%	1.00	1.97	0.50	1.93
After 1979	10	40%	1.63	4.95	0.50	37.43

[1] LBP is defined as the maximum XRF value exceeding 1.0 mg/cm^2.

3.3.2 Characterizing the Population of Children in the Nation's Housing Stock

Tables 3-4 through 3-10 in Section 3.3.1.1 contained summaries of estimated environmental-lead levels in the nation's occupied housing stock prior to implementing interventions that would occur under the proposed §403 rule. These summaries were based on data from the HUD National Survey with sampling weights revised to represent the 1997 national occupied housing stock. To characterize the extent to which these environmental-lead levels provide exposures to children and to characterize the benefits associated with §403, it was

necessary to estimate numbers of children of specific age groups who reside within the housing units represented in Table 3-3 of Section 3.3.1.1.

Methods used to obtain numbers of children in the national housing stock are presented in Section C.1.2. These methods used estimates of the 1997 birth rate, average number of children per 1,000 people, and average number of residents per housing unit. While this risk assessment focused on characterizing the blood-lead concentrations and associated health effects for children aged 12-35 months (i.e., 1-2 years), the sensitivity analysis (Section 5.4) also considered children aged 12 to 71 months (1 to 5 years). Therefore, the methods in Appendix C were applied to both age groups.

Table 3-24 provides the number of children residing in the 1997 housing stock according to age of unit and age of child. Numbers of children associated with the 1997 sampling weights for each HUD National Survey unit are displayed in Table C-7 of Appendix C.

Table 3-24. Estimated Number of Children in the 1997 National Housing Stock, by Age of Child and Year-Built Category

Year-Built Category	Age of Child	
	12-35 Months	12-71 Months
Prior to 1940	1,578,000	4,043,000
1940-1959	1,581,000	4,051,000
1960-1979	2,805,000	7,188,000
After 1979	1,996,000	5,115,000
Entire Nation[1]	7,961,000	20,397,000

[1] Value may differ from sum of previous rows due to rounding.

3.4 DISTRIBUTION OF CHILDHOOD BLOOD-LEAD

The §403 risk assessment, as described in Section 2.4, characterizes health effects and blood-lead concentrations for children aged 1 to 2 years (i.e., 12 to 35 months). The national distribution of blood-lead concentration for children aged 1-2 years is based on NHANES III. This information is summarized in Section 3.4.1. Supporting information on children's blood-lead concentrations is provided through summary statistics from the Baltimore R&M Study and

the Rochester Study. These summaries are presented in Section 3.4.2. While blood-lead concentrations in these two epidemiological studies are not representative of lead exposure on a national scale, they provide additional evidence on the prevalence of elevated childhood blood-lead concentrations.

3.4.1 NHANES III

The National Health and Nutrition Examination Surveys, conducted by the CDC's National Center for Health Statistics (NCHS), trace the health and nutritional status of the noninstitutionalized, civilian U. S. population. The surveys consist of adult, youth, and family questionnaires, followed by standardized physical examinations.

The Third National Health and Nutrition Examination Survey (NHANES III), conducted from 1988 to 1994, was the seventh in a series of national examination studies conducted by NCHS since 1960. The target population for NHANES III included the civilian noninstitutionalized population 2 months of age and older. The primary objectives of NHANES III were the following (CDC, 1992):

> *"To produce national population health parameters; to estimate the national prevalence of selected diseases and disease risk factors; to investigate secular trends in selected diseases and risk factors; to contribute to the understanding of disease etiology; and to investigate the natural history of selected diseases."*

Approximately 40,000 persons were sampled in NHANES III, including approximately 3,000 children aged 1 to 2 years. Phase 1 of NHANES III, conducted from 1988-1991, provided the most recently-collected data on blood-lead concentrations that were available for analysis in the §403 risk assessment. These data included 1341 children aged 1 to 2 years, of which 924 had blood-lead concentrations reported.

Study participants in NHANES III were subjected to a physical examination conducted by a physician, a dentist, and health technicians. For participants aged 12 months and older, these examinations included taking a blood sample via venipuncture. This sample was analyzed for lead content by graphite furnace atomic absorption spectrophotometry.

To provide for a nationally representative sample, a complex survey design was employed in NHANES III (CDC, 1992; CDC, 1994). Although estimates of national population health and nutrition parameters were the primary objectives of the survey, suitably precise estimates for certain age and race groups were obtained through oversampling. As a result, the NHANES III provides national and subpopulation estimates of the distribution of childhood blood-lead concentrations.

In NHANES III, each subject was assigned a series of sampling weights. Each weight, determined from the 1990 Current Population Survey (CPS), indicates the total number in the U.S. population represented by a subject at a given stage of the survey. Because the risk assessment is using the blood-lead concentrations, the sampling weight assigned to subjects at the time of the physical examination is utilized in the risk assessment.

Tables 3-25 and 3-26 summarize the blood-lead concentrations from NHANES III for children aged 1-2 years (12-35 months) and aged 1-5 years (12-59 months). According to Table 3-25, the estimated geometric mean blood-lead concentration for children aged 1-2 years is 4.046 μg/dL, with a geometric standard deviation of 2.057. The geometric mean declines to 3.571 μg/dL for children aged 1 to 5 years. Table 3-26 contains probabilities of observing elevated blood-lead concentrations within these two age groups. Slightly over 10% of U.S. children aged 1-2 years are estimated to have blood-lead concentrations greater than 10 μg/dL (the current action level established by the CDC). Blood-lead concentrations greater than 25 μg/dL are relatively rare. The probability of elevated blood-lead concentration and the geometric mean blood-lead concentration declines as children aged 3-5 years are also considered, supporting the hypothesis that blood-lead concentration tends to peak at some age between 1 and 2 years.

For children aged 1 to 2 years, Table 3-27 presents geometric mean blood-lead concentration and percentage of children with a blood-lead concentration above 10 μg/dL, for selected subgroups of the U.S. population (family income, race, and urban status). These results illustrate that socioeconomic status is an important factor in the incidence rate of elevated lead exposure. Low-income families, especially those containing non-Hispanic African-Americans, have the highest percentage of children with blood-lead concentration exceeding 10 μg/dL. Urbanicity is also an important factor, with children residing in urban centers having the highest probability of exhibiting elevated blood-lead concentrations. Urban centers are usually

associated with high environmental-lead exposure, due to the high density of older buildings containing lead-based paint and remaining fallout of leaded gasoline emissions from urban traffic. These results confirm that while childhood blood-lead concentrations may have declined over the years, childhood lead exposure remains a real public health threat for certain subgroups of the U.S. population.

Table 3-25. Summary of Blood-Lead Concentration Data for Children Aged 1-2 Years and Aged 1-5 Years, Based on NHANES III (Phase 1)

Age Range (years)	# Children with Blood-Lead Conc. Reported	Sum of NHANES Sample Weights[1]	Blood-Lead Concentration (μg/dL)			
			Minimum	Maximum	Geometric Mean	Geometric Standard Deviation
1-2	924	7,812,631	0.70	56.6	4.046	2.057
1-5	2232	19,206,122	0.70	72.0	3.571	2.084

[1] Weights assigned at the time of the physical examination. Included in this sum are the weights for all children who were examined, including those who did not have a blood-lead concentration reported.

Table 3-26. Estimated Probabilities of Elevated Blood-Lead Concentrations in Children Aged 1-2 Years and Aged 1-5 Years, Based on NHANES III (Phase 1)

Age Range (years)	# Children with Blood-Lead Conc. Reported	Sum of NHANES Sample Weights[1]	Percentages with Elevated Blood-Lead Concentration			
			≥10 μg/dL	≥15 μg/dL	≥20 μg/dL	≥25 μg/dL
1-2	924	7,812,631	10.5%	3.5%	1.3%	0.6%
1-5	2232	19,206,122	8.0%	2.5%	0.9%	0.4%

[1] Weights assigned at the time of the physical examination. Included in this sum are the weights for all children who were examined, including those who did not have a blood-lead concentration reported.

Table 3-27. Estimated Percentage of Children With Blood-Lead Concentrations Exceeding 10 μg/dL, and the Geometric Mean and Geometric Standard Deviation of Blood-Lead Concentration, for Children Aged 1 to 2 Years Within Selected Subgroups

Percentage Geometric Mean (GSD)		All Children[1]	Selected Race Groups		
			Non-Hispanic White	Non-Hispanic African-American	Mexican-American
Family Income Level[2]	Low	17.4% 5.00 (2.04)	12.2% 4.17 (2.02)	30.0% 6.86 (2.05)	7.4% 4.39 (1.81)
	Mid	8.0% 3.73 (2.01)	7.7% 3.58 (2.00)	7.5% 4.27 (2.07)	6.9% 3.94 (1.87)
	High	7.1% 3.31 (2.02)	7.9% 3.33 (2.06)	2.6% 3.54 (1.69)	< 1% 2.69 (1.63)
Urban Status[3]	Central City, ≥1,000,000 Persons	10.7% 4.04 (2.08)	6.5% 3.60 (1.96)	26.0% 5.53 (2.44)	9.5% 4.00 (1.95)
	Central City, <1,000,000 Persons	20.4% 4.92 (2.05)	18.6% 4.21 (2.12)	16.9% 5.76 (1.78)	9.5% 4.70 (1.88)
	Non-Central City	3.7% 3.46 (1.93)	3.4% 3.25 (1.97)	5.2% 5.23 (1.66)	6.6% 4.35 (1.78)

[1] Includes race/ethnicity groups not shown separately.
[2] Income level was defined by poverty income ratio (PIR) categorized as low (0<PIR<1.30), mid (1.30≤PIR<3.0), and high (PIR≥3.0). Persons with missing information on income level are not included in summaries by income level.
[3] Persons with missing information on urban status are not included in summaries by urban status.

Source: National Center for Health Statistics, NHANES III, 1988-91.

The blood-lead concentration summaries in Tables 3-25 through 3-27 may differ from what is reported in published literature on NHANES III results (Pirkle et al., 1994; Brody et al., 1994). The summaries in these three tables were calculated using the public-use dataset for Phase 1 of NHANES III. Reasons why these summaries would differ from published results include:

- When subjects received two physical examinations in Phase I of the NHANES III, the public-use dataset reported results only for the first examination, regardless of whether a blood-lead concentration was available for the subject from this examination. In contrast, published results included blood-lead information from the second examination when information from the first examination was not reported.

- If a subject did not have a detectable blood-lead concentration, the NHANES III set the blood-lead concentration equal to a small, positive value. This value differed between the dataset used to produce the published results and the public-use dataset.

- The sampling weights were refined after the published results were produced. The refined weights are included in the public-use dataset.

Data from Phase 1 of NHANES III are used to characterize the distribution of blood-lead concentration for children aged 1 to 2 years in 1997, prior to any intervention activities that occur as a result of implementing the proposed §403 rules. More details on this distribution are presented in Section 5.1.

3.4.2 Baltimore Repair and Maintenance (R&M) Study

Childhood blood-lead concentrations collected prior to any interventions performed in the Baltimore R&M Study (Section 3.2.1) provide evidence of elevated blood-lead concentrations for children in high-exposure environments. Units slated for R&M interventions were documented to contain lead-based paint and elevated lead levels in household dust. Modern urban units acted as negative controls, being free of lead-based paint. Previously-abated units were abated for lead-based paint previous to this study, and therefore reflect a post-abatement environment. The urban setting of this study indicates an increased potential for lead exposures.

Table 3-28 summarizes the pre-intervention blood-lead concentrations. The overall geometric mean blood-lead concentration for children aged 1-2 years is 9.94 μg/dL, which is over twice the value of 4.05 μg/dL for NHANES III. In particular, geometric mean blood-lead concentrations for children aged 1-2 years in previously-abated units and units slated for R&M intervention (11.86 μg/dL and 10.56 μg/dL, respectively) are high, while for 1-2 year old children residing among the modern urban units, where potential for lead exposure was reduced, the geometric mean was 2.82 μg/dL.

The probabilities that children's blood-lead concentrations exceed 10, 15, 20, or 25 μg/dL are high for all but the modern urban units (Table 3-28). Approximately 58% of 1-2 year olds in the R&M Study had blood-lead concentrations greater than 10 μg/dL, compared to the NHANES III estimate of 10.5% (Table 3-26). Again, this is likely due to the increased lead exposure associated with these children compared to the national population. Of the different unit groups

Table 3-28. Summary Statistics on Blood-Lead Concentration Measured Prior to Intervention in the Baltimore Repair and Maintenance Study

Age Range (years)	Sample Size	Blood-Lead Concentration (μg/dL)				Percentages with Elevated Blood-Lead Concentration			
		Minimum	Maximum	Geometric Mean	Geometric Standard Deviation	≥10 μg/dL	≥15 μg/dL	≥20 μg/dL	≥25 μg/dL
All Children with Measured Pre-Intervention Blood-Lead Concentration[1]									
All[2]	163	0.9	65.5	10.42	2.12	58.3	35.0	14.7	10.4
1-2	93	0.9	65.5	9.94	2.29	53.8	33.3	16.1	12.9
Children in Previously-Abated Units									
All[3]	23	3.65	28.8	12.70	1.60	73.9	43.5	13.0	4.3
1-2	12	3.65	24.15	11.86	1.71	66.7	50.0	8.3	0.0
Children in Units Slated for Repair and Maintenance									
All[2]	69	1.75	65.5	10.20	1.87	49.3	27.5	10.1	5.8
1-2	41	1.75	65.5	10.56	1.99	51.2	26.8	14.6	9.8
Children in Modern Urban (control) Units									
All[4]	19	0.9	10.15	3.04	1.74	5.3	0.0	0.0	0.0
1-2	14	0.9	5.8	2.82	1.67	0.0	0.0	0.0	0.0

[1] Includes children who did not complete the study and children in non-study units targeted to move into vacant study units after intervention. As a result, numbers of children within the four specified housing groups at pre-intervention do not total the numbers of children with pre-intervention blood-lead concentrations.
[2] Children aged 6-57 months.
[3] Children aged 10-57 months.
[4] Children aged 16-43 months.

in the study, the highest percentage of children with blood-lead concentrations above 10 μg/dL occurred for previously-abated units (67%). Recall from Section 3.1.3.2 that environmental-lead levels were high in these units as well, suggesting that lead exposures were not removed as a result of the abatements performed previous to this study. No children aged 1-2 years who resided in modern urban units had elevated blood-lead concentrations.

3.4.3 Rochester Study

Blood-lead concentration data were collected in the Rochester Study (Section 3.2.2) for 205 children aged 12-30 months of age. While units having recent major renovations or the potential for lead contamination from exterior sources were not considered in this study, no

attempt was made to include units based on environmental-lead levels or the presence of lead-based paint. Therefore, blood-lead concentrations in this study likely reflect typical lead exposure conditions in an urban setting.

Table 3-29 summarizes the blood-lead concentrations from the Rochester study. The geometric mean blood-lead concentration was 6.38 μg/dL (geometric standard deviation, 1.85). Twenty-three percent of the children had blood-lead concentrations above 10 μg/dL, 8% above 15 μg/dL, and 3% above 20 μg/dL. These numbers are higher than those for NHANES III, but lower than those for the Baltimore R&M study.

Table 3-29. Summary Statistics on Blood-Lead Concentration Measured in the Rochester Lead-in-Dust Study

Number of Children	Blood-Lead Concentration (μg/dL)				Percentages with Elevated Blood-Lead Concentration			
	Minimum	Maximum	Geometric Mean	Geometric Standard Deviation	≥10 μg/dL	≥15 μg/dL	≥20 μg/dL	≥25 μg/dL
205	1.4	31.7	6.38	1.85	23.4	7.8	2.9	1.5

4.0 DOSE-RESPONSE ASSESSMENT

CHAPTER 4 SUMMARY

Chapter 4 presents the approach for characterizing the relationship between environmental lead exposure and the resulting adverse health effects. The relationship is established in two stages. First, the relationship between environmental lead levels and blood-lead concentration is characterized by two models, the IEUBK and EPI models. Then the relationship between blood-lead concentration and representative health effects is established. This relationship is applied in the integrated risk assessment, using environmental data from the HUD National Survey to estimate the number of children who will benefit from the §403 rule. Additional topics covered in this chapter describe assumptions required for this application, including 1) methods for converting environmental lead levels measured by different sampling methods; 2) methods for incorporating the effect of paint pica, and 3) development of a range of options for the standards.

This chapter presents the approach for characterizing the relationship between children's exposure to lead in dust, soil, and paint and the resulting adverse health effects. It would be ideal to relate particular health outcomes directly to environmental lead levels. However, most studies of lead in the environment relate residential measures of lead exposure (e.g., floor or window sill dust-lead loadings) to measures of body lead burden (e.g. blood-lead concentration), rather than directly to health effects. In addition, most studies of health effects of lead exposure relate specific health outcomes (e.g. IQ point loss) to measures of body lead burden, rather than directly to environmental lead levels.

Thus, the dose-response relationship between environmental lead levels and health outcomes for this risk assessment will be established in two stages. First, the relation between environmental lead levels and blood-lead concentration will be estimated via quantitative models. Then, the relationships between blood-lead concentration and health effects documented in the scientific literature will be used to establish the link between environmental lead exposure and health outcomes.

Sections 4.1 through 4.3 discuss the methodology associated with characterizing the dose-response relationship between environmental lead levels and blood-lead concentration.

Section 4.1 describes two models used to relate environmental lead levels to blood-lead concentrations. While the goal of each model is similar, the modeling approaches are markedly

distinct. The first, described in Section 4.1.1, is a biokinetics-inspired model that attempts to simulate how lead is absorbed, processed, and eliminated by the human body. The second model, described in Section 4.1.2, is a regression model that seeks to relate observed environmental lead levels to observed blood-lead concentrations.

Each of these models is used to predict a national distribution of blood-lead concentrations in both a pre- and post-§403 environment. Environmental lead levels in the HUD National Survey homes are used as input to the models to predict the geometric mean blood-lead concentration of children exposed to environmental lead conditions evidenced in HUD National Survey homes. The national distribution of blood-lead concentrations is then developed by allowing each home to represent a proportion of the total number of 1-2 year old children (Section 3.3).

Environmental dust-lead levels in the HUD National Survey were measured by the blue nozzle vacuum sampling method. However, §403 dust-lead standards will be specified in terms of wipe sampling, and the EPI model dust-lead parameters are based on wipe sampling. Section 4.2 discusses conversion equations developed to relate different dust sampling methods that will be used, for example, to convert blue nozzle vacuum dust loadings to wipe dust loadings.

Section 4.3 describes the methodology used to an account for the effect of a child's pica for paint on the distribution of blood-lead concentrations developed using each model.

Methodology discussed in this chapter related to characterizing the relationship between a national distribution of environmental lead levels and a national distribution of blood-lead levels is illustrated in Figure 4-1 Additional details on methodology related to predicting the national distribution of blood-lead concentrations is provided in Chapter 5.

Section 4.4 presents the approach for relating blood-lead concentration to the representative health effects identified in Chapter 2: IQ deficits and elevated blood-lead concentrations. Lead-related IQ deficits are measured directly as decrements in IQ scores and through the increased incidence of IQ scores less than 70. Incidence of elevated blood-lead concentrations is estimated at 10 μg/dL and 25 μg/dL, based on CDC guidelines (CDC, 1991), as described in Section 2.4.1.

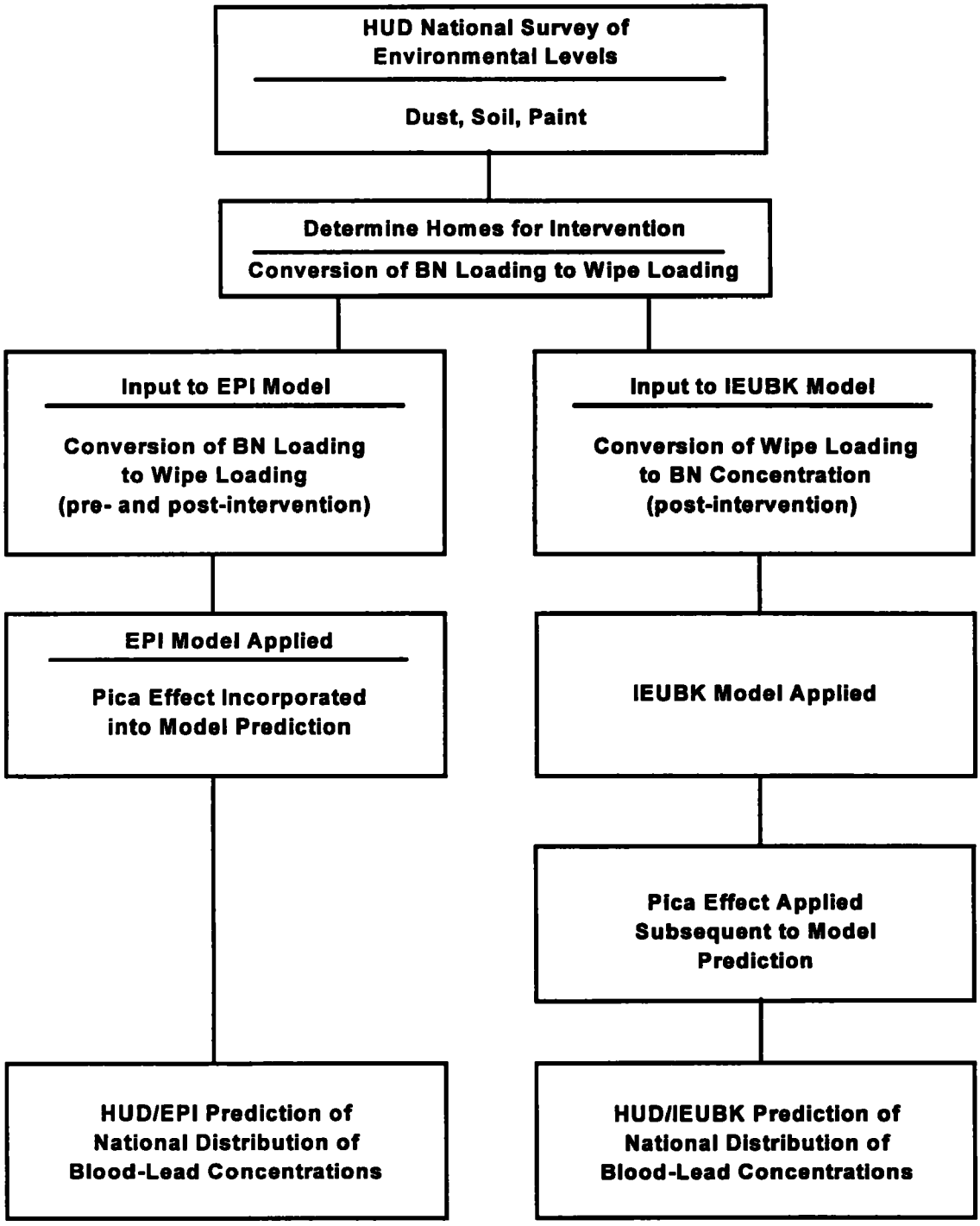


Figure 4-1. Methodology Associated With Characterizing the Relationship Between a National Distribution of Environmental Lead Levels and a National Distribution of Blood-Lead Concentrations

The tools and methodology presented in Sections 4.1 through 4.4 are used in Chapter 5 to help characterize the reduction in childhood health risks and blood-lead concentrations for various options for the §403 standards. Because standards are set for multiple media it was not possible to look at all sets of standards. Section 4.5 presents a range of options for standards that will be evaluated in the integrated risk analysis in Chapter 5 and discusses the methodology used to specify the ranges.

4.1 ESTIMATION OF MEAN BLOOD LEAD CONCENTRATION

This section describes the first stage of the dose-response relationship, estimation of the relationship between environmental lead levels and blood-lead concentration. Two modeling approaches will be used to estimate blood-lead concentrations from environmental lead exposure. In the first approach, blood-lead concentrations are estimated using EPA's Integrated Exposure, Uptake, and Biokinetic Model for Lead (version 0.99D), hereafter referred to as the IEUBK model (EPA, 1994a; 1995e). The second approach estimates blood-lead concentrations using a multiple regression model fitted to epidemiological data.

4.1.1 IEUBK Model

The IEUBK model is a biokinetics-inspired simulation model designed to predict the probability of elevated blood-lead levels in children given a set of environmental lead levels. The model addresses three components of environmental risk assessment: 1) multi-media nature of exposures to lead, 2) lead pharmacokinetics, and 3) inter-individual variability in blood-lead levels, through the estimation of the probability distribution of blood-lead levels for children exposed to similar environmental lead concentrations.

Specifically, the model uses lead concentrations measured in dust, soil, air, water, diet, and other ingested media as inputs to estimate a longitudinal exposure pattern from birth to seven years of age (EPA, 1995e). The model then estimates a distribution of blood lead levels for a population of children exposed to that exposure pattern. The center of this distribution, the geometric mean, is predicted by the model. A constant empirical estimate is used by the model to represent the variability about the geometric mean. In statistical terminology, this variation is referred to as the geometric standard deviation (GSD). A value of 1.6 for the GSD was estimated

from residential community blood-lead studies and is assumed by the model. The GSD pertains to the inter-individual and biological variability in blood-lead levels of individual children exposed to similar environmental lead levels. It must be recognized here that the IEUBK model is not intended to predict the blood-lead level of an individual child, and therefore cannot substitute for a medical evaluation of an individual child.

It is beyond the scope of this document to describe the IEUBK model in detail. Very briefly, the model has three distinct functional components that work together in series: exposure, uptake, and biokinetic components. Each model component is a set of complex equations and parameters. The Technical Support Document (EPA, 1995e) provides the scientific basis of the parameters and equations used in the model and the Guidance Manual (EPA, 1994a) includes a detailed description of the exposure pathways, absorption mechanism, and biokinetic compartments and associated compartmented transfers of lead.

The IEUBK model can be implemented by running a PC-compatible software program. The program provides the user access to input values for the exposure and uptake parameters. The biokinetic parameter values, however, are not accessible. The Guidance Manual (EPA, 1994a) recommends using the input values specified by the software for the exposure and uptake parameters whenever more representative data are not available. In the absence of better information, these default input values are used in the risk characterization to predict blood-lead levels via the IEUBK model. In addition, blood-lead concentrations are predicted at age 24 months. Table 4-1 presents the default input values for the exposure and uptake parameters.

For purposes of illustration, Figure 4-2 displays the relationship between blood-lead concentration predicted by the IEUBK model and soil- or dust-lead concentration for children aged 2 years.

The dust-lead concentration was set to 200 ppm for the soil curve as the HUD National Survey data report a geometric mean dust-lead concentration of 192 ppm. Similarly, the soil-lead concentration was set to 100 ppm for the dust curve because the HUD National Survey data estimated a geometric mean soil-lead concentration of 78 ppm. Air, water, diet, and other ingested media were set to model default values. From the dust curve in Figure 4-2, the predicted blood-lead concentration is 3 μg/dL for a dust-lead concentration of 100 ppm and a

Table 4-1. Summary of Default Parameter Values Used in the IEUBK Model (Version 0.99D).

Air Parameters

Parameter	Vary air concentration by year?	Outdoor air lead concentration	Indoor air lead concentration
Setting*	No	0.10 $\mu g/m^3$	30% of outdoor value

* All air parameters use default values

Diet Intake Parameters

	Lead intake in diet, by age of child						
Parameter	0-1 yrs	1-2 yrs	2-3 yrs	3-4 yrs	4-5 yrs	5-6 yrs	6-7 yrs
Setting*	5.53 µg/day	5.78 µg/day	6.49 µg/day	6.24 µg/day	6.01 µg/day	6.34 µg/day	7.00 µg/day

* All diet intake parameters use default values

Water Intake Parameters

	Lead Conc. in Water	Drinking water consumption, by age of child						
Parameter		0-1 yrs	1-2 yrs	2-3 yrs	3-4 yrs	4-5 yrs	5-6 yrs	6-7 yrs
Setting*	4 µg/L	0.20 L/day	0.50 L/day	0.52 L/day	0.53 L/day	0.55 L/day	0.58 L/day	0.59 L/day

* All water intake parameters use default values

Soil and Dust Intake Parameters

	Soil/Dust Ingestion Weighting Factor	Total soil + dust intake, by age of child						
Parameter		0-1 yrs	1-2 yrs	2-3 yrs	3-4 yrs	4-5 yrs	5-6 yrs	6-7 yrs
Setting*	45% soil; 55% dust	0.085 g/day	0.135 g/day	0.135 g/day	0.135 g/day	0.1 g/day	0.09 g/day	0.085 g/day

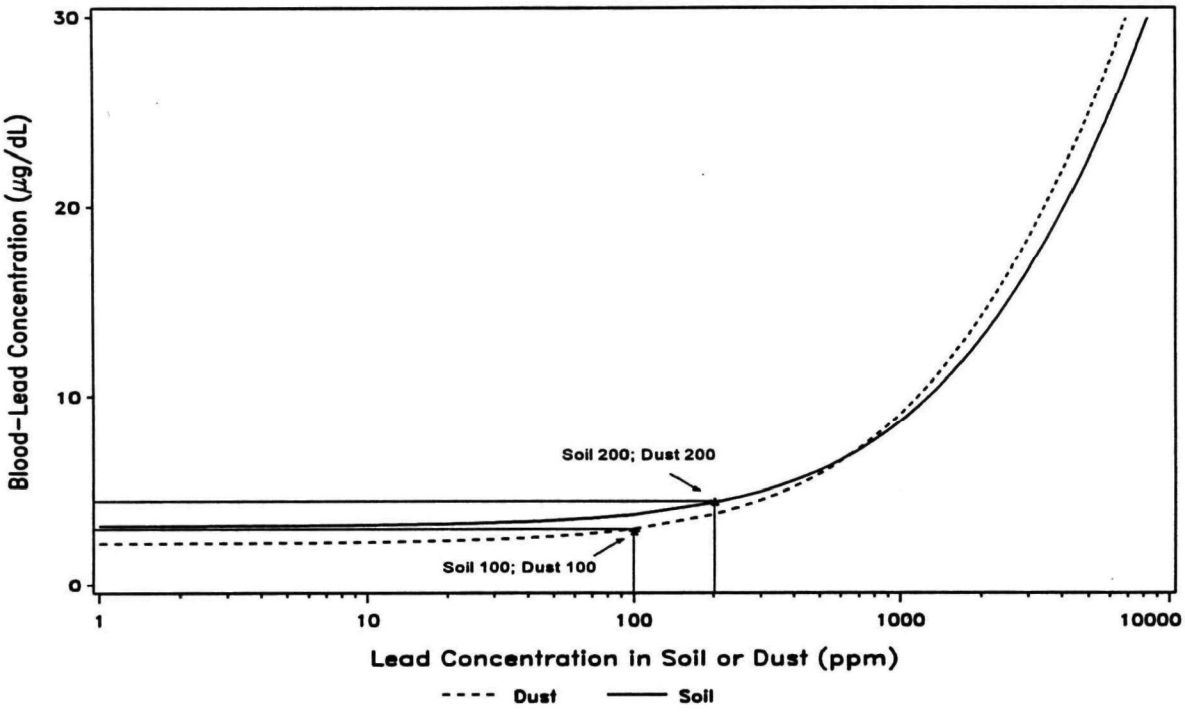
* Soil and dust lead concentrations are input. All other parameters use default values.

Absorption Method Parameters

	Half Saturation Level	Total Absorption					Fraction of Total Assumed Passive Absorption				
Parameter		Soil	Dust	Water	Diet	Alt.	Soil	Dust	Water	Diet	Alt.
Setting	100 µg/day	30%	30%	50%	50%	0%	0.20	0.20	0.20	0.20	0.20

Blood Lead Parameter

Parameter	Geometric Standard Deviation (GSD)
Setting	1.6



Dust-lead concentration was set to 200 ppm for the soil curve and soil-lead concentration was set to 100 ppm for the dust curve.

Figure 4-2. IEUBK Model Predicted Blood-Lead Concentration for Children Two Years Old Plotted Separately Versus Soil-Lead Concentration and Dust-Lead Concentration for Fixed Default Values of the Remaining Model Parameters

soil-lead concentration of 100 ppm. Similarly, from the soil curve, the predicted mean blood-lead concentration for 200 ppm for lead concentrations of both soil and dust is 4.5 μg/dL. It is important to recognize that each point on the predicted curve represents a geometric mean blood-lead level for children exposed to similar environmental lead levels. The blood-lead levels for individual children will vary about the predicted geometric mean. The variation about the geometric mean is captured by the GSD.

4.1.2 Epidemiological Model

An epidemiological (EPI) model is another model to estimate blood-lead levels in young children stemming from exposure to environmental lead. The EPI model was developed from the Rochester Study (Lanphear et al., 1995; HUD, 1995a) data. Measures that were in both the

Rochester data and the HUD National Survey data or measures in the Rochester data that could be closely approximated by measures in the HUD National Survey data were considered for inclusion in the model. Variables whose definition provided a convenient translation when applied to the National Survey, whose predictive power in Rochester was high, and whose spread in the National Survey population covered a wide range of values, were used in the EPI model. Appendix W provides further details on development of the EPI model.

The EPI model addresses three components of environmental risk assessment: 1) multi-media nature of exposures to lead, 2) intercorrelations among the environmental lead exposure variables as they relate to blood-lead levels, and 3) inter-individual variability in blood-lead levels. As with the IEUBK model, the EPI model is used to estimate a probability distribution of blood-lead levels for children exposed to similar environmental lead levels. The center of the distribution, the geometric mean, is predicted by the model. As with the IEUBK, a GSD of 1.6 is used to represent the variability about the geometric mean.

The main difference between the EPI model and the IEUBK model is that the EPI model relates blood-lead concentration to dust-lead loading (rather than dust-lead concentration) and two other environmental variables based on a multiple regression equation fitted to data from a single locality. In contrast, the IEUBK model relates blood-lead concentration to dust-lead concentration, was not developed for data form a single locality, and is based on a simulation of the lead biokinetics within a child's body.

The EPI model applies to children aged 12-30 months. The model may be written as:

$$\log(PbB)=\beta_o+\beta_1\cdot\log(DripSoil)+\beta_2\cdot PaintPica+\beta_3\cdot\log(FloorLoad)+\beta_4\cdot\log(SillLoad)+\epsilon$$

where DripSoil is the dripline soil-lead concentration at the house; PaintPica is an indicator variable equal to 1.5 if there is a child with paint pica in the house and the house has deteriorated lead based paint, and zero otherwise; FloorLoad is the area-weighted floor dust-lead loading for all surfaces in the house; and SillLoad is the area-weighted average of dust-lead loading from window sills in the house. In the equation β_0, β_1, β_2, β_3, and β_4 are model parameters, ϵ is a random error term that represents the residual error left unexplained by the model and log is the natural logarithm function. In the Rochester data, the extent of paint pica was coded as a 0, 1

or 2, where 0 represented no paint pica, 1 was for children who exhibited paint pica only rarely, and 2 was for children who exhibited paint pica at least sometimes. A value of 1.5 was used for the EPI model when applied to the HUD National Survey data, since it was the average of the two values used for children who exhibited any amount of paint pica in the Rochester data.

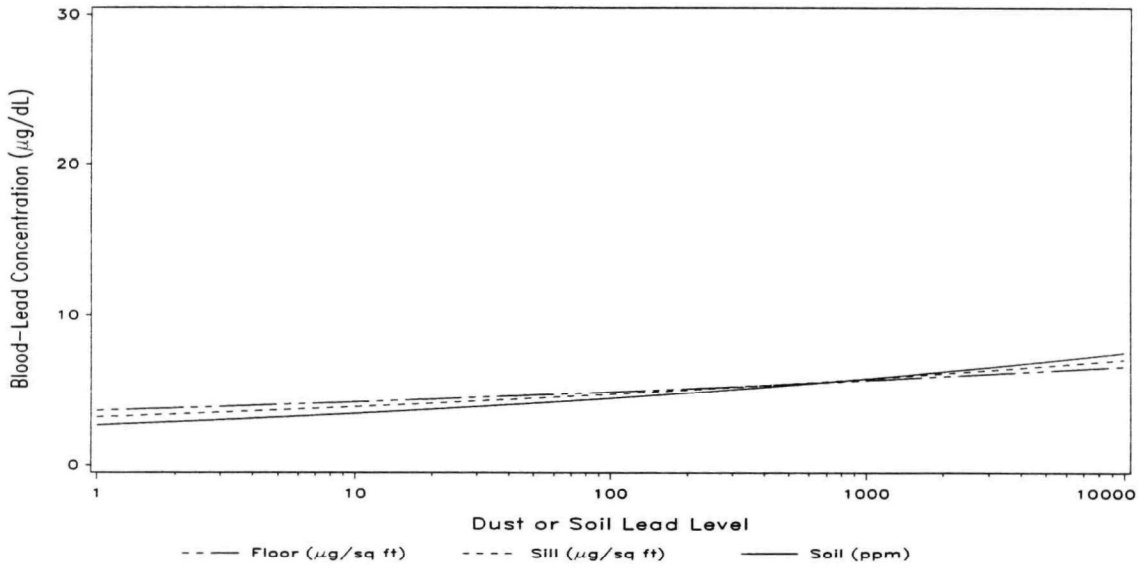
The model was fit to the Rochester data using the SAS® System. Table 4-2 presents the resulting parameter estimates and associated standard errors. For example, the EPI model predicts a geometric mean blood-lead concentration of 5.3 μg/dL for children with no paint pica or damaged lead-based paint exposure but who are exposed to a floor dust-lead loading of 40 $\mu g/ft^2$, a drip line soil-lead concentration of 200 ppm, and a window sill dust-lead loading of 100 $\mu g/ft^2$. It is important to note that the EPI model predicts the geometric mean blood-lead level for children in similar exposure environments; it should not be used to predict the blood-lead level for an individual child. The variation in blood-lead concentration about the geometric mean is captured by the GSD. A GSD of 1.6 is assumed by the EPI model, to be consistent with the IEUBK model.

Table 4-2. Parameter Estimates and Associated Standard Errors for the Multimedia Exposure Model Based on Data from the Rochester Lead-In Dust Study.

Parameter	Predictor Variable	Estimated Effect	Standard Error
β_0	Intercept	0.418	0.240
β_1	log(DripSoil)	0.114	0.035
β_2	PaintPica	0.248	0.100
β_3	log(DustLoad)	0.066	0.040
β_4	log(SillLoad)	0.087	0.036

For purposes of illustration, Figure 4-3 displays the blood-lead concentration, predicted by the EPI model, as a function of drip line soil-lead concentration, floor dust-lead loading, and window sill dust-lead loading. Each medium is represented by a different line on the plot. For the prediction of blood-lead concentration as a function of soil-lead concentration, the floor dust-lead loading is held constant at 25 $\mu g/ft^2$, window sill dust-lead loading is held constant at 50

$\mu g/ft^2$, and paint pica is set equal to zero. For predicting blood-lead concentration as a function of floor dust-lead loading, soil-lead concentration is held constant at 100 ppm, window sill dust-lead loading is held constant at 50 $\mu g/ft^2$, and again paint pica is set equal to zero. Finally, for the sill dust-lead loading curve, soil-lead concentration was held constant at 100 ppm, floor dust-lead loading was held constant at 25 $\mu g/ft^2$, and paint pica was set equal to zero. The values at which variables were held constant in Figure 4-2 were chosen to be approximately equal to the variable's weighted geometric mean estimated from the HUD National Survey data. The blood-lead levels for individual children will vary about the predicted geometric mean. This variation is captured by the assumed GSD of 1.6.



Sill dust-lead loading was set to 50 $\mu g/ft^2$ and soil-lead concentration was set to 100 ppm for the floor dust curve.
Floor dust-lead loading was set to 25 $\mu g/ft^2$ and soil-lead concentration was set to 100 ppm for the sill dust curve.
Floor dust-lead loading was set to 25 $\mu g/ft^2$ and sill dust-lead loading was set to 50 $\mu g/ft^2$ for the soil lead curve.

Figure 4-3. EPI Model Predicted Blood-Lead Concentration Plotted Separately Against Floor Dust-Lead Loading, Sill Dust-Lead Loading and Soil Lead Concentration for Fixed Values of the Remaining Model Inputs

As any of the environmental lead levels increase, the geometric mean blood-lead concentration increases gradually. This is similar to the same plot for the IEUBK model except that there is no point at which blood-lead concentration begins to rise rapidly as environmental lead increases.

4.2 UTILIZING DUST LEAD LOADINGS

The HUD National Survey is the only national survey of environmental lead levels and therefore will be used for prediction of a national distribution of blood-lead concentrations. Dust lead measurements in the HUD National Survey were collected by the blue nozzle vacuum method. However, §403 standards for dust will be expressed as a measured lead loading collected by a dust wipe sample. This discrepancy leads to several instances where conversion factors will be used in the risk assessment methodology. These include:

- Convert blue nozzle loading to wipe loading in the HUD National survey to determine the extent to which homes in the United States are impacted by various options for the §403 dust-lead standard.
- Convert blue nozzle loadings to wipe loadings in the HUD National Survey data before input into the EPI model.
- Convert post-intervention wipe loadings to blue nozzle concentrations for input to the IEUBK model.

In addition, conversion factors were used to convert different sampling methods to wipe sampling for production of prevalence tables in Chapter 3 and for sensitivity/specificity analyses in Section 4.5 of this chapter.

This section presents the equations developed to perform the conversions described above. The conversion equations are presented for samples collected from floors and window sills, since §403 will set standards for those housing components. Appendix X provides detailed information concerning the development of all the conversion equations discussed in this section.

4.2.1 Wipe versus Blue Nozzle (BN) Vacuum Conversions

Three studies reported side-by-side wipe and BN vacuum dust-lead measurements:

1. CAPS Pilot Study [EPA, 1995]
2. National Center for Lead-Safe Housing (NCLSH)/Westat Study [Westat, 1995]
3. Baltimore Repair and Maintenance (R&M) Pilot Study [Battelle, 1992]

Log-linear regression models were fitted to each data set separately. Weighted averages of the parameter estimates from each model were used to obtain the following conversion equations (written in the scale of the original data) for predicting a wipe dust-lead loading from a BN vacuum dust-lead loading:

Uncarpeted Floors: $Wipe_{load} = 11.4\ (BN_{load})^{0.690}$
Window Sills: $Wipe_{load} = 5.79\ (BN_{load})^{1.08}$

For example, a BN dust-lead loading of 100 $\mu g/ft^2$ on an uncarpeted floor would be converted to a wipe dust-lead loading of 273 $\mu g/ft^2$ (confidence intervals and prediction intervals are provided in Appendix X to describe the uncertainty associated with these conversions).

The same statistical procedure produced the following equations for predicting a BN vacuum dust-lead concentration from a wipe dust-lead loading:

Uncarpeted Floors: $BN_{conc} = 34.2\ (Wipe_{load})^{0.613}$
Window Sills: $BN_{conc} = 115\ (Wipe_{load})^{0.451}$

Thus, a wipe dust-lead loading of 100 $\mu g/ft^2$ on an uncarpeted floor would be converted to a BN dust-lead concentration of 576 $\mu g/g$.

4.2.2 Wipe versus Baltimore Repair and Maintenance (BRM) Vacuum Conversions

Four studies reported side-by-side wipe and BRM vacuum dust-lead measurements:

1. R&M Mini Study
2. Rochester Lead-In-Dust Study
3. NCLSH 5-Method Comparison Study
4. Milwaukee Low-Cost Intervention Study

These studies are described in Appendix X.

An analogous approach to that presented in Section 4.2.1 for developing the conversions resulted in the following equations for predicting a wipe dust-lead loading from a BRM vacuum dust-lead loading:

Uncarpeted Floors:	$\text{Wipe} = 8.79\ \text{BRM}^{0.313}$
Carpeted Floors:	$\text{Wipe} = 2.21\ \text{BRM}^{0.271}$
Window Sills:	$\text{Wipe} = 17.0\ \text{BRM}^{0.421}$

For instance, a BRM dust-lead loading of 100 $\mu g/ft^2$ on an uncarpeted floor would be converted to a wipe dust-lead loading of 37.1 $\mu g/ft^2$.

Note that the floor dust-lead samples in the Baltimore R&M Study were collected as composite samples, which eliminates the ability to distinguish uncarpeted floor samples from carpeted floor samples. However, the number of uncarpeted and carpeted subsamples within each composite sample can be determined. Therefore, floor samples from the Baltimore R&M Study were converted using the following heuristic approach to obtain the summary statistics provided in Chapter 3, and the sensitivity specificity analyses results in Section 4.5:

$$\text{Wipe} = p \cdot 8.79\ \text{BRM}^{0.313} + (1-p) \cdot 2.21\ \text{BRM}^{0.271},$$

where p represents the proportion of the composite sample obtained from uncarpeted floors, and BRM represents the dust-lead loading measured with the BRM sampler. For example, a composite floor BRM dust-lead loading of 100 $\mu g/ft^2$ made up of 3 uncarpeted and 2 carpeted subsamples would be converted to a floor wipe dust-lead loading of 25.4 $\mu g/ft^2$ from $(0.6 \cdot 8.79 \cdot 100^{0.313}) + (0.4 \cdot 2.21 \cdot 100^{0.271})$. Confidence and prediction intervals were not derived for the conversion of composite floor samples.

4.3 ESTIMATING THE EFFECT OF PICA FOR PAINT ON CHILDHOOD BLOOD-LEAD LEVELS

The exposure pathway from lead-based paint to childhood blood-lead concentration can be both direct and indirect. Indirect exposure takes place when deteriorated lead-based paint

contaminates residential dust or soil, which is then ingested by the child. Direct exposure takes place through the ingestion of paint chips. The two models described in Section 4.1 differ in their handling of direct and indirect exposure to lead-based paint. The IEUBK model estimates the geometric mean blood-lead concentration for children receiving indirect exposure to lead-based paint, through the soil- and dust-lead concentrations used as model inputs. The IEUBK model does not include a direct mechanism for estimating the contribution of paint chip ingestion to childhood blood lead. The EPI model does include a mechanism for estimating the effect of lead-based paint ingestion, as well as the effects of indirect exposure. This section describes how the effect of pica for paint is applied to the HUD National Survey homes for each model.

4.3.1 IEUBK Model

As described in Section 4.1, environmental conditions observed in the HUD National Survey are used as input to the IEUBK model. For each home in the HUD National Survey, the IEUBK model is used to predict the geometric mean blood-lead concentration of children exposed to those environmental conditions at age 24 months. The distribution of blood-lead levels in the population of children is then developed by allowing each home in the HUD National Survey to represent a proportion of the total number of children in the country. For homes without damaged lead-based paint, the IEUBK model predicted geometric mean blood-lead concentration and the assumed geometric standard deviation of 1.6 μg/dL are used to model the distribution of blood-lead levels in children represented by each home.

For each home with damaged lead-based paint (defined as greater than 0 ft^2 of interior or exterior deteriorated lead-based paint), the children represented by that home are divided into three groups: 1) children who have recently ingested paint chips (0.03%), 2) children who ingested paint chips at some time (8.97%), and 3) children without pica for paint (91%). The distribution of blood-lead levels for children in the three groups is estimated as follows:

1. Children who have recently ingested paint chips (0.03%) – Blood-lead concentration is assigned the value 63 μg/dL with no variation.

2. Children who ingested paint chips at some time (8.97%) – Geometric mean blood-lead concentration is 3.0 μg/dL greater than the geometric mean blood-lead

concentration predicted by the IEUBK model. The adjusted geometric mean blood-lead concentration and the assumed geometric standard deviation of 1.6 μg/dL is used to model the distribution of blood-lead levels for these children.

3. Children without pica for paint (91.0%) – The IEUBK model predicted geometric mean blood-lead concentration and the assumed geometric standard deviation of 1.6 μg/dL is used to model the distribution of blood-lead levels for these children.

The scientific evidence and assumptions used to select percentages of children assigned to each group and the adjustments to blood-lead concentrations for children who have ingested paint chips are described in Appendix D1.

4.3.2 EPI Model

Because the EPI model incorporates the effect of pica for paint, EPI model predicted values are used to estimate the distribution of blood-lead concentrations both for children who do and do not ingest paint chips, as described in Section 4.1.2. For HUD National Survey homes with no damaged lead-based paint, the predicted geometric mean blood-lead concentration for children who do not ingest paint chips and the assumed geometric standard deviation of 1.6 μg/dL are used to model the distribution of blood-lead levels for all children represented by each home. For homes with damaged lead-based paint, children represented by that home are divided into two groups: 1) children who have ingested paint chips (9%), and 2) children who do not ingest paint chips (91%). EPI model predicted geometric mean blood-lead concentrations and the assumed geometric standard deviation of 1.6 μg/dL are used to estimate the distribution of blood-lead concentrations for both groups.

4.4 HEALTH OUTCOMES

This section presents the approach for determining the incidence of adverse health outcomes resulting from lead exposure in young children. Two representative health effects were identified in Chapter 2: IQ deficits and elevated blood-lead concentrations. These effects are measured through: 1) decrements in IQ scores, 2) increased incidence of IQ scores less than 70, and 3) incidence of blood-lead concentrations greater than 10 μg/dL, and 4) incidence of

blood-lead concentrations greater than 25 μg/dL. These effects were chosen to represent the spectrum of health effects of lead exposure. In this section, the relationship of each representative health effect to blood-lead concentration is characterized. These relationships are applied to the IEUBK and EPI model predicted blood-lead concentrations to relate environmental lead exposure to health effects. The integrated risk analysis estimates specific health outcomes. For example, the relationship between blood-lead concentration and IQ scores is used to estimate the average IQ point loss due to lead exposure and the percentage of children with IQ point decrements greater than one, two, or three IQ points. Total health risks were not obtained by summing over the specific health outcomes. While the estimation of economic benefits was considered when the primary health effects were selected, this report does not convert health outcomes to monetary values. Economic benefits associated with health outcomes are estimated in the §403 Regulatory Impacts Analysis.

4.4.1 Decrements in IQ Scores

The IQ point loss health effect represents the neurological loss due to low level lead exposure. The association between blood-lead levels and IQ scores has been consistently reported in the scientific literature, as described in Section 2.3.1. Lower IQ scores are associated with a lower level of educational attainment and lower life-time earnings.

Schwartz (1994) conducted a meta-analysis to combine the findings of multiple studies in determining the effect that blood-lead concentration has on full-scale IQ score in primary school age children. The results from seven studies were employed to characterize the decrease in IQ score associated with a 1 μg/dL increase in blood-lead concentration. The three longitudinal and four cross-sectional studies included in the meta-analysis are summarized in Table 4-3. Additional details are provided in Appendix D2, Tables D2-1 and D2-2. A summary of the Schwartz (1994) article and a comparison of the results to those reported in other, similar papers (Schwartz, 1993; Pocock, et al, 1994) are also presented in Appendix D2.

Table 4-3. Summary Information for Studies Included in the Schwartz (1994) Meta-Analysis.

Study	Number of Children	Blood-Lead Concentration (μg/dL)		Estimated Effect[1] (SE)	Other Study Information
		Range	Mean (SD)		
Hawk, et al (1986)	75	6.2 - 47.4	20.9 (9.7)	2.55 (1.5)	Cross-sectional study of children age 3-7 in Lenoir and New Hanover counties, NC
Hatzakis, et al (1987)	509	7.4 - 63.9	23.7 (9.2)	2.66 (0.7)	Cross-sectional study of primary school age children in a lead smelter community (Lavrion, Greece)
Fulton, et al (1987)	501	3.3 - 34.0	11.5[2]	2.56 (0.9)	Cross-sectional study of primary school age children in Edinburgh, Scotland
Yule, et al (1981)	166	7.0 - 33.0	13.5 (4.1)	5.6 (3.2)	Cross-sectional study of primary school age children in London, England
Bellinger, et al (1992)	147	na	6.5 (4.9)	5.8 (2.1)	Longitudinal study in Boston, MA; Blood lead at age 2; IQ measured at school age
Dietrich, et al (1993)	231	na	15.2 (11.3)	1.3 (0.9)	Longitudinal study in Cincinnati, OH; Integrated blood lead up to age 3; IQ measured at school age
Baghurst, et al (1992)	494	<12.2 - >28.2	20 (na)	3.33 (1.5)	Longitudinal study in Port Pirie, Australia; Integrated blood lead up to age 3; IQ measured at school age

[1] Effect was estimated for a doubling of blood-lead concentrations from 10 μg/dL to 20 μg/dL.

[2] Geometric Mean was reported for this study.

The seven studies used linear, or log-linear, regression models to model the relationship between IQ scores and childhood blood-lead levels, along with other potentially important covariates. A log-linear regression model is a regression model fitted to the logarithm of the independent variables; in this application the independent variable is blood-lead concentration. The modeled relationships reported for each study were used to estimate that a 1 μg/dL increase in blood-lead concentration results in a loss of 0.257 IQ points, on average. This relationship is most applicable for blood-lead concentrations between 10 and 20 μg/dL, due to the modeling assumptions for those studies that used log-linear models. However, the relationship is applied over a much broader range of blood-lead concentrations in the risk assessment. This use is justified because a similar effect was observed in the two studies (Hawk, et al., 1986; Hatzakis, et al., 1987) that employed linear models, where the interpretation of model parameters is similar to the manner in which the relationship is applied in the risk assessment. The blood-lead concentrations in these two studies ranges from 6.2 μg/dL to 63.9 μg/dL, as shown in Table 4-3.

The relationship between blood lead and IQ point loss is illustrated in Figure 4-4. For example, children with blood-lead concentrations of 4 μg/dL are expected to have IQ scores approximately one point lower, on average, compared to children who are not exposed to lead. For an individual child, a greater or lesser IQ point loss may be observed.

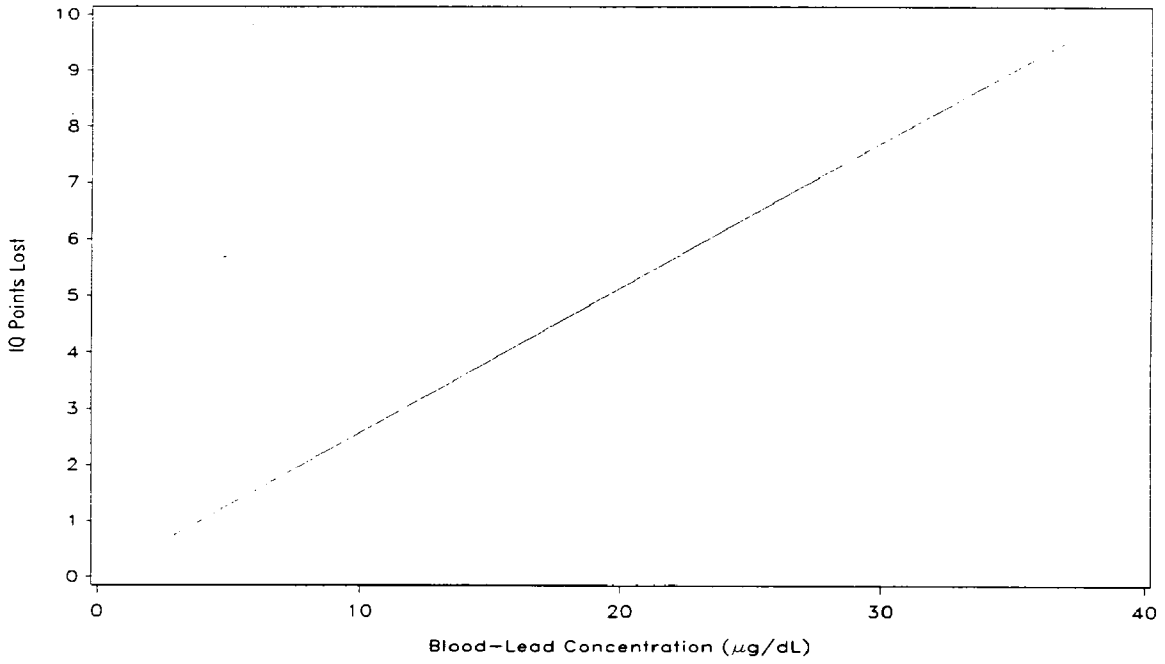
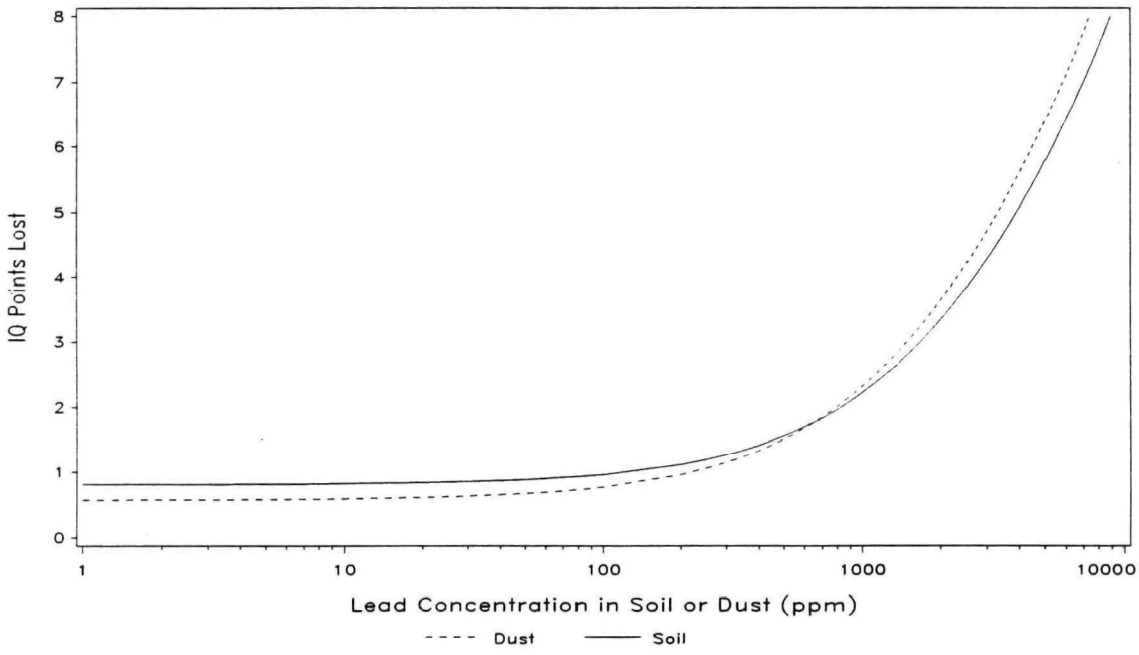


Figure 4-4. Estimated IQ Point Loss Due to Lead Exposure Plotted Against Blood-Lead Concentration

The relationship between IQ scores and blood lead was used in the risk assessment to estimate the average IQ point decrement for children exposed to environmental lead and to estimate the benefit, measured as IQ points not lost, following promulgation of the §403 rule. In addition, the percentage of children with decrements of >1, >2, and >3 IQ points due to lead exposure were calculated. Both the EPI and the IEUBK models were used to estimate the distribution of blood-lead concentrations of children exposed to a given set of environmental conditions, based on homes in the HUD National Survey. The blood-lead levels estimated across all children were multiplied by 0.257 to estimate the IQ point loss due to lead exposure.

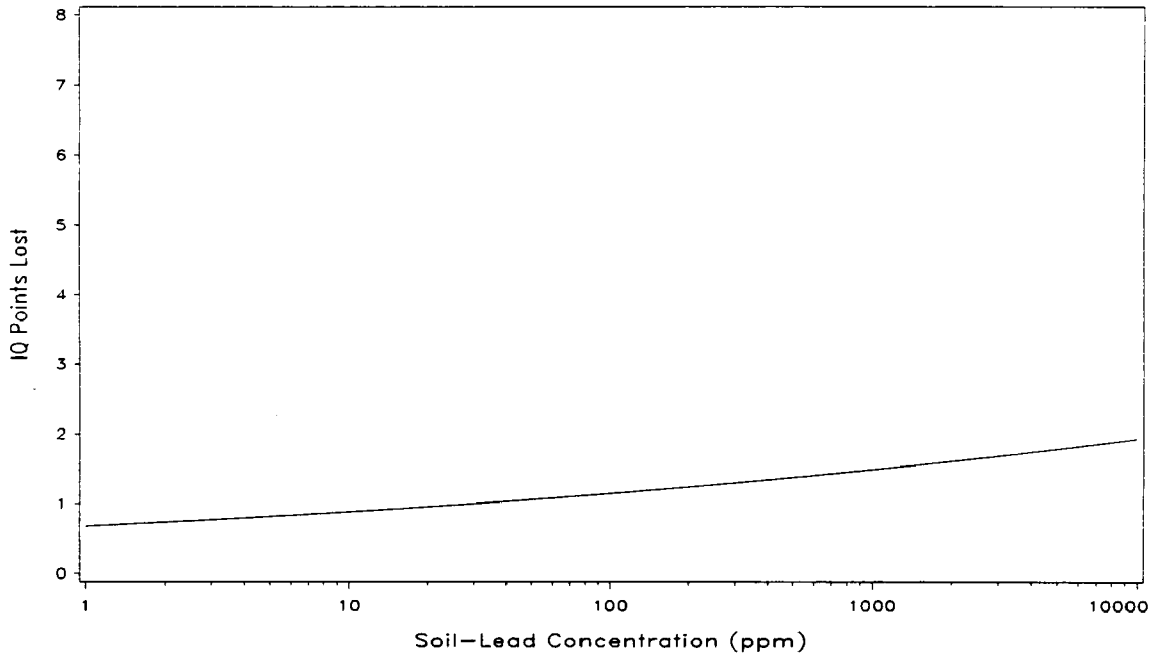
The relationship between environmental lead levels and IQ point loss are presented in Figures 4-5 and 4-6 for the IEUBK and EPI models, respectively. For each curve, the soil- or

dust-lead levels were varied over a range of values, while all other parameters were held fixed as described in Section 4.1. The predicted blood-lead level was used to estimate the average IQ point loss in the manner described above. For example, for a soil or floor dust-lead concentration of 1000 ppm, the IEUBK model predicts that 2 IQ points will be lost. The EPI model predicts that 1.5 IQ points will be lost for a dripline soil-lead concentration of 1100 ppm. Note that for Figure 4-5 and subsequent figures in this section illustrating predictions from the EPI model only the soil-lead concentration curve is illustrated.



The IEUBK model was used to relate environmental lead to blood lead.
Dust-lead concentration was set to 100 ppm for the soil curve and soil-lead concentration was set to 200 ppm for the dust curve.

Figure 4-5. Estimated IQ Point Loss Due to Lead Exposure Plotted Against Concentration of Lead in Soil and Dust, Utilizing IEUBK Model Predictions



The EPI model was used to relate environmental lead to blood lead. Floor dust-lead loading was set to 25 $\mu g/ft^2$, window sill dust-lead loading was set to 50 $\mu g/ft^2$ and it was assumed that there was no paint pica.

Figure 4-6. Estimated IQ Point Loss Due to Lead Exposure Plotted Against Concentration of Lead in Soil, Utilizing EPI Model

4.4.2 Increased Incidence of IQ Scores Less Than 70

The increased incidence of IQ scores less than 70 represents the increased likelihood of mental retardation resulting from lead exposure. An IQ of 70 is two standard deviations below the population mean IQ of 100 and can be used as an indicator of mental retardation. Children who are mildly mentally retarded require special education classes in school. Children who are severely mentally retarded may require life-long institutional care. This health effect may be used to estimate the number of children who will benefit under the proposed rule and the societal benefit through reduced costs of caring for the mentally retarded.

There is limited data available to estimate the increased likelihood of mental retardation resulting from lead exposure. Because of the lack of data, Wallsten and Whitfield (1986) used judgmental probability encoding methods to assess health risks due to lead exposure, particularly in the area of lower IQ scores. The results of their analyses are worth summarizing. As part of

this assessment, the increased percentage of children having IQ scores less than 70 was estimated for populations of children with elevated blood-lead levels.

In the Wallsten and Whitfield study, care was taken to select experts whose opinions spanned the range of respected opinion. The six experts who participated in the assessment of the relationship between IQ scores and blood-lead levels are listed in Table 4-4. These experts were asked to consider a hypothetical experiment in which a large number of children were randomly assigned at birth to either a control group, or one of six lead-exposure groups. Lead exposure was to remain fixed until the children reached age seven, at which time the Wechsler Intelligence Scale for Children – Revised (WISC-R) IQ test would be administered. Blood-lead levels were to be measured at age three. The lead exposure levels were such that at age three, members of each of the lead-exposure groups had blood-lead levels of 5, 15, 25, 35, 45, and 55 μg/dL. The experts were asked to estimate the mean and standard deviation of IQ scores in the control group. The experts also estimated the expected mean IQ differences between the control group and each exposure group. Each expert assumed that the IQ standard deviation in exposure groups was the same as that of the control group. This information was used to estimate the increased percentage, due to lead exposure, of children having IQ scores less than 70.

Table 4-4. Experts Who Participated in the Assessment of the Relationship Between IQ Scores and Blood-lead Levels by Wallsten and Whitfield.

Expert	Affiliation
Kim Dietrich	University of Cincinnati
Claire Ernhart	Cleveland Metropolitan General Hospital
Herbert Needleman	University of Pittsburgh
Michael Rutter	Institute of Psychiatry, London, UK
Gerhard Winneka	University of Dusseldorf, Dusseldorf, West Germany
William Yule	Institute of Psychiatry, London, UK

If the expert thought it necessary, separate judgements were made for children in low and high socioeconomic (SES) groups. For this purpose, the low SES group was defined as children living in households with incomes at, or below, the fifteenth percentile; the high SES group was

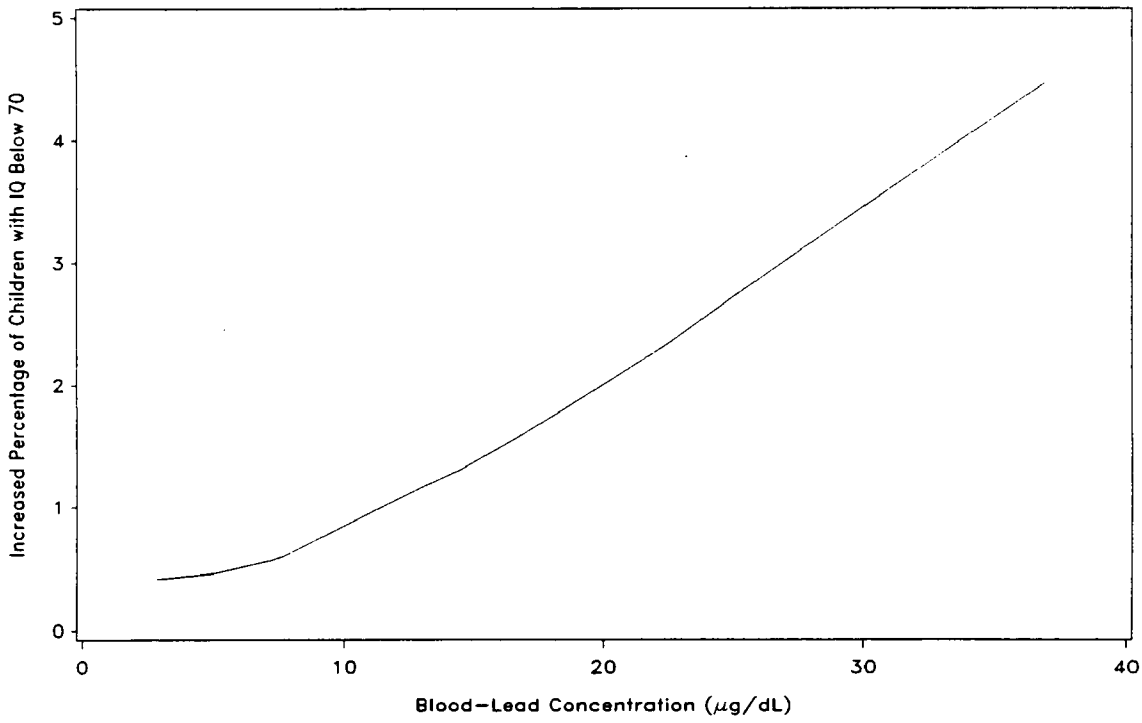
defined as children living in households with incomes above the fifteenth percentile. Five of the six experts chose to make separate judgements based on socioeconomic status.

At blood-lead levels ranging from 2.5 to 27.5 μg/dL, the distribution of increased percentage of children having IQ scores less than 70 was reported by Wallsten and Whitfield (Table D3-4), for each expert and SES (low and high). These distributions were combined by calculating the weighted average of the low income median (15% of weight) and high income SES medians (85%). The increased percentage of children having IQ scores less than 70, due to lead exposure, was estimated from the weighted average of the medians as a piecewise linear function of blood-lead concentration. This function is reported in Table 4-5 and illustrated in Figure 4-7, over a range of blood-lead concentrations. For example, 1.06% = -0.193 + 0.1044 x 12 of children with blood-lead concentrations of 12 μg/dL are expected to have IQ scores less than 70 due to lead exposure above and beyond those whose IQ would naturally fall below that level.

Table 4-5. Piecewise Linear Function for Estimating the Increased Percentage of Children Having IQ Scores less than 70 Due to Lead Exposure.

Range of Blood-Lead (PbB) Levels (μg/dL)	Function for Estimating Increased Percentage of Children Having IQ Scores less than 70 (IQ<70)
0 - 5.0	IQ<70 = 0.360 + 0.0204 PbB
5.1 - 7.5	IQ<70 = 0.218 + 0.0488 PbB
7.6 - 10.0	IQ<70 = 0.217 + 0.1068 PbB
10.1 - 12.5	IQ<70 = 0.193 + 0.1044 PbB
12.6 - 15.0	IQ<70 = 0.108 + 0.0976 PbB
15.1 - 17.5	IQ<70 = 0.534 + 0.126 PbB
17.6 - 22.5	IQ<70 = 0.653 + 0.1328 PbB
22.6 - 25.0	IQ<70 = 1.112 + 0.1532 PbB
> 25.0	IQ<70 = 0.942 + 0.1464 PbB

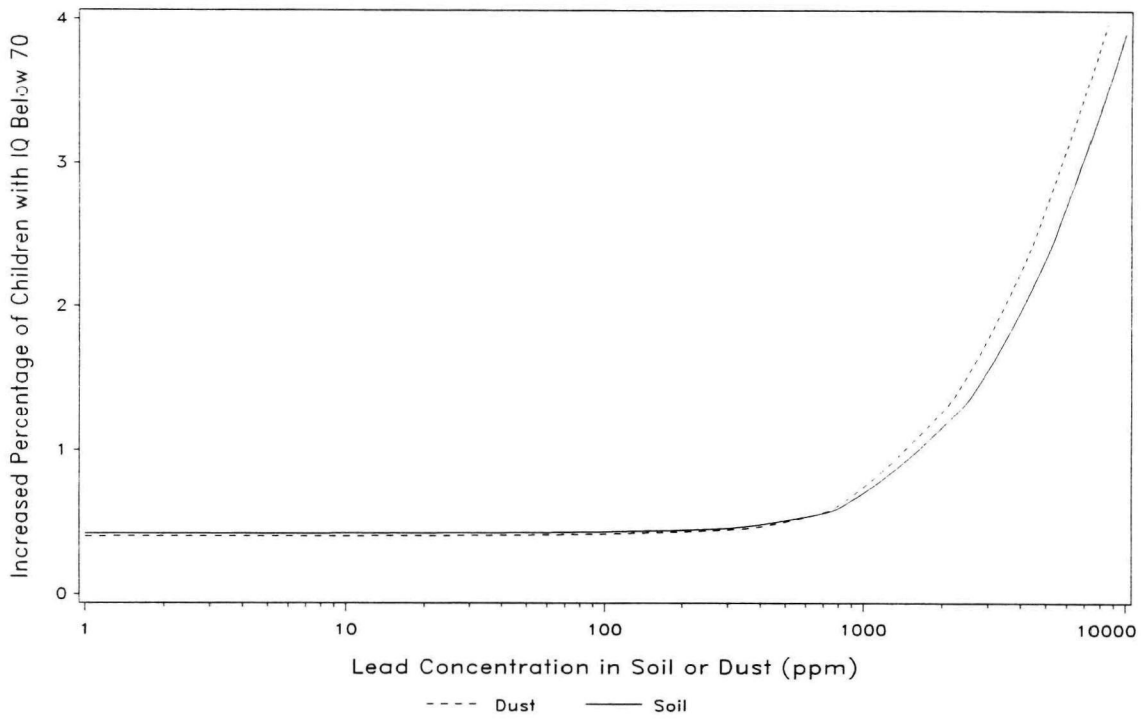
Note to Reader: The piecewise linear function displayed in Table 4-5 is being revised for the next draft of this report.



EPI model predictions for a fixed dust-lead loading of 100 μg/sq.ft, no pica or paint exposure, and African American race were used to relate soil-lead concentrations to blood lead.

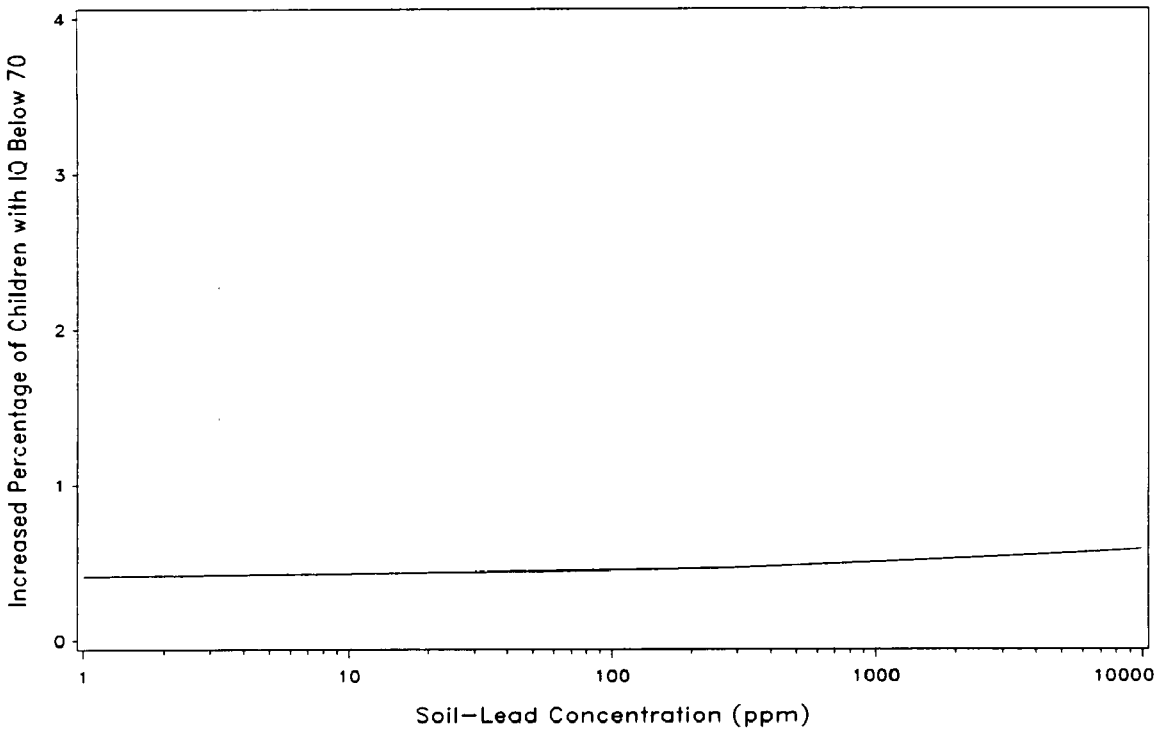
Figure 4-7. Increase in Percentage of Children with IQ Below 70 Due to Lead Exposure Plotted Against Blood-Lead Concentration

The relationships between environmental lead levels and the increased percentage of children having IQ scores less than 70 are presented in Figures 4-8 and 4-9 for the IEUBK and EPI models, respectively. For each curve, the soil or dust-lead levels were varied over a range of values, while all other parameters were held fixed. The predicted blood-lead level was used to estimate the increased percentage of children with IQ scores less than 70 due to lead exposure. For example, if the soil- or floor dust-lead concentration were 1000 ppm, the IEUBK model predicts that an additional 0.8% of children will have IQS less than 70. For a dripline soil-lead concentration of 1000 ppm, the EPI model predicts that 0.5% more children will have IQS less than 70.



The IEUBK model was used to relate environmental lead to blood lead.
Dust-lead concentration was set to 100 ppm for the soil curve and soil-lead concentration was set to 200 ppm for the dust curve.

Figure 4-8. Increase in Percentage of Children with IQ Below 70 Due to Lead Exposure Plotted Against Concentration of Lead in Soil and Dust, Utilizing IEUBK Model Predictions



The EPI model was used to relate environmental lead to blood lead. Floor dust-lead loading was set to 25 $\mu g/ft^2$, window sill dust-lead loading was set to 50 $\mu g/ft^2$ and it was assumed that there was no paint pica.

Figure 4-9. Increase in Percentage of Children with IQ Below 70 Due to Lead Exposure Plotted Against Concentration of Lead in Soil, Utilizing EPI Model

4.4.3 Incidence of Elevated Blood-Lead Levels

Two endpoints are utilized to estimate the incidence of elevated blood-lead concentrations. Children with blood-lead levels above 10 µg/dL are considered lead poisoned according to CDC (CDC, 1991), although the extent of recommended medical and environmental interventions varies as the blood-lead concentration increases. Blood-lead levels in excess of 25 µg/dL represent the level at which medical intervention is necessary. The extent of the intervention may vary from closely monitoring the child's behavior and blood-lead level for children with moderate elevations, to multiple courses of chelation therapy for children with severely elevated blood-lead levels.

Figure 4-10 illustrates the relationship between geometric mean blood-lead concentration and the percentage of children with a blood-lead level greater than 25 μg/dL, over a range of geometric mean blood-lead levels. This relationship was computed using a geometric standard deviation of 1.6 μg/dL, assuming that blood-lead concentrations may be characterized by using a log-normal distribution. The same assumptions were applied in the risk characterization to calculate the percentage of children having a blood-lead concentration greater than 10 μg/dL. Relationships between environmental lead levels and the incidence of blood-lead levels greater than 25 μg/dL are illustrated in Figures 4-11 and 4-12. For each curve, the soil- or dust-lead levels were varied over a range of values, while all other parameters were held fixed. The predicted blood-lead concentration was used to calculate the percentage of children having a blood-lead concentration greater than 25 μg/dL for the environmental conditions. For a soil-lead concentration of 2000 ppm the IEUBK model predicts that 10% of children will have blood-lead concentrations greater than 25 μg/dL. The EPI model, by contrast, predicts that less than 1% of children will have blood-lead concentrations greater than 25 μg/dL for a dripline soil-lead concentration of 2000 ppm.

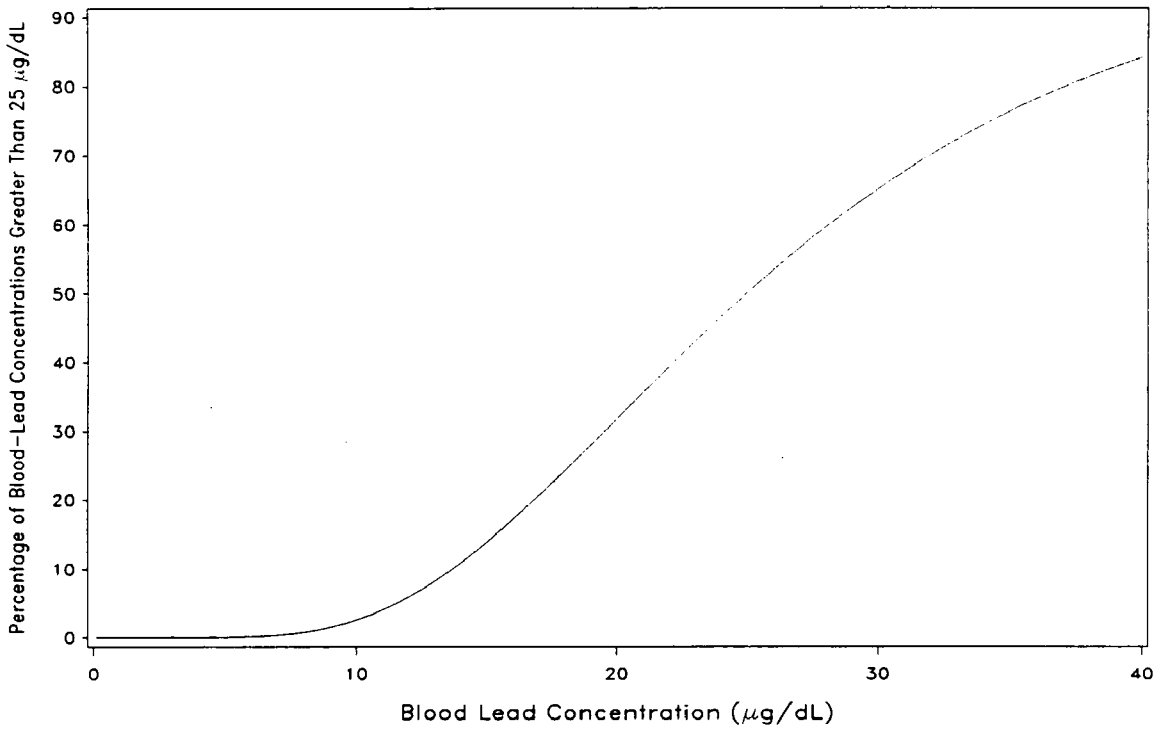
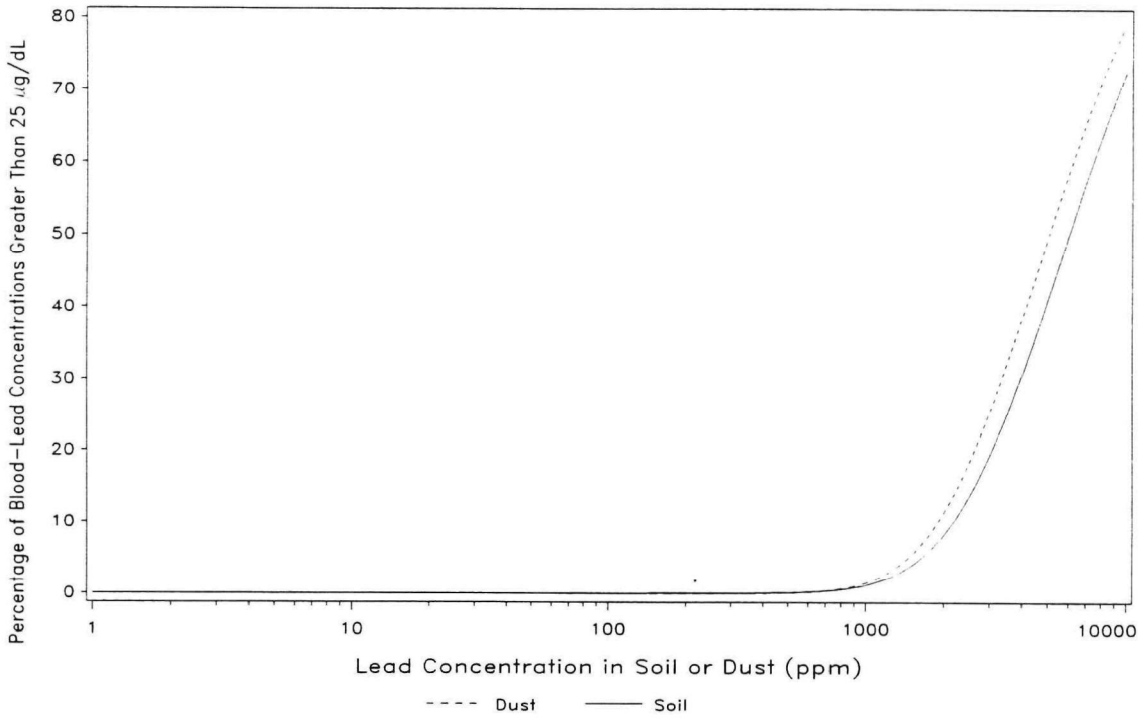
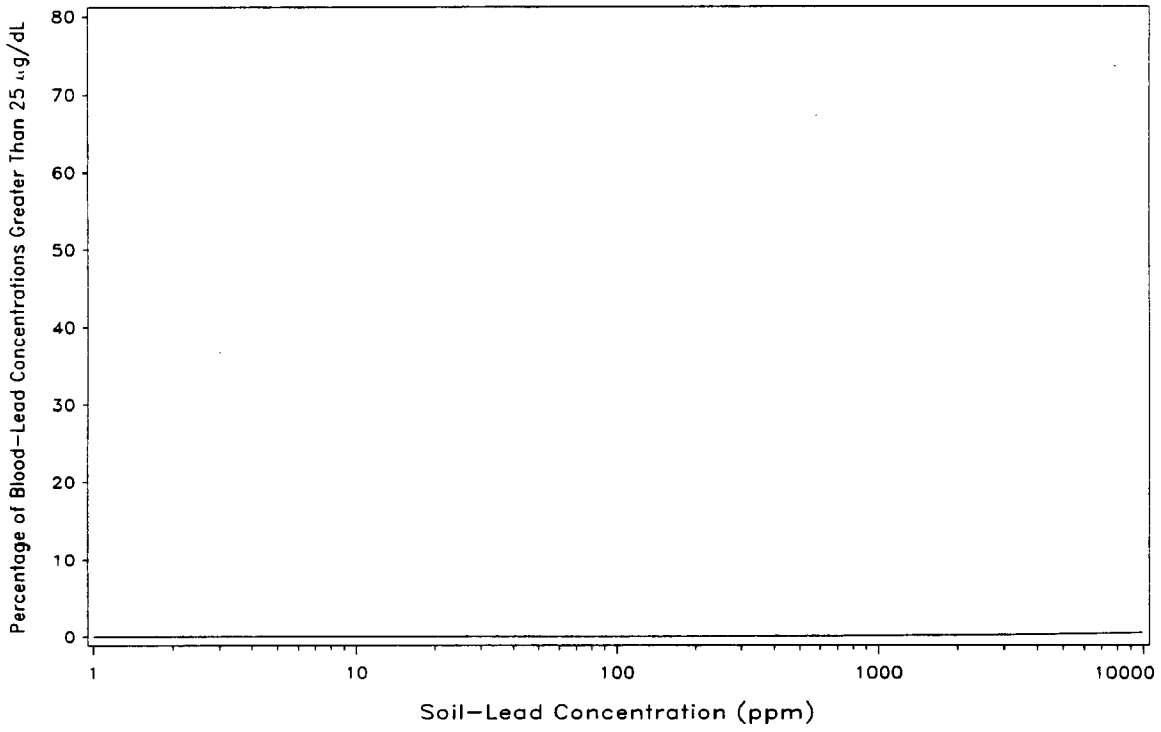


Figure 4-10. Percentage of Children with Blood-Lead Concentration Greater than 25 μg/dL Due to Lead Exposure Plotted Against Geometric Mean Blood-Lead Concentration, Assuming a GSD of 1.6



The IEUBK model was used to relate environmental lead to blood lead. Dust-lead concentration was set to 100 ppm for the soil curve and soil-lead concentration was set to 200 ppm for the dust curve.

Figure 4-11. Percentage of Children with Blood-Lead Concentration Greater than 25 µg/dL Due to Lead Exposure Plotted Against Concentration of Lead in Soil and Dust, Utilizing IEUBK Model Predictions



The EPI model was used to relate environmental lead to blood lead. Floor dust-lead loading was set to 25 $\mu g/ft^2$, window sill dust-lead loading was set to 50 $\mu g/ft^2$ and it was assumed that there was no paint pica.

Figure 4-12. Percentage of Children with Blood-Lead Concentration Greater than 25 $\mu g/dL$ Due to Lead Exposure Plotted Against Concentration of Lead in Soil, Utilizing EPI Model

4.5 OPTIONS FOR STANDARDS

The risk characterization in Chapter 5 will characterize risk reduction associated with implementing different options for the §403 standards. Because multiple standards will be set for different media it is not possible to evaluate all possible combinations of standards. The purpose of this section is to provide a range of options for the standards which can be further evaluated in the risk assessment and economic analysis.

Options for the standards were developed from modeling analyses of two epidemiological studies, the Baltimore R&M and the Rochester Study. The Rochester study was the primary source for defining the options for §403 standards in this section.

4.5.1 Analysis Methods

Two main analysis methods were used to estimate options for the §403 standards from the epidemiological data: 1) regression models; and 2) sensitivity/specificity analyses. Each method was applied to individual standards separately (single media analysis) and to a combination of standards jointly (multi-media analysis). Summarized final results from the statistical analyses are presented in this section, while specific details on the statistical analyses are provided in Appendix Y.

Regression Models

Both single and multi-media regression models were explored for estimating options for the §403 standards based on data from the Rochester Study. The single media models related childhood blood-lead concentrations to measures of lead from each media (floor dust, window sill dust, soil, and paint) separately as follows:

$$\ln(PbB_i) = \beta_0 + \beta_1 * PbE_i + \Sigma_i$$

where PbB_i represents the blood-lead level of the child living in the ith home, PbE_i represents a measure of environmental lead (either on the original scale or log transformed) from the ith home, β_0 and β_1 are intercept and slope parameters which describe the modeled relationship, and Σ_i is the residual error in $\ln(PbB_i)$ left unexplained by the model. For each model, an estimate of the environmental lead level at which 95 percent of the population of children would be expected to be below 10 μg/dL was provided to help develop ranges of options for the §403 Standards.

The multi-media regression model related childhood blood-lead concentrations to measures of lead from each media (dust, soil and paint) simultaneously as follows:

$$\ln(PbB_i) = \beta_0 + \beta_1 * \ln(Dust_i) + \beta_2 * \ln(Soil_i) + \beta_3 * Paint_i + \Sigma_i$$

For this model, a joint estimate of environmental lead levels in paint, dust, and soil at which 95 percent of the population of children would be expected to be below 10 μg/dL was provided.

Sensitivity/Specificity Analyses

Table 4-6 describes the performance characteristics that were estimated as part of the sensitivity/specificity analyses. The single media analysis focused on media standards that each corresponded to a single measure of lead at the primary residence, whereas in the multi-media analysis, the media standard corresponded to measures of environmental lead in several media (dust, soil and paint). Thus, if any single measure of environmental lead was above a standard in the multi-media analysis, the residence was categorized as being above the standard.

Table 4-6. Definitions of Performance Characteristics Used to Characterize the Performance of Options for the §403 Standards Based on Empirical Data from Lead Exposure Studies

		Media Standard	
		Below	Above
Blood Lead Concentration Standard	Above	a	b
	Below	c	d

In the above table, the letter 'a' represents the number of children which have a blood lead concentration above a given blood-lead standard and who live in a residence with an environmental lead level below a standard for that environmental medium. Letters 'b', 'c', and 'd' represent similar counts. From these counts the following performance characteristics are calculated

Performance Characteristic	Definition	Calculation
Sensitivity (or True Positive Rate)	Probability of a dwelling being above the soil lead standard given that there is a resident child with an elevated blood concentration.	b/(a+b)
Specificity (or True Negative Rate)	Probability of a dwelling being below the soil lead standard given that a resident child has a low blood lead concentration.	c/(c+d)
Positive Predictive Value (PPV)	Probability of a resident child having an elevated blood lead concentration given that the observed soil lead in the dwelling is above the standard.	b/(b+d)
Negative Predictive Value (NPV)	Probability of a resident child having a low blood lead concentration given that the observed soil lead in the dwelling is below the standard.	c/(a+c)

4.5.2 Assumptions

It was assumed that individual standards will be set for each media, location, or surface addressed in the rulemaking, and that action will be recommended if any individual standard is exceeded. Therefore, ranges are provided for each media, location, or surface separately. It is also assumed that dependencies between media will be addressed in the guidance provided to risk assessors and in the recommended actions associated with each standard.

Ranges for the options of the standards presented in this report are based on estimated effects of lead exposure on blood-lead concentration. The high and low values for each range result from different analyses. The low values are generally taken from the conservative, single media analyses and the high values from the less conservative, joint analyses. Still, all options proposed are estimated to provide a minimum 95% probability that a child living in a home with environmental levels below each of the §403 standards will have a blood-lead concentration less than 10 μg/dL.

4.5.3 Results

Table 4-7 presents a summary of estimates of standard levels which achieved (for homes that meet the standard) either:

1. A negative predictive value of 95% from a single media or multi-media sensitivity/specificity analysis, or

2. An estimated 95% probability that a child's blood-lead concentration is below 10 μg/dL from the single media or multi-media regression analyses.

The above two criteria were the current target health effects to be considered in choosing the §403 standards.

As seen in Table 4-7, based on the single media analyses, i.e., each §403 standard considered separately, options for standards must be set very low to meet the target health effects. However, Table 4-7 also indicates that if the target health effects are associated with meeting all §403 standards simultaneously, then the options for standard levels can rise significantly, even well above the levels proposed in the Interim Guidance (EPA, 1995h). In the

Table 4-7. Summary of Estimated Standards Which Achieved a Negative Predictive Value of 95% or an Estimated 95% Probability of a Child's Blood-Lead Concentration Below 10 μg/dL in a Dwelling that is at or Below the Standard.

Media	Single Media Analyses[1]			Multi-Media Analyses						
		Sensitivity/Specificity			Sensitivity/Specificity[2]					
					Joint Soil and Dust Rochester[4]		Joint Soil, Dust, and Paint Rochester[5]		Joint Soil, Dust, Paint, and Sills Rochester	
	Regression Models Rochester	R&M	Rochester	Regression Models[3]	Joint Standard 1	Joint Standard 2	Joint Standard 1	Joint Standard 2	Joint Standard 1	Joint Standard 2
Dripline Soil (μg/g)	<50	<50	50	<50	<200		1000		500	1500
Play Area Soil (μg/g)	<50		<50	<50		<200		600		1000
Uncarpeted Floor (μg/ft²)	<25		<25	<25	<50	<50	400	50	400	400
Carpeted Floor (μg/ft²)	<25		<25							
Carpeted and Uncarpeted Floor (μg/ft²)	<25	<25	<25							
Window Sills (μg/ft²)	<25	25	25						800	500
Window Troughs (μg/ft²)	<25	200	<50							
Percent of Interior Components with Deteriorated LBP	0%	0%	0%							
Average Percent of Deteriorated LBP per Interior Component	0%		0%							
Percent of Exterior Components with Deteriorated LBP	0%	0%	0%							
Average Percent of Deteriorated LBP per Exterior Component	0%		0%							
Maximum of Interior/ Exterior Percent of Components with Deteriorated LBP		0%	0%				20%	20%	20%	20%
Maximum of Interior/ Exterior Average Percent of Deteriorated LBP per Component			0%							

1 No paint standard was identified in single media analyses which achieved the criterion of an associated 95% probability that a child's blood-lead concentration would be below 10 μg/dL. Even with zero percent deteriorated lead-based paint, the probability of a blood-lead concentration below 10 μg/dL was estimated to be less than 95%.

2 Multi-media sensitivity specificity results for the R&M study were not included because 1) they were not conducted for the three-way (soil, dust, paint) or four-way (soil, dust, paint, sills) analyses since only 8 homes were available for analysis, and 2) they do not change the results given for the two-way (soil, dust) analysis.

3 The multi-media regression model included a variable that indicated a combination paint/pica hazard which was set to zero for these analyses.

4 The lowest dust lead standard assessed in the two-way joint soil and dust sensitivity/specificity analyses was 50 μg/ft². The lowest soil lead standard assessed was 200 μg/g. No combination of soil and dust for either the Rochester or R&M data achieved an NPV of 95% with those lower limits on soil and dust standards.

5 In the three-way (soil, dust, paint) multi-media analyses and higher (four-way and five-way), the combination of standards with the highest values that achieve the target health criteria were chosen for presentation in this table

Rochester study for any single media/surface/location standard alone there were homes with children with blood-lead concentrations above 10 μg/dL, even at very low media lead levels. However, when all media/surface/location standards were considered jointly, even at relatively high levels, all homes with a child with a blood-lead concentration above 10 μg/dL were identified. In other words, any home with a child with a blood-lead concentration above 10 μg/dL exhibited a relatively high lead level in at least one media/surface/location being considered for a §403 standard. This is a significant finding related to the §403 rulemaking as it illustrates the effectiveness of using multiple standards applied in a single risk assessment to identify a home with a lead-based paint hazard.

It should be noted that the above conclusion is based upon the multi-media sensitivity/specificity analyses from Rochester alone. The multi-media regression analyses do not show the same effect. There could be a number of reasons for this including the fact that the form of the regression model fitted is not sensitive to interactions between the effect of the different environmental media. In addition, many blood-lead concentrations are close to the sensitivity/specificity target value of 10 μg/dL.

Based on Table 4-7, a range of options for a standard for each media/surface/location can be specified. Table 4-8 below lists the proposed range of options for each standard. For each standard the upper limit of the range is specified as the maximum estimated level in one of the multi-media analyses.

The lower limit of the standard is more problematic. Very low levels of standards (e.g. less than 25 $\mu g/ft^2$ for dust and less than 50 μg/g for soil) were estimated by the single media analyses to be associated with the target health effect. From a practical standpoint, these standards are not necessarily achievable. Therefore, the lower limits presented in Table 4-8 for the range of options for a standard are based on practicality.

For dust-lead standards, this lower limit is defined as 25 $\mu g/ft^2$ because a dust-lead standard below 25 $\mu g/ft^2$ may be problematic due to laboratory analysis issues. Many currently accredited laboratories have levels of quantification between 10 $\mu g/ft^2$ and 25 $\mu g/ft^2$.

Table 4-8. Proposed Options for §403 Standards To Be Evaluated in the Risk Assessment and Economic Analysis.

Standard	Range	
	Low Limit	High Limit
Uncarpeted Floor Dust-Lead Loading ($\mu g/ft^2$)	25	400
Window Sill Dust-Lead Loading ($\mu g/ft^2$)	25	800
Dripline Soil-Lead Concentration ($\mu g/g$)	50	1500
Play Area Soil-Lead Concentration ($\mu g/g$)	50	1000
Maximum of Percent of Interior Components with Deteriorated Lead-Based Paint and Percent of Exterior Components with Deteriorated Lead-Based Paint	0%	20%

Note to EPA: The amount of damaged lead-based paint is currently being expressed in terms of square footage rather than percentage of components in the integrated risk assessment.

The lower limit for a soil-lead standard(s) is defined as 50 µg/g. This level was chosen since national background levels of lead in soil have been estimated in the neighborhood of 20 µg/g to 35 µg/g.

The lower limit for a paint-lead standard was chosen as zero percent deteriorated lead-based paint, the level suggested by the single media analyses. Single media analyses indicate that no single standard for paint can achieve the target health criteria of a 95% probability that a child's blood-lead concentration when exposed to paint lead will be below 10 µg/dL. There are no practical difficulties associated with setting a lower limit of zero percent for deteriorated lead-based paint.

The ranges in Table 4-8 were used as bounds on options for standards that were evaluated in Chapter 5.

5.0 INTEGRATED RISK ANALYSIS

CHAPTER 5 SUMMARY

This chapter provides the estimated health risks and blood-lead concentrations for children aged 1-2 years associated with current residential lead exposures (pre-§403). These risks are then compared to the projected health risks and blood-lead concentration associated with the predicted residential lead exposures in the post-§403 environment. Baseline risk is computed based on NHANES survey data; post-§403 risk is estimated separately using the IEUBK model and an EPI model applied to environmental-lead levels observed in the HUD National Survey. Post-§403 environmental-lead levels are adjusted for the assumed effects of intervention initiated by §403, under various options of standards for lead in dust, soil, and paint. The results of this chapter are projected childhood health risks and blood-lead concentrations for a wide range of options for standards.

A sensitivity analysis was performed to characterize how the estimated risk reductions results differ when alternatives are considered for the most important assumptions and approaches in the procedure. Alternative procedures were considered for characterizing baseline and post-intervention blood-lead distributions. Alternative assumptions were considered on post-intervention environmental-lead levels, the IQ decrement associated with increased blood-lead concentration, and the age group of interest. The largest differences in results, especially those representing the most extreme health effects, tended to appear when making alternative assumptions on post-intervention environmental-lead levels and on the IQ decrement associated with increased blood-lead concentration.

The goals of the integrated risk analysis are to

- identify the baseline distribution of blood-lead concentrations in the nation's children,
- use this distribution to characterize adverse health effects associated with elevated blood-lead concentration in children,
- characterize the post-intervention distribution of children's blood-lead concentrations and associated adverse health effects adjusted for the assumed effects of performing interventions in response to various options for the §403 standards,
- compare the post-§403 distributions of childhood health effects and blood-lead concentrations to their respective pre-§403 baselines, noting any risk reduction that may result for the §403 standards, and

- Characterize how sensitive the estimated risk reductions are to the uncertainty present in key assumptions, parameters, data sources, and analysis tools.

The risk assessment targets children aged 12 to 35 months. As discussed in Section 2.3 and 4.4, the health effect endpoints of interest are:

- The number and percentage of children with blood-lead concentrations at least 10 μg/dL
- The number and percentage of children with blood-lead concentrations at least 25 μg/dL
- The average of IQ points lost per child, due to exposure to lead-based paint (LBP) hazards
- The number and percentage of children with IQ scores less than 70
- The number and percentage of children with IQ decrements of at least 1
- The number and percentage of children with IQ decrements of at least 2
- The number and percentage of children with IQ decrements of at least 3

In addition, in order to understand the impact of §403 on the nation's housing, the number and percentage of housing units in which some action might be required is predicted for various options of the standard for each medium.

Section 5.1 presents a characterization of the baseline (i.e., pre-§403) distribution of children's blood-lead concentrations and associated health effect endpoints. The intervention activities, expected reductions in environmental-lead levels, and intervention triggers are discussed in Section 5.2. Section 5.3 characterizes the childhood health risks and blood-lead concentrations predicted to exist after performing the interventions in response to §403. Finally, Section 5.4 presents the results of a sensitivity analysis on the effects of the uncertainty present in key assumptions, parameters, data sources, and analysis tools on the risk assessment.

5.1 BASELINE CHARACTERIZATION OF CHILDREN'S BLOOD-LEAD CONCENTRATIONS AND HEALTH EFFECTS

For purposes of this Risk Assessment, interventions in response to the proposed §403 rules are assumed to begin in 1997. In this section, the national distribution of children's blood-lead concentrations in 1997 is estimated to characterize the distribution prior to the enactment of §403. This distribution is then used to estimate selected health endpoints. These characterizations serve as the baseline for evaluating the risk of environmental-lead exposure to children. Post-intervention distributions are compared to these baselines to assess risk reduction resulting from §403.

As discussed in Section 2.4, the target age for characterizing a baseline distribution of blood-lead concentrations and estimating health effects is from 1 to 2 years (12 to 35 months). Information from NHANES III, phase I was used to characterize the 1997 baseline distribution of blood-lead concentrations in children aged 1 to 2 years. The NHANES III weights, for children aged 1 to 2 years with non-missing blood-lead concentrations, add up to 5,272,000, which is less than 7,961,000, the total number estimated for 1997 in Section 3.3. Therefore, the sampling weights in NHANES III were scaled by a factor of 7,961/5,272 so that the distribution determined by the NHANES III represents the same total number of children projected for 1997 as the subsequent post-§403 estimates.

There are many ongoing local, state, and federal initiatives to reduce childhood blood-lead concentrations. Therefore, the actual distribution of childhood blood-lead concentrations in 1997 may differ from that reported in NHANES III which was conducted between 1988 and 1991. Specifically, if the government strategies already in place are effective, the distribution reported may assign slightly higher probabilities to elevated blood-lead concentrations than will actually occur in 1997. Nevertheless, the distribution of childhood blood-lead concentrations in NHANES III is the best available data for estimating the baseline distribution of blood-lead concentrations in 1997.

Figure 5-1 contains two plots presenting the estimated baseline distribution of blood-lead concentrations in 1997 for children aged 1-2 years. The top plot presents the estimated relative frequency distribution based on a lognormal model. The bottom plot presents the estimated cumulative frequency distribution. There are two curves in this plot. The jagged

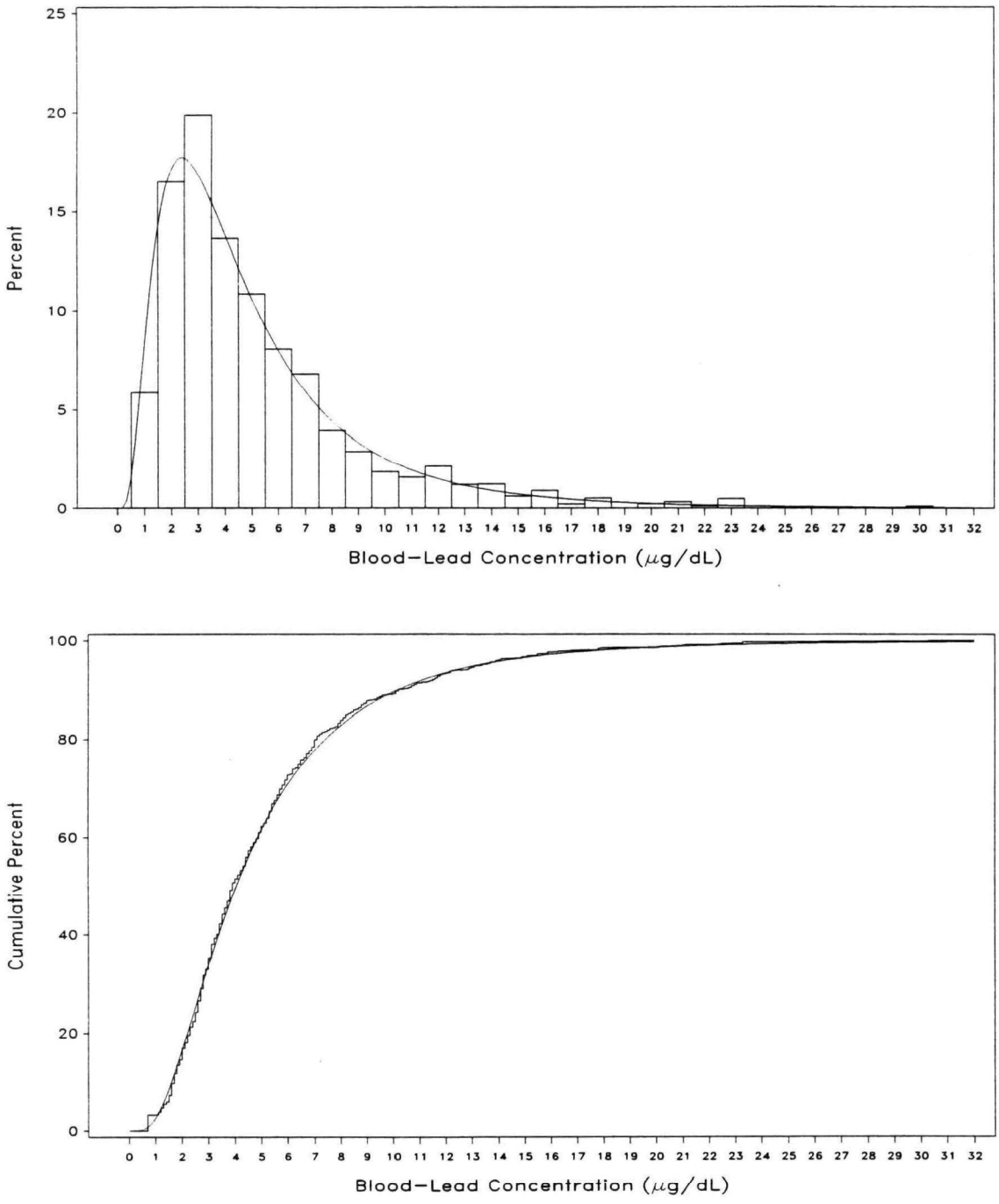


Figure 5-1. Baseline Distribution of Blood-Lead Levels Based on NHANES III, Phase 1 (0.2 Percent of Children Had Blood-Lead Concentration Greater than 32 μg/dL)

curve represents the empirical distribution observed in NHANES III; the smooth curve represents the lognormal model approximation to the data. The plot illustrates that the lognormal model fits the data well.

This graph is useful for understanding the percentage of children aged 1-2 years having a blood-lead concentration greater than a specified value. For example, 10.5% = 100% - 89.5% of children are estimated to have a blood-lead concentration greater than 10 μg/dL, and 0.6% = 100% - 99.4% are estimated to have a blood-lead concentration greater than 25 μg/dL.

The estimated geometric mean blood-lead concentration was 4.1 μg/dL, and the estimated geometric standard deviation was 2.1 μg/dL. The distributions in Figure 5-1 were translated to the distribution of number of IQ points lost due to blood-lead concentration using the methods discussed in Section 4.4.1. Figure 5-2 displays this translated distribution using the same format as in Figure 5-1. The lower plot presents the percentage of children with anticipated IQ point losses within particular ranges. For example, the percentage of children with at least a 2 point decrement in IQ, due to blood-lead concentration, is estimated to be about 18 percent.

Table 5-1 displays estimated 1997 baseline probabilities for the various adverse health effects in children aged 1-2 years. These estimates were calculated from the information summarized in Figures 5-1 and 5-2 and are employed to characterize the pre-§403 risk of lead exposure in 1997. Thus, it is assumed that this risk will prevail if §403 is not implemented. The method for estimating the probability of children having IQ less than 70 is described in Section 4.4.2. Each of these endpoints is estimated from the geometric mean and geometric standard deviation, assuming a lognormal distribution. The mathematical approach used to make these inferences is described in Step (5) of Appendix E2.

5.2 INTERVENTION ACTIVITIES

Once promulgated, §403 will prompt intervention activities targeting residential lead hazards. These interventions will be conducted on behalf of children already exposed to the targeted lead hazards, as well as children who would otherwise be exposed if the hazards are not abated or controlled. For the purposes of the Risk Assessment, a lead hazard intervention is defined as any non-medical activity that seeks to prevent a child from being exposed to the

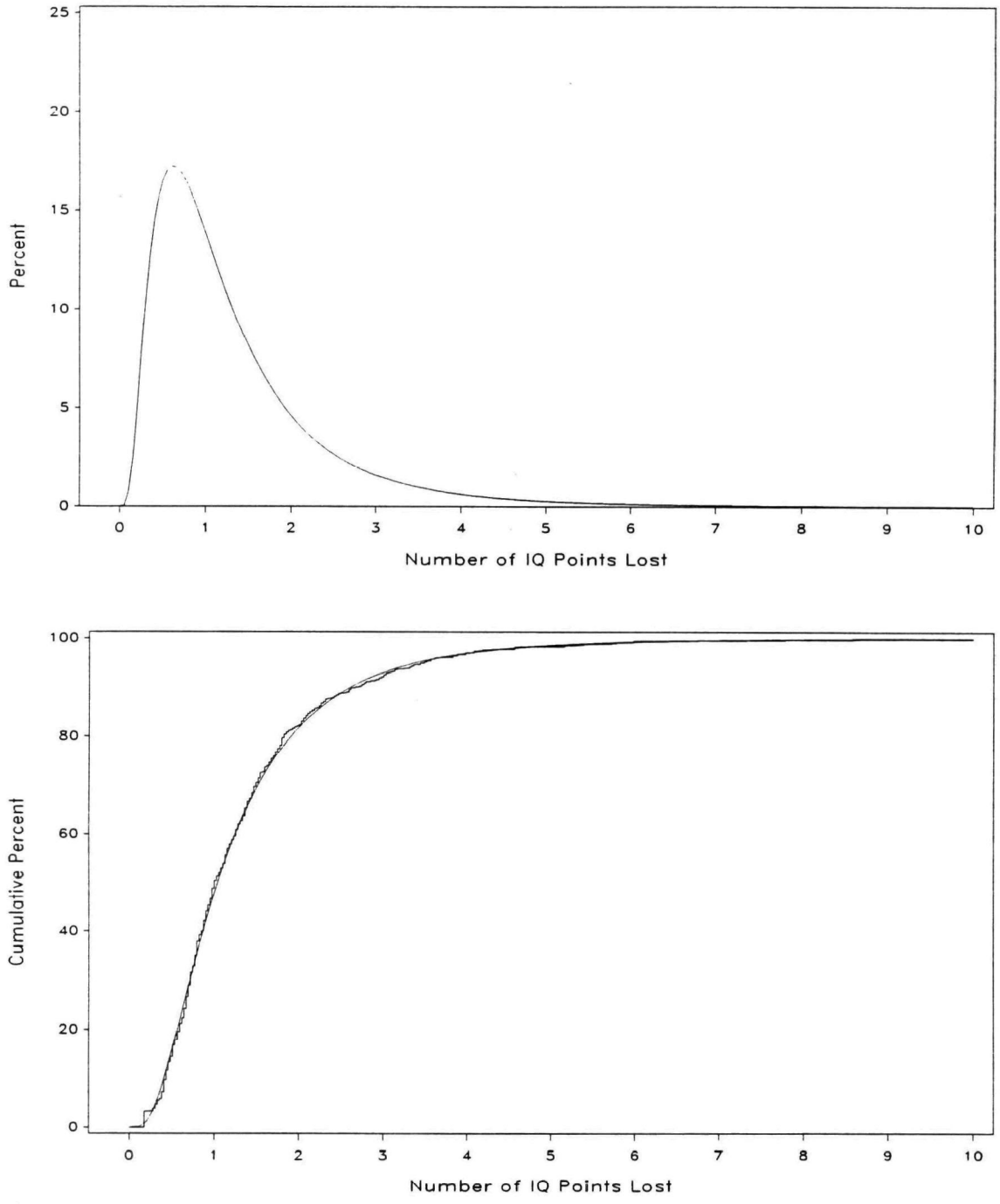


Figure 5-2. Baseline Distribution of IQ Decrements Due to Elevated Blood-Lead Concentration Based on NHANES III, Phase 1 (0.03 Percent of Children Had in Excess of 10 IQ Points Lost)

Table 5-1. Estimated Baseline (1997, Pre-Intervention) Number and Percentage of Children Aged 1 to 2 Years Having Specific Health Effects.

Health Effect	Estimated Baseline Number of Children	Estimated Baseline Percentage of Children
Blood-lead concentration of at least 25 μg/dL[1]	46,000	0.58
Blood-lead concentration of at least 10 μg/dL[1]	834,000	10.5
IQ score less than 70[2]	45,000	0.57
IQ score decrement of greater than 1[3]	4,152,000	52.2
IQ score decrement of greater than 2[3]	1,451,000	18.2
IQ score decrement of greater than 3[3]	564,000	7.09
	Average	**SD**
IQ decrement	1.35	1.11

[1] Determined from Figure 5-1.
[2] Determined from methods in Section 4.4.2
[3] Determined from Figure 5-3.

lead in his or her surrounding environment. An intervention, therefore, may range from the in-home education of parents regarding the dangers of a young child's hand-to-mouth activity, to the abatement of lead-based paint.

An intervention conducted on behalf of children already exposed to the targeted hazard is termed *secondary prevention* (e.g., paint abatement in the home of a child who has a blood-lead concentration exceeding 20 μg/dL). A *primary prevention* intervention prevents exposure before it occurs (e.g., paint abatement in a home before a new family with children moves in). The distinction between primary and secondary prevention efforts is one of the population targeted rather than the activity conducted. In fact, a given intervention can have primary and secondary prevention benefits.

One objective of §403 is to prompt primary prevention interventions targeting lead hazards in residential soil, dust, and paint. (Secondary prevention will, of course, also take place.) As the risk assessment needs to model the expected benefits following promulgation of §403, measures of the effectiveness of these lead hazard interventions are required.

Unfortunately, there is no information currently available in the scientific literature regarding the efficacy (as measured by either health outcomes or by changes in children's blood-lead concentrations) of primary prevention interventions targeting paint, dust, or soil. There are limited data on the effectiveness of secondary prevention interventions (EPA, 1995b).

Research suggests that primary prevention interventions will produce greater efficacy than secondary prevention interventions (Gulson et al., 1995). Bone-lead stores accumulated by exposed children continue to mobilize into the blood following an intervention and may mask the intervention's full effectiveness. The effectiveness of interventions studied in the literature, therefore, have shortcomings as estimates of the efficacy stemming from primary interventions. Thus, these blood lead declines from a secondary prevention situation are not used as our primary mechanism for assessing health benefits of the §403 rule. However, a method which uses the changes in blood-lead concentrations from secondary prevention settings with a modeled effect of the bone lead stores is examined in the Section 5.4 sensitivity analysis.

Data on the effectiveness of lead hazard interventions in terms of change in environmental lead levels following interventions targeting paint, dust and soil were used to estimate environmental-lead levels following interventions conducted as a result of §403. It is important to note, however, that only some of the interventions considered viable under current standards have been studied in the literature. Where available, the reported post-intervention environmental-lead levels may then be translated into blood-lead concentrations representing the benefit of primary prevention interventions. The translation is accomplished using both epidemiological models and the IEUBK Lead Model. Where little or no environmental effectiveness information is available about a particular intervention, EPA has used its current understanding of the intervention to develop an estimated effectiveness.

Fully characterizing an intervention requires addressing four questions:

1. What "triggers" the intervention?

2. What procedures are conducted during the intervention?

3. How effective is the intervention at reducing environmental lead levels?

4. How effective is the intervention at reducing blood-lead concentrations (for both primary and secondary prevention)?

The interventions and their associated procedures utilized in the risk assessment are discussed in Section 5.2.1. The effectiveness of these methods in reducing environmental lead levels and blood-lead concentrations are documented in Section 5.2.2 and 5.2.3, respectively. Finally, Section 5.2.4 discusses the circumstances under which each of the defined interventions are triggered.

5.2.1 Interventions

For the purposes of the Risk Assessment, a total of seven interventions were defined for lead in paint, dust, and soil. The seven interventions are dust cleaning, exterior LBP maintenance, exterior LBP abatement, interior LBP maintenance and abatement, soil cover, and soil removal. For interior paint, exterior paint, and soil, two intervention approaches were defined. These two approaches are intended to reflect the viable range in scope achieved by interventions of the targeted media. For residential dust, only a dust-cleaning method was included as a one-time activity to follow LBP interventions. Table 5-2 presents these seven interventions by defining the procedures conducted and the expected duration of the intervention's benefits.

The procedures defined in Table 5-2 for each of the interventions are consistent with intervention practices currently recommended by EPA (and mandated in some communities). For example, paint removal must be conducted using appropriate precautions, and LBP encapsulation must utilize materials approved as encapsulants (i.e., remain effective for 20 years). The procedures exclude interventions previously utilized but now considered hazardous, such as open-flame burning or abrasive sanding of lead-based paint.

Table 5-2. Interventions Defined for the §403 Risk Assessment Effort

Intervention		Procedures Defining the Intervention	Expected Duration[1]
Dust Cleaning		Cleaning the unit using HEPA vacuums and wet mopping.	5 years
Exterior LBP	Maintenance	Painted surfaces with deteriorated LBP are repaired by feathering the edges of deteriorating paint and repainting with new, lead-free paint.	5 years for paint
	Encapsulation/ Abatement	Painted surfaces with deteriorated LBP are encapsulated, enclosed, or removed using currently acceptable practices and materials.	20 years for paint
Interior LBP	Maintenance	Painted surfaces with deteriorated LBP are repaired by feathering the edges of deteriorating paint and repainting with new, lead-free paint. Window sills are covered with permanent barrier. A *Dust Cleaning* of the affected area follows the intervention.	5 years for paint, 5 years for dust
	Encapsulation/ Abatement	Painted surfaces with deteriorated LBP are encapsulated, enclosed, or removed using currently acceptable practices and materials. A *Dust Cleaning* of the housing unit follows the intervention.	20 years for paint, 8 years for dust
Soil	Cover	Areas of bare soil are reseeded, resodded or covered with mulch, gravel, etc.	5 years
	Removal	Soil from areas with elevated lead concentrations are removed and replaced with clean soil, or the areas are permanently covered. A *Dust Cleaning* of the housing unit follows the intervention.	Permanent

[1] Duration is defined as the length of time before the lead levels in the targeted medium or conditions of the medium require further intervention.

The specified durations of the interventions reflect the length of time before the targeted media returns to levels or conditions requiring further intervention. For example, the duration of a paint intervention represents the estimated period of time before formerly intact or repaired surfaces deteriorate. When defining the duration of interior lead-based paint abatements, the duration of reduced interior residential dust-lead levels is also defined. Since paint interventions target only deteriorated lead-based paint, it would be unrealistic to assume that dust-lead levels remain low permanently. The once intact lead-based paint could, over time, deteriorate and produce elevated lead levels in residential house dust.

Unfortunately, there were only limited data available for estimating the duration of the methods defined in Table 5-2. The HUD Regulatory Impact Analysis (pages 3-21 through 3-22) utilized 4 and 8 years as the duration of reduced dust-lead levels following interim paint controls and paint abatements, respectively. These durations were based on estimates of the rate of increased dust-lead loading ($\mu g/ft^2$ per year) stemming from residential recontamination reported in studies of LBP interventions conducted in Baltimore and Cincinnati (page 3-22). These durations were the starting point for determining the dust durations for the paint interventions reported in Table 5-2. The efficacy duration for paint in the paint encapsulation/abatement intervention is consistent with HUD's definition of a LBP abatement practice: requiring the abatement to be effective for at least 20 years in order to be called an abatement. The five-year duration for paint maintenance intervention was intentionally set equal to the estimated dust duration, as the dust intervention is designed to follow paint interventions. For the soil cover intervention, a 5 year duration was utilized. This duration is reasonable given the duration of exterior paint repair (which would presumably reduce soil-lead levels) cited in the HUD RIA (page 3-23). Finally, the soil removal intervention was assumed to have permanent effectiveness in that the soil exhibiting elevated lead concentrations had been either removed or permanently covered.

5.2.2 Reductions in Environmental Lead Levels Following Interventions

The effectiveness of the interventions outlined in Table 5-2 is defined in terms of reductions in environmental-lead levels following conduct of the intervention. More particularly, the post-intervention environmental-lead levels may be specified for each of the interventions. Table 5-3 presents the post-intervention environmental-lead levels for each of the interventions described in Table 5-2. For each intervention, the post-intervention lead levels are defined for those media expected to be affected by the intervention. For example, interior paint abatement can be expected to prompt reductions in interior dust-lead loadings as well as in paint-lead loadings. Where relevant, additional details are provided regarding the effectiveness of the interventions.

The interventions outlined in Tables 5-2 and 5-3 are intended to include state-of-the-art practices. As a result, defining the effectiveness of these interventions as measured by reduced

environmental-lead levels is difficult. Though numerous intervention studies are documented in the literature, many utilized methods that today would be considered inappropriate. The available information on intervention effectiveness too often is of little relevance. Where possible, however, the available data were utilized.

Encapsulation/abatement of interior paint is assumed to reduce residential floor and window sill dust-lead loadings to 40 and 100 $\mu g/ft^2$, respectively, while effectively eliminating (for the duration outlined in Table 5-2) the hazard from deteriorated lead-based paint. The same degree of effectiveness with regard to residential dust was assumed for maintenance of interior paint (but for a shorter duration), soil removal, and one-time dust cleaning. This value was selected after considering the efficacy reported for housing units in the Denver Comprehensive Abatement Performance (CAP) Study and in the Baltimore Experimental Paint Abatement Study. The geometric mean floor vacuum dust-lead loading measured in abated units studied by the Denver CAP Study was 29.0 $\mu g/ft^2$ approximately two years following extensive paint abatements; the geometric mean window sill vacuum dust-lead loading was 91.6 $\mu g/ft^2$ among the same units (page 34 of EPA, 1995f). Similarly, the Baltimore Experimental Paint Abatement Study reported a geometric mean floor wipe dust-lead loading of 40.9 $\mu g/ft^2$ among 13 housing units 18-42 months following complete paint abatements; a geometric mean of 103 $\mu g/ft^2$ was reported for the unit's window sill wipe dust-lead loadings at the same time (page 62 of EPA, 1995c).

The complete effectiveness in terms of paint levels assumed for the four paint interventions is consistent with the procedures defined for the interventions and their assumed durations. These interventions are defined as utilizing practices consistent with ensuring that the surfaces with deteriorated paint remain intact following the intervention for the specified duration. Recall that the durations were defined to recognize the potential for paint, intact at the time of the intervention, becoming deteriorated over time. Thus, the potential hazard posed by deteriorated paint is assumed to be completely eliminated by each of the interventions (both interior and exterior) for the durations specified in Table 5-2.

Table 5-3. Expected Post-Intervention Lead Levels Associated With Performing §403 Interventions.

Intervention		Post-Intervention Lead Level	Comments on Performing the Intervention
Dust Cleaning		Dust-lead loading = min{40 $\mu g/ft^2$, pre} for floors min{100 $\mu g/ft^2$, pre} for window sills Dust-lead concentration = min{328 $\mu g/g$, pre} for floors	It is assumed that this intervention would occur only if dust-lead levels were above the standard, and if no sources of lead exposure remain in the housing unit.
Exterior LBP	Maintenance	Paint = 0	Deteriorated LBP is eliminated as a potential exposure source for the duration specified in Table 5-2.
	Encapsulation / Abatement	Paint = 0	Deteriorated LBP is eliminated as a potential exposure source for the duration specified in Table 5-2.
Interior LBP	Maintenance	Paint = 0 Dust-lead loading = min{40 $\mu g/ft^2$, pre} for floors min{100 $\mu g/ft^2$, pre} for window sills Dust-lead concentration = min{328 $\mu g/g$, pre} for floors	Deteriorated LBP is eliminated as a potential exposure source for the duration specified in Table 5-2.
	Encapsulation / Abatement	Paint = 0 Dust-lead loading = min{40 $\mu g/ft^2$, pre} for floors min{100 $\mu g/ft^2$, pre} for window sills Dust-lead concentration = min{328 $\mu g/g$, pre} for floors	Deteriorated LBP is eliminated as a potential exposure source for the duration specified in Table 5-2.
Soil	Cover	Soil = 50% of Pre-Intervention Levels	Residential dust is assumed unaffected by the intervention.
	Removal	Soil = 150 ppm Dust-lead loading = min{40 $\mu g/ft^2$, pre} for floors min{100 $\mu g/ft^2$, pre} for window sills Dust-lead concentration = min{328 $\mu g/g$, pre} for floors	Residential dust is not recontaminated by the intervention

The post-intervention soil-lead concentration assumed following soil removal was derived from the HUD National Survey and the Boston 3-City data. Among 257 HUD National Survey housing units with interior and exterior LBP in good condition (e.g., no deteriorated paint), a geometric mean soil-lead concentration of 61 ppm was reported. The Boston 3-City Soil Abatement Project reported an average soil-lead concentration of 160 ppm among 34 housing units 20 months following soil abatements (page 62 of EPA, 1995c). The same study documented arithmetic average soil-lead concentrations of 171 ppm and 180 ppm among two other groups (sample sizes of 32 and 26, respectively) of housing units 6 to 12 months following soil abatements (page A-24 of EPA, 1995c). Unfortunately, limited data

were available regarding the effectiveness of soil cover. The one available study that examined soil cover as an intervention strategy (Mielke et al., 1994) also included paint stabilization and interior dust control in the interventions performed at each residence. Foundation and mid-yard soil-lead levels, as measured by 2.5 cm core samples pre- and post-intervention, were reduced by approximately a factor of 3 to 4, four months following the interventions (as noted in Table 5-3 a factor of 2 was assumed in the analyses).

5.2.3 Intervention Triggers

An intervention is triggered if the housing unit exhibits environmental-lead levels in excess of those defined in §403. The findings of the risk assessment will not depend upon when the intervention occurs, only on its effectiveness once the intervention is conducted. It is assumed that the specific interventions conducted are those targeting the environmental media exhibiting the elevated levels. If either dust, soil, or paint exhibit levels in excess of those specified by the §403 standards, selecting appropriate interventions that target the problematic media are assumed. However, since two approaches to intervention are defined for paint and soil, a question remains as to which of the two is to be selected when the relevant environmental medium exceeds the §403 standard.

Table 5-4 summarizes the circumstances under which each of the defined interventions in Table 5-2 would be conducted. The choice of a soil-removal intervention versus a soil-cover intervention is made strictly on the measured lead concentration for specific areas in the yard. In contrast, the choice of an encapsulation/abatement approach versus a maintenance approach to paint intervention is based on the extent to which deteriorated lead-based paint is present. As noted earlier, dust cleaning is only prompted as a clean-up activity following an interior paint intervention or soil removal, or as a one-time activity where elevated dust-lead levels are observed despite the absence of residential sources of lead exposure (e.g., soil or paint). In such a case, it is assumed that the source of the lead has been abated due to activities conducted under §403 in the residence, neighboring residences, and the neighborhood in general.

Table 5-4. Intervention Triggers Defined for the Risk Assessment of §403.

Intervention		Circumstances Prompting Conduct of the Intervention
Dust Cleaning		Follows any interior paint intervention or soil removal, and when dust-lead loadings are elevated despite absence of residential sources of lead exposure (e.g., no deteriorated LBP or elevated soil lead).
Exterior LBP	Maintenance	When deteriorated exterior LBP is present, but not extensive (e.g., confined to a limited area).
	Encapsulation/ Abatement	When deteriorated exterior LBP is present and extensive (e.g., not confined to a limited area).
Interior LBP	Maintenance	When deteriorated interior LBP is present, but not extensive (e.g., confined to one area of the housing unit).
	Encapsulation/ Abatement	When deteriorated interior LBP is present and extensive (e.g., greater than one area of the housing unit).
Soil	Cover	When residential soil-lead concentrations exceed lower soil standard, but do not exceed the higher, emergency standard.
	Removal	When residential soil-lead concentrations exceed the higher, emergency soil standard. It is assumed this degree of intervention would only be warranted in specific areas of the yard.

5.2.4 Reductions in Blood-Lead Levels Following Interventions

For each home in the National Survey, the post-intervention environmental-lead levels presented in Table 5-3 were employed to predict the blood-lead concentrations of resident children. If an intervention was triggered by one or more of the standards, then the environmental-lead levels in the post-intervention time frame were set equal to those displayed in Table 5-3. The dose-response models (IEUBK and EPI) for predicting blood-lead concentration discussed in Section 4.1 were employed to predict childhood blood lead in the residence following the intervention activity. In this manner, the Risk Assessment estimated the impact of various options for the §403 standards on environmental-lead levels and childhood blood-lead concentrations in the nation's housing and children.

5.3 CHARACTERIZING THE RISKS FOLLOWING INTERVENTION

This section describes the predicted distributions of blood-lead concentrations and health effects following promulgation of §403. To enable evaluation of various options for standards, risks are characterized for different sets of standards, each set affecting a different number of houses nationwide. A four-step process was employed to characterize these risks for each set of standards:

1. §403 Intervention: Predict post-§403 environmental-lead levels
2. IEUBK/EPI PbB Prediction Model: Apply IEUBK and EPI models to environmental-lead levels to predict post-§403 blood-lead concentrations
3. Calibrate Predicted Blood-Lead Concentrations Using NHANES III: Estimate the change in modeled pre- and post-§403 blood-lead concentrations and apply that change to the NHANES III blood-lead concentration distribution
4. Summarize Risk: Predict health effects and blood lead endpoints for children aged 1-2 years.

This process is illustrated in Figure 5-3. In this figure, boxes with rectangular corners represent datasets or tables of results. Boxes with rounded corners represents steps in the process that transform the data being fed to it -- either through a predictive model (e.g., the IEUBK and EPI models are used to predict blood-lead concentrations from environmental-lead levels) or computation.

The four numbered steps in the process are illustrated by the four boxes with rounded corners in Figure 5-3. The remaining text in this section describes each of these steps in more detail.

Step 1: Predict Post-§403 Environmental Lead Levels. The HUD National Survey data were used to predict health risks after §403 in the following manner.

1. Environmental-lead levels observed at each home were compared to various options for the §403 standards.
2. These levels are projected to be reduced as a result of an intervention required by the rule or to remain unchanged if an intervention is not required as described in Section 5.2.

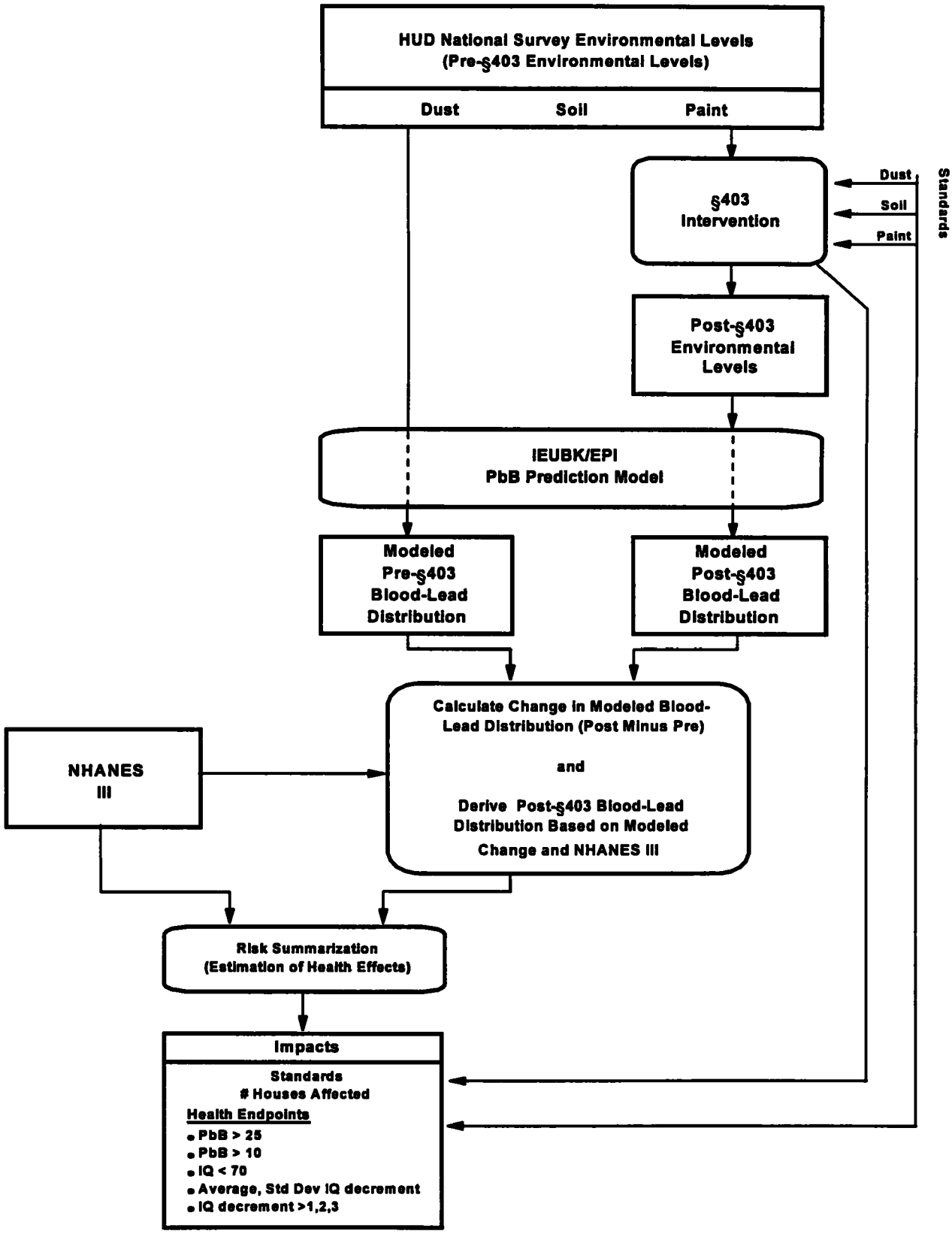


Figure 5-3. Post-§403 Risk Characterization Process

For example, Table 5-5 illustrates the assumed impact of a §403 intervention on environmental-lead levels at a particular house in the HUD National Survey (ID 1011501) for the following set of standards:

floor dust-lead loading:	200 μg/ft^2
window sill dust-lead loading:	500 μg/ft^2
soil (cover):	400 μg/g
soil (removal):	3000 μg/g
paint (maintenance):	Greater than 5 ft^2 of damaged LBP
paint (abatement):	Greater than 20 ft^2 of damaged LBP

Table 5-5. Projected Impact of §403 on House 1011501 in the National Survey

Environmental Lead Level	Pre-§403	Post-§403
Floor dust-lead loading (μg/ft^2)	32.4	32.4
Floor dust-lead concentration (μg/g)	623	328
Window sill dust-lead loading (μg/ft^2)	65.3	65.2
Soil-lead concentration (μg/g)	4,619	150
Maximum XRF (mg/cm^2)	1.4	1.4
Damaged LBP (ft^2)	0	0

Notice that the soil-lead concentration (4619 μg/g) was above the 3000 μg/g standard for soil removal. This would have triggered soil removal. As pointed out in Table 5-3, this causes post-§403 soil-lead concentration to go down to 150 μg/g, and dust-lead concentrations to be reduced to the minimum of pre-intervention levels and 328 μg/g. Because the pre-§403 floor dust-lead concentration was 623 μg/g, levels are reduced to 328 μg/g. The floor dust-lead loading is not affected because pre-intervention loading was less than 40 μg/ft^2. Similarly, the

window sill dust-lead loading is unchanged because the pre-intervention loading was below 100 μg/ft².

Selection of ranges for the proposed standards was discussed in Section 4.5. The ranges assessed in the risk characterization for lead in dust and soil and lead-based paint are displayed in Table 5-6. The anticipated effects of different types of interventions were documented in Section 5.2.

Table 5-6. Ranges of Standards Considered.

Medium	Range of Standards
Dust	Floors: 25-400 μg/ft² Window Sills: 25-800 μg/ft²
Soil	Cover: 50-1500 μg/g Removal: 1000-5000 μg/g
Paint	Maintenance: 0-10 ft² of damaged LBP Abatement: 5-100 ft² of damaged LBP

Step 2: Use IEUBK/EPI models to predict blood-lead concentrations. The second step in the process is the translation of post-intervention environmental-lead levels and pica tendencies into blood-lead concentration in children. Post-§403 blood-lead levels are being inferred using two different blood-lead models applied to projected environmental-lead levels anticipated as a result of promulgating §403 (Step 1 above).

The first of these models is an epidemiologically-based (EPI) model introduced in Section 4.1. EPA's IEUBK model, is also used to project blood-lead levels following promulgation of the §403 ruling. The IEUBK model is being used to predict blood-lead levels for children aged 24 months, as discussed in Section 4.1. For example, for the case illustrated under Step 1, the geometric mean blood-lead concentration estimated from pre-§403 environmental-lead levels is 22.9 μg/dL and the post-§403 geometric mean blood-lead level is predicted to be 5.0 μg/dL based on the IEUBK model.

Both the EPI model and the IEUBK model predict geometric mean blood-lead levels for the subpopulation of children exposed to the specified environmental conditions. However,

these geometric means are not sufficient to characterize the national distribution of children's blood-lead levels. Not every child exposed to floor dust-lead loading of 100 $\mu g/ft^2$, soil-lead concentration of 250 μg/g, and maximum paint lead loading of 1.1 mg/cm^2 with 5 ft^2 of damaged LBP will have the same blood-lead concentration. The model-predicted geometric means need to be supplemented by some measure of variability that reflects differences in blood-lead levels observed under the same measured environmental-lead levels. This is accomplished by use of the GSD of 1.6 μg/dL presented in the guidance manual for the IEUBK model (EPA, 1994a) (see Section 4.3.1). Appendix E1 describes the approach taken to characterize the variability in blood-lead levels about the estimated geometric means and how this information is used to determine a distribution of PbB over a population of children. These additional steps are required to infer the arithmetic average blood-lead concentration or the proportion of children with a blood-lead concentration are above a specified concentration.

Step 3: Adjust predicted blood-lead concentrations using (NHANES) baseline information. Step 2 in this process estimates the pre- and post-§403 distribution of blood-lead concentrations from environmental-lead levels. Step 3 determines the change in blood-lead concentrations resulting from the intervention (post-§403 minus pre-§403), and applies this change to the distribution of blood-lead concentrations inferred from NHANES III. This step is necessary because the NHANES results are regarded as the most reliable baseline characterization of children's blood-lead concentration available. The IEUBK and EPI models applied in Step 2, however, are the best tools available for estimating the change in PbB associated with an intervention. Thus, there are three inputs to this step in the process:

1. A model-predicted, pre-§403 distribution of PbB
2. A model-predicted, post-§403 distribution of PbB
3. A baseline distribution of PbB from NHANES

In this step, the difference between pre-§403 modeled PbB and post-§403 modeled PbB is applied to the baseline distribution of PbB inferred from NHANES III. The details of this step are described in Steps (1) through (4) of Appendix E2. The result is an estimate of the

geometric mean and the geometric standard deviation of blood-lead levels in the nation following §403. This information is used to predict health risks to children in the next step.

Step 4: Predict health effects and blood lead endpoints for children 1-2 years old. The last step in the process is the summarization of health risks associated with the baseline and the predicted post-§403 distributions of blood-lead concentrations. This step estimates the proportion of children with blood-lead levels above specified thresholds, the proportion of children anticipated to experience IQ decrements of specified amounts due to elevated blood lead concentrations, the proportion of children with IQ levels below 70 due to elevated blood lead concentrations, and the average and standard deviation of IQ point losses, due to elevated blood lead concentrations. Each of these endpoints is estimated from the geometric mean and geometric standard deviation, assuming a lognormal distribution. The mathematical approach used to make these inferences is described in Step (5) of Appendix E2.

The remainder of this section presents the estimated health risks due to lead-based paint following the rule-making for various options of the standard. Section 5.3.1 summarizes the specific endpoints listed in Section 5.0 for various options considered for the §403 standards (see Section 4.5). Section 5.3.2 displays in more detail the estimated risks associated with a particular "central" set of standards and environmental conditions observed in the HUD National Survey. Please note that Section 5.3.2 does not promote a set of standards as being the best to implement; it is merely provided to present a more in-depth summary of the predicted reduction in risks associated with an option for the standard.

5.3.1 Characterization of Risks for Various Sets of Standards

Chapter 3 of this report presented numbers of children aged 1-2 years associated with each housing unit in the HUD National Survey in 1997. These numbers reflect the estimated numbers of children who will reside in housing units with similar dust-, soil-, and paint-lead levels, and demographic variables in 1997. The HUD National Survey environmental-lead levels as modified by the proposed §403 interventions, are being used to predict blood-lead concentrations for the children assigned to each house. Predicted blood-lead concentrations are in turn used to characterize the national distribution of blood-lead concentrations as a result of §403.

For each medium, several different standards were considered. The ranges of different standards for each medium were introduced in Section 4.5 based on an analysis of health effects. Table 5-6 (above) presents a list of these ranges for the standards. There is a tremendous number of combinations of standards options that could be evaluated for the various media being considered. To reduce the complexity and reduce the number of options to consider, dust options are evaluated first with soil and paint standards fixed at central values within their specified ranges. Then soil options are considered with dust and paint standards fixed at central values. Options for paint standards are presented in an analogous fashion.

For example, Table 5-7 presents a range of options for floor and window sill dust standards. In this table, the soil cover standard is set at 400 μg/g, the soil removal standard is set at 3000 μg/g, the paint maintenance standard is set at 5 ft^2 of damaged LBP, and the paint abatement standard is set at 20 ft^2 of damaged LBP. The options for floor dust-lead loading standards range from 25 to 400 $\mu g/ft^2$ (in reverse order), and from 25 to 800 $\mu g/ft^2$ for window sills. Each column is devoted to a specific pair of standards for floor and window sill dust-lead loading. For each set of standards, the top part of Table 5-7 indicates the percentage of homes that would be affected specifically by each of the floor and window sill dust-lead loading standards, the percentage of homes that would be affected by either the floor or window sill dust-lead loading standards and the percentage of homes that would be affected by any one of the standards for dust, soil, or paint specified in this table.

For example, in the first column of standards in Table 5-7, we see that only 0.30 percent of houses in the nation would be expected to exceed a floor dust lead loading standard of 400 $\mu g/ft^2$. 10.6 percent of the nation's homes would be expected to have window sill dust-lead loading exceeding 800 $\mu g/ft^2$. 10.9 percent of the nation's homes would be expected to exceed either of these two standards. Note that in this case, the percentage exceeding the floor dust-lead standard (0.3) and the percentage exceeding the window sill dust-lead standard (10.6) added to equal the percentage of homes exceeding either dust-lead standard (10.9).

Table 5-7. Characterization of Impact of Various Options for Dust Standards: Soil and Paint Standards fixed (400 μg/g for Soil Cover, 3000 μg/g for Soil Removal, 5 ft² damaged LBP for Paint Maintenance, 20 ft² damaged LBP for Paint Abatement).

Options for Dust Lead Loading Standard (μg/ft²)						
Floors	400	200	100	100	50	25
Window Sills	800	500	500	200	100	25
Percentage of Homes Exceeding Floor Dust Standard	0.297	1.98	8.94	8.94	16.7	33.4
Percentage of Homes Exceeding Window Sill Dust Standard	10.6	14.1	14.1	26.9	37.0	54.6
Percentage of Homes Exceeding Any Dust Standard	10.9	15.4	18.0	29.0	43.1	62.9
Percentage of Homes Exceeding Any Standard	24.3	26.1	28.2	35.7	47.1	64.6
Health Effects Projected by EPI Model						
PbB > 25 μg/dL (%)	0.27	0.25	0.24	0.22	0.21	0.21
PbB > 10 μg/dL (%)	7.8	7.5	7.3	7.1	7.0	6.9
IQ < 70 (%)	0.53	0.53	0.52	0.52	0.52	0.52
IQ decrement > 1 (%)	49	49	49	48	48	48
IQ decrement > 2 (%)	15	14	14	14	14	14
IQ decrement > 3 (%)	4.9	4.7	4.6	4.4	4.3	4.3
Avg. IQ decrement	1.24	1.23	1.22	1.21	1.20	1.20
SD of IQ decrement	0.94	0.92	0.91	0.90	0.89	0.89
Health Effects Projected by IEUBK Model						
PbB > 25 μg/dL (%)	0.071	0.031	0.026	0.023	0.015	0.011
PbB > 10 μg/dL (%)	4.8	3.6	3.4	3.2	2.7	2.5
IQ < 70 (%)	0.49	0.48	0.48	0.48	0.47	0.47
IQ decrement > 1 (%)	46	45	44	44	43	42
IQ decrement > 2 (%)	11	8.8	8.4	8.1	7.2	6.7
IQ decrement > 3 (%)	2.7	1.9	1.8	1.7	1.4	1.2
Avg. IQ decrement	1.13	1.09	1.08	1.07	1.05	1.04
SD of IQ decrement	0.75	0.67	0.65	0.64	0.61	0.60

However, this is not always the case because there is often overlap between these two sets of homes. That is, often a house exceeding the sill dust-lead standard will also exceed the floor dust-lead standard. For example, for the third set of dust-lead standards listed in Table 5-7, the floor and window sill dust-lead standards are 100 and 500 μg/ft², respectively. 8.94 percent of homes are expected to exceed the floor dust-lead standard, and 14.1 percent are expected to exceed the window sill dust-lead standard. Eighteen percent are expected to exceed either standard. This means that 23.04 (=8.94 + 14.1) - 18.0 = 5.04 percent are expected to exceed both floor and window sill standards.

Continuing down the rows of Table 5-7, we see that 24.3 percent of the nation's homes would be expected to exceed any of the standards considered in this column. These are:

floor dust-lead loading:	400 μg/ft²
window sill dust-lead loading:	800 μg/ft²
soil (cover):	400 μg/g
soil (removal):	3000 μg/g
paint (maintenance):	Greater than 5 ft² of damaged LBP
paint (abatement):	Greater than 20 ft² of damaged LBP

As mentioned above, only 10.9 percent of the homes were projected to exceed either of the dust-lead standards. This means that 24.3 - 10.9 = 13.4 percent of the nation's homes are projected to exceed 400 μg/ft in soil-lead concentration, or exceed 5 ft² of damaged LBP but not exceed either of the dust-lead standards.

Estimates of the selected health effects projected after implementation of these standards, based on the EPI model are presented in the middle section of Table 5-7. For example, if §403 were formulated with the standards associated with the first column of Table 5-7 (described above), 0.27 percent of the nation's children would be projected to have PbB above 25 μg/dL using the EPI model. Approximately 8 percent of children would be projected to have PbB exceeding 10 μg/dL. Only 0.53 percent of kids would be expected to have IQ scores below 70 due to elevated blood-lead concentration. The next three lines describe the proportion of kids expected to have IQ decrements of 1 or more, 2 or more, and 3 or more, based on the EPI model, after intervention on the basis of these standards. The predictions are 49 percent, 15 percent, and 4.9 percent, respectively. The next two lines describe the distribution of IQ

decrements associated with elevated PbB. Interventions triggered by the first set of standards would be projected to result in (arithmetic) average IQ decrement of 1.24 with standard deviation of 0.94. Note that the distribution of IQ decrements is not symmetric. This was illustrated in Figure 5-2 for NHANES III. In fact, it is well described by a lognormal distribution. Therefore, for estimating the proportion of children with IQ decrement of specified levels, the three previous lines in the table should be used.

The bottom part of Table 5-7 presents the same information but with the projected health effects determined using the IEUBK model to predict blood-lead concentrations instead of the EPI model.

5.3.1.1 Varying Dust Standard Options

Table 5-7 examines the impact of various options for the dust-lead standard on childhood health effects and blood-lead concentration. Options for floor and window sill dust-lead loadings were varied simultaneously from 400 and 800 μg/ft² for floor dust-lead loading and window sill dust-lead loading, respectively, to 25 and 25 μg/ft², respectively. A total of six combinations were assessed. The first few rows of Table 5-7 predict that the number of houses that would be affected by any of the selected standards represented in these tables ranges from 24 percent to 65 percent. Examining the associated health effect and blood-lead concentration endpoints reveals that the most dramatic improvement is achieved between the least stringent sets of standards considered, with diminishing returns evident between successive reductions in the standards. This is most clearly evident for the percentage of children with blood-lead concentration exceeding 25 μg/dL or 10 μg/dL, the percentage of children that will have an IQ decrement of at least 2 or 3, and the average IQ decrement. For example, based on the EPI model, 7.8 percent of the nation's children would be anticipated to have PbB exceeding 10 μg/dL if the least stringent set of standards (floor: 400 μg/ft²; window sill: 800 μg/ft²). The IEUBK model predicts 4.8 percent. These percentages can be compared with the baseline (current) estimate of 10.5 percent. Under the second option for dust standards (floor 200 μg/ft²; window sill 500 μg/ft²) without changing the options for the soil and paint standards, the projections come down to 7.5 percent (EPI) and 3.6 percent (IEUBK). By reducing both of the dust standards to the lowest considered in this analysis (floor 25 μg/ft²,

window sill 25 μg/ft²), the estimates of the proportion of children with blood-lead concentration exceeding 10 μg/dL are reduced to 6.9 percent (EPI) and 2.5 percent (IEUBK).

Figures 5-3a and 5-3b display the eight health endpoints presented in Table 5-7 in graphical form. The top left graph in Figure 5-3a presents the projected percentage of children that would have PbB greater than 25 μg/dL as a result of implementing §403 for each set of standards considered for floor and window sill dust versus the percentage of homes exceeding any of these standards. There are two curves in the graph, the top curve reflects predictions based on the EPI model, and the bottom curve is based on the IEUBK model. Note that on each curve there are six dots, corresponding to the six sets of standards considered for dust-lead loadings. A reference line is drawn at the top of the graph to indicate the baseline levels for this response determined from NHANES III, (0.58 percent of children are currently estimated to have PbB above 25 μg/dL).

The top right graph in Figure 5-3a presents the analogous information for the projected percentage of children that would have PbB greater than 10 μg/dL as a result of implementing §403 with these standards. The lower left graph presents the average IQ decrement resulting from elevated blood-lead concentration, and the lower right graph presents the standard deviation of IQ decrements resulting from elevated blood-lead concentration. All of these are plotted versus the percentage of homes anticipated to exceed any of the standards over the range of standards considered. Figure 5-3b presents, in the same format, the percentage of children with IQ below 70, and the percentage of children expected to have IQ decrements of at least 1, 2, and 3 points as a result of elevated blood-lead concentration.

These graphs show the impact of various options for the dust standards on health effects, children's blood-lead concentrations, and the number of homes impacted by the standards. Note the generally consistent shape of each of the curves in these figures. A sharp decline in the curve indicates a large change in the health effects or blood-lead concentrations relative to a small difference in the number of homes requiring an intervention. A less steep decline indicates either a large increment in the number of homes requiring an intervention, or

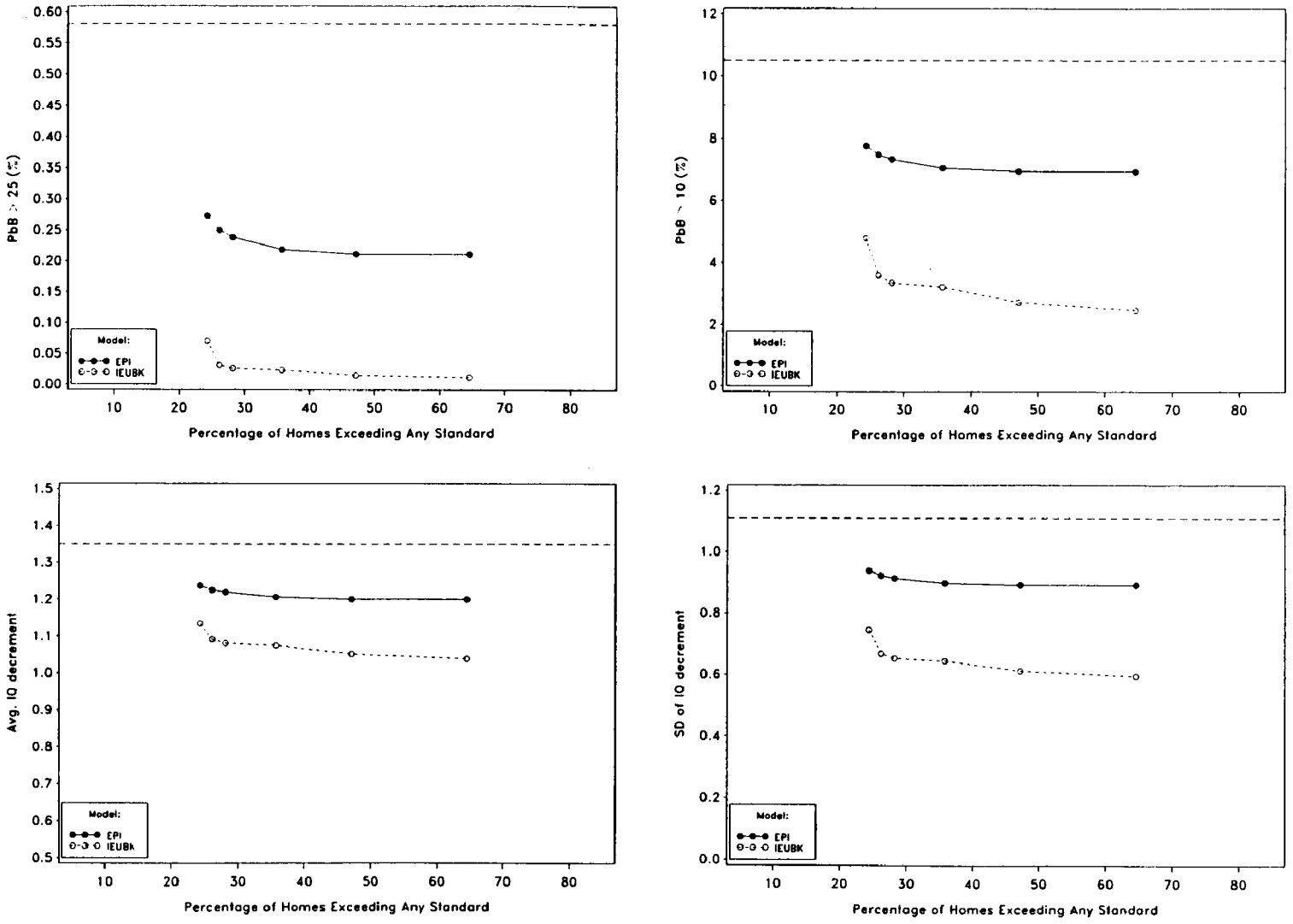


Figure 5-3a. Projected Health Endpoints Based on Various Options for Dust Standards, Part 1; Soil Cover 400 μg/g, Soil Removal 3000 μg/g, Paint Maintenance 5 ft² Paint Abatement 20 ft². (Dashed reference line represents baseline risk.)

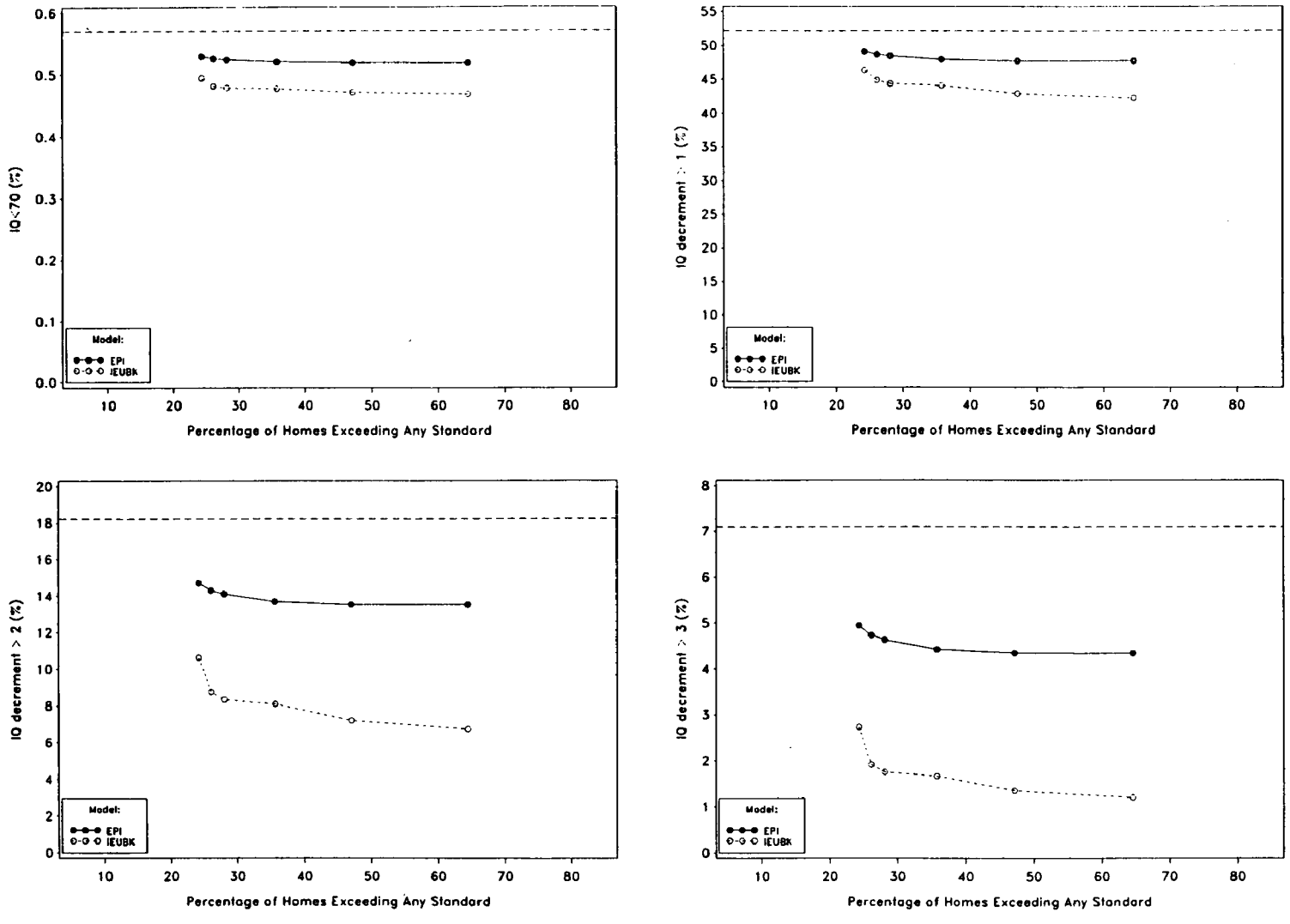


Figure 5-3b. Projected Health Endpoints Based on Various Options for Dust Standards, Part 2; Soil Cover 400 μg/g, Soil Removal 3000 μg/g, Paint Maintenance 5 ft², Paint Abatement 20 ft². (Dashed reference line represents baseline risk.)

a small net health benefit. In each case, the steepest drop occurs between the two least-stringent sets of standards (floor: 400 μg/ft²; window sill: 800 μg/ft²) and (floor: 200 μg/ft²; window sill: 500 μg/ft²), and then gradually levels off as the standards affect greater and greater numbers of homes. This pattern is consistent between the EPI and IEUBK models, and across health effects, with some endpoints reflecting the pattern more drastically than others.

This suggests that with regard to changes in dust-lead standards, there does not appear to be much benefit to enforcing dust-lead loading standards more stringent than perhaps the second or third most stringent standards considered in this analysis. Although the number of additional houses affected by the more stringent standards is very large, the incremental gains in health benefits are small.

The projected health effects as a result of implementing §403 with the various standards can be compared to the baseline (current estimated) health effects and blood-lead concentrations using the reference line in each graph. For example, each of the sets of standards considered for dust would result in a substantial improvement relative to the baseline for the percentage of children exceeding 25 μg/dL and 10 μg/dL, and the percentage of children anticipated to have an IQ decrement of at least 2 or 3 resulting from elevated blood-lead concentration. The improvement from baseline is much less for IQ < 70 and IQ decrements of at least 1. Note that the graph of average IQ decrement has a lower axis bound of 0.5.

There is little reduction in the percentage of children predicted to have IQ below 70 or in the percentage of children expected to have IQ decrements greater than 1 over the range of standards considered.

5.3.1.2 Varying Soil Standard Options

Table 5-8 presents results for a range of options for the §403 soil cover and soil removal standards with the floor dust-lead loading standard set at 100 μg/ft², the window sill dust-lead loading set at 500 μg/ft², the paint maintenance standard set at 5 ft² of damaged LBP, and the paint abatement standard set at 100 ft² of damaged LBP. The options for requiring soil covering range from 50 to 1500 μg/g and the options for requiring soil abatement range from 1000 to 5000 μg/g. For each of these options, the top portion of Table 5-8 indicates the percentage of homes that would be affected specifically by the soil covering standard, the

percentage that would be affected by the soil abatement standard, and the percentage of homes that would be affected by any one of the standards for dust, soil, or paint specified in the table. The remaining rows of Table 5-8 are analogous to those displayed in Table 5-7. Projected health effects are predicted first based on the EPI model, then based on the IEUBK model.

Table 5-8 predicts that the number of houses that would be affected by any of the selected standards represented in these tables ranges from 25 percent to 52 percent. The least stringent standards considered were 1500 μg/g for soil cover and 5000 μg/g for soil removal. The most stringent standards considered were 50 μg/g and 1000 μg/g for soil cover and soil removal, respectively. Over this range of standards, the projected post-§403 proportion of children with blood-lead concentration exceeding 25 μg/dL ranges from 0.27 percent to 0.16 percent based on the EPI model and from 0.074 percent to 0.005 based on the IEUBK model. The corresponding projected proportions for blood-lead concentration exceeding 10 μg/dL range from 7.8 to 6.2 percent based on the EPI and 4.9 to 1.8 percent based on the IEUBK. Thus, although for each set of standards the IEUBK projects lower incidence of elevated blood-lead concentrations, both models project substantial reductions in this incidence over the range of standards.

The proportion of children projected to have IQ scores below 70 due to elevated blood-lead concentration only ranges from 0.53 percent to 0.51 percent based on the EPI model, and from 0.50 percent to 0.46 percent based on the IEUBK model. Thus, little benefit is anticipated from implementation of these standards for this health effect. However, of the three sets of standards considered (dust, soil, and paint), varying soil standards has the greatest potential impact on this endpoint.

For IQ decrements, the EPI model projects reductions in the proportions of children with the greatest relative reductions seen for the larger decrements (IQ decrement >2, 3). The IEUBK predicts greater relative reductions for each of the three thresholds (>1, >2, >3).

Table 5-8. Characterization of Impact of Various Options for Soil Standards: Dust and Paint Standards fixed (100 $\mu g/ft^2$ for Dust Lead Loading, 500 $\mu g/ft^2$ for Window Sill Dust Lead Loading, 5 ft^2 damaged LBP for Paint Maintenance, 20 ft^2 damaged LBP for Paint Abatement).

Options for Soil Lead Concentration Standard ($\mu g/g$)						
Soil Cover	1500	800	400	200	100	50
Soil Removal	5000	4000	3000	3000	2000	1000
Percentage of Homes Exceeding Soil Cover Standard	3.27	8.11	12.8	20.1	27.1	42.8
Percentage of Homes Exceeding Soil Removal Standard	0.215	0.746	0.746	0.746	2.71	6.14
Percentage of Homes Exceeding Any Standard	25.2	25.9	28.2	30.9	36.6	52.1
Health Effects Projected by EPI model						
PbB > 25 $\mu g/dL$ (%)	0.27	0.25	0.24	0.22	0.19	0.16
PbB > 10 $\mu g/dL$ (%)	7.8	7.5	7.3	7.1	6.7	6.2
IQ < 70 (%)	0.53	0.53	0.52	0.52	0.52	0.51
IQ decrement > 1 (%)	49	49	49	48	47	46
IQ decrement > 2 (%)	15	14	14	14	13	12
IQ decrement > 3 (%)	4.9	4.7	4.6	4.5	4.1	3.8
Avg. IQ decrement	1.24	1.23	1.22	1.21	1.19	1.16
SD of IQ decrement	0.94	0.92	0.91	0.9	0.88	0.85
Health Effects Projected by IEUBK model						
PbB > 25 $\mu g/dL$ (%)	0.074	0.034	0.026	0.020	0.010	0.0050
PbB > 10 $\mu g/dL$ (%)	4.9	3.7	3.4	3.1	2.3	1.8
IQ < 70 (%)	0.50	0.48	0.48	0.47	0.47	0.46
IQ decrement > 1 (%)	47	45	44	44	41	39
IQ decrement > 2 (%)	11	9.0	8.4	7.8	6.5	5.2
IQ decrement > 3 (%)	2.8	2.0	1.8	1.6	1.1	0.81
Avg. IQ decrement	1.14	1.10	1.08	1.06	1.03	0.99
SD of IQ decrement	0.75	0.68	0.65	0.63	0.59	0.55

Figures 5-4a and 5-4b display the eight health endpoints presented in Table 5-8 in a graphical form similar in format to Figures 5-3a and 5-3b. There are two curves in each graph representing the predictions from the EPI and IEUBK models, and the baseline level of each endpoint (as determined from NHANES) is drawn as a reference line for comparison on each graph.

As with the predicted health effects associated with changes in dust-lead standards, the health effects and blood-lead concentrations predicted as a result of various options for soil standards indicate that the greatest improvement in health effects is achieved between the two least stringent sets of standards considered. The first and least stringent set of soil standards considered is (soil cover: 1500 μg/g; soil removal: 5000 μg/g) and the second set was (soil cover: 800 μg/g; soil removal: 4000 μg/g). There are reduced incremental benefits achieved for more stringent soil standards, but there are still gains to be made between successive reductions in the standards up to about the fifth set of standards (soil cover: 100 μg/g; soil removal: 2000 μg/g).

Again, note the generally consistent shape of each of the curves in these figures. In each case, the steepest drop occurs between the two least-stringent sets of standards, the next four points follow almost a straight line, and then the most stringent set of standards results in only a small incremental health benefit. This pattern is consistent between the EPI and IEUBK models and across responses, with some endpoints reflecting the pattern more drastically than others. The endpoints most sensitive to changes in soil standards based on the EPI and IEUBK models are the proportion of children projected to have blood-lead concentrations exceeding 10 and 25 μg/dL and the proportion of children expected to have IQ decrements at least 2 or 3. The health effect least sensitive to changes in soil standards is the projected proportion of children with IQ less than 70 due to elevated blood-lead concentration.

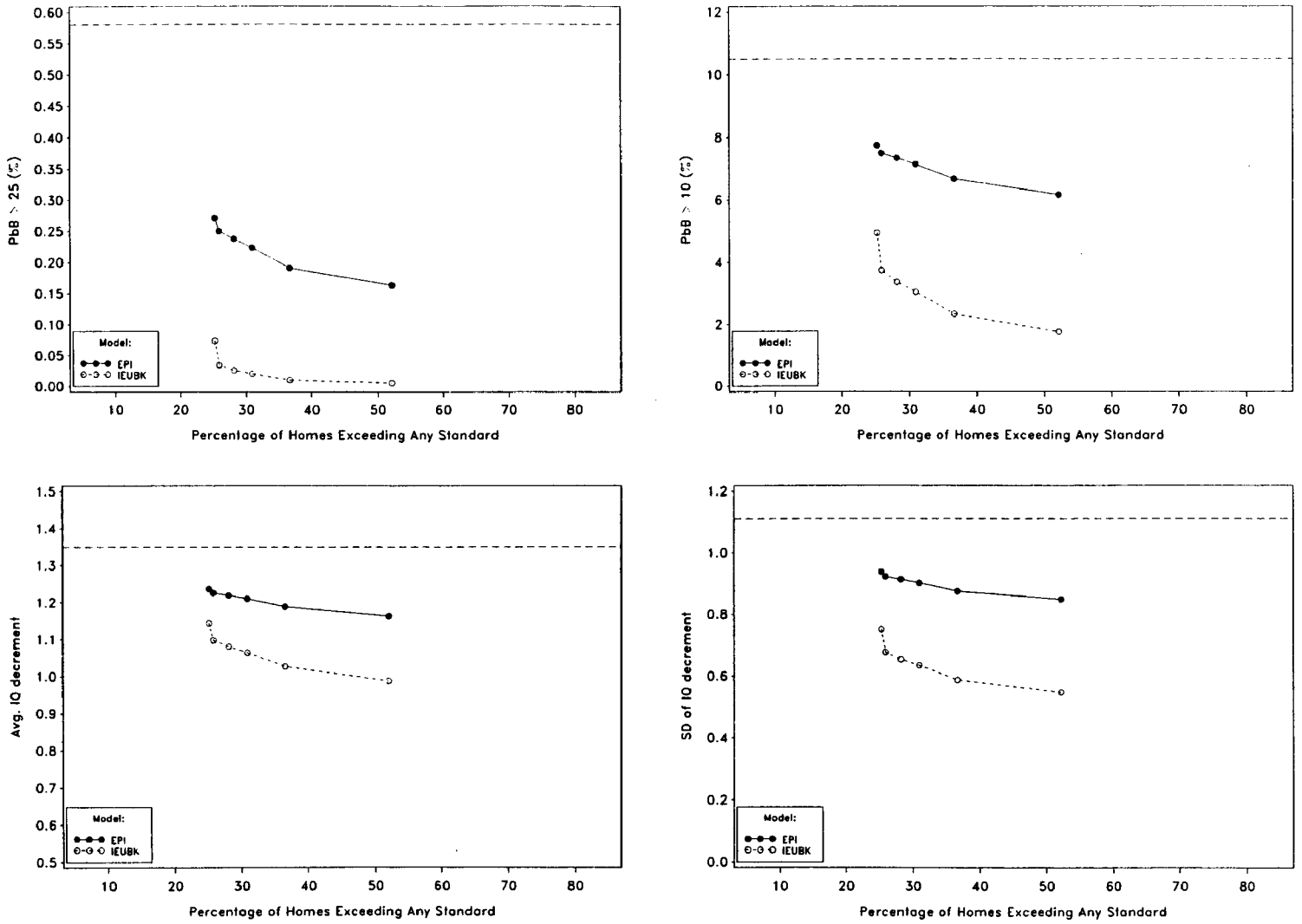


Figure 5-4a. Projected Health Endpoints Based on Various Options for Soil Standards, Part 1; Floor Dust 100 μg/ft², Window Sill Dust 500 μg/ft², Paint Maintenance 5 ft², Paint Abatement 20 ft². (Dashed reference line represents baseline risk.)

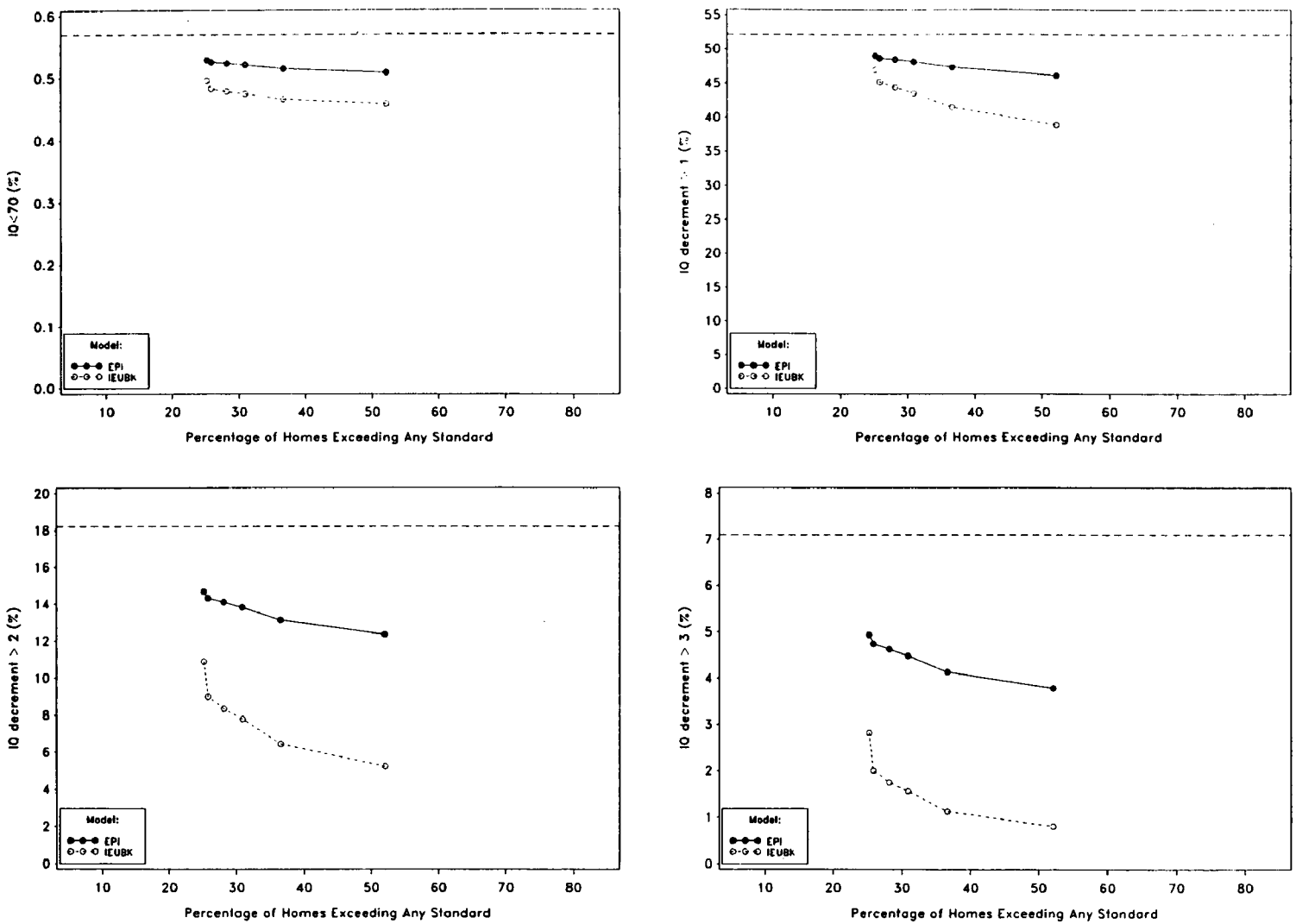


Figure 5-4b. Projected Health Endpoints Based on Various Options for Soil Standards, Part 2; Floor Dust 100 μg/ft², Window Sill Dust 500 μg/ft², Paint Maintenance 5 ft², Paint Abatement 20 ft². (Dashed reference line represents baseline risk.)

The projected health effect and blood-lead concentration endpoints as a result of implementing §403 with the various standards can be compared to the baseline (current estimated) effects using the reference line in each graph. Each of the sets of standards considered for soil would result in a substantial improvement relative to the baseline for the percentage of children exceeding 25 μg/dL and 10 μg/dL, and the percentage of children anticipated to have an IQ decrement of at least 2 or 3 resulting from elevated blood-lead concentration.

There is little reduction in the percentage of children predicted to have IQ below 70 or in the percentage of children expected to have IQ decrement greater than 1 over the range of standards considered.

There is a clear benefit projected for even the least stringent soil-lead standards, and there is some additional benefit predicted for the more stringent standards. There are gains to be made in health benefits for more stringent standards as low as (soil cover: 100 μg/g; soil removal: 2000 μg/g). The most stringent set of standards (soil cover: 50 μg/g; soil removal: 1000 μg/g) affects a large number of houses with little incremental gains in health effects.

5.3.1.3 Varying Paint Standard Options

Table 5-9 presents results for a range of options for paint intervention standards with the floor dust-lead loading standard set at 100 μg/ft^2, the window sill dust-lead loading standard set at 500 μg/ft^2, the soil covering standard set at 400 μg/g, and the soil abatement standard set at 3000 μg/g. The options for requiring a paint maintenance range from 0 to 10 ft^2 of damaged LBP, and options for requiring a paint abatement range from 5 to 100 ft^2 of damaged LBP. For each of these options, the top portion of Table 5-9 indicates the percentage of homes that would be affected specifically by the standard for either interior or exterior paint maintenance standard, the percentage that would be affected specifically by either interior or exterior paint abatement standard, and the percentage of homes that would be affected by any one of the standards for dust, soil, or paint specified in the table. The remaining rows of Table 5-9 are analogous to those displayed in Tables 5-7 and 5-8. Results are first presented based on the EPI model, then based on the IEUBK model.

Table 5-9 predicts that the number of houses that would be affected by any of the selected standards represented in these tables ranges from 27 percent to 29 percent. The standards for paint intervention are defined in terms of a specified amount of damaged LBP. The least stringent standards considered were 10 square feet and 100 square feet, respectively. The most stringent standards considered for paint maintenance and paint abatement were zero square feet of damaged LBP and five square feet of damaged LBP, respectively.

Figures 5-5a and 5-5b display the eight health endpoints presented in Table 5-9 in graphical form in the same format as Figures 5-3a and 5-3b. There are two curves in each graph representing the predictions from the EPI and IEUBK models. The baseline level of each endpoint (as determined from NHANES III) is drawn as a reference line for comparison on each graph.

These figures highlight an important fact: The amount of damaged LBP is not a very useful discriminant for determining whether an intervention should be performed. The range of the percentage of houses affected by different thresholds of damaged LBP is not very wide. In fact, 24 percent of the nation's houses have dust- or soil-lead levels exceeding the standards for these media regardless of the amount of damaged LBP present in the houses. Imposing the standards considered for paint increases this number by only 3 to 5 percent. However, the tools available for assessing the impact of damaged lead-based paint are limited. Both the EPI and IEUBK models for predicting blood-lead concentrations based on environmental-lead levels are limited in their usage of paint-lead measurements. Paint-lead is incorporated into the IEUBK model by considering paint ingested due to pica as discussed in Section 4.3. The EPI model is based on the Rochester study data which does not have damaged LBP variables similar to the HUD National Survey damaged LBP variables. Pica for paint also plays a role in this model. Our estimate of the prevalence of pica for paint may not be accurate.

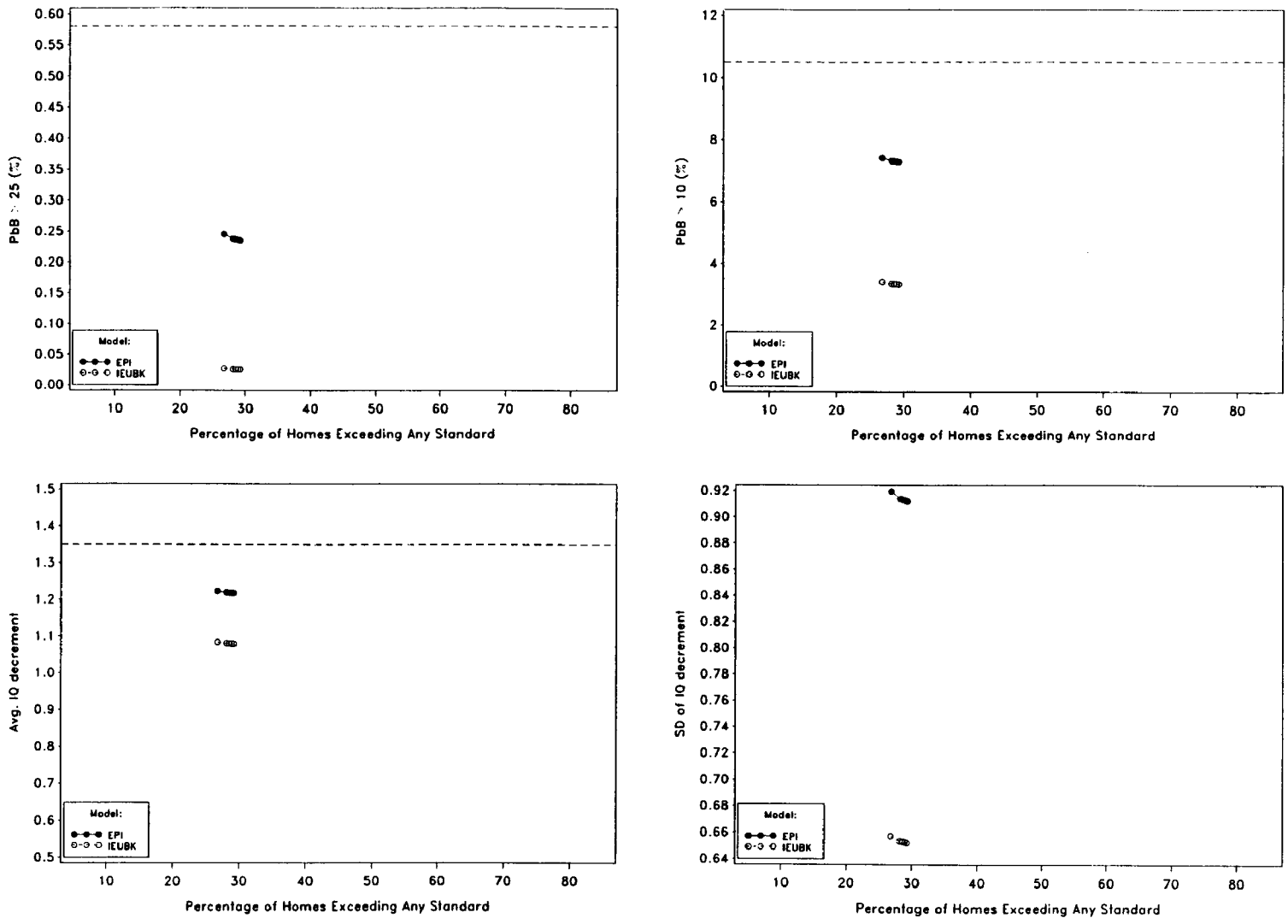


Figure 5-5a. Projected Health Endpoints Based on Various Options for Paint Standards, Part 1; Floor Dust 100 μg/ft², Window Sill Dust 500 μg/ft², Soil Cover 400 μg/g, Soil Removal 3000 μg/g. (Dashed reference line represents baseline risk.)

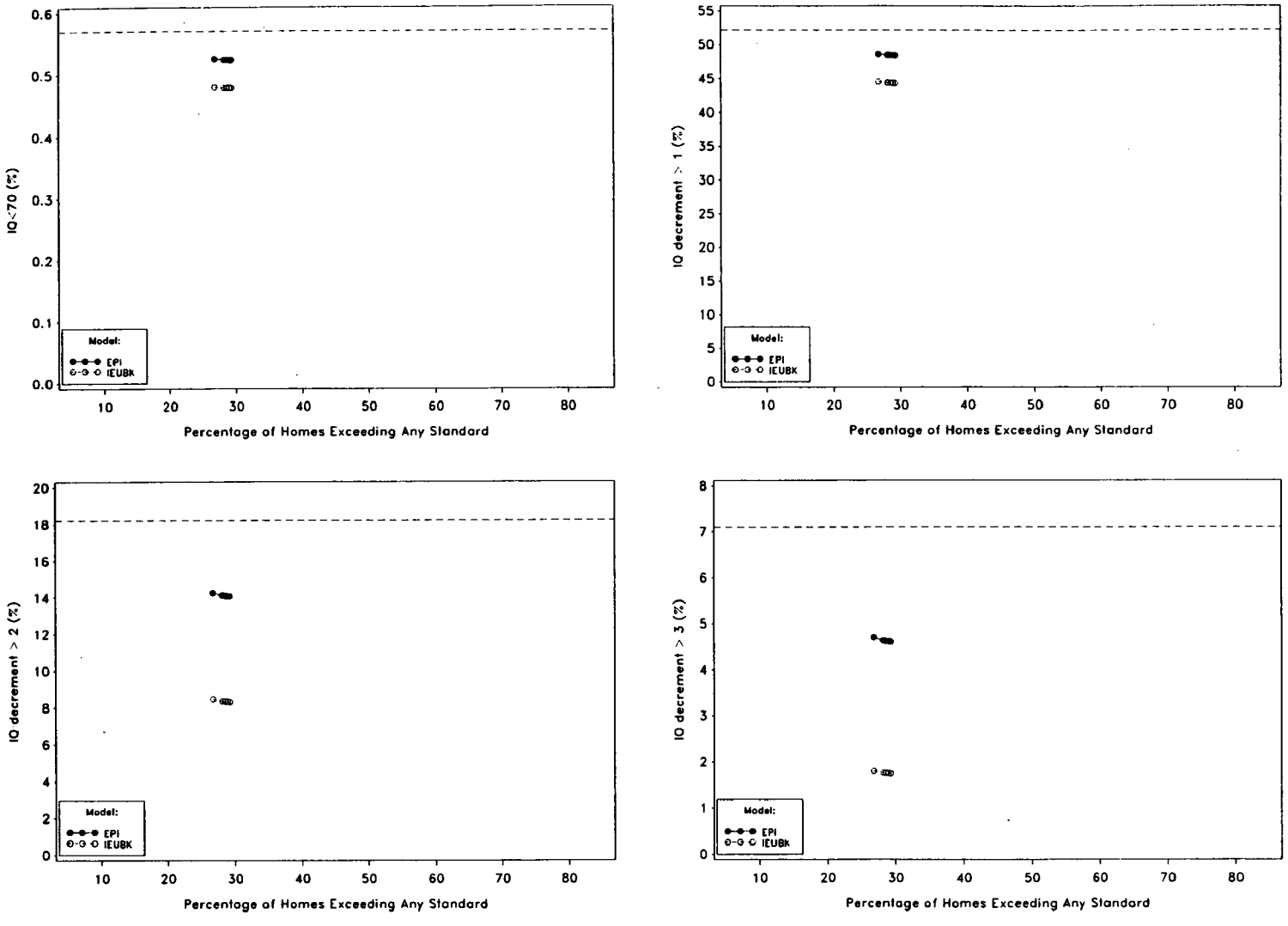


Figure 5-5b. Projected Health Endpoints Based on Various Options for Paint Standards, Part 2; Floor Dust 100 μg/ft², Window Sill Dust 500 μg/ft², Soil Cover 400 μg/g, Soil Removal 3000 μg/g. (Dashed reference line represents baseline risk.)

Table 5-9. Characterization of Impact of Various Options for Paint Standards: Dust and Soil Standards fixed (100 $\mu g/ft^2$ for Dust Lead Loading, 500 $\mu g/ft^2$ for Window Sill Dust Lead Loading, 400 $\mu g/g$ for Soil Covering, 3000 $\mu g/g$ for Soil Removal).

Options for Paint Standard (ft^2 damaged LBP)					
Paint Maintenance	**10**	**5**	**2**	**1**	**0**
Paint Abatement	**100**	**40**	**20**	**10**	**5**
Percentage of Homes Exceeding Interior Paint Maintenance Standard	2.80	4.37	3.03	2.75	1.08
Percentage of Homes Exceeding Exterior Paint Maintenance Standard	3.84	4.80	4.20	3.22	1.15
Percentage of Homes Exceeding Interior Paint Abatement Standard	0.453	0.980	2.43	3.25	5.35
Percentage of Homes Exceeding Exterior Paint Abatement Standard	3.03	4.46	5.77	6.87	9.26
Percentage of Homes Exceeding Any Standard	26.8	28.2	28.6	28.9	29.2
Health Effects Projected by EPI model					
PbB > 25 (%)	0.25	0.24	0.24	0.24	0.24
PbB > 10 (%)	7.4	7.3	7.3	7.3	7.3
IQ < 70 (%)	0.52	0.52	0.52	0.52	0.52
IQ decrement > 1 (%)	49	49	49	48	48
IQ decrement > 2 (%)	14	14	14	14	14
IQ decrement > 3 (%)	4.7	4.6	4.6	4.6	4.6
Avg. IQ decrement	1.22	1.22	1.22	1.22	1.22
SD of IQ decrement	0.92	0.91	0.91	0.91	0.91
Health Effects Projected by IEUBK model					
PbB > 25 (%)	0.027	0.026	0.026	0.026	0.025
PbB > 10 (%)	3.4	3.4	3.4	3.3	3.3
IQ < 70 (%)	0.48	0.48	0.48	0.48	0.48
IQ decrement > 1 (%)	45	44	44	44	44
IQ decrement > 2 (%)	8.5	8.4	8.3	8.3	8.3
IQ decrement > 3 (%)	1.8	1.8	1.7	1.7	1.7
Avg. IQ decrement	1.08	1.08	1.08	1.08	1.08
SD of IQ decrement	0.66	0.65	0.65	0.65	0.65

5.3.1.4 Varying All Standard Options

Analyses summarized in Tables 5-7, 5-8, and 5-9 permit an assessment of the impact on the nation's housing and health effects of children for various standard options for each individual environmental medium. However, those results do not show the effect of varying the levels simultaneously for dust, soil, and paint. Table 5-10 presents the results of analyses when the standards for all media are varied. The table is structured similarly to Tables 5-7, 5-8, and 5-9. Each column represents a unique combination of standards displayed at the very top in the shaded rows. For instance, the second column represents an option for the standards of 400 $\mu g/ft^2$ for floor dust-lead loading, 500 $\mu g/ft^2$ for window sill dust-lead loading, 800 $\mu g/g$ for soil cover, 4000 $\mu g/g$ for soil removal, 10 ft^2 of damaged lead-based paint for paint maintenance, and 40 ft^2 for paint abatement. Below these rows, the table displays the estimated number of homes affected by each standard. The first row in the middle portion provides the estimated number of homes above the option for the floor dust standard. Analogous information are provided in the next seven rows for window sill dust, soil cover, soil removal, interior and exterior paint maintenance, and interior and exterior paint abatement. Finally, the estimated number of housing units that would be affected by any one of the standards is presented.

The bottom portion of Table 5-10 provides the estimated health and blood-lead concentration effects, first based on the EPI model, then based on the IEUBK model, for each selected health endpoint in the post-§403 environment. For instance, health endpoints are shown in the second column for the combination of standards defined in the second column in the top half of the table. Presentation of the health effect results is in the same format as that employed in Tables 5-7 through 5-9.

A total of seven complete options for the standards were assessed. The least stringent option, given in the first column, is (floor: 400 $\mu g/ft^2$; window sill: 800 $\mu g/ft^2$; soil cover: 1500 $\mu g/g$; soil removal: 5000 $\mu g/g$; paint maintenance: 10 ft^2 damaged LBP; paint abatement: 100 ft^2 damaged LBP). The most stringent option, given in column 7, is (floor: 25 $\mu g/ft^2$; window sill: 25 $\mu g/ft^2$; soil cover: 50 $\mu g/g$; soil removal: 1000 $\mu g/g$; paint maintenance: 0 ft^2 damaged LBP; paint abatement: 5 ft^2 damaged LBP). In addition, an option corresponding to the interim standards presented in the interim rule (floor: 100 $\mu g/ft^2$; window sill: 500 $\mu g/ft^2$;

soil cover: 400 μg/g; soil removal: 5000 μg/g; paint maintenance: 2 ft^2 damaged LBP; paint abatement: 10 ft^2 damaged LBP), is assessed in the last column. Table 5-10 illustrates, in a rough sense, the costs and benefits that would be realized as a result of implementing §403 with various sets of standards. The cost is measured as the number of housing units affected and the benefits are expressed as probabilities of observing various health effects in children residing in these housing units. The number of units affected and the proportion of children with health effects were estimated using the methods presented in Chapter 3.

Table 5-10 predicts that the number of houses that would be affected by the sets of standards selected ranges from 19 percent to 74 percent. This is a wider range than was observed for any of the individual media. This is because the options considered in these tables represent the broadest range of standards considered in this risk assessment.

Over this range of standards, the proportion of children expected to have PbB exceeding 25 μg/dL ranged from 0.32 to 0.14 percent based on the EPI model and 0.16 to 0.002 percent based on the IEUBK model. The proportion of children expected to exceed 10 μg/dL ranged from 8.3 to 5.8 percent for the EPI model and from 6.5 to 3.7 percent based on the IEUBK model. The proportion of kids expected to have IQ below70 only ranged from 0.54 to 0.50 percent based on the EPI model and 6.5 to 11 percent based on the IEUBK model.

Figures 5-5a and 5-5b display the eight health endpoints presented in Table 5-10 in graphical form in the same format as Figures 5-3a and 5-3b. There are two curves in each graph representing the predictions from the EPI and IEUBK models, and the baseline level of each endpoint (as determined from NHANES III) is drawn as a reference line for comparison on each graph. On each curve, a diamond is overlaid to represent the health effects that would be projected if the interim standards were used for §403.

The incremental improvement in aggregate health effects per house affected can be judged by the slope of the curve between two points on each of the graphs. The slope is steepest on the left side of each of these graphs, between the first, second, and third sets of standards. This property was generally present in the graphs illustrating the effects of changes in standards for the individual media. However, allowing each of the standards to vary from the greatest option considered to the lowest, these graphs illustrate that greater benefits are achievable than those reflected in the graphs for the individual media.

Table 5-10. Characterization of Impact of Various Sets of Dust, Soil, and Paint Standards.

STANDARDS							Current Interim Guidance
Floor Dust Lead Loading ($\mu g/ft^2$)	400	400	200	100	50	25	100
Window Sill Dust Lead Loading ($\mu g/ft^2$)	800	500	500	200	100	25	500
Soil Cover ($\mu g/g$)	1500	800	400	200	100	50	400
Soil Removal ($\mu g/g$)	5000	4000	3000	3000	2000	1000	5000
Paint Maintenance (ft^2 damaged LBP)	10	10	5	2	1	0	2
Paint Abatement (ft^2 damaged LBP)	100	40	20	10	10	5	10
PERCENTAGE OF HOMES EXCEEDING STANDARDS							
Floor Dust	0.297	0.297	1.98	8.94	16.7	33.4	8.94
Window Sill Dust	10.6	14.1	14.1	26.9	37.0	54.6	14.1
Soil Cover	3.27	8.11	12.8	20.1	27.1	42.8	13.4
Soil Removal	0.215	0.746	0.746	0.746	2.71	6.14	0.215
Interior Paint Maintenance	2.80	2.27	2.92	2.22	2.75	1.08	2.22
Exterior Paint Maintenance	3.84	2.41	3.49	3.09	3.22	1.15	3.09
Interior Paint Abatement	0.453	0.980	2.43	3.25	3.25	5.35	3.25
Exterior Paint Abatement	3.03	4.46	5.77	6.87	6.87	9.26	6.87
Percentage of Homes Exceeding Any Standard	18.6	22.0	26.1	38.3	52.9	74.2	28.6
HEALTH EFFECTS PROJECTED BY EPI MODEL							
PbB>25 (%)	0.32	0.27	0.25	0.20	0.17	0.14	0.24
PbB>10 (%)	8.3	7.8	7.5	6.9	6.3	5.8	7.4
IQ<70 (%)	0.54	0.53	0.53	0.52	0.51	0.50	0.52
IQ decrement>1 (%)	50	49	49	48	47	45	49
IQ decrement>2 (%)	15	15	14	13	13	12	14
IQ decrement>3 (%)	5.3	4.9	4.7	4.3	3.9	3.5	4.7
Avg. IQ decrement	1.26	1.24	1.23	1.20	1.17	1.15	1.22
SD of IQ decrement	0.97	0.94	0.92	0.89	0.85	0.83	0.92
HEALTH EFFECTS PROJECTED BY IEUBK MODEL							
PbB>25 (%)	0.16	0.081	0.031	0.018	0.0052	0.0017	0.035
PbB>10 (%)	6.5	5.1	3.6	2.9	1.8	1.1	3.7
IQ<70 (%)	0.51	0.50	0.48	0.47	0.46	0.45	0.48
IQ decrement>1 (%)	49	47	45	43	40	36	45
IQ decrement>2 (%)	13	11	8.8	7.5	5.4	3.9	9.0
IQ decrement>3 (%)	4.0	2.9	1.9	1.5	0.83	0.48	2.0
Avg. IQ decrement	1.20	1.15	1.09	1.06	1.00	0.95	1.10
SD of IQ decrement	0.85	0.76	0.67	0.63	0.55	0.50	0.68

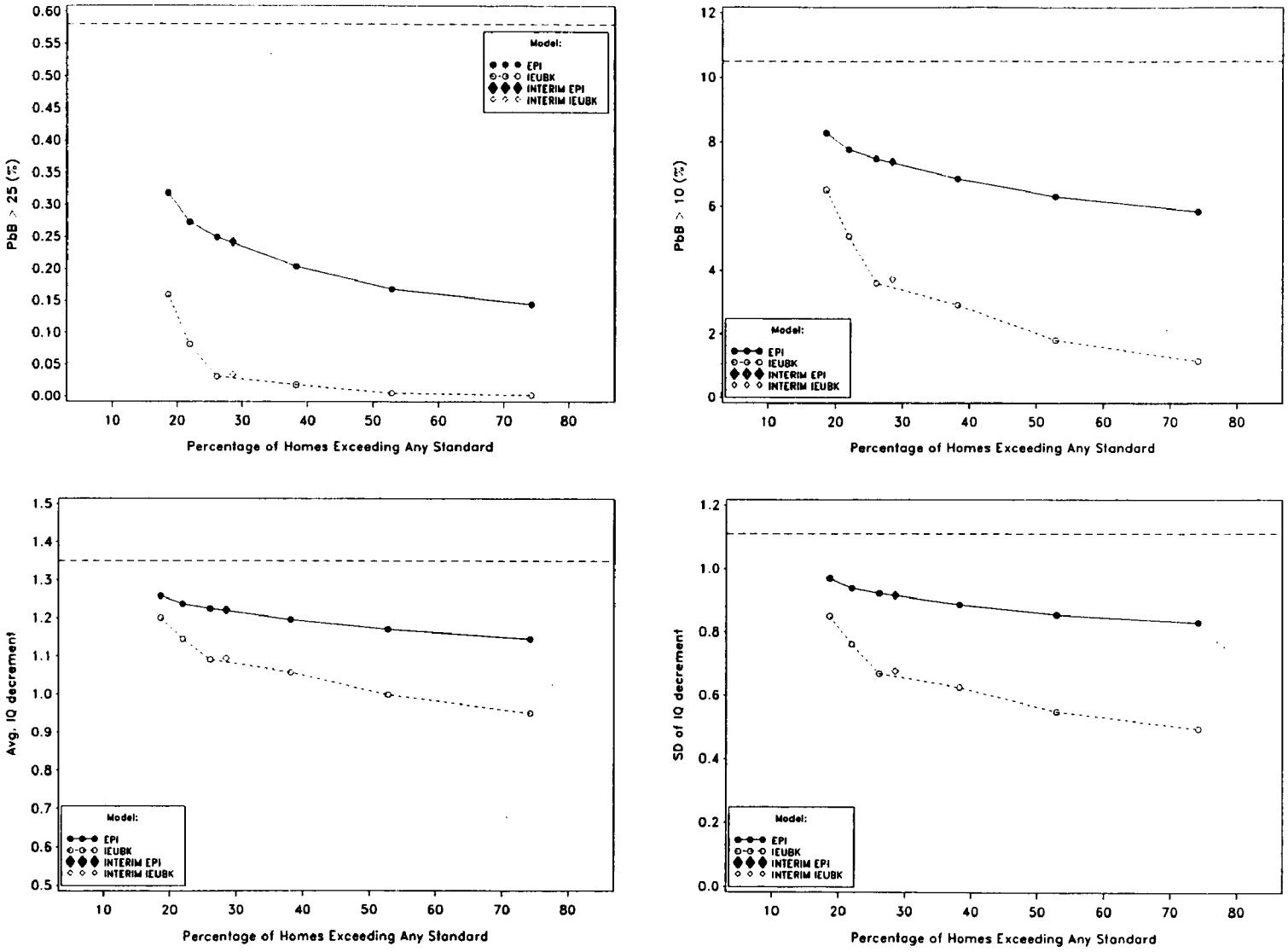


Figure 5-6a. Projected Health and Blood-Lead Endpoints Based on Various Sets of Options for Dust, Soil, and Paint, Part 1. (Dashed reference line represents baseline risk.)

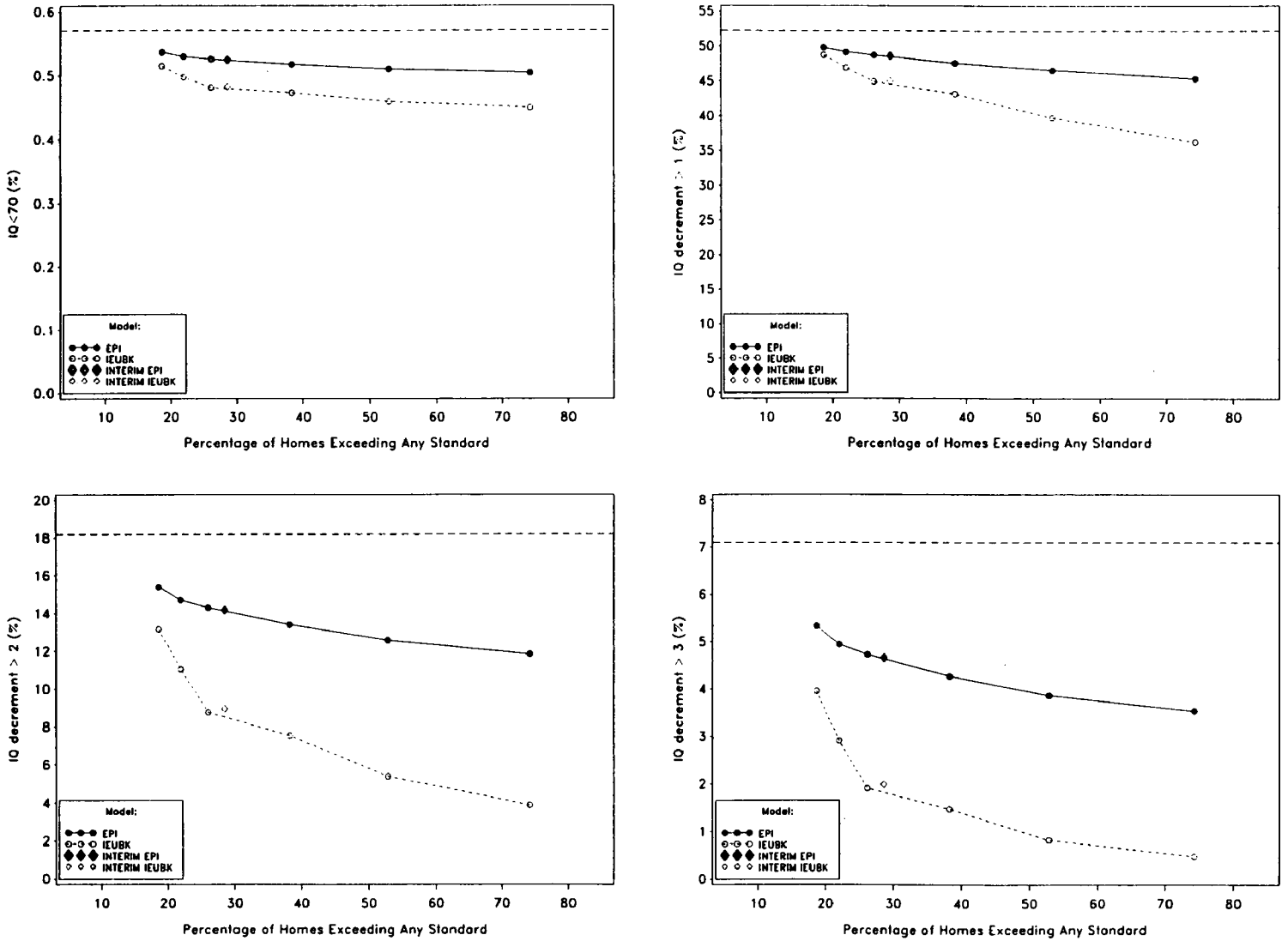


Figure 5-6b. Projected Health Endpoints Based on Various Sets of Options for Dust, Soil, and Paint, Part 2. (Dashed reference line represents baseline risk.)

There is, again, a generally consistent shape of each of the curves in these figures. In each case, the steepest drop occurs between the three least-stringent sets of standards, the next four points follow almost a straight line, and then a gradual reduction in the slope of each curve. This pattern is consistent between the EPI and IEUBK models. In each case, the projected health endpoints associated with the interim standards (black and white diamonds in the graphs) appear just to the right of the point representing the third set of standards, affecting about 29 percent of the homes. The estimated health and blood-lead concentration effects for the interim standards are always slightly above the line connecting the third and fourth set of options considered. This implies that slightly larger benefits might be achieved without affecting more houses if standards slightly different from the interim guidelines were employed.

Similarly to the results for the individual media standards, each of the sets of standards considered would result in a substantial improvement relative to the baseline for the percentage of children exceeding 25 μg/dL and 10 μg/dL, and the percentage of children anticipated to have an IQ decrement of at least 2 or 3 resulting from elevated blood-lead concentration. Even by varying all standards, there is little reduction in the percentage of children predicted to have IQ below 70 due to elevated blood-lead concentration or in the percentage of children expected to have IQ decrement greater than 1 due to elevated blood-lead concentration over the range of standards considered.

In general, the IEUBK model predicts a larger impact of Section 403 on health effects and children's blood-lead concentrations than the EPI model. This is evident, first, in that it predicts a greater reduction from the baseline. For instance, for incidence of PbB>25 μg/dL, the EPI model predicts 0.32 percent for the first set of standards, whereas the IEUBK model predicts 0.16. Both of these are compared to a baseline prediction of 0.58 percent. Second, the IEUBK generally predicts a greater reduction in health effects over the range of standards. For example, for the percentage of children exceeding 25 μg/dL, the drop was

56 percent $\left(= \frac{0.32 - 0.14}{0.32} \right)$ over the range of standards for the EPI model, and the drop was

99 percent $\left(= \frac{0.16 - 0.002}{0.16} \right)$ over the range of standards for the IEUBK model. This pattern persists across all ranges of standards considered in this risk assessment.

5.3.2 Detailed Characterization for a Particular Set of Standards

This section provides a more detailed characterization of projected blood-lead concentrations and health effects associated with a particular option for the §403 standards for dust, soil, and paint. These particular levels of the standards were chosen as "central" values, not to promote a single set of standards as optimal. Those standards are 200 $\mu g/ft^2$ for dust-lead loading, 500 $\mu g/ft^2$ for window sill dust lead loading, 400 $\mu g/g$ for soil cover, 3000 $\mu g/g$ for soil removal, 5 ft^2 damaged LBP for paint repair, and 20 ft^2 damaged LBP for paint abatement.

Figure 5-7 displays the projected post-§403 distribution of blood-lead levels based on the EPI model and the IEUBK model in both histogram and cumulative distribution function (cdf) format. The former allows the reader to clearly understand the general shape of the distribution. The cdf displays the probability that a child has blood-lead level below a specified value. This also enables the reader to infer the proportion of children projected to have blood-lead concentrations within a particular interval.

Qualitatively, the curve associated with the IEUBK-predicted, post-intervention blood-lead levels appears slightly to the left of the corresponding EPI curve, which is slightly to the left of the baseline curve determined from NHANES III. This means that the decrease in blood-lead concentrations predicted by the IEUBK model is greater than the decrease predicted by the EPI model.

Table 5-11 compares blood-lead concentrations and health effects as reported in NHANES III and those projected post-§403 based on both the EPI model and the IEUBK model for the third set of standards assessed in Section 5.3.2: 200 $\mu g/ft^2$ for dust-lead loading, 500 $\mu g/ft^2$ for window sill dust-lead loading, 400 $\mu g/g$ for soil cover, 3000 $\mu g/g$ for soil removal, 5 ft^2 damaged LBP for paint repair, and 20 ft^2 damaged LBP for paint abatement. The top half of the table characterizes the distribution of children's blood-lead concentrations.

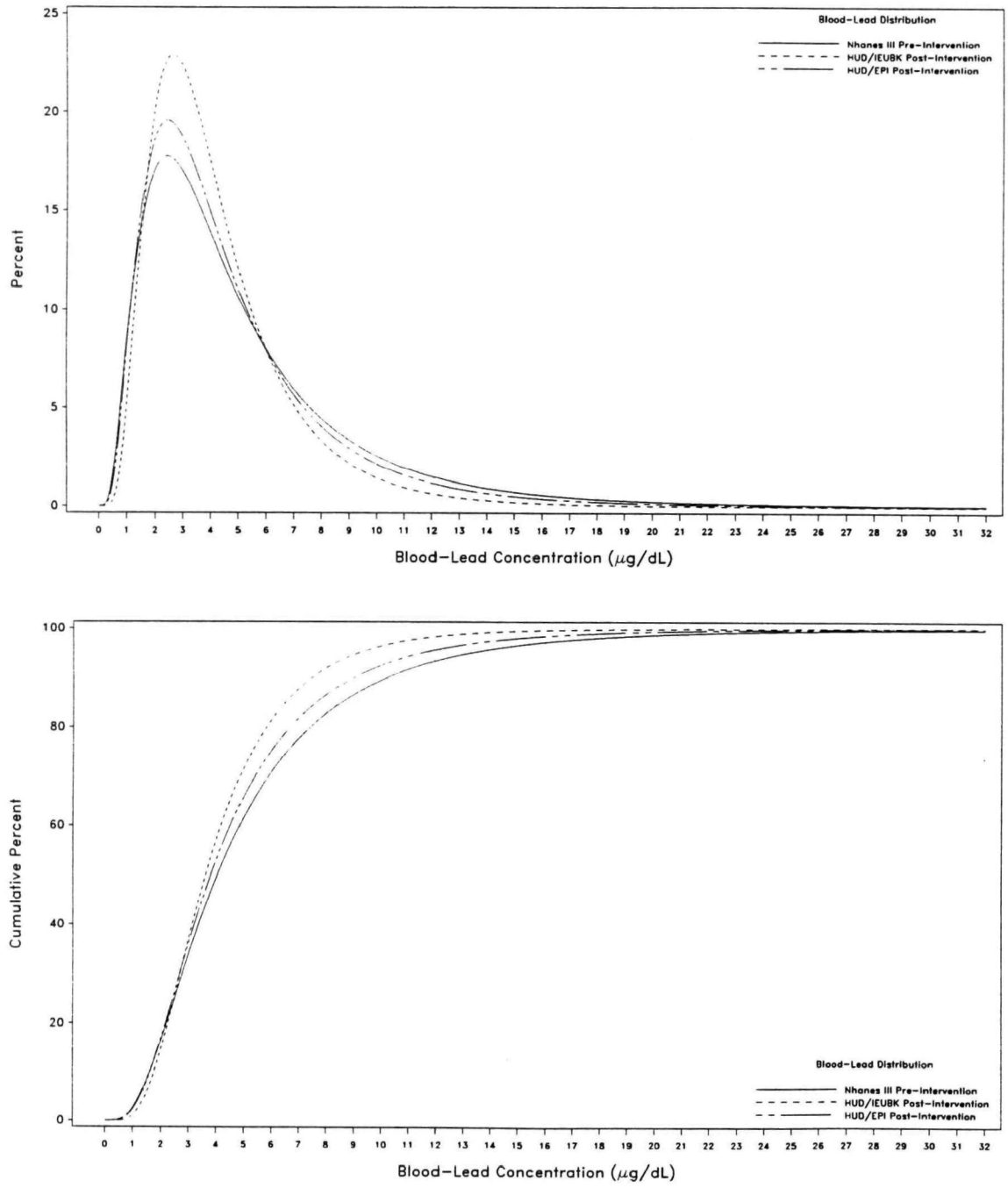


Figure 5-7. Projected Post-§403 Blood-Lead Concentration Distributions Based on EPI and IEUBK Models at Standards of Floor Dust-Lead –200 μg/ft²; Window Sill Dust-Lead – 500 μg/ft²; Soil Cover – 400 μg/g; Soil Removal – 3000 μg/g; Paint Maintenance – 5 ft²; Damaged LBP, and Paint Abatement – 20 ft² Damaged LBP

Contained are the estimated numbers and proportions of children with blood-lead concentration in various intervals. The bottom half of the table displays the various health endpoints discussed thus far in this risk assessment for the baseline and post-§403 projections based on this set of standards.

Table 5-11. Comparison of Blood-Lead Concentrations Before and After §403.

	NHANES III		EPI Model[1]		IEUBK Model[2]	
PbB (μg/dL)	# Children[3]	Percent	# Children	Percent	# Children	Percent
Total	7,961,000	100	7,961,000	100	7,961,000	100
[0,1)	209,000	2.6	183,000	2.3	90,000	1.1
[1,3)	2,490,000	31	2,689,000	34	2,850,000	36
[3,5)	2,200,000	28	2,363,000	30	2,760,000	35
[5,10)	2,227,000	28	2,129,000	27	1,974,000	25
[10,15)	559,000	7.0	434,000	5.5	240,000	3.0
[15,20)	169,000	2.1	109,000	1.4	37,000	0.5
[20,25)	60,000	0.8	33,000	0.4	7,000	0.1
≥25	46,000	0.6	20,000	0.3	2,000	0.03
Inferred Health Effects						
IQ < 70	45,000	0.6	42,000	0.5	38,313	0.5
IQ decrement >1	4,153,000	52	3,879,000	49	3,577,914	45
IQ decrement >2	1,451,000	18	1,140,000	14	698,810	8.8
IQ decrement >3	564,000	7.1	377,000	4.7	152,099	1.9
Average IQ decrement	1.35		1.23		1.09	
S.D. of IQ decrements	1.11		0.92		0.67	
	# Houses	Percent	# Houses	Percent	# Houses	Percent
Houses Affected	0	0	26,210,000	26	26,210,000	26

[1]Predicted distribution of blood-lead concentration following the rule-making for standards of 200 μg/ft² for dust lead loading, 500 μg/ft² for window sill dust-lead loading, 400 μg/g for soil cover, 3000 μg/g for soil removal, 5 ft² damaged LBP for paint repair, and 20 ft² damaged LBP for paint abatement.

[2]Predicted distribution of blood-lead concentration and health effects following the rule-making for standards of 200 μg/ft² for dust lead loading, 500 μg/ft² for window sill dust-lead loading, 400 μg/g for soil cover, 3000 μg/g for soil removal, 5 ft² damaged LBP for paint repair, and 20 ft² damaged LBP for paint abatement.

[3]Numbers of children in thousands

5.4 SENSITIVITY AND UNCERTAINTY ANALYSES

The results presented in this risk assessment are dependent on a number of factors, including the various assumptions and data analysis approaches taken, the outcomes of supporting data analyses, and the availability of sufficient data. Sensitivity analyses address the extent to which variations in key assumptions and approaches affect the outcome of the risk assessment. These variations are associated with overall uncertainty. Thus, sensitivity analysis evaluates how sensitive the results and conclusions of the risk assessment are to the uncertainty present in the analysis.

There are numerous procedures and assumptions discussed and presented in Chapters 3 through 5 that contribute to the final results of the risk assessment. As it was not feasible to consider variations in all aspects of the risk assessment data analysis, the sensitivity analysis considered approaches and assumptions which had the potential for producing the largest expected deviation from the final results. The alternative approaches considered in the sensitivity analysis and the comparison of their findings with the final results had to be manageable within the context of the sensitivity analysis. Table 5-12 summarizes the six factors addressed by the sensitivity analysis and the alternative approach(es) considered for each factor.

5.4.1 Components of the Sensitivity Analysis

This subsection discusses each component of the sensitivity analysis portrayed in Table 5-12. Justification for each alternative approach considered in the analysis is provided, and reasons for not including certain factors of the risk assessment in the sensitivity analysis are discussed.

One aspect of sensitivity analysis on predicting post-intervention blood-lead concentrations from environmental-lead levels does not appear within Table 5-12, as it has been incorporated directly within the risk assessment. Two models were used to predict post-intervention blood-lead concentration: the IEUBK model and the EPI model. As these two models represent different approaches for predicting blood-lead concentration from environmental-lead levels, their results can be compared as part of a sensitivity analysis. In

addition, the use of two models led to the decision to not consider modifying parameter estimates or model forms within either the IEUBK or EPI models in the sensitivity analysis.

Table 5-12. Procedures and Their Alternatives That Were Included in the Sensitivity Analysis

Procedure	Approach Taken in the Risk Assessment	Alternative(s) Considered in the Sensitivity Analysis
Determine an appropriate age group of children to consider for risk assessment	Age group = 1 to 2 years (i.e., 12 to 35 months)	Age group = 1 to 5 years (i.e., 12 to 71 months)
Determine an average IQ point loss associated with every 1 microgram of lead per deciliter of blood in children	Average IQ point loss = 0.257.	Alt. #1: Average IQ point loss = 0.185 Alt. #2: Average IQ point loss = 0.323
Determine a baseline (pre-intervention) distribution of blood-lead concentrations from NHANES III data	Assume lognormal distribution (See Section 5.1)	Use the empirical distribution reported in the NHANES III without any type of modeling.
Convert Blue Nozzle vacuum dust-lead loadings reported in the National Survey to wipe dust-lead loadings, so their area-weighted geometric mean can be compared to §403 environmental-lead standards	Convert each sample result using the following formulas: Floors: Wipe = $11.4*(Vac)^{0.690}$ Window Sills: Wipe = $5.79*(Vac)^{1.079}$ where "Wipe" is the estimated wipe dust-lead loading and "Vac" is the measured vacuum dust-lead loading (see Section 4.2)	Alt. #1 (low estimate): Assign the lower 90% confidence bound on the estimated wipe dust-lead loading obtained from the adjacent formula to each sample result. Alt. #2 (high estimate): Assign the upper 90% confidence bound on the estimated wipe dust-lead loading obtained from the adjacent formula to each sample result.
Determine a post-§403 blood-lead concentration distribution as a function of post-intervention environmental-lead levels (Section 5.2)	Consider post-intervention environmental-lead levels summarized in Table 5-3 of Section 5.2.	Consider the following alternative post-intervention environmental-lead levels: – 20 $\mu g/ft^2$ for floor dust-lead loading and 50 $\mu g/ft^2$ for window sill dust-lead loading – 100 $\mu g/ft^2$ for floor dust-lead loading and 250 $\mu g/ft^2$ for window sill dust-lead loading – 20% and 80% decline in pre-intervention soil-lead concentration when soil cover is performed.
Determine a method for characterizing the post-§403 distribution of blood-lead concentration, and comparing health effects between pre- and post-§403.	Apply the methods in Section 5.3 to obtain pre- and post-intervention distributions.	Alt. #1: Apply the alternative method detailed in Approach #1 in Section 5.4.1.6. Alt. #2: Rather than predicting post-§403 blood-lead concentration as a function of environmental-lead levels, conduct the prediction based on efficacy seen in abatement studies with an adjustment for bone-lead stores.

The sensitivity analysis does not consider other options for obtaining estimated numbers of housing units in the 1997 housing stock or numbers of children residing in the housing stock (presented in Chapter 3). In preliminary analyses, it was observed that regardless of the method used to obtain an estimated number of units (or children) within the four categories determined by housing age, the percentage of the total housing stock (or the total population of children) within each group remained relatively constant. Therefore, it was not deemed necessary to consider alternative methods for determining numbers of housing units or children.

Note that the sensitivity analysis does not address various options for the §403 standards. Results to be used in evaluating these options are included within the risk assessment presented in Section 5.3.

5.4.1.1 Alternative Age Range of Children

For reasons discussed in Section 2.4, the §403 risk assessment characterized lead exposures and health effects for children aged 12-35 months (i.e., 1-2 years). However, as the interventions that result from §403 are expected to benefit young children of other ages as well, the sensitivity analysis calculated health effects associated with children in a broader age range: children aged 12-71 months (i.e., 1-5 years). This alternative age range is considered as Title X has defined target housing as housing built prior to 1978 in which children less than six years of age may reside. Broadening the age range to include older children will likely result in an emphasis on lower blood-lead concentrations in the overall distribution.

5.4.1.2 Alternative Assumptions on Average IQ Score Decline Per Unit Increase in Blood-Lead Concentration

As discussed in Chapter 4, results of the meta-analysis documented in Schwartz (1994) indicate that an average IQ point loss of 0.257 is predicted for every 1.0 μg/dL increase in blood-lead concentration. This relationship was used in Sections 5.1 and 5.3 to characterize health effects associated with elevated blood-lead concentration. In the sensitivity analysis, two alternative average IQ point loss estimates were considered when calculating pre-intervention reduction in IQ points associated with blood-lead concentration: 0.185 and 0.323.

The lower value of 0.185 was selected based on findings of a prospective study of blood-lead concentration for children approximately two years of age, as reported in Pocock et al. (1994). The higher value of 0.323 corresponds to an examination in Schwartz (1994) on the existence of a threshold in the relationship between IQ score and blood-lead concentration. In studies that involve primarily children with blood-lead concentrations of 15 μg/dL or lower, the estimated average IQ point loss was reported to be 0.323. The estimates of 0.185 and 0.323 result in a lower and higher estimate, respectively, of the benefits associated with §403. The sensitivity analysis did not consider alternative methods for estimating other health effect endpoints, such as the probability of observing IQ scores less than 70 or the probability of observing elevated blood-lead concentrations.

5.4.1.3 Alternative Approach to Characterizing a Baseline Blood-Lead Distribution from NHANES III Data

As discussed in Section 5.1, the baseline, pre-§403 distribution of blood-lead concentrations in children aged 12 to 35 months was assumed to be lognormal, with geometric mean and standard deviation calculated from NHANES III for this age group. Health endpoints were then calculated from this distribution. An alternative approach to characterizing the baseline distribution using the NHANES III data considered an empirical distribution. This alternative approach was applied in the sensitivity analysis.

The NHANES III database contained blood-lead concentrations for 924 children aged 12-35 months at the time of their survey interview. Each child in the survey was assigned a sampling weight corresponding to the number of children in the country being represented by the child. This combination of blood-lead concentration and sample weight for each surveyed child provided an empirical distribution of blood-lead concentration for children aged 12-35 months. Percentiles, such as the probability of observing a blood-lead concentration less than 10 μg/dL, were calculated from this distribution by summing the sample weights for children with blood-lead concentrations less than 10 μg/dL, then dividing by the total of all sampling weights. Percentiles were used to obtain the probability of elevated blood-lead concentration and the probability of observing a specific decrement in IQ score. The probability of observing an IQ score less than 70 was calculated for each surveyed child based on his/her blood-lead

concentration (see Section 4.4), then was multiplied by the child's sample weight and summed across children to obtain an expected number (and percentage) of children in the nation with IQ less than 70.

Providing an alternative approach to characterizing the pre-§403 blood-lead concentration distribution and the resulting health effects provided a means of evaluating the lognormality assumption placed on this distribution.

5.4.1.4 Uncertainty in Converting Dust-Lead Loadings for Comparison to Standards

Because the §403 dust-lead standards will be defined in terms of a lead loading of a wipe sample, and because dust samples in the HUD National Survey were collected using a Blue Nozzle vacuum, methods in Section 4.2 were used to convert the National Survey dust-lead loadings (for both floors and window sills) to wipe dust-lead loadings. In the risk assessment, two formulas were used (Table 5-12) to predict a wipe dust-lead loading from a Blue Nozzle vacuum dust-lead loading, depending on whether a floor or window sill was sampled. These formulas indicate that the expected value of the log-transformed wipe dust-lead loading (log(Wipe)) takes the form

$$\alpha + \beta * \log(\text{Vac})$$

where α and β are parameter estimates. Therefore, assuming lognormality, upper and lower one-sided 90% confidence bounds on the expected value of log(Wipe) are

$$\log(\text{Wipe}) \pm 1.3 * \text{SE}(\alpha + \beta * \log(\text{Vac}))$$

where $\text{SE}(\alpha + \beta * \log(\text{Vac}))$ is the standard error of the predicted average wipe dust-lead loading of a vacuum sample with a dust-lead loading of Vac. Upper and lower 90% confidence bounds on the untransformed wipe dust-lead loadings are obtained by exponentiating the bounds for the log-transformed loading.

The confidence bounds were used to define two alternative sets of converted dust-lead loadings in the sensitivity analysis:

Alternative set #1: Wipe dust-lead loading equals the lower 90% confidence bound on the expected value of wipe dust-lead loading

Alternative set #2: Wipe dust-lead loading equals the upper 90% confidence bound on the expected value of wipe dust-lead loading

Note that alternative set #1 is a low estimate of the converted loading value, while alternative set #2 is a high estimate. Under both sets, area-weighted arithmetic mean dust-lead loadings for both floors and window sills were calculated for each National Survey unit. The means were used to determine whether dust-lead loading standards were exceeded for a given unit. In the sensitivity analysis, numbers and percentages of units exceeding various combinations of environmental-lead standards were calculated under each set of converted dust-lead loadings.

5.4.1.5 Alternative Assumptions on Post-Intervention Environmental-Lead Levels

Estimates of expected post-intervention environmental-lead levels were provided in Table 5-3. The sensitivity analysis considered alternatives to the post-intervention dust-lead loading following dust cleaning, interior LBP intervention, or soil removal; as well as in the soil-lead concentration following soil cover, in order to observe how the health effect estimates were affected by assumptions on post-intervention environmental-lead levels. Two sets of alternative post-intervention dust-lead loadings for floors and window sills were considered as a result of dust cleaning:

- 20 $\mu g/ft^2$ for floors and 50 $\mu g/ft^2$ for window sills, and
- 100 $\mu g/ft^2$ for floors and 250 $\mu g/ft^2$ for window sills.

(The loadings used in the risk assessment were 40 $\mu g/ft^2$ for floors and 100 $\mu g/ft^2$ for window sills.) As post-intervention soil-lead concentration following soil cover was assumed to be 50% of the pre-intervention concentration in the risk assessment, the sensitivity analysis considered a low alternative value of 20% and a high alternative value of 80%. The sensitivity analysis did not address alternative soil-lead concentration values following soil removal (150 $\mu g/g$), or amounts of deteriorated lead-based paint following paint interventions (0 ft^2). The

same approaches to determining post-§403 blood-lead distributions using either the IEUBK model or the EPI model were repeated in this analysis.

5.4.1.6 Alternative Methods to Observing Differences in Health Effects Between Pre- and Post-Intervention

As presented in Section 5.3 and Appendix E2, the method for characterizing a post-§403 distribution of blood-lead concentration used in the risk assessment effort involved the following: 1) obtain a predicted distribution for both pre- and post-§403 by applying either the IEUBK or the EPI model to environmental-lead levels; 2) calculate the ratio of the post-§403 geometric mean to the pre-§403 geometric mean; 3) multiply the ratio by the geometric mean observed for the pre-§403 distribution obtained from NHANES III data, resulting in an estimated geometric mean for the post-§403 distribution; and 4) assume that the distribution is lognormal. This method yielded a post-§403 distribution that was directly comparable with the pre-§403 distribution. Two alternative approaches to obtaining a post-§403 blood-lead concentration distribution were considered in the sensitivity analysis.

Approach #1: Alternative approach to obtaining comparable pre- and post-§403 blood-lead distributions

In the first alternative approach, two predicted distributions were obtained (one for pre-§403 one for post-§403) simply by applying the same model (IEUBK or the EPI model) to either pre- or post-§403 environmental-lead levels. This approach is step #1 in the preceding paragraph. Note, that the resulting distributions are purely model-based; the NHANES III data are not used as a basis for characterizing the distributions. In the sensitivity analysis, the health endpoints were calculated using the risk assessment approach directly on the model-based blood-lead distributions.

Approach #2: Alternative approach to determining a post-intervention blood-lead distribution using directly-measured blood lead changes

A second approach to characterizing the post-§403 blood-lead concentration distribution was performed utilizing published results on the effectiveness of lead hazard

intervention strategies among children exposed to residential lead hazards. This approach is desirable since blood-lead concentrations are a more direct measure of intervention effectiveness than environmental lead levels. The scientific literature reports the results of a range of non-medical intervention strategies conducted to reduce the lead exposure of children residing at the targeted residences (EPA, 1995b). The strategies studied included lead-based paint abatement, interior dust abatement via routine cleaning procedures, elevated soil lead abatement, and intensive educational efforts (EPA, 1995b). The effectiveness of these strategies as measured by declines in children's blood-lead concentrations may be used to estimate the post-§403 blood-lead concentration distribution. As such, this approach represents a somewhat independent (of many of the procedures and data used for the risk assessment) estimation of the post-§403 distribution.

As summarized in the EPA technical report, "Review of Studies Addressing Lead Abatement Effectiveness," the intervention strategies reported 18–34% declines in the blood-lead concentrations of exposed children six to twelve months following the conduct of the intervention (EPA, 1995b). Lead-based paint abatement (of all deteriorated LBP), biweekly dust abatement (of areas with elevated dust lead), soil abatement (removal and replacement of top 6"), and intensive education (visit by semi-professional outreach worker) reported comparable declines of approximately 25% one year following conduct of the intervention (EPA, 1995b). Each of these four intervention studies reported significantly greater declines among the study population than among a suitable control population—no control population was studied for the educational intervention associated with the 34% decline—providing reassurance that the interventions themselves were responsible for much of the reported declines. For the purpose of this sensitivity analysis, therefore, the average decline in children's blood-lead concentration resulting from an intervention was taken to be 25%[4].

This degree of effectiveness may not be suitable for estimating the post-§403 blood-lead distribution since the reported declines were for children already exposed (i.e., already exhibiting elevated blood-lead concentrations due to exposure to the targeted lead source). By

[4] In all four studies, the control population did exhibit some decline which may be attributed to increased awareness of environmental lead and its hazards. As similar awareness may be expected to accompany §403 prompted interventions, it was not deemed necessary to adjust the reported study population declines by the declines associated with the control populations.

contrast, the promulgation of §403 will prompt preventive interventions (primary prevention) conducted prior to any lead exposure to resident children. Measures of secondary prevention effectiveness may not be representative of primary intervention effectiveness because lead present in blood is a combination of current environmental exposure and internal reservoirs of lead stored in bone and soft tissue (Gulson et al., 1995; Smith et al., 1996; Rabinowitz et al., 1976; Manton, 1985). The reported declines in exposed children's blood-lead concentrations, therefore, may underestimate the primary prevention effectiveness of a intervention (Gulson et al., 1995).

A methodology was developed to estimate the impact of body lead burdens on measures of secondary intervention effectiveness in order to adjust the reported secondary prevention effectiveness (see Appendix E3). For a two-year-old child (the target population of interest), it is estimated that an intervention prompting 25% declines one year following intervention among exposed children would actually prompt 33% declines were the intervention primary prevention in character. Based on this result a 33% efficacy will be utilized for the purposes of this portion of the sensitivity analysis.

As a comparison, the IEUBK model indicates a 54% primary prevention efficacy were lead-based paint hazards eliminated and dust- and soil-lead levels lowered to background levels. Specifically, the geometric mean blood-lead concentration reported by NHANES III for children 1-2 years of age (4.1 μg/dL) (Brody et al., 1994) was contrasted with the geometric mean predicted by the IEUBK model with inputted environmental lead levels at national background levels (1.9 μg/dL). Shacklette et al. reported a background national geometric mean soil-lead concentration of 20 ppm. The background dust-lead concentration corresponded to the default dust lead assumed by the IEUBK Multiple Source Analysis (dust-lead concentration = 0.70 x soil-lead concentration + 100 μg/g / μg/m^3 x air-lead concentration [air-lead concentration = 0.01 μg/m^3]). All other default values defined by the IEUBK model were used in these analyses.

It is worth noting that the scientific literature also includes two recent journal articles regarding the percentage of lead in blood that may be attributed to body lead stores (Gulson et al., 1995; Smith et al., 1996). Such results, of course, have relevance to this aspect of the sensitivity analysis. Both articles indicate that between 40-70% of an adult woman's blood

lead may be attributed to mobilized bone-lead stores. The fact that these studies examined adult women is critical because the percentage of blood lead attributable to bone-lead stores varies considerably with age (Rabinowitz, 1991). Higher percentages are associated with older individuals (Rabinowitz, 1991). Thus, the target population considered by §403 may have lower percentages of their blood lead attributable to mobilized bone lead. Greater primary prevention efficacy is reported for, say, 7 year old children than for 2 year old children (see Table 1 in Appendix E3). If the methodology used in this alternative approach were extended to adults, it would also suggest that 40-70% of blood lead is attributable to mobilized bone-lead stores.

This alternate approach to estimating a post-§403 national distribution of blood-lead concentrations for 1997 children aged 12 to 35 months (1 to 2 years) was implemented based on the estimated 33% decline in blood-lead concentration following an intervention. This alternative estimate of primary prevention effectiveness, which adjusts the blood-lead changes for body-lead stores and hereafter is denoted the 'adjusted blood lead effects model', was then compared to post-§403 distribution using the IEUBK model and the HUD National Survey data.

The methodology for this comparison is summarized as follows:

1. Environmental lead levels for each HUD National Survey unit were used as input to the IEUBK model to predict the geometric mean blood-lead concentration for children aged 1-2 years old exposed to environmental lead levels similar to that in the National Survey unit. The contribution of pica was estimated using the methodology documented in Section 4.3.

2. For each unit in the National Survey, lead levels in paint, dust, and soil were compared to the following options for standards:

    - 100 $\mu g/ft^2$ as an interior floor dust-lead loading and 500 $\mu g/ft^2$ as an interior window sill dust-lead loading; and,
    - 400 $\mu g/g$ as a soil-lead concentration for soil cover and 3000 $\mu g/g$ for soil abatement.
    - 0 ft^2 of deteriorated lead-based paint;

    If environmental lead levels exceeded the standard for at least one media, then an intervention will be conducted in the unit.

3. For each National Survey unit, if an intervention was triggered then the geometric mean blood-lead concentration for the post-§403 result was set equal to 67% of the geometric mean computed in (1). If an intervention was not triggered then the geometric mean blood-lead concentration for the post-§403 result equaled the geometric mean calculated in (1).

4. The geometric mean blood-lead concentration and the geometric standard deviation of 1.6 μg/dL were used to generate a frequency distribution of blood-lead concentrations for each unit in the National Survey. The frequency distributions were then combined over all of the National Survey units to yield the HUD/IEUBK post-§403 blood-lead distribution. The details for generating the frequency distribution of blood-lead concentration at each unit and over all units in the National Survey are presented in Appendix E1.

5.4.2 Results of the Sensitivity Analysis

The six factors considered in the sensitivity analysis and presented in Table 5-12 address various segments of the risk assessment effort. The results of the sensitivity analysis are presented in this subsection and are grouped according to these segments.

5.4.2.1 Alternative Age Range of Children

Table 5-1 of Section 5.1 presented baseline estimates (pre-§403) of numbers and percentages of children in the U.S. aged 12-35 months in 1997 who exhibited the health effects of interest. Tables 5-13a and 5-13b present these estimates, along with estimates for the age group 12-71 months (i.e., 1-5 years). Table 5-13a presents estimated percentages of children with elevated blood-lead levels, IQ less than 70, and specified decrements in IQ score. The percentages for the 12-71 month age group are approximately 15%-25% lower than those for the 12-35 month age group. For example, Table 5-13a indicates that the expected percentage of children aged 12-35 months having blood-lead concentration of at least 10 μg/dL is 10.5%, compared to approximately 8% for children aged 12-71 months. Table 5-13b presented estimated average IQ score loss; similar declines are observed here. The declines are the result of lower blood-lead concentrations introduced to the distribution by including older children.

Table 5-13a. Estimated Baseline[1] Number and Percentage of Children Having Specific Health Effects, for Two Age Groups of Children and Under Three Assumptions on Average Decline in IQ Score per Unit Increase in Blood-Lead Concentration

Health Effect[2]		Children Aged 12-35 Months Having the Given Health Effect		Children Aged 12-71 Months Having the Given Health Effect	
		Number (millions)	Percentage	Number (millions)	Percentage
Blood-lead concentration of at least 10 μg/dL		0.83	10.5	1.64	8.04
Blood-lead concentration of at least 25 μg/dL		0.046	0.578	0.082	0.403
IQ score less than 70		0.045	0.566	0.11	0.531
IQ score decrement of greater than 1	0.185 decline/ 1μg/dL increase	2.74	34.4	5.84	28.6
	0.257 decline/ 1μg/dL increase	4.15	52.2	9.25	45.3
	0.323 decline/ 1μg/dL increase	5.13	64.5	11.77	57.7
IQ score decrement of greater than 2	0.185 decline/ 1μg/dL increase	0.69	8.65	1.34	6.57
	0.257 decline/ 1μg/dL increase	1.45	18.2	2.95	14.4
	0.323 decline/ 1μg/dL increase	2.21	27.8	4.63	22.7
IQ score decrement of greater than 3	0.185 decline/ 1μg/dL increase	0.22	2.71	0.40	1.97
	0.257 decline/ 1μg/dL increase	0.56	7.09	1.09	5.34
	0.323 decline/ 1μg/dL increase	0.99	12.5	1.97	9.65

[1] "Baseline" refers to projected 1997 conditions prior to implementing any interventions under Section 403 rules. For a given age group, the baseline blood-lead distribution used to determine health effects was characterized using methods in Section 5.1.

[2] For IQ score decrement, this column also includes the assumption on average IQ score decline per 1 μg/dL increase in blood-lead concentration.

Shaded cells correspond to results that were presented in Table 5-1.

Table 5-13b. Estimated Baseline[1] Average (and Standard Deviation) IQ Loss, for Two Age Groups of Children and Under Three Assumptions on Average Decline in IQ Score per Unit Increase in Blood-Lead Concentration

Assumption on Average IQ Score Decline per 1.0 μg/dL Increase in Blood-Lead Concentration	Average IQ Loss (Standard Deviation)	
	Children Aged 12-35 Months	Children Aged 12-71 Months
0.185	0.97 (0.80)	0.87 (0.73)
0.257	1.35 (1.11)	1.20 (1.02)
0.323	1.70 (1.40)	1.51 (1.28)

[1] "Baseline" refers to projected 1997 conditions prior to implementing any interventions under Section 403 rules. For a given age group, the baseline blood-lead distribution used to determine health effects was characterized using methods in Section 5.1.

Shaded cell corresponds to results that were presented in Table 5-1.

5.4.2.2 Alternative Assumptions on Average IQ Score Decline Per Unit Increase in Blood-Lead Concentration

In Tables 5-13a and 5-13b, the percentage of children with IQ score decrements of a certain magnitude, and average IQ score loss across all children, were calculated under the assumption of a decline of 0.257 in IQ score for each 1 μg/dL increase in blood-lead concentration. The tables also include these percentages as calculated under the alternative assumptions of an average decline of 0.185 and 0.323 in IQ score (Section 5.4.1.2).

The low and high estimates for the decline in IQ score associated with a 1 μg/dL increase in blood-lead concentration has a considerable impact on the likelihood that a child will experience a specific decrement in IQ score, with this effect increasing as the decrement of interest increases. As seen in Table 5-13a, the estimated percentage of children with an IQ score decrement of greater than one, as calculated using the low estimate of IQ point loss (0.185), nearly doubles when the high estimate (0.323) is used instead (from 34% to 65%). When the decrement is greater than three, this difference is over four times the result associated with the low estimate (from 2.7% to 12.5%). In Table 5-13b, average IQ point loss increases from 0.97 to 1.70 for children aged 12-35 months, with a similar increase for children aged 12-71 months.

5.4.2.3 Alternative Approach to Characterizing a Baseline Blood-Lead Distribution from NHANES III Data

Using the empirical distribution method in Section 5.4.1.3, an alternative baseline blood-lead distribution based on NHANES III data was derived. The values of the health endpoints under this alternative distribution, as well as for the baseline distribution used in the risk assessment, are provided in Table 5-14. Between the two distributions, the values differ by small amounts (e.g., from one to ten percent). While the alternative method estimates a higher percentage of children with blood-lead concentrations exceeding 10 μg/dL (11.1% versus 10.5% for the risk assessment baseline distribution), it estimates a slightly smaller percentage of children with blood-lead concentrations exceeding 25 μg/dL (0.52% versus 0.58%). Estimated average IQ score loss is nearly identical between the two distributions.

Table 5-14. Estimated Baseline Health Effects, As Calculated Under Two Approaches to Calculating the Baseline Distribution of Blood-Lead Concentration Using NHANES III Data

Health Effect	Approach Used in the Risk Assessment	Alternative Approach
Percent with blood-lead concentration of at least 10 μg/dL	10.5	11.1
Percent with blood-lead concentration of at least 25 μg/dL	0.58	0.52
Percent with an IQ score less than 70	0.566	0.56
Percent with an IQ score decrement of greater than 1	52.2	51.1
Percent with an IQ score decrement of greater than 2	18.2	17.9
Percent with an IQ score decrement of greater than 3	7.09	7.96
Average IQ Loss (Standard Deviation)	1.35 (1.11)	1.35 (1.12)

5.4.2.4 Uncertainty in Converting Dust-Lead Loadings for Comparison to Standards

As discussed in Section 5.4.1.4, the dust-lead loadings (both floors and window sills) measured in the HUD National Survey were converted to wipe equivalent dust-lead loadings. In the sensitivity analysis, two alternative sets of converted dust-lead loadings were considered for the dust samples:

Alternative set #1: Wipe dust-lead loading equals the lower 90% confidence bound on the predicted average value of wipe dust-lead loading

Alternative set #2: Wipe dust-lead loading equals the upper 90% confidence bound on the predicted average value of wipe dust-lead loading

Table 5-15 contains estimates of the total number of 1997 housing units which exceed various environmental-lead standards, given that the wipe-converted dust-lead loadings for the units were determined based on the risk assessment method, alternative set #1, or alternative set #2. Table 5-15 considered numbers of units exceeding the floor-dust standard of 200 $\mu g/ft^2$, exceeding the window sill dust standard of 500 $\mu g/ft^2$, any of these two standards, or any of the standards for dust, soil, or paint.

The largest variation between the two alternative sets of dust-lead loadings occurred when considering only the floor-dust standard. When a high conversion value is used for each dust-lead loading, over four million units fail the floor-dust standard, compared to nearly two million units under the risk assessment conversion, and 1.5 million units under the low conversion values. This finding implies that the risk assessment may be underestimating the numbers of homes affected by the §403 floor dust standard by a factor of two if the average predicted wipe equivalent dust-lead loadings are being underestimated. However, a dust-cleaning intervention is triggered if either the floor or window sill dust-lead loading standard is exceeded. The impact of the uncertainty in the dust-lead loading conversion equation was much less for the number of homes affected by either the §403 floor dust or window sill dust standard. The number of units triggering an intervention because of either dust standard ranged from a low estimate of 12.7 million to a high estimate of 16.3 million, which is a range of about 30%.

Table 5-15. Number (and Percentage) of Units in the 1997 National Housing Stock Projected to Exceed Various Combinations of Environmental-Lead Standards Under Section 403 Rules, As Determined from Three Different Sets of Converted Dust-Lead Loadings

Environmental-Lead Standards Exceeded	Number (%) of Units		
	Using Risk Assessment Estimates for Converted Dust-Lead Loading[1]	Using Low Alternative Estimates for Converted Dust-Lead Loading[2]	Using High Alternative Estimates for Converted Dust-Lead Loading[2]
Floor-dust standard of 200 $\mu g/ft^2$	1,968,000 (1.98%)	1,509,000 (1.52%)	4,270,000 (4.30%)
Window sill-dust standard of 500 $\mu g/ft^2$	13,979,000 (14.1%)	11,803,000 (11.9%)	14,597,000 (14.7%)
Floor- or window sill- dust standard	15,269,000 (15.4%)	12,743,000 (12.8%)	16,275,000 (16.4%)
At least one dust, soil, or paint standard[3]	25,957,000 (26.1%)	24,918,000 (25.1%)	26,780,000 (27.0%)

[1] See Section 4.2 on the methods for performing conversions from Blue Nozzle vacuum to wipe dust-lead loadings.
[2] Low and high estimates correspond to the lower 90% confidence bound and upper 90% confidence bound, respectively, of the risk assessment estimates considered in the second column of this table.
[3] Soil and paint standards are as follows: soil-lead concentration of 400 $\mu g/g$ for soil cover, soil-lead concentrations of 3000 $\mu g/g$ for soil removal, 5 ft^2 of deteriorated lead-based paint for paint repair, and 20 ft^2 of deteriorated lead-based paint for paint removal.

5.4.2.5 Alternative Assumptions on Post-Intervention Environmental-Lead Levels

Tables 5-16a and 5-16b summarize the childhood health and blood lead effects post-§403 based on the IEUBK and EPI models, respectively, for alternative post-intervention environmental-lead levels. These tables show the impact of alternative assumptions on the efficacy of the interventions on the risk assessment. Results in these two tables were calculated assuming the following §403 standards:

- Dust-lead loading (under wipe sampling techniques) of 200 $\mu g/ft^2$ for floors and 500 $\mu g/ft^2$ for window sills
- Soil-lead concentration of 400 $\mu g/g$ for soil cover and 3000 $\mu g/g$ for soil removal
- 5 ft^2 of deteriorated lead-based paint for paint repair, and 20 ft^2 for paint removal

Table 5-16a. Estimated Percentages of Children Aged 12-35 Months Having Specific Health and Blood-Lead Effects, Based on the IEUBK Model, for Various Options for Post-Intervention Environmental-Lead Levels

	0 ft² Deteriorated Lead-Based Paint after all Paint Interventions Soil-Lead Concentration after Soil Removal Intervention = 150 μg/g								
	Dust-Lead Loading after Dust Cleaning Intervention: Floors = 20 μg/ft² Window Sills = 50 μg/ft²			Dust-Lead Loading after Dust Cleaning Intervention: Floors = 40 μg/ft² Window Sills = 100 μg/ft²			Dust-Lead Loading after Dust Cleaning Intervention: Floors = 100 μg/ft² Window Sills = 250 μg/ft²		
	Soil-Lead Conc. after Soil Cover Intervention (% of Pre-Intervention Conc.)			Soil-Lead Conc. after Soil Cover Intervention (% of Pre-Intervention Conc.)			Soil-Lead Conc. after Soil Cover Intervention (% of Pre-Intervention Conc.)		
Health Effect	20%	50%	80%	20%	50%	80%	20%	50%	80%
Percent with blood-lead concentration of at least 10 μg/dL	2.01	3.10	4.22	2.44	3.60	4.74	3.29	4.51	5.66
Percent with blood-lead concentration of at least 25 μg/dL	0.007	0.022	0.049	0.011	0.031	0.066	0.024	0.056	0.104
Percent with an IQ score less than 70	0.462	0.475	0.488	0.468	0.481	0.494	0.478	0.492	0.505
Percent with an IQ score decrement of greater than 1	40.5	43.6	45.6	42.2	44.9	46.8	44.4	46.7	48.2
Percent with an IQ score decrement of greater than 2	5.81	7.88	9.73	6.70	8.78	10.6	8.26	10.3	12.0
Percent with an IQ score decrement of greater than 3	0.940	1.60	2.32	1.19	1.91	2.68	1.71	2.51	3.31
Average IQ Loss (Standard Deviation)	1.01 (0.56)	1.07 (0.64)	1.11 (0.71)	1.04 (0.59)	1.09 (0.67)	1.14 (0.74)	1.08 (0.65)	1.13 (0.72)	1.17 (0.79)

This analysis assumes the following environmental-lead standards determine whether or not a particular intervention is performed in a housing unit:

- Dust-lead loading (under wipe techniques) of 200 μg/ft² for floors and 500 μg/ft² for window sills for dust cleaning
- Soil-lead concentration of 400 μg/g for soil cover and 3000 μg/g for soil removal
- 5 ft² of deteriorated lead-based paint for paint repair and 20 ft² for paint removal

Shaded cells correspond to results that were presented in Table 5-7.

Table 5-16b. Estimated Percentages of Children Aged 12-35 Months Having Specific Health and Blood-Lead Effects, Based on the EPI Model, for Various Options for Post-Intervention Environmental-Lead Levels

Health Effect	0 ft² Deteriorated Lead-Based Paint after all Paint Interventions; Soil-Lead Concentration after Soil Removal Intervention = 150 μg/g								
	Dust-Lead Loading after Dust Cleaning Intervention: Floors = 20 μg/ft², Window Sills = 50 μg/ft²			Dust-Lead Loading after Dust Cleaning Intervention: Floors = 40 μg/ft², Window Sills = 100 μg/ft²			Dust-Lead Loading after Dust Cleaning Intervention: Floors = 100 μg/ft², Window Sills = 250 μg/ft²		
	Soil-Lead Conc. after Soil Cover Intervention (% of Pre-Intervention Conc.)			Soil-Lead Conc. after Soil Cover Intervention (% of Pre-Intervention Conc.)			Soil-Lead Conc. after Soil Cover Intervention (% of Pre-Intervention Conc.)		
	20%	50%	80%	20%	50%	80%	20%	50%	80%
Percent with blood-lead concentration of at least 10 μg/dL	6.46	6.94	7.22	6.96	7.49	7.79	7.63	8.20	8.53
Percent with blood-lead concentration of at least 25 μg/dL	0.177	0.210	0.231	0.209	0.249	0.275	0.260	0.310	0.342
Percent with an IQ score less than 70	0.512	0.519	0.522	0.519	0.526	0.529	0.527	0.535	0.539
Percent with an IQ score decrement of greater than 1	47.0	47.8	48.2	47.9	48.7	49.1	49.0	49.8	50.2
Percent with an IQ score decrement of greater than 2	12.8	13.5	13.9	13.6	14.3	14.7	14.5	15.3	15.7
Percent with an IQ score decrement of greater than 3	3.97	4.33	4.54	4.34	4.73	4.96	4.84	5.28	5.53
Average IQ Loss (Standard Deviation)	1.18 (0.86)	1.20 (0.89)	1.21 (0.91)	1.20 (0.89)	1.23 (0.92)	1.24 (0.94)	1.23 (0.93)	1.26 (0.97)	1.27 (0.99)

This analysis assumes the following environmental-lead standards determine whether or not a particular intervention is performed in a housing unit:

- Dust-lead loading (under wipe techniques) of 200 μg/ft² for floors and 500 μg/ft² for window sills for dust cleaning
- Soil-lead concentration of 400 μg/g for soil cover and 3000 μg/g for soil removal
- 5 ft² of deteriorated lead-based paint for paint repair and 20 ft² for paint removal

Shaded cells correspond to results that were presented in Table 5-7.

The following alternative of post-intervention environmental-lead levels, in addition to the levels assumed in the risk assessment effort, were evaluated:

- two combinations of post-intervention dust-lead loadings for floors and window sills (20 $\mu g/ft^2$ for floors and 50 $\mu g/ft^2$ for window sills; and 100 $\mu g/ft^2$ for floors and 250 $\mu g/ft^2$ for window sills)
- two settings of soil-lead concentrations following soil cover (20% and 80% of pre-intervention levels).

Table 5-16a indicates that the health effects and blood-lead most affected by uncertainty in the post-intervention environmental-lead levels are those indicating the most extreme effects (e.g., IQ decrement greater than 3, blood-lead concentrations of at least 25 μg/dL). The uncertainty in the assumed efficacy of the soil cover intervention has a larger impact on the predicted health endpoints than the uncertainty in the efficacy of the dust cleaning. For example, the percentage of children predicted with an IQ decrement greater than three varies from 1.19 to 2.68% (columns 5, 6, 7) when the assumed efficacy of soil cover is varied from 20 to 80% of pre-intervention levels. On the other hand, the percentage of children predicted with an IQ decrement greater than three varies from 1.6 to 2.5% (columns 3, 6, and 9) when the assumed efficacy of the dust cleaning is varied from 20 to 100 $\mu g/ft^2$ for floor dust-lead loadings and from 50 to 250 $\mu g/ft^2$ for window sill dust-lead loadings.

A slightly different conclusion is made when considering the EPI model (Table 5-16b). The uncertainty in the assumed efficacies of the interventions has little impact on the predicted childhood health and blood-lead effects. For instance, the percentage of children predicted to have an IQ decrement greater than three varies from 4.34 to 4.96% (columns 5, 6, and 7) when the assumed efficacy of soil cover is varied from 20 to 80% of pre-intervention levels.

5.4.2.6 Alternative Methods to Observing Differences in Health Effects Between Pre- and Post-Intervention

Approach #1: Alternative approach to obtaining comparable pre- and post-intervention blood-lead concentration distributions

Section 5.4.1.6 described an alternative approach to obtaining comparable pre- and post-§403 blood-lead distributions. This approach used the same modeling techniques (using either the IEUBK or the EPI model) to obtain a geometric mean blood-lead distribution and associated geometric standard deviation for both the pre- and post-§403 distributions, and then assumed lognormality to characterize the distribution.

Table 5-17a presents estimated health and blood-lead endpoints at both pre- and post-§403 for the risk assessment approach and for this alternative approach, based on the IEUBK model. The results for the EPI model are presented in Table 5-17b. The following standards were employed in the analyses:

- Dust-lead loading of 200 $\mu g/ft^2$ for floors and 500 $\mu g/ft^2$ for window sills
- Soil-lead concentration of 400 $\mu g/g$ for soil cover and 3000 $\mu g/g$ for soil removal
- 5 ft^2 of deteriorated lead-based paint for paint repair and 20 ft^2 for paint removal

Table 5-17a shows that for the IEUBK model, the estimated reduction in health and blood-lead concentration risks for the two approaches are very similar. For example, the risk assessment approach calculated an approximate 95% drop in the incidence of blood-lead concentration exceeding 25 $\mu g/dL$, compared to an 88.5% drop under the alternative approach.

In contrast, more substantial differences between the two approaches were observed when the EPI model was used, as indicated in Table 5-17b. The estimated risk reduction is greater for the alternative approach employed in the risk assessment. However, the differences in the estimated risk reduction for the two approaches are not substantial. For instance, the reduced risk of a blood-lead concentration greater than 25 $\mu g/dL$ are 57% and 75% based on the risk assessment and alternative approaches, respectively.

Table 5-17a. Estimated Percentages of Children Aged 12-35 Months Having Specific Health and Blood-Lead Effects, Based on the IEUBK Model Under the Approach Used in the Risk Assessment and an Alternative Approach

Health Effect	Approach Used in the Risk Assessment			Alternative Approach		
	Pre-Int.	Post-Int.	% Change	Pre-Int.	Post-Int.	% Change
Percent with blood-lead concentration of at least 10 μg/dL	10.5	3.60	-65.6	12.3	5.37	-56.4
Percent with blood-lead concentration of at least 25 μg/dL	0.578	0.031	-94.6	1.08	0.124	-88.5
Percent with an IQ score less than 70	0.566	0.481	-15.0	0.598	0.498	-16.8
Percent with an IQ score decrement of greater than 1	52.2	44.9	-13.8	50.6	44.0	-13.1
Percent with an IQ score decrement of greater than 2	18.2	8.78	-51.8	19.9	11.1	-44.2
Percent with an IQ score decrement of greater than 3	7.09	1.91	-73.0	8.83	3.22	-63.5
Average IQ Loss (Standard Deviation)	1.35 (1.11)	1.09 (0.67)	-19.1	1.40 (1.33)	1.12 (0.81)	-20.1
Geometric Mean Blood-Lead Concentration (Geometric Standard Deviation)	4.05 (2.06)	3.62 (1.76)	-10.5	3.94 (2.23)	3.53 (1.91)	-10.5

This analysis assumes the following environmental-lead standards determine whether or not a particular intervention is performed in a housing unit:

- Dust-lead loading (under wipe techniques) of 200 μg/ft² for floors and 500 μg/ft² for window sills for dust cleaning
- Soil-lead concentration of 400 μg/g for soil cover and 3000 μg/g for soil removal
- 5 ft² of deteriorated lead-based paint for paint repair and 20 ft² for paint removal

Table 5-17b. Estimated Percentages of Children Aged 12-35 Months Having Specific Health Effects, Based on Blood-Lead Concentrations at Pre- and Post-Intervention as Determined from the EPI Model Under the Approach Used in the Risk Assessment and an Alternative Approach

Health Effect	Approach Used in the Risk Assessment			Alternative Approach		
	Pre-Int.	Post-Int.	% Change	Pre-Int.	Post-Int.	% Change
Percent with blood-lead concentration of at least 10 μg/dL	10.5	7.49	-28.6	6.65	3.97	-40.4
Percent with blood-lead concentration of at least 25 μg/dL	0.578	0.249	-56.9	0.117	0.029	-75.2
Percent with an IQ score less than 70	0.566	0.526	-7.19	0.520	0.489	-5.84
Percent with an IQ score decrement of greater than 1	52.2	48.7	-6.59	53.5	49.3	-7.70
Percent with an IQ score decrement of greater than 2	18.2	14.3	-21.4	14.0	9.80	-30.0
Percent with an IQ score decrement of greater than 3	7.09	4.73	-33.2	3.90	2.07	-46.9
Average IQ Loss (Standard Deviation)	1.35 (1.11)	1.23 (0.92)	-9.15	1.26 (0.82)	1.15 (0.67)	-8.56
Geometric Mean Blood-Lead Concentration (Geometric Standard Deviation)	4.05 (2.06)	3.81 (1.95)	-5.87	4.10 (1.81)	3.86 (1.72)	-5.87

This analysis assumes the following environmental-lead standards determine whether or not a particular intervention is performed in a housing unit:

- Dust-lead loading (under wipe techniques) of 200 μg/ft² for floors and 500 μg/ft² for window sills for dust cleaning
- Soil-lead concentration of 400 μg/g for soil cover and 3000 μg/g for soil removal
- 5 ft² of deteriorated lead-based paint for paint repair and 20 ft² for paint removal

Approach #2: Alternative approach to determining a post-intervention blood-lead distribution using directly-measured blood lead changes

An alternative approach to estimating a post-§403 national distribution of blood-lead concentrations for 1997 children aged 12 to 35 months (1 to 2 years) assumed an estimated 33% decline in blood-lead concentration following an intervention, based on results of published intervention efficacy studies and information on mobilized bone-lead stores (see Section 5.4.1.6). This alternative estimate of primary prevention effectiveness is denoted the 'adjusted blood-lead effects model', and was compared to post-§403 distribution using the IEUBK model and the HUD National Survey data.

In this approach, the summarized environmental-lead levels within each National Survey unit (Table C-7) were compared to the following to determine necessary intervention strategies:

- 100 $\mu g/ft^2$ as an interior floor dust-lead loading and 500 $\mu g/ft^2$ as an interior window sill dust-lead loading;
- 400 $\mu g/g$ as a soil-lead concentration for soil cover and 3000 $\mu g/g$ for soil removal; and
- 0 ft^2 of deteriorated lead-based paint.

(Note that other analyses within the sensitivity analysis considered a floor dust-lead loading standard of 200 $\mu g/ft^2$).

Table 5-18 summarizes the pre-§403 blood-lead distribution and the post-§403 distribution generated under the adjusted blood-lead effects model. This table also presents the post-§403 blood-lead distribution generated in Section 5.3 for candidate standards of 100 $\mu g/ft^2$ for floor dust, 500 $\mu g/ft^2$ for window sill dust, 400 ppm for soil cover, 3000 ppm for soil removal, 0 ft^2 for paint maintenance, and 20 ft^2 for paint abatement. The table presents the geometric mean and geometric standard deviation of the distributions, and the probabilities of exceeding certain concentration threshold values.

According to Table 5-18, the post-intervention geometric mean blood-lead concentration under both the adjusted blood-lead effects model and the post-§403 risk assessment method was 11% lower than the pre-intervention geometric mean. Also, as compared to the adjusted blood-lead effects model, a smaller percentage of children exceeded the various blood-lead levels under the approach used in the risk assessment.

Figure 5-8 contains a plot of the three blood-lead distributions documented in Table 5-18. As noted in the accompanying legend, distinct line types are utilized for each of the three distributions (e.g., the solid line denotes the pre-intervention distribution). The difference between the geometric standard deviation estimated for both post-§403 approaches is evident in this figure. The adjusted blood-lead effects model suggests a wider post-§403 distribution than does the post-§403 risk assessment method.

Table 5-18. Estimated Distribution of Post-§403 Blood-Lead Concentrations for Children 1-2 Years Old Based on the IEUBK Model for Both the Risk Assessment and the Adjusted Blood-Lead Effects Model Approach

Distribution Estimation Procedure	Geometric Mean PbB	Geometric Std. Dev. PbB	% with PbB > 5 μg/dL	% with PbB > 10 μg/dL	% with PbB > 15 μg/dL	% with PbB > 20 μg/dL	% with PbB > 25 μg/dL
Baseline (Pre-§403)	4.05	2.06	38.5	10.5	3.5	1.3	0.6
Post-§403 Under the Adjusted Blood Lead Effects Model[1]	3.60	1.89	30.2	5.4	1.2	0.3	0.1
Post-§403 Under the Risk Assessment Method[1]	3.60	1.75	27.8	3.4	0.5	0.1	0.03

[1] Based on the IEUBK model with the following options for standards: 100 μg/ft² for floor dust-lead loading; 500 μg/ft² for window sill dust-lead loading; 400 μg/g for soil cover; 3000 μg/g for soil removal; and 0 ft² of deteriorated lead-based paint for paint maintenance.

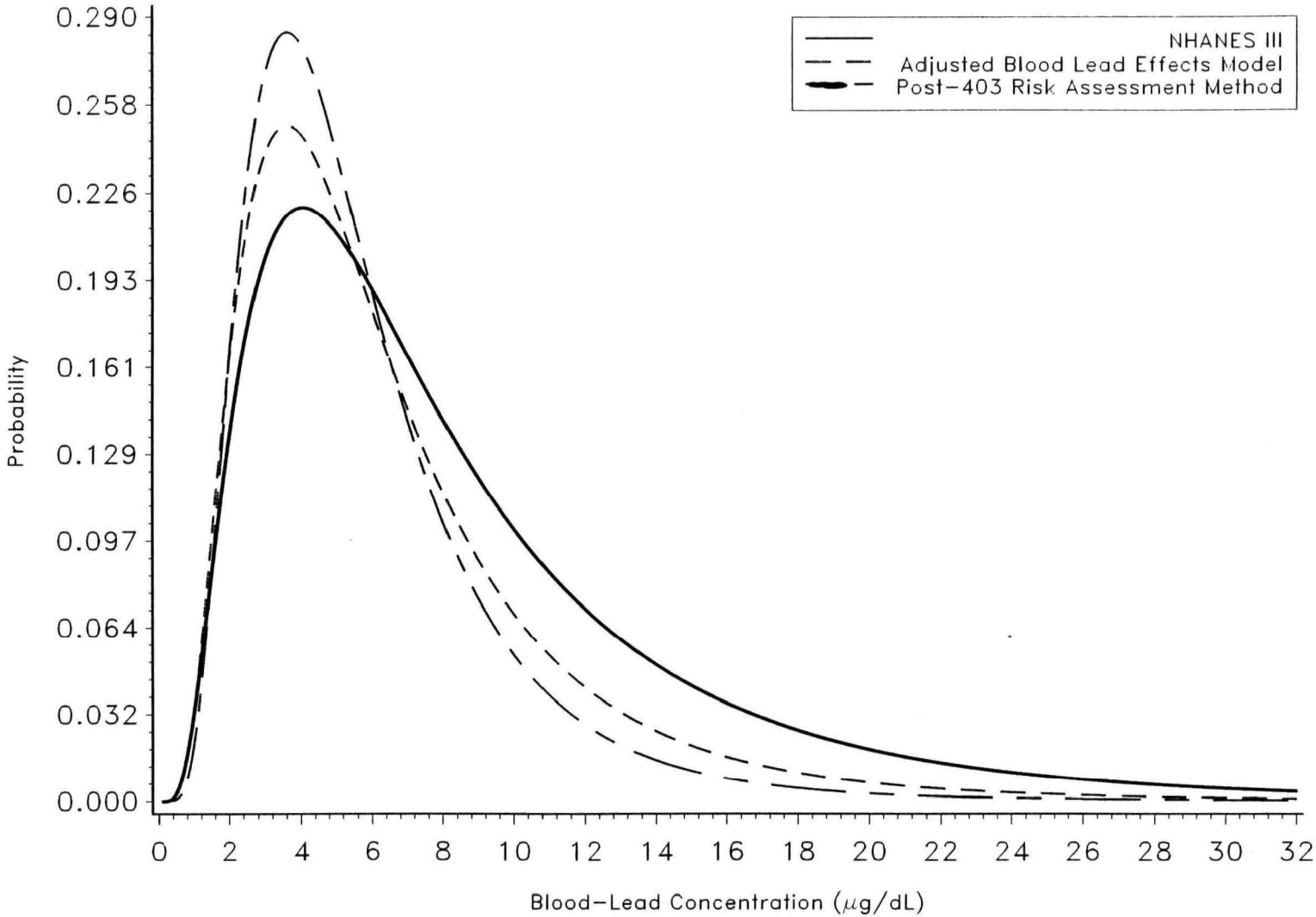


Figure 5-8. Comparison of NHANES III Blood-Lead Concentration Distribution to Distributions Estimated Using the Adjusted Blood Lead Effects Model and the Post-§403 Risk Assessment Method

6.0 CONFIRMATION STUDIES FOR IEUBK MODEL

CHAPTER 6 SUMMARY

Chapter 6 evaluates the IEUBK model for its implementation in the Risk Assessment by using relevant data from 3 studies: NHANES III / HUD National Survey, Rochester Lead-In-Dust study, and Baltimore Repair and Maintenance study. Model performance is evaluated by comparing the geometric mean of the model-predicted blood-lead levels across all the children to the observed geometric mean blood-lead level. The proportions of children exceeding threshold concentrations, such as 10 µg/dL, 15 µg/dL, 20 µg/dL, and 25 µg/dL are also examined. The differences between observed and model-predicted percentages of children having high blood-lead levels ranged from 0 to 1% when using national data from the NHANES III / HUD National Survey study. Results confirm IEUBK model's ability to predict a national distribution of blood-lead levels based on a national distribution of environmental lead levels.

As described in Chapter 4, the IEUBK model is a biological simulation model designed to predict the probability of elevated blood-lead levels in children. For the §403 Risk Assessment, the IEUBK model (version 0.99D) was applied to the environmental-lead measurements collected in the HUD National Survey to estimate a national distribution of blood-lead levels in children, assuming promulgation of the §403 health-based standards. The model predicts children's blood-lead levels using information on their multimedia exposure to environmental lead. The model has been developed using data from many different scientific studies of lead biokinetics, contact rates of children exposed to contaminated media, and data on the presence of environmental lead in residences. As part of applying this model to the §403 Risk Assessment, it is important to understand the extent to which model predictions are supported by comparisons with real-world data.

This chapter presents a comparison of IEUBK model predictions against data from relevant epidemiological studies:

- Phase I of the Third National Health and Nutrition Examination Survey (NHANES III) in combination with the HUD National Survey (Brody, et al., 1994; EPA, 1995a);

- Rochester Study (HUD, 1995a);
- Baltimore R&M Study (EPA, 1994a).

As stated in Chapters 3 and 5, the NHANES III Survey and the HUD National Survey are the fundamental sources of baseline data for the §403 Risk Assessment. The NHANES III Survey was used to estimate a baseline national distribution of blood-lead concentrations in children. The HUD National Survey (the only national survey of environmental lead levels) was employed to estimate a distribution of environmental lead levels, which were then inputted to the IEUBK model to predict a national distribution of children's blood-lead levels. Comparison of model-predicted blood-lead levels based on the HUD National Survey data versus the baseline levels from the NHANES III Survey data provides a basis for a general assessment of model predictions.

The Rochester and Baltimore R&M studies are blood-lead studies that provide useful information from urban residential communities over a range of environmental conditions. A comparison of IEUBK model predictions against data in these studies is relevant to the examination of model performance for urban lead sources. However, there are some difficulties in using Rochester and Baltimore R&M studies in evaluating IEUBK model:

- *Environmental measurements in Rochester and Baltimore may not have been representative of a child's cumulative lead exposure.* Given representative measurements of all relevant lead sources at a residence, the model estimates a plausible distribution of blood-lead levels associated with full-time exposure to those measured sources. Environmental measurements collected in childhood lead exposure studies are usually selected in a systematic fashion across residences. This may involve looking for areas containing high lead levels (dripline soil or window wells), or surfaces with ample medium to sample (sides of rooms where more dust may collect). While such samples may indeed represent real hazards to some children, they can be over-represented in exposure assessments.

- *Model predictions were based on a generalized activity assessment.* Residence-specific soil- and dust-lead concentrations should come from areas where children are most likely to play or spend their time and water-lead concentrations should reflect the water consumed by the children. Because environmental lead levels can vary considerably within a child's exposure unit and children vary in their activities, it would be preferable to weight individual dust or soil measurements by information on

childhood activity patterns. However, this information is not available. The particular environmental measures (average of play area and bedroom dust, and bare/play area fine fraction soil from the Rochester study and composite floor dust and dripline soil from the Baltimore R&M study) were evaluated because they may represent typical locations where most children are exposed to lead. This generalized approach to activity assessment will overestimate exposure for some children and underestimate it for others, and therefore, predictions of blood-lead concentrations are best assessed on an overall group basis.

- *Variability in environmental lead samples.* House dust, in particular, has substantial spatial variability and temporal variability. These sources of measurement error are difficult to assess quantitatively without more extensive data. In the Rochester data set, bedroom and play area dust-lead concentrations differed by at least 200 ppm for 50% of the residences. Although the selected model inputs of environmental exposure may represent the average lead exposure for an average child, there is considerable variability in their application to individual children.

- *Relation between blood and environmental lead in Rochester and Baltimore may be biased due to study selection criteria.* In Rochester, children in lower income families living in older homes were purposely over-sampled. Children were excluded if 1) medical treatment for an elevated blood-lead level or an environmental intervention was conducted 2) child had taken an iron supplement in the past 2 months, 3) any major renovation of residence occurred during past 12 months, or 4) an adult employed in an industry or involved in a hobby that would expose to lead lived in the household. Of 1536 families who were interviewed, about 75% were considered ineligible by these criteria. On one hand, the Rochester study may have targeted older homes with the potential for containing higher concentrations of lead in dust and soil. On the other hand, the selection criteria for children may have eliminated children with the potential for higher blood lead. Therefore, the study design may have undersampled children with a potential for both higher lead exposures and blood-lead concentrations. Therefore, it is not unexpected that the IEUBK model predicted a larger number of children with higher blood-lead concentrations compared to that observed in the study. In Baltimore, children were included because their homes were chosen for intervention or for control/baseline measurements. This design comes closer to a control versus treatment framework than a random sampling approach.

The validation approach used is the same as that recommended in an EPA guidance document, Validation Strategy for the Integrated Exposure Uptake Biokinetic Model for Lead in Children (EPA, 1994b). EPA developed this document to outline a working strategy for conducting IEUBK model empirical comparisons which includes the data requirements for generating and interpreting model predictions. Specifically, three steps are involved:

1. Identify the children that have adequate data to characterize exposure or dose (approximately). This requires a review and the assessment of available data on each child relative to observed measurements on lead in dust, soil, and water.

2. Calculate the inputs to the IEUBK model. Because the model requires single estimates of lead concentrations in dust, soil, and water for each child, multiple measurements available at the target locations for a medium need to be combined into a composite measurement.

3. Compare the model predictions of central values (e.g., geometric mean) and population percentiles (e.g., percent above 10 μg/dL) to those observed in the studies. These comparisons are made across all the children and for subsets of children according to variables (e.g., behavioral) that may influence or qualify their exposure characterization.

Descriptive measures used to evaluate model performance and the criteria for selecting data from each study for IEUBK model input are described in Section 6.1. Section 6.2 presents the results of comparing observed blood-lead levels to IEUBK model predictions. Conclusions are drawn in Section 6.3.

6.1 METHODS

6.1.1 Descriptive Measures

For each of the studies, model performance is evaluated by comparing the geometric mean of the model-predicted blood-lead levels across all the children to the observed geometric mean blood-lead level. In addition, the proportions of children exceeding threshold concentrations, such as, 10 μg/dL, 15 μg/dL, 20 μg/dL, and 25 μg/dL are examined.

While the observed proportion of children exceeding a threshold is estimated directly from the actual blood-lead levels for all studies, the procedure used to calculate the predicted proportion for the HUD National Survey is different from that used for the other two studies. For the HUD National Survey, the procedure is the same as that described in Chapter 5. This procedure takes into account the HUD National Survey weights which represent the number of children in the nation associated with a specific set of environmental lead measurements. For each study, the predicted proportion is estimated by calculating the exceedance probability for

each hypothetical child, and averaging them over the entire group. The exceedance probability for each hypothetical child is calculated using

$$\text{Prob}\left(Z > \frac{[\ln(T_c) - \ln(GM)]}{\ln(GSD)}\right)$$

where Z is a standard normal variate, T_c is a threshold concentration, GM is the predicted blood-lead level, GSD is the geometric standard deviation assumed equal to the IEUBK default value of 1.6, and ln is the natural logarithm. The GSD pertains to the inter-individual and biological variability in the blood-lead levels of children exposed to similar environmental lead levels. The IEUBK default value of 1.6 was estimated by EPA using several epidemiological studies, as described in the Guidance Manual (EPA, 1994a).

Predicted blood-lead levels are also compared to the observed levels graphically. For the NHANES III Survey, histograms and probability density functions are used to compare the observed and predicted distributions of blood-lead levels. For the Rochester and Baltimore R&M studies, scatter plots are used to display the observed and predicted individual blood-lead levels in combination with the line of perfect agreement and the 95% prediction intervals based on the model. The prediction intervals represent ranges which are intended to encompass with 95% confidence the true blood-lead levels of children exposed to similar environmental levels.

6.1.2 Input Data Selection

The IEUBK model is a dose-response model and therefore requires environmental lead levels that represent children's typical lead exposure or dose as inputs. As none of the studies was designed to measure children's exposure, the exposure information available varied across children. Therefore, subsets of children that had data judged to reasonably characterize exposure were selected from the Rochester and R&M studies. As recommended in both the Guidance Manual and Validation Strategy, only those children whose exposure to lead is relatively well characterized were included in the analysis. Children without dust-, soil-, or water-lead measurements and those who did not live for at least three months in their residence prior to blood collection were excluded from the analysis. These children were not expected to have enough information to predict blood-lead levels reliably. Environmental lead levels

corresponding to each child having sufficient exposure data were provided as input to the model. Note that the small sample size in the input data sets for the Rochester study is because play yard soil-lead concentration was reported for only approximately 40% of the 205 study children. Similarly, for the Baltimore R&M study, only one-third of the 163 study children from the pre-intervention round had dripline soil-lead concentration measurements. Due to the availability of only dripline soil and BRM (HVS-3 vacuum sampler) dust measurements in the Baltimore R&M study, IEUBK model predictions may be biased in estimating the lead exposures. For the HUD National Survey, we used environmental lead levels for each housing unit in the survey as input to the model. The model was then used to predict the blood-lead concentration for a hypothetical child of age 24 months in each housing unit.

Rochester

Two different analysis data sets were constructed to analyze the data from Rochester study based on different groups of children having adequate exposure to lead. These two data sets were labeled as Input Data Sets A and B.

(1) Input Data Set A

From the Rochester study, only 87 children had play yard fine-sieved soil concentration measurement. Among those children, 84 children had at least one floor dust-lead concentration measurement from the bedroom or principal play area collected by the DVM (Dust Vacuum Method). Of the three dust collection methods used in this study, wipe sampling, DVM, and BRM, the DVM method was the closest to that used in calibrating the IEUBK model. Therefore, only 84 of the 205 children in the Rochester study were considered to have sufficient exposure data and were included in the IEUBK model empirical comparisons. These children's blood-lead concentration measurements and environmental lead concentration measurements (dust-lead, soil-lead, water-lead) constitute Input Data Set A.

Dust-lead, soil-lead, and water-lead concentrations were computed or selected as parameters to be input into the IEUBK model. The model used a composite dust-lead concentration which was computed as a dust mass-weighted arithmetic mean of the floor

measurements from the bedroom floor and principal play area. A weighted average is meaningful if the precision with which lead is measured increased with large sample masses. The play yard fine-sieved soil-lead concentration and one-minute flushed water-lead concentration were also provided as inputs to the IEUBK model. Fine-sieved soil fraction was selected rather than coarse soil because fine soil is more likely to be ingested by children.

(2) Input Data Set B

Among 87 children who had play yard fine soil-lead concentration measurements, 82 children also had foundation fine soil-lead concentration measurements in the Rochester data set. Among those, 80 children had at least one floor dust-lead concentration measurement from all sampled indoor locations (bedroom, principal play area, living room, kitchen, and entryway). These 80 children make up Input Data Set B.

The composite dust-lead concentration was computed as a dust mass-weighted arithmetic mean of the floor measurements from all sampled indoor locations. An arithmetic average of play yard find soil-lead concentration and foundation fine soil-lead concentration was calculated and used in the IEUBK model. One-minute flushed water lead concentration also provided input to the lead model computation.

Baltimore R&M

Similar to the Rochester study, two data sets were constructed to analyze the data from Baltimore R&M study. These two data sets were labeled as Input Data Sets C and D.

(1) Input Data Set C

Only 54 children from the Baltimore R&M study had dripline soil-lead concentration measurements. Among those, 7 children were excluded from the IEUBK model empirical comparisons due to children living in the current residence less than three months or because the surveyed houses were vacant at the time of measurement collection. Therefore, only 47 of the 163 children from the pre-intervention round in the Baltimore R&M study who had blood-lead concentration measurements were considered to have sufficient exposure data and were used in the IEUBK model comparison. These 47 children constitute Data Set C in Table 6-1.

Dripline soil-lead concentration measurements were used in the IEUBK model validation due to lack of other data representing outdoor lead exposure. Almost all soil samples were collected from the driplines in the pre-intervention round for this study. Floor dust samples collected by the BRM method were composited over multiple rooms and a single composite floor dust-lead concentration based on all samples was computed using a dust mass-weighted arithmetic mean for the input to the model. The two-hour stagnation water-lead concentration was also used in the IEUBK model.

(2) Input Data Set D

Input Data Set D was constructed using the 47 children from Input Data Set C. The composited dust-lead concentration employed in Input Data Set D included entryway dust-lead concentration together with floor dust-lead concentration. The composite dust-lead concentration was calculated using a dust mass-weighted arithmetic mean. The dripline soil-lead concentration and the two-hour stagnation water-lead concentration were also included in the Input Data Set D.

NHANES III/HUD

Soil-lead concentrations and dust-lead concentrations were generated from the HUD National Survey database for use in calculating blood-lead levels by the IEUBK model. Because water-lead measurements were not reported in the survey, default values assigned by the IEUBK model were used (Table 4-1). Soil samples collected at the dripline, entryway, and a remote location were used to calculate a weighted soil-lead concentration average for each house. The soil-lead concentration average was calculated by giving the remote location sample twice the weight as the weight given to each of the samples collected near the house.

Floor dust samples were used to calculate a dust mass-weighted average of dust-lead concentrations from each house. The weighting of the samples in the average was based on the reported tap weights of the samples.

6.2 RESULTS

The IEUBK model performance was analyzed using the input data sets containing soil, dust, and water lead concentrations for children with adequate exposure data as described in Section 6.1. Default values defined by the model were used for all other required data in these runs. Table 6-1 presents the observed and predicted geometric mean blood-lead levels and proportions of children exceeding 10 μg/dL, 15 μg/dL, 20 μg/dL, and 25 μg/dL for each of the studies. The geometric mean blood-lead levels and exceedance proportions for each input data set were calculated by combining the data across all the children in that data set. Results of the comparison are separately discussed below for each study.

Rochester

As indicated in Table 6-1, the observed geometric mean blood-lead level for Input Data Set A is 6.3 μg/dL, while the model predicted geometric mean blood-lead level is 6.4 μg/dL. Thus, utilizing soil-lead levels from the play yard and dust-lead levels from the bedroom and principal play area as inputs to the IEUBK model results in predicted blood-lead levels that on the average agree with the observed blood-lead levels. The observed and predicted proportions of children exceeding 10 μg/dL in blood-lead, when using Input Data Set A, are 17% and 26%, respectively; the observed and predicted proportions of children exceeding 25 μg/dL are 1% and 6%, respectively.

The results of using Input Data Set B show that the observed geometric mean blood-lead level is 6.3 μg/dL and the predicted geometric mean blood-lead level is 9.2 μg/dL. The observed and predicted proportions of children exceeding 10 μg/dL in blood-lead are 18% and 44%, respectively; the observed and predicted proportions of children exceeding 25 μg/dL are 1% and 11%, respectively.

For both input data sets, the predicted proportions of children exceeding 10 μg/dL, 15 μg/dL, 20 μg/dL, and 25 μg/dL in blood-lead are all higher than the corresponding observed proportions. Figure 6-1 presents a scatter plot of the observed blood-lead levels against model predictions for the same set of children from Input Data Set A. Each symbol in the figure represents an individual child. Note from the figure that 83% of the observed blood-lead levels

Table 6-1. Comparison of Observed and IEUBK Model Predicted Blood-Lead Levels

Study	Input Data Set	No. of Children	Geometric Mean Blood-Lead (μg/dL)			Percent of Blood-Lead > 10 μg/dL		Percent of Blood-Lead > 15 μg/dL		Percent of Blood-Lead > 20 μg/dL		Percent of Blood-Lead > 25 μg/dL	
			Observed	Predicted	Relative Difference (Pred-Obs)/Obs	Observed	Predicted	Observed	Predicted	Observed	Predicted	Observed	Predicted
Rochester	A	84	6.3	6.4	0%	17%	26%	2%	14%	2%	8%	1%	6%
	B	80	6.3	9.2	46%	18%	44%	3%	26%	3%	17%	1%	11%
Baltimore	C	47	6.6	9.1	38%	34%	53%	17%	35%	9%	21%	6%	12%
	D	47	6.6	9.7	46%	34%	54%	17%	38%	9%	25%	6%	17%
NHANES III/HUD		5,272,068 (NHANES) 7,960,614 (HUD)	4.1 (GSD = 2.1)	3.9 (GSD = 2.2)	6%	11%	12%	4%	5%	1%	2%	1%	1%

lie within the 95% prediction intervals, and there is a pattern for those outside of the 95% prediction intervals. In six of the seven pairs where the observed blood-lead level is greater than the upper prediction bound, predicted blood-lead levels were less than 10 μg/dL. Similarly, for four of the seven pairs where the observed blood-lead level is less than the lower prediction bound, predicted blood-lead levels were greater than 10 μg/dL.

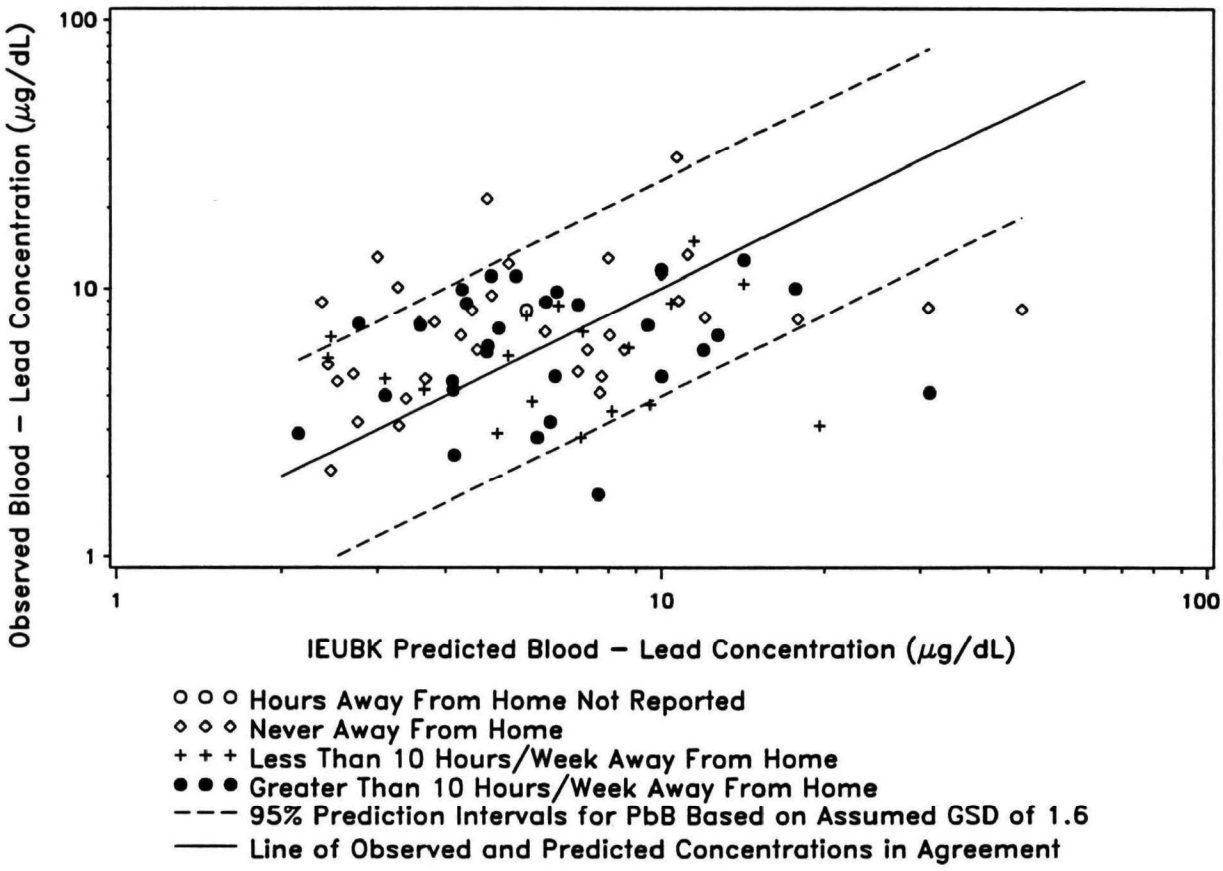


Figure 6-1. Comparison of Predicted and Observed Blood-Lead Levels for the Rochester Study.

(Symbols are Coded for Time Away from Home in Hours/Week)

Baltimore R&M

When using Input Data Set C, the observed geometric mean blood-lead level is 6.6 μg/dL, compared to the predicted geometric mean blood-lead level of 9.1 μg/dL . The observed and predicted proportions of children exceeding 10 μg/dL in blood-lead are 34% and 53%, respectively; the observed and predicted proportions of children exceeding 25 μg/dL are 6% and 12%, respectively.

For Input Data Set D, the observed and predicted geometric mean blood-lead levels are 6.6 μg/dL and 9.7 μg/dL respectively. The observed and predicted proportions of children exceeding 10 μg/dL in blood-lead are 34% and 54%, respectively; the observed and predicted proportions of children exceeding 25 μg/dL are 6% and 17%, respectively.

As before, the predicted proportions are higher than the observed proportions. Figure 6-2 displays the observed blood-lead levels plotted against the predicted blood-lead levels from Input Data Set C with 95% prediction intervals. More than 80% of the observed blood-lead levels lie within the 95% prediction intervals. Note that in one of the three pairs where the observed blood-lead level is greater than the upper prediction bound, predicted blood-lead levels were less than 10 μg/dL. Similarly, for five of the six pairs where the observed blood-lead level is less than the lower prediction bound, predicted blood-lead levels were greater than 10 μg/dL. Also note that a higher percentage of children in the Control Modern Urban homes are bracketed by the prediction limits. These children's blood-lead levels span the range of blood-lead levels which §403 aims to achieve.

NHANES III/HUD

Table 6-1 shows that the predicted geometric mean blood-lead level is 3.9 μg/dL using IEUBK/HUD data, and the observed value is 4.1 μg/dL for the NHANES III data. The predicted and observed geometric standard deviations are 2.2 and 2.1, respectively. The observed and predicted percentages of children having blood-lead levels exceeding 10 μg/dL are 11% and 12%, respectively; and the observed and predicted proportions of children exceeding 25 μg/dL, are both 1%. Figure 6-3 displays the distribution of blood-lead levels for

children aged 1-2 years as reported in NHANES III. A smooth probability density function for blood-lead levels is overlaid on the histogram. Similarly, Figure 6-4 displays the corresponding projected distribution of blood-lead concentrations for children aged 1-2 years based on the IEUBK model. In Figure 6-5, the probability density functions based on NHANES III and the IEUBK model are presented simultaneously for purposes of easier comparison. These two curves are comparable.

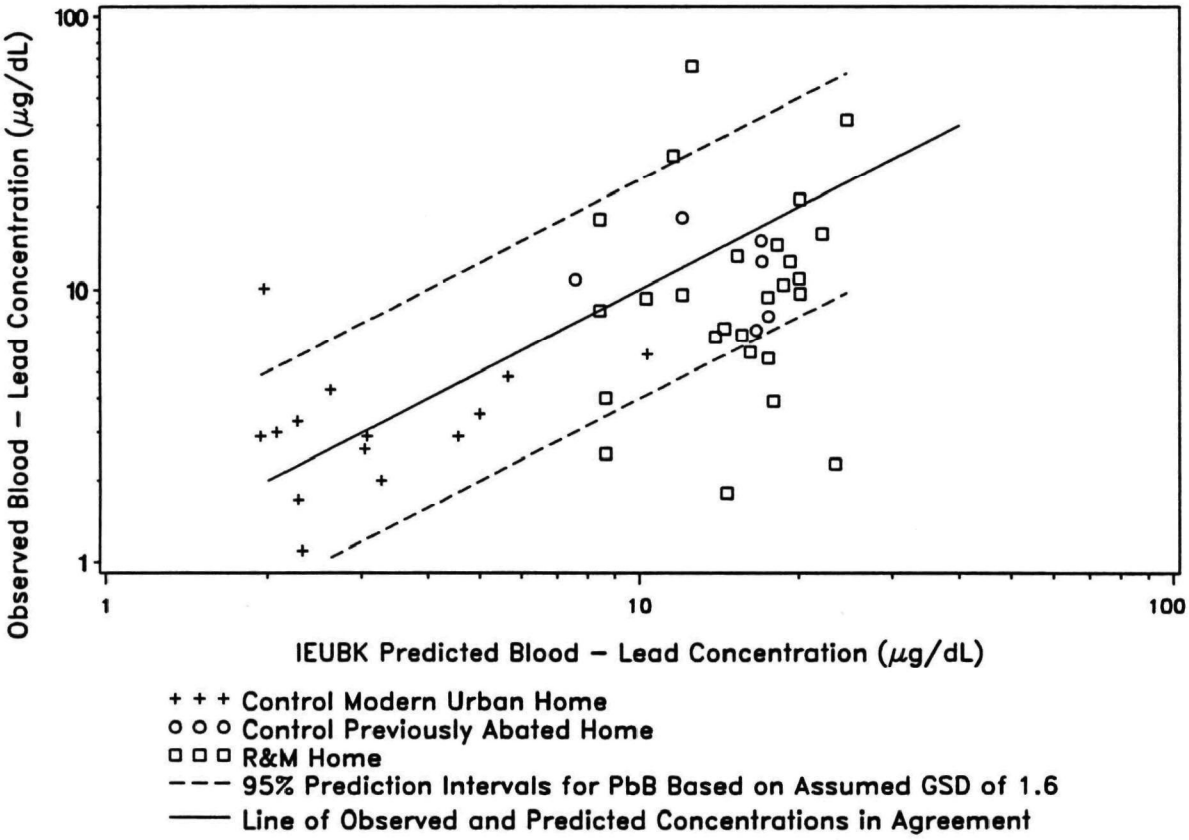


Figure 6-2. Comparison of Predicted and Observed Blood-Lead Levels for the Baltimore R&M Study.

(Symbols are Coded for Study Group)

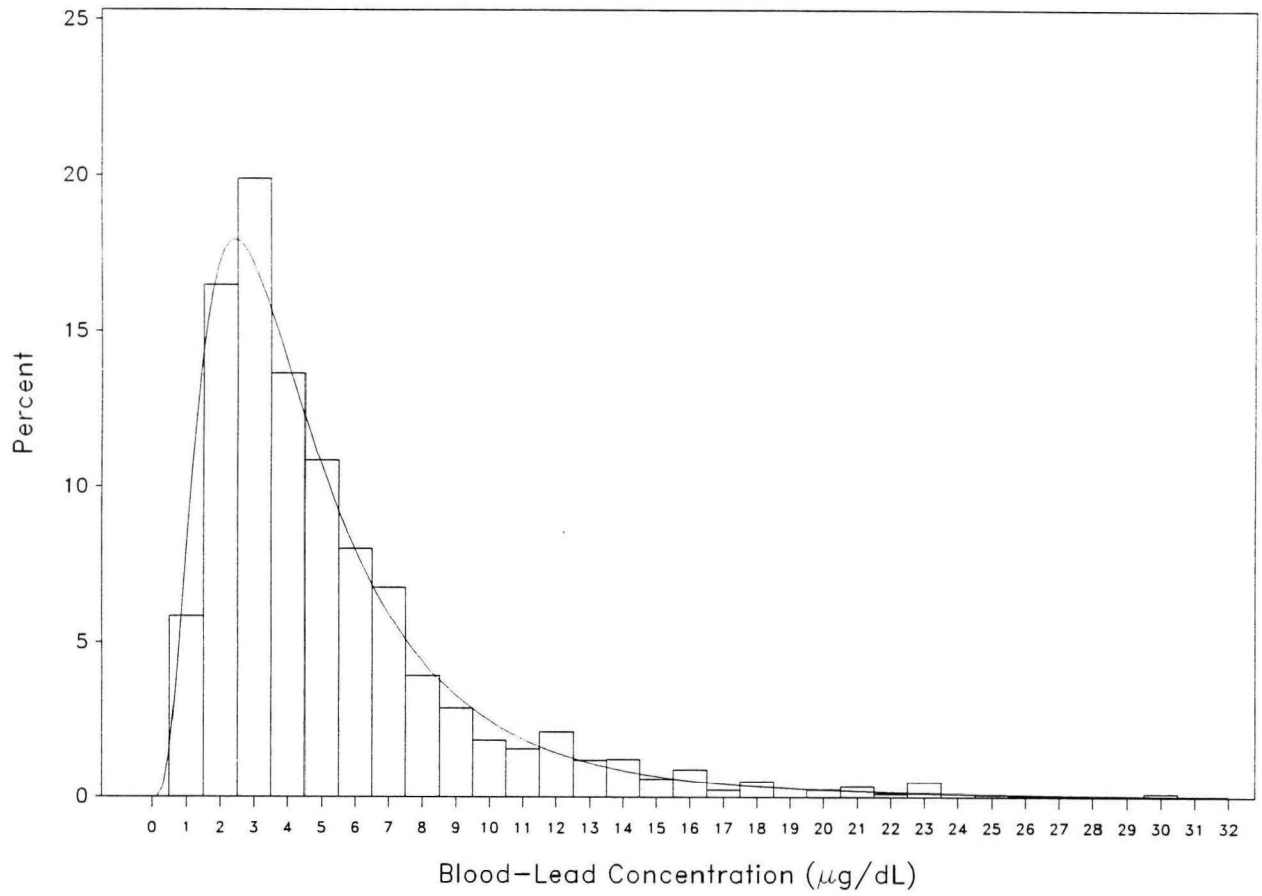


Figure 6-3. Distribution of Blood-Lead Concentrations (μg/dL) for NHANES III (1-2 Year Old Children).

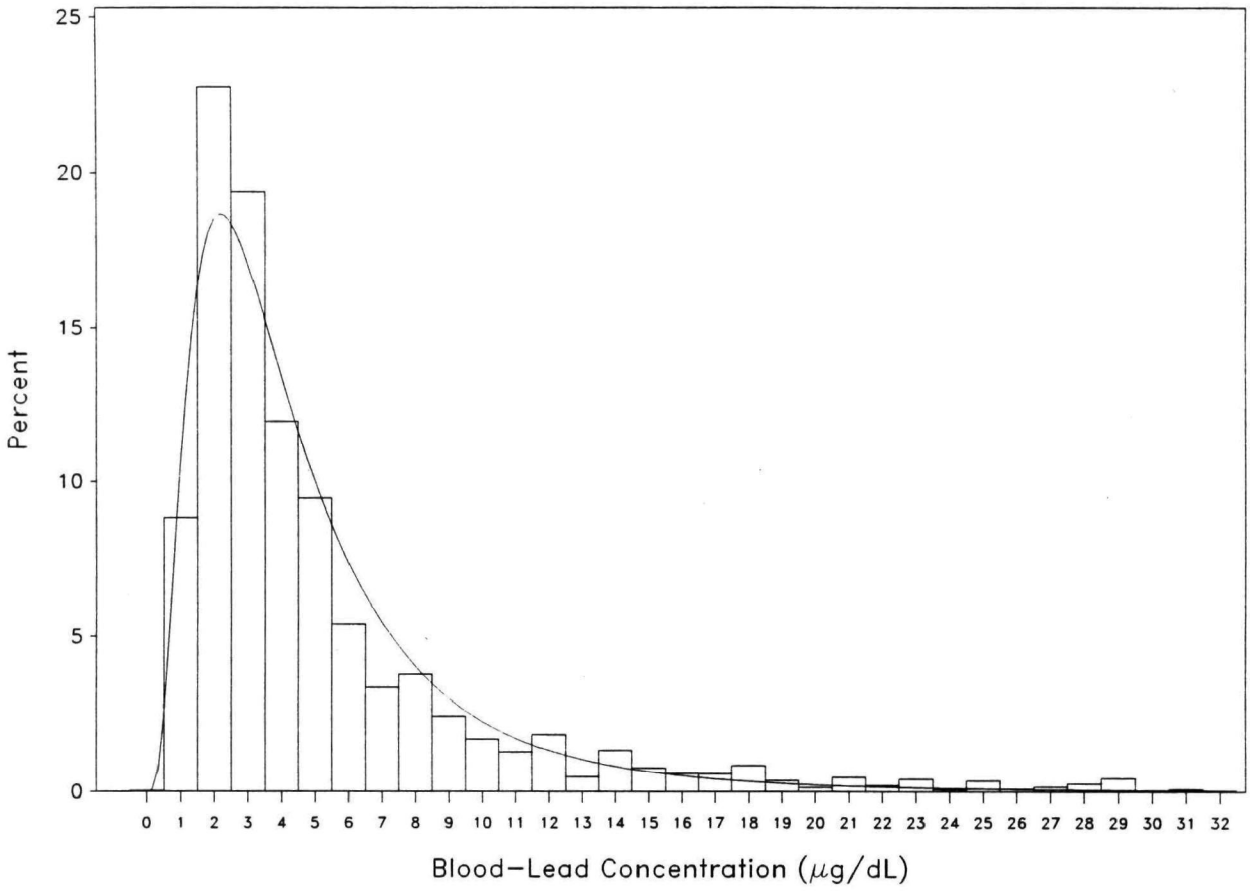


Figure 6-4. Distribution of Blood-Lead Concentrations (μg/dL) for HUD National Survey Data (1-2 Year Old Children) Predicted from IEUBK Model.

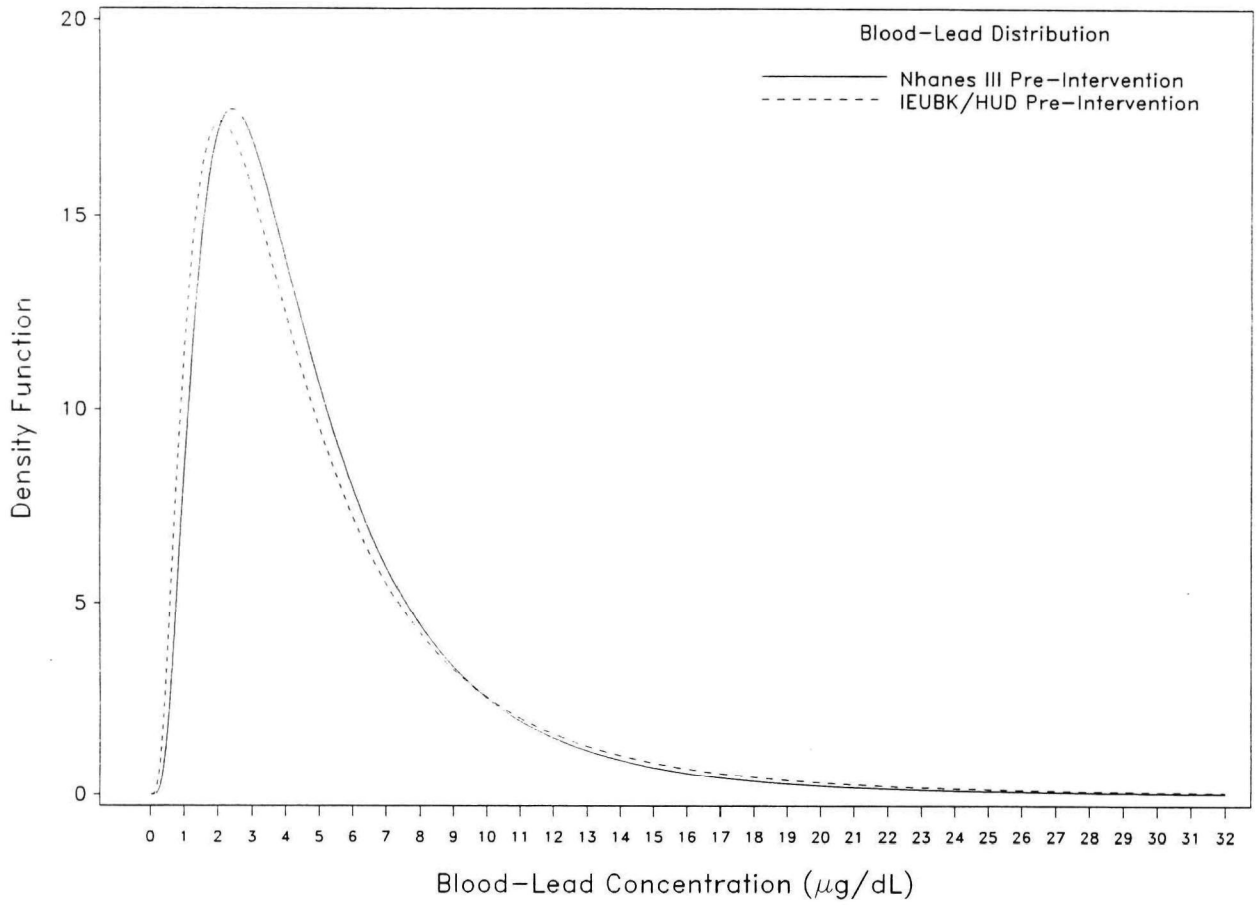


Figure 6-5. Distribution of Blood-Lead Concentrations (μg/dL) Based on NHANES III and IEUBK Model (1-2 Year Old Children).

6.3 CONCLUSIONS

The IEUBK model-predicted percentages of children exceeding 10 μg/dL, 15 μg/dL, 20 μg/dL, and 25 μg/dL are consistently higher than the observed percentages. Observed and Predicted percentages of children having blood-lead levels exceeding 25 μg/dL are 1% vs 6% for Rochester study Input Data Set A; 1% vs 11% for Rochester Input Data Set B; 6% vs 12% for Baltimore R&M study Input Data Set C; 6% vs 17% for Baltimore R&M study Input Data Set D; and 1% vs 1% for NHANES III/HUD.

Differences between observed and model-predicted geometric means ranged from 1.6 to 47%. Observed and model predicted geometric means are 6.3 and 6.4 μg/dL for Rochester study Input Data Set A; 6.3 and 9.2 μg/dL for Rochester study Input Data Set B; 6.6 and 9.1 μg/dL for Baltimore R&M study Input Data Set C; 6.6 and 9.7 μg/dL for Baltimore R&M study Input Data Set D; and 4.1 and 3.9 μg/dL for NHANES III/HUD.

As shown in Table 6-1, differences between observed and model-predicted percentages of children having high blood-lead levels ranged from 0 to 1% when using national data from the HUD survey and NHANES III. The statistical design employed in the HUD National Survey produced a sample of environmental lead levels which was representative of actual levels in U.S. housing. The IEUBK model is designed to predict distributions of blood-lead levels from environmental lead levels. Results here confirm its ability to predict a national distribution of blood-lead levels based on a national distribution of environmental lead levels. In conclusion, the analyses conducted to compare IEUBK model predicted blood-lead concentrations to those of observed blood-lead concentrations did not provide any evidence that use of the IEUBK model in the analyses conducted for §403 is inappropriate.

7.0 RISK SUMMARY

This risk assessment documents the scientific basis for setting regulatory standards for lead-based paint hazards, lead-contaminated dust, and lead-contaminated soil under §403 and provides estimates of health risk reductions (for children under six) and numbers of children and housing units affected for various §403 regulatory standards options. This risk assessment does not include selection of the §403 standards.

Figures included in Chapter 5, plotting quantitative health risks against percentages of housing units in which interventions are required, illustrate the main conclusions of this report. Varying options for standards for lead-contaminated paint, dust, and soil from intermediate values to more stringent values provides diminishing gains in health risk reduction while requiring interventions at many more housing units. Although health risks continue to decline as the standards for lead-based paint hazards, lead-contaminated dust, and lead-contaminated soil are made more stringent, the rate of decline diminished relative to the number of interventions required. Conversely, standards less stringent than the intermediate options provide only marginal decreases in the numbers of housing units requiring intervention, but significantly decrease gains in health risk reduction.

To summarize, the greatest quantitative reduction in health risks is always achieved by the most stringent standards. However, these reductions require intervention in the largest number of housing units. At intermediate values for the standards, many of the health risk endpoints are nearly at their lowest levels, but many fewer housing units are estimated to be affected by these standards in comparison to the number of units affected by the more stringent standards.

Sensitivity analyses are performed in this risk assessment to gauge the robustness of the risk analysis methodology employed. These results suggest relative insensitivity to some changes in some assumptions, but strong dependence on other assumptions. The applicability of this risk assessment to risk managers making policy decisions to set appropriate §403 standards hinges on the robustness of the risk assessment methodology.

Section 7.1 includes a summary of the scientific evidence, presented in Chapters 2, 3, and 4, identifying the need for §403 standards. Tools developed for implementation of the risk

assessment methodology are also summarized in this section. Section 7.2 provides conclusions on the potential health risk reductions predicted by this risk assessment and numbers of children and housing units affected for various standards options. Comments on the robustness of the methodology employed are made in Section 7.3.

7.1 SCIENTIFIC BASIS FOR §403 AND TOOLS FOR THE RISK ASSESSMENT

The toxicologic effects of lead exposure on young children were documented based on numerous studies reported in the scientific literature. Typically, in studies assessing adverse health effects associated with lead exposure, relationships between health effects and exposure are established using a measure of internal rather than external exposure. Blood-lead concentration is the most common measure of internal exposure.

Though lead causes a wide array of adverse health effects, particularly at high dose levels, lead is best known for its adverse effects on the central nervous system. IQ score decrements and incidence of IQ scores less than 70 due to lead exposure were examined as health endpoints in this risk assessment. As surrogates for the wide array of other, non-IQ related health risks to both the central nervous system and other organs, incidence of elevated blood-lead concentrations were estimated for specified thresholds.

Children aged 1-2 were targeted for estimation of health risks in this risk assessment for two reasons. The first is related to the increased vulnerability of this age group due to their rapidly developing central nervous system. One to two year old children may be the most appropriate age group for relating blood-lead concentrations to adverse IQ effects. This is documented in Chapter 2. Second, both the normal hand-to-mouth activities of this age group and pica tendencies observed in some children may put children aged 1-2 most at risk to lead exposure.

It was shown, based on a number of lead studies, that elevated lead levels continue to exist in residential environments (Chapter 3) providing an on-going threat of childhood lead exposure. Lead is particularly a threat in older homes. The HUD National Survey, discussed in detail in Section 3.3, provides nationally representative estimates of numbers of homes exceeding certain exposure levels. For instance, 10.4% and 6.4% of all homes are predicted to

have some deteriorated lead-based paint in the exterior and interior, respectively. At least 10% of homes are estimated to have lead loadings above the Interim Guidance standards.

NHANES III data indicate that concentrations of lead in children's blood are still high. Section 3.4 documents these elevated levels. There is also evidence in the existing scientific literature of a dose-response relationship between residential environmental lead (external exposure) and elevated blood-lead concentrations of resident children (internal exposure). Section 3.2 documents the overwhelming evidence from epidemiologic studies on the existence of a positive relationship between environmental-lead levels and blood-lead concentrations.

In this risk assessment, the connection between environmental-lead levels and adverse health effects (dose-response) was estimated in two steps because there is little scientific data for estimating this relationship directly. First blood-lead concentrations were estimated based on environmental-lead levels, and then health risks were estimated from those blood-lead concentrations. This was necessary because the majority of the existing scientific evidence for the relationship between lead exposure and adverse health effects was available in this form.

Two approaches were taken to mapping environmental-lead levels to blood-lead concentrations, an EPI model and the IEUBK model. The EPI model was constructed based on data from the Rochester study. This study is presented in Section 3.2, and the EPI model is discussed in Section 4.1. The EPI and IEUBK models reflect qualitatively different relationships between environmental-lead levels and blood-lead concentrations. Estimating blood-lead concentrations (and thus adverse health effects) using these two different models strengthens the analysis by providing two separate estimates of health risk reductions associated with §403.

Analyses were conducted to select appropriate ranges of options for the §403 standards. The selected ranges are presented in Table 4.6.

Decisions regarding the appropriate interventions for §403 and the efficacy of these interventions are documented in Section 5.2. In this risk assessment, in which *potential* health risk reductions achievable under various standards options are evaluated, interventions are assumed to occur whenever a housing unit exceeds a proposed standard. One dust intervention

was proposed; two levels of soil intervention; and two levels of exterior and interior paint intervention. Abatement efficacy assumptions are detailed in Table 5.3.

7.2 HEALTH RISK REDUCTIONS AND NUMBERS OF CHILDREN AND HOUSING UNITS EFFECTED

Risk comparisons between baseline and predicted post-§403 values of seven health endpoints (IQ related health effects and elevated blood-lead concentration incidence) for 1997 were conducted for each set of standards evaluated. Baseline health risks were estimated using NHANES III. Post-§403 health endpoints were estimated by modeling blood-lead concentrations, both before and after interventions triggered by §403, and applying that difference to the estimated baseline blood-lead concentration distribution.

This summary focuses on four of the health endpoints: blood-lead concentrations greater than 25 μg/dL or 10 μg/dL and IQ score decrements of greater than 2 or 3 points. The pre-§403 (baseline) health risks, as quantified by these four measures, were presented in Table 5-1. Over 10% of children aged 1-2 years are predicted to have blood-lead concentrations over 10 μg/dL. 10 μg/dL is the CDC level of community concern. These estimates represent current predicted health risks for the United States in 1997.

Chapter 5 presents post-§403 adverse health effects estimates for a wide array of standards options. First, results for varying dust-lead standard options are presented with soil and paint standards held fixed at central values. Likewise results for different soil and paint standard options are presented with the other media held fixed at central values. Lastly, post-§403 adverse health effects estimates are presented for different combinations of options. In this case, the standards for all media are simultaneously varied for lead-contaminated paint, dust, and soil from options at the upper end of each media's range down to the more stringent end. This summary focuses on this last set of results (Table 5-10 and Figures 5-6a and 5-6b).

The IEUBK model predicts larger reductions in adverse health effects due to promulgation of §403 than the EPI model in all cases. The IEUBK model also predicts greater incremental reduction in health risks across the range of standards.

The characterization of post-§403 health risks (particularly those based on the IEUBK model) and the numbers of housing units requiring interventions, as plotted in Figures 5-6a and

5-6b, are well described by a piecewise linear function with two pieces; the first piece is steeper than the second. In summarizing the results of the integrated risk assessment, these conclusions focus on the *intermediate set of standards*, where the two linear pieces meet. Performance of the intermediate set of standards is compared to performance of sets of standards at the most stringent and least stringent end of the range of options.

The incremental reductions in health risks per number of housing units requiring interventions between the least stringent set of standards and the intermediate set is greater than the incremental reductions between the intermediate and most stringent set of standards.

Table 7-1 summarizes results for these three selected sets of standards, both in terms of the numbers of interventions that would be required by each set of standards, and the resulting risk reductions. At the intermediate set of standards, 26% of all housing units require at least one of the interventions considered. Less than 1% of all housing units need costly Soil Removal interventions with an additional 13% requiring Soil Cover interventions; 6% need costly Exterior Paint Abatement interventions with an additional 2% requiring Exterior Paint Maintenance and; 2% require the more intensive treatment of Interior Paint Abatement while less than 1% require Interior Paint Maintenance.

The bottom portion of Table 7-1 describes the estimated health risk reductions anticipated after promulgation of §403 based on the selected standards. Results are presented as the percentage decline in numbers of children affected by each health endpoint. According to the EPI model only half as many children (20,000 as compared to 46,000) would have blood-lead concentrations greater than 25 μg/dL if the standards are set at the intermediate levels identified earlier. The reduction is even greater for the IEUBK model, 95% (2,500 as compared to 46,000). Similarly, for the EPI model, elevated blood-lead concentrations above 10 μg/dL would be reduced from 834,000 to 600,000 or approximately 28%. The reduction is predicted to be even greater using the IEUBK model, with approximately 548,000 (≈66%) fewer children above 10 μg/dL.

To summarize, the EPI model predicts 57% declines in blood-lead concentrations above 25 μg/dL and 28% declines in blood-lead concentrations above 10 μg/dL. Based on the IEUBK model, declines of 95% and 67% are predicted. According to the EPI model, the number (1.45 million) of IQ decrements greater than 2 would be reduced by 23% with the

Table 7-1. Percent of Housing Units Requiring §403 Interventions and Estimated Reduction from Baseline Health Risks Due to §403 for Three Sets of Standards Options

Intervention		Percent of Housing Requiring Intervention for Three Sets of §403 Standards					
		Least Stringent[1]		Intermediate[2]		Most Stringent[3]	
Total		19		26		74	
Soil	Cover	3		13		43	
	Removal	<0.5		1		6	
Interior Paint	Maintenance	3		3		1	
	Abatement	<0.5		2		5	
Exterior Paint	Maintenance	4		3		1	
	Abatement	3		6		9	
Dust	Cleaning	13		18		64	
Health Effects		Estimated Percent Reduction in Risk from Baseline					
		EPI	IEUBK	EPI	IEUBK	EPI	IEUBK
Blood-Lead Concentration Exceeding 10 μg/dL (%)		45	72	57	95	76	100
Blood-Lead Concentration Exceeding 25 μg/dL (%)		21	38	28	66	45	90
IQ Decrement Exceeding 2 (%)		18	29	23	52	34	79
IQ Decrement Exceeding 3 (%)		25	44	34	73	51	93

[1] These standards are floor dust-lead loading 400 $\mu g/ft^2$, window sill dust-lead loading 800 $\mu g/ft^2$, soil cover soil-lead concentration 1500 μg/g, soil removal soil-lead concentration 5000 μg/g, paint maintenance square footage of deteriorated lead-based paint 10 ft^2, and paint abatement square footage of deteriorated lead-based paint 100 ft^2.

[2] These standards are floor dust-lead loading 200 $\mu g/ft^2$, window sill dust-lead loading 500 $\mu g/ft^2$, soil cover soil-lead concentration 400 μg/g, soil removal soil-lead concentration 3000 μg/g, paint maintenance square footage of deteriorated lead-based paint 5 ft^2, and paint abatement square footage of deteriorated lead-based paint 20 ft^2.

[3] These standards are floor dust-lead loading 25 $\mu g/ft^2$, window sill dust-lead loading 25 $\mu g/ft^2$, soil cover soil-lead concentration 50 μg/g, soil removal soil-lead concentration 1000 μg/g, paint maintenance square footage of deteriorated lead-based paint 0 ft^2, and paint abatement square footage of deteriorated lead-based paint 5 ft^2.

intermediate set of standards; the number of cases of IQ decrements of greater than 3 would be reduced by 34%. Predicted reductions based on the IEUBK model are 52% and 73% for IQ decrements of at least 2 and 3, respectively.

Moving to the most stringent standards, slight reductions in health risks can be achieved compared to the intermediate standards but 74% of housing units would require intervention. This is a threefold increase in total number of interventions required from the intermediate set of standards. In comparison, the increased reductions in health risks gained by moving to the most stringent set of standards are only marginal.

Decreasing to the least stringent standards yields smaller reductions in health risk from the baseline without greatly decreasing the number of housing units which would be affected. For example, at the least stringent set of standards, 18.6% of housing units require interventions. This is nearly three quarters as many interventions as are estimated to be required at the intermediate standards.

Based on our analysis of health risks reductions, it may be possible to discover some slight changes to the standards, i.e., shifting the standard for a single media, which further reduces health risks without increasing numbers of housing units requiring interventions. However, given the uncertainties associated with estimates of health risk reductions, discussed in the context of sensitivity analyses in the subsequent section, it is doubtful that this will generate additional information to aid risk managers in selection of standards.

7.3 ROBUSTNESS OF RISK ASSESSMENT DATA SOURCES AND METHODOLOGY

Several analyses were conducted to assess the sensitivity of the estimated reductions in risks to the uncertainty in the underlying assumptions and methods utilized in the risk assessment.

1. Using both the IEUBK and EPI models to predict blood-lead concentrations from environmental-lead levels.

2. Estimating baseline numbers and percentages of children having specific health effects for two age groups, 1-2 year olds and 1-5 year olds, using three different coefficients to quantify the relationship between decline in IQ score and increases in blood-lead concentration.

3. Estimating baseline health effects using both an empirical and a model-based approach.

4. Estimating numbers and percentage of housing units expected to exceed a 200 $\mu g/ft^2$ floor dust-lead standard and/or a 500 $\mu g/ft^2$ window sill dust-lead standard using three different approaches to converting dust-lead loadings to wipe equivalent dust-lead loadings.

5. Estimating post-§403 health effects using three different assumptions for the effectiveness of the Dust Cleaning Intervention and three different assumptions for the effectiveness of the Soil Cover Intervention.

6. Comparing pre- and post-§403 estimates of health effects using the methodology employed in the risk assessment and an alternative approach that makes direct comparison of model-predicted pre- and post-§403 distributions of blood-lead concentrations (approach #1 in Section 5.4.2.6).

7. Comparing pre- and post-§403 estimates of health and blood-lead effects using the methodology employed in risk assessment approach which relies on assumptions of the effectiveness of interventions on environmental-lead levels to an alternative approach which relies on assumptions of the effectiveness of interventions on childhood blood-lead concentrations (adjusted blood-lead effects model in Section 5.4.2.6).

It may be concluded, based on the results of the sensitivity analysis presented in Section 5.4.2, that the reductions in health risks predicted in this risk assessment are sensitive to at least three sources of uncertainty:

1. Uncertainty in the relationship between declines in IQ score and increases in blood-lead concentration (Section5.4.2.2).

2. Uncertainty in converting vacuum dust-lead loadings to wipe equivalent dust-lead loadings (Section 5.4.2.4).

3. Uncertainty in the assumed efficacy of the environmental interventions on the environmental-lead levels (Section 5.4.2.5).

The assumption of an 0.257 decrease in IQ score for an increase of one μg/dL in blood-lead concentration has considerable impact on the estimates of numbers of children with specified IQ decrements and average decline in IQ due to lead exposures. However, even if the

decline is less severe (0.185 vs. 0.257), approximately 700,000 children 1-2 years old, and 1.3 million children 1-5 years old suffer IQ decrements greater than 2 points due to exposures to lead-based paint hazards, lead-contaminated dust, and lead-contaminated soil.

The number and percentage of housing units expected to exceed a 200 $\mu g/ft^2$ floor dust-lead standard varies considerably among the three conversion approaches taken to estimate wipe dust-lead loading. Over twice as many housing units' floor dust-lead loadings exceed 200 $\mu g/ft^2$ using the "high" alternative as do using the risk assessment approach. Exceedance proportions for window sill wipe dust-lead loadings are considerably less sensitive. The impact of uncertainty in the dust-lead loadings conversion on the number of homes requiring Dust Cleaning interventions based on either the floor or window sill dust standard is much less. This number ranges from 12.7 million to 16.3 million when the standards are set at 200 $\mu g/ft^2$ for floor dust-lead loadings and 500 for window sill dust-lead loadings.

Estimated reductions in health and blood-lead concentration risks are dependent on the assumed efficacy of interventions performed under §403. Under the alternative efficacies considered, which varied post-intervention soil-lead concentrations following soil cover intervention and dust-lead loading following Dust Cleanings, estimates of reductions in health risks varied considerably. The range of health risk reductions observed in the intervention effectiveness sensitivity analysis, calculated as percent reduction in numbers of children affected for each health endpoint, are presented in Table 7-2. These reductions are all calculated with standards set at the intermediate option for the standards.

Although the results in Table 7-2 indicate that there is considerable variability in the estimated reduction in health risks due to the uncertainty in the assumed efficacy of the interventions, the estimated risk reductions are nevertheless considerable and would impact the health of hundreds of thousands of children, even if the assumed efficacy of the intervention were decreased by a factor of 2.5.

Two analyses were performed to evaluate the risk assessment methodology. The first method provides an alternative characterization of the pre-§403 health risks based on mapping the HUD National Survey environmental-lead levels to a blood-lead concentration distribution rather than using NHANES III to characterize pre-§403 health risks. The second method

Table 7-2. Reductions From Baseline Health Risks (in percent) Obtained by Varying Intervention Effectiveness Due to §403 Intermediate Standards For Selected Health Endpoints and Risk Assessment Methodology

	Dust and Soil Intervention Effectiveness						Direct Comparison of Model-Predicted Distribution[4]		Adjusted Blood-Lead Effects Model[5]
	Reduced[1]		RA[2]		Enhanced[3]				
Health Effects	EPI	IEUBK	EPI	IEUBK	EPI	IEUBK	EPI	IEUBK	IEUBK
Blood-lead concentration exceeding 25	41	82	57	95	71	99	75	89	83
Blood-lead concentration exceeding 10	19	46	28	66	38	80	40	56	49
IQ decrement exceeding 2	14	34	23	52	30	68	30	44	
IQ decrement exceeding 3	22	53	34	73	44	87	47	64	

[1] Soil Cover intervention is assumed to reduce soil-lead concentration to 80% of pre-intervention concentration. Dust Cleaning intervention is assumed to reduce floor dust-lead loading to 100 $\mu g/ft^2$ and window sill dust-lead loading to 250 $\mu g/ft^2$.

[2] Soil Cover intervention is assumed to reduce soil-lead concentration to 50% of pre-intervention concentration. Dust Cleaning intervention is assumed to reduce floor dust-lead loading to 40 $\mu g/ft^2$ and window sill dust-lead loading to 100 $\mu g/ft^2$.

[3] Soil Cover intervention is assumed to reduce soil-lead concentration to 20% of pre-intervention concentration. Dust Cleaning intervention is assumed to reduce floor dust-lead loading to 20 $\mu g/ft^2$ and window sill dust-lead loading to 50 $\mu g/ft^2$.

[4] This risk assessment methodology involves comparing environmentally predicted pre- and post-§403 blood-lead concentration distributions without considering NHANES III data.

[5] An alternative approach to determining intervention effectiveness based on observed changes in blood-lead concentration following an intervention and adjusted to be reflective of primary prevention interventions. Note that the floor dust-lead loading standard used in this analysis was 100 $\mu g/ft^2$ rather than 200 $\mu g/ft^2$ which was used in all other sensitivity analyses. The paint standards were also different. Paint maintenance interventions were conducted at hourly units exceeding 0 ft^2 (rather than 5 ft^2) of deteriorated LBP.

provides an alternative characterization of the post-§403 blood-lead concentration distribution based on the adjusted blood-lead effects model (Section 5.4.1.6). Predicted reductions in health risks based on these methodologies are also presented in Table 7-2.

Results of directly comparing model-predicted post-§403 distribution of blood-lead concentration to model-predicted pre-§403 distributions are shown in columns 8 and 9 of the table. Estimated risk reduction are comparable to those based on the risk assessment methodology. Estimates are slightly larger for the EPI model and slightly less for the IEUBK model than those generated by the risk assessment methodology.

Estimated reductions in blood-lead concentration based on the adjusted blood-lead effects model are shown in the last column of Table 7-2. They are remarkably similar to those

estimated based on the main risk assessment methodology, suggesting that estimates of post-intervention environmental-lead levels are reasonable.

Obviously, estimates of health risk reductions vary when the methodology used to estimate them changes. The results presented in Table 7-2 reflect this variability and provide a range of reasonable estimates for potential reductions in health risks due to §403 at the intermediate standards.

7.4 CONCLUSIONS OF RISK ASSESSMENT

On an overall basis after performing, integrating, and assessing the uncertainty in the hazard identification, exposure assessment, and dose-response assessment, the following conclusions were made:

1. The health risks of young children from exposure to lead-based paint hazards, lead contaminated dust, and lead-contaminated soil are severe. Forty-six thousand children aged 1-2 years, and 82,000 children aged 1-5 years have a blood-lead concentration exceeding 25 μg/dL.

2. The health risks of children can be reduced.

3. The standards defined by §403 will help reduce the health risks to our nation's children. Depending on the methodology implemented (assumptions on intervention efficacy, predictive model used, and methodology for computing risk reductions) the reduction in the number of children with a blood-lead concentration exceeding 25 μg/dL for the intermediate option for the §403 standards ranged from 41% to 99%.

8.0 REFERENCES

Adebonojo, F.O. (1974) "Hematologic Status of Urban Black Children in Philadelphia: Emphasis on the Frequency of Anemia and Elevated Blood Lead Levels." *Cli Pediatr.* 13:874-888.

Alessio, L., Bertazzi, P.A., Monelli, O., et al. (1976) "Free Erythrocyte Protoporphyrin as an Indicator of the Biological Effect of Lead in Adult Males: II. Comparison Between Free Erythrocyte Protoporphyrin and Other Indicators of Effect." *International Archives of Occupational and Environmental Health.* 37:89-105.

Alomran, A.H., Shleamoon, M.N. (1988) "The Influence of Chronic Lead Exposure on Lymphocyte Proliferative Response and Immunoglobulin Levels in Storage Battery Workers." *Journal of Biological Science Research.* 19:575-585.

Alvares, A.P., Kapelner, S., Sassa, S., et al. (1975) "Drug Metabolism in Normal Children, Lead-Poisoned Children, and Normal Adults." *Clin Pharmacol Ther.* 17:179-183.

Amitai, Y., Brown, M.J., Graef, J.W., Cosgrove, E. (1991) "Residential Deleading: Effects on the Blood Lead Levels of Lead-Poisoned Children." *Pediatrics.* 88(5):893-897.

Angle, C. R., McIntire, M. S., Colucci, A. V. (1974) "Lead in Air, Dustfall, Soil, House Dust, Milk and Water: Correlation with Blood Lead of Urban and Suburban School Children." In: Trace Substances in Environmental Health - VIII, D. D. Hemphill, Ed., 23-29.

Angle, C.R., McIntire, M.S. (1978) "Low Level Lead and Inhibition of Erythrocyte Pyrimidine Nucleotidase." *Environmental Research.* 17:296-302.

Angle, C.R., McIntire, M.S. (1979) "Environmental Lead and Children: The Omaha Study." *Journal of Toxicological and Environmental Health.* 5:855-870.

Angle, C.R., McIntire, M.S., Swanson, M.S., et al. (1982) "Erythrocyte Nucleotides in Children--Increased Blood Lead and Cytidine Triphosphate." *Pediatr Res.* 16:331-334.

Angle, C.R., Marcus, A., Cheng, I-H., et al. (1984) "Omaha Childhood Blood Lead and Environmental Lead: A Linear Total Exposure Model." *Environmental Research.* 35:160-170.

Angle, C.R., Kuntzelman, D.R. (1989) "Increased Erythrocyte Protoporphyrins and Blood Lead — A Pilot Study of Childhood Growth Patterns." *J Toxicol Environ Health.* 26:149-156.

Annest, J.L. (1983) "Trends in the Blood-Lead Levels of the U.S. Population: The Second National Health and Nutrition Examination Survey (NHANES II) 1976-1980." In: Lead Versus Health: Sources and Effects of Low Level Lead Exposure, Rutter, M., Russell Jones, R., eds. New York: John Wiley and Sons, 33-58.

Araki, S., Honma, T., Yanagihara, S., et al. (1980) "Recovery of Slowed Nerve Conduction Velocity in Lead-Exposed Workers." 46:151-157.

Arnvig, E., Grandjean, P., Beckmann, J. (1980) "Neurotoxic Effects of Heavy Lead Exposure Determined with Psychological Tests." *Toxicol Lett.* 5:399-404.

Aschengrau, A., Beiser, A., Bellinger, D., Copenhafer, D., Weitzman, M. (1994) "The Impact of Soil Lead Abatement on Urban Children's Blood Lead Levels: Phase II Results from the Boston Lead-in-Soil Demonstration Project." *Environmental Research.* 67:125-148.

Assennato, G., Baser, M., Molinini, R., et al. (1987) "Sperm Count Suppression Without Endocrine Dysfunction in Lead-Exposed Men." *Archives of Environmental Health.* 42:124-127.

ATSDR (1988a) "The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress." U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry.

ATSDR (1988b) "The Silver Creek Mine Tailings Exposure Study, Park City, Utah." Final Report of the Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, June 1988.

ATSDR (1991a) "Child Lead Exposure Study, Leeds, Alabama." Final Report of the Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, September 1991.

ATSDR (1991b) "Philadelphia Neighborhood Lead Study." Final Report of the Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, September 1991.

ATSDR (1992) "ClearCreek/Central City Mine Waste Exposure Study, Part I: Smuggler Mountain Site." Final Report of the Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, September 1992.

ATSDR (1993) "Toxicological Profile for Lead." Final Report of the Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, April, 1993.

Awad El Karim, M.A., Hamed, A.S., Elhaimi, Y.A.A., et al. (1986) "Effects of Exposure to Lead among Lead-Acid Battery Factory Workers in Sudan." *Archives of Environmental Health.* 41:261-265.

Baghurst, P.A., Robertson, E.F., McMichael, A.J., et al. (1987) "The Port Pirie Cohort Study: Lead Effects on Pregnancy Outcome and Early Childhood Development." *Neurotocicology.* 8:395-401.

Baghurst, P.A., McMichael, A.J., Wigg, N.R., Vimpani, G., Robertson, E.F., Roberts, R.J., Tong, S. (1992) "Life-long Exposure to Environmental Lead and Children's Intelligence at Age Seven: The Port Pirie Cohort Study." *New England Journal of Medicine*. 327:1279-1284.

Baker, E.L. Jr., Landrigan, P.J., Barbour, A.G., et al. (1979) "Occupational Lead Poisoning in the United States: Clinical and Biochemical Findings Related to Blood Lead Levels." *British Journal of Industrial Medicine*. 36:314-322.

Baker, E.L., Feldman, R.G., White, R.F., et al. (1983) "The Role of Occupational Lead Exposure in the Genesis of Psychiatric and Behavioral Disturbances." *Acta Phsychiatr Scand Suppl*. 67:38-48.

Baloh, R.W., Spivey, G.H., Brown, C.P., et al. (1979) "Subclinical Effects of Chronic Increased Lead Absorption — A Prospective Study: II. Results of Baseline Neurologic Testing." *Journal of Occupational Medicine*. 21:490-496.

Barry, P.S.I., Mossman, D.B. (1970) "Lead Concentration in Human Tissues." *British Journal of Industrial Medicine*. 27:339-351.

Barry, P.S.I. (1975) "A Comparison of Concentrations of Lead in Human Tissues." *British Journal of Industrial Medicine*. 32:119-139.

Barry, P.S.I. (1981) "Concentrations of Lead in the Tissues of Children." *British Journal of Industrial Medicine*. 38:61-71.

Batschelet, E., Brand, L., Steiner, A. (1979) "On the Kinetics of Lead in the Human Body." *J. Math Biology*. 8:15-23.

Battelle (1995) "Studies of the Lead Problem in Paint, Dust, and Soil - Volume II: Appendices." Final Report, September 1995.

Battelle (1995) "A Summary of the Relationships Between Blood Lead and Lead-Contaminated Soil and Lead-Contaminated Dust, as Reported in the Scientific Literature." November 1995.

Batuman, V., Maesaka, J.K., Haddad, B., et al. (1981) "The Role of Lead in Gout Nephropathy." *New England Journal of Medicine*. 304:520-523.

Batuman, V., Landy, E., Maesaka, J.K., et al. (1983) "Contribution of Lead to Hypertension with Renal Impairment." *New England Journal of Medicine*. 309:17-21.

Bauchinger, M., Dresp, J., Schmid, E., et al. (1977) "Chromosome Analyses of Children After Ecological Lead Exposure." *Mut. Res.* 56:75-79.

Bellinger, D.C., Needleman, H.L. (1983) "Lead and the Relationship Between Maternal and Child Intelligence." *Journal of Pediatrics*. 102:523-527.

Bellinger, D.C., Needleman, H.L., Leviton, A., et al. (1984) "Early Sensory-Motor Development and Prenatal Exposure to Lead." *Neurobehav Toxicol Teratol*. 6:387-402.

Bellinger, D.C., Leviton, A., Waternaux, C., et al. (1985a) "A Longitudinal Study of the Developmental Toxicity of Low-Level Lead Exposure in the Prenatal and Early Postnatal Periods." In: Lekkas TD, ed. International Conference on Heavy Metals in the Environment, Athens, Greece, September, Vol 1. Edinburgh, United Kingdom: CEP Consultants, Ltd, 32-34.

Bellinger, D.C., Leviton, A., Waternaux, C., et al. (1985b) "Methodological Issues in Modeling the Relationship Between Low-Level Lead Exposure and Infant Development: Examples from the Boston Lead Study." *Environmental Research*. 38:119-129.

Bellinger, D.C., Leviton, A., Needleman, H.L., et al. (1986a) "Low-Level Lead Exposure and Infant Development in the First Year." *Neurobehav Toxicol Teratol*. 8:151-161.

Bellinger, D.C., Leviton, A., Rabinowitz, M., et al. (1986b) "Correlates of Low-Level Lead Exposure in Urban Children at Two Years of Age." *Pediatrics*. 77:826-833.

Bellinger, D., Needleman, H.L., Bromfield, R., Mikntz, M. (1986c) "A Follow-up Study of the Academic Attainment and Classroom Behavior of Children with Elevated Dentine Lead Levels." *Bio Trace Elem Res*. 6:207-23.

Bellinger, D.C., Leviton, A., Waternaux, C., et al. (1987a) "Longitudinal Analyses of Prenatal and Postnatal Lead Exposure and Early Cognitive Development." *New England Journal of Medicine*. 316:1037-1043.

Bellinger, D., Sloman, J., Leviton, A., et al. (1987b) "Low Level Lead Exposure and Child Development: Assessment at Age 5 of a Cohort Followed from Birth." In: Lindberg, S.E., Hutchinson, T.C., eds. International Conference on Heavy Metals in the Environment. New Orleans, LA, September, Vol. 1. Edinburgh, UK: CEP Consultants, Ltd., 49-53.

Bellinger, D., Leviton, A., Waternaux, C., et al. (1989) "Low-Level Lead Exposure and Early Development in Socioeconomically Advantaged Urban Infants." In: Smith M., Grant L.D. Sors, A., eds. Lead Exposure and Child Development: An International Assessment. Lancaster, UK: Kluwer Academic Publishers.

Bellinger, D.C., Leviton, A., Waternaux, C., et al. (1989b) "Low-level Lead Exposure, Social Class, and Infant Development." *Neurotoxicol Teratol*. 10:497-504.

Bellinger, D., Sloman, J., Leviton, A., Rabinowitz, M., Needleman H.L., Waternaux, C. (1991) "Low-level Lead Exposure and Children's Cognitive Function in the Preschool Years." *Pediatrics*. 87(2):219-227.

Bellinger, D., Stiles, K.M., Needleman, H. (1992) "Low-level Lead Exposure, Intelligence, and Academic Achievement: a Long-term Follow-up Study." *Pediatrics*. 90(6):855-861.

Bellinger, D.C. (1995) "Interpreting the Literature on Lead and Child Development: The Neglected Role of the 'Experimental System'." *Neuratoxicology and Teratology*. 17(3):201-212.

Bergomi, M., Borella, P., Fantuzzi, G., et al. (1989) "Relationship Between Lead Exposure Indicators and Neuropsychological Performance in Children." *Dev Med Child Neurol.* 31:181-190.

Bernard, B.P., Becker, C.E. (1988) "Environmental Lead Exposure and the Kidney." *Clin Toxicol.* 26:1-34.

Bert, J.L., van Dusen, L.J., Grace, J.R. (1989) "A Generalized Model for the Prediction of Lead Body Burdens." *Environmental Research.* 48:17-127.

Betts, P.R., Astley, R., Raine, D.N. (1973) "Lead Intoxication in Children in Birmingham." *British Medical Journal.* 1:402-406.

Biagini, G., Caudarella, R., Vangelista, A. (1977) "Renal Morphological and Functional Modification in Chronic Lead Poisoning." In: Brown, S.S., ed. Clinical Chemistry and Chemical Toxicology of Metals. Elsevier/North-Holland Biomedical Press, 123-126.

Bornschein, R.L., Hammond, P.B., Dietrich, K.N., Succop, P.A., Krafft, K.M., Clark, C.S., Pearson, D., Que Hee, S. (1985a) "The Cincinnati Prospective Study of Low-Level Lead Exposure and Its Effects on Child Development: Protocol and Status Report." *Environmental Research* 38:4-18.

Bornschein, R.L., Succop, P.A., Dietrich, R.N., Clark, C.S., Que Hee, S., Hammond, P.B. (1985b) "The Influence of Social and Environmental Factors on Dust Lead, Hand Lead, and Blood Lead Levels in Young Children." *Environmental Research* 38:108-118.

Bornschein, R.L., Succop, P.A., Krafft, K.M., Clark, C.S., Peace, B., Hammond, P.B. (1986). "Exterior Surface Dust Lead, Interior House Dust Lead and Childhood Lead Exposure in an Urban Environment." In: Trace Substances in Environmental Health II, 1986, A Symposium, D.D. Hemphill, ed. Columbia, MO: University of Missouri.

Bornschein, R.L., Clark, S., Grote, J., Peace, B., Roda, S., Succop, P. (1988) "Soil Lead-Blood Lead Relationship in a Former Lead Mining Town." In: Lead in Soil: Issues and Guidelines, Supplement to Volume 9 of Environmental Geochemistry and Health, Devis, B.E., and Wixson, B.G., Eds., 149-160.

Bornschein, R.L., Grote, J., Mitchell, T., et al. (1989) "Effects of Prenatal Lead Exposure on Infant Size at Birth." In: Smith, M., Grand, L.D., Sors, A., eds. Lead Exposure and Child Development: An International Assessment. Lancaster, UK: Kluwer Academic Publishers.

Bornschein, R., Clark, S., Pan, W., Succop, P. (1990) "Midvale Community Lead Study." Final Report of the University of Cincinnati Medical Center, July 1990.

Bradley, J.E., Powell, A.E., Niermann, W., et al. (1956) "The Incidence of Abnormal Blood Levels of Lead in a Metropolitan Pediatric Clinic: With Observation on the Value of Coproporphyrinuria as a Screening Test." *Journal of Pediatrics*. 49:1-6.

Bradley, J.E., Baumgartner, R.J. (1958) "Subsequent Mental Development of Children with Lead Encephalopathy, as Related to Type of Treatment." *Journal of Pediatrics*. 53:311-315.

Braunstein, G.D., Dahlgren, J. Loriaux, D.L. (1978) "Hypogonadism in Chronically Lead-poisoned Men." *Infertility*. 1:33-51.

Brody, D. J., Pirkle, J.L., Kramer, R. A., Flegal, K.M., Matte, T. D., Gunter, E.W., Paschal, D.C. (1994) "Blood Lead Levels in the US Population: Phase I of the Third National Health and Nutritional Examination Survey (NHANES III, 1988 to 1991)," *Journal of the American Medical Association*. 272(4):277-283.

Buc, H.A., Kaplan, J.C. (1978) "Red-cell Pyrimidine 5'-nucleotidase and Lead Poisoning." *Clin Chim Acta*. 87:49-55.

Bureau of the Census, HUD (1995). *American Housing Survey for the United States in 1993*. Current Housing Reports, No. H150/93. Bureau of the Census, U.S. Department of Commerce; and the Office of Policy Development and Research, U.S. Department of Housing and Urban Development, February, 1995.

Bureau of the Census, HUD (1996). *Housing Completions*, December 1995/January 1996. C22/96-1, March 1996.

Butte-Silver Bow Department of Health, and Department of Environmental Health, University of Cincinnati. (1991) "The Butte-Silver Bow Environmental Health Lead Study." Draft Final Report, 10 June 1991.

Campara, P., D'Andrea, F., Micciolo, R., et al. (1984) "Psychological Performance of Workers With Blood-lead Concentration Below the Current Threshold Limit Value." *International Archives of Occupational Environmental Health*. 53:233-246.

Centers for Disease Control (1985) "Preventing Lead Poisoning in Young Children." Altanta, GA: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control. Publication No. 99-2230, 7-19.

Centers for Disease Control (1991) "Preventing Lead Poisoning in Young Children: A Statement by the Centers for Disease Control." Public Health Service, Centers for Disease Control and Prevention, U.S. Department of Health and Human Services, October 1991.

Centers for Disease Control (1992) "Sample Design: Third National Health and Nutrition Examination Survey. Series 2: Data Evaluation and Methods Research." National Center for Health Statistics, Centers for Disease Control and Prevention, U.S. Department of Health and Human Services, DHHS Publication No. (PHS) 92-1887.

Centers for Disease Control (1994) "Plan and Operation of the Third National Health and Nutrition Examination Survey, 1988-94." National Center for Health Statistics, Centers for Disease Control and Prevention, U.S. Department of Health and Human Services, DHHS Publication No. (PHS) 94-1308.

Charney, E., Kessler, B., Farfel, M., Jackson, D. (1983) "Childhood Lead Poisoning: a Controlled Trial of the Effect of Dust-Control Measures on Blood Lead Levels." *New England Journal of Medicine*. 309:1089-1093.

Chisolm, J.J., Jr., Harrison, H.E. (1956) "The Exposure of Children to Lead." *Pediatrics*. 18:943-958.

Chisolm, J.J., Jr. (1962) "Aminoaciduria as a Manifestation of Renal Tubular Injury in Lead Intoxication and a Comparison with Patterns of Aminoaciduria Seen in Other Diseases." *Journal of Pediatrics*. 60:1-17.

Chisolm, J.J., Jr. (1965) "Chronic Lead Intoxication in Children." *Dev Med Child Neurol*. 7:529-536.

Chisolm, J.J., Thomas, D.J., Hammill, T.G. (1985) "Erythrocyte Porphobilinogen Synthase Activity as an Indicator of Lead Exposure to Children." *Clin Chem*. 31:601-605.

Chowdhury, A.R., Chinoy, N.J., Gautam, A.K., et al. (1986) "Effect of Lead on Human Semen." *Adv Contracept Deliv Syst*. 2:208-211.

Christoffersson, J.O., Ahlgren, L., Schutz, A., Skerfving, S., Mattsson, S. (1986) "Decrease of Skeletal Lead Levels in Man after End of Occupational Exposure." *Archives of Environmental Health*. 41:5:312-318.

Clark, C.S., Bornschein, R.L., Succop, P., Que Hee, S.S., Hammond, P.B., Peace, B. (1985) "Condition and Type of Housing as an Indicator of Potential Lead Exposure and Pediatric Blood Lead Levels." *Environmental Research*. 38:46-53.

Coate, D., Fowles, R. (1989) "Is There Statistical Evidence for a Blood Lead-Blood Pressure Relationship?" *Journal of Economics*. 8:173-184.

Cohen, N., Jaakkola, T., Wrenn, M.E. (1973) "Lead-210 Concentrations in the Bone, Blood, and Excreta of a Former Uranium Miner." *Health Physics*. 24:601-609.

Colorado Department of Health, University of Colorado at Denver, and ATSDR (1990) "Leadville Metals Exposure Study." Final Report by the Colorado Department of Health, Division of Disease Control and Environmental Epidemiology; University of Colorado at Denver, Center for Environmental Sciences; and the Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, April, 1990.

Cooney, G.H., Bell, A., McBride, W., et al. (1989a) "Low-Level Exposures to Lead: The Sydney Lead Study." *Dev Med Child Neurol*. 31:640-649.

Cooney, G., Bell, A., Stavron, C. (1991) "Low Level Exposures to Lead and Neurobehavioural Development: the Sydney Study at Seven Years." *Heavy Metals in the Environment.* Edinburgh: CEP Consultants, 1991:16-9.

Cooper, W.C., Gaffey, W.R. (1975) "Mortality of Lead Workers." *Journal of Occupational Medicine*. 17:100-107.

Cooper, W.C. (1976) "Cancer Mortality Patterns in the Lead Industry." Ann, NY *Acad Sci.* 271:250-259.

Cooper, W.C., Wong., O., Kheifets., L. (1985) "Mortality Among Employees of Lead Battery Plants and Lead Producing Plants, 1947-1980." *Scand J Work Environ Health.* 11:331-345.

Cooper, W.C. (1988) "Deaths from Chronic Renal Disease in U.S. Battery and Lead Production Workers." *Environmental Health Perspectives*. 78:61-63.

Copley, C. G. (1983) "The Effect of Lead Hazard Source Abatement and Clinic Appointment Compliance on the Mean Decrease of Blood Lead and Zinc Protoporphyrin Levels. Mimeo." City of St. Louis, Department of Health and Hospitals, Division of Health, Office of the Health Commissioner, St. Louis, MO.

Cramer, K., Goyer, R.A., Jagenburg, R., et al. (1974) "Renal Ultrastructure, Renal Function, and Parameters of Lead Toxicity in Workers with Different Periods of Lead Exposure." *British Journal of Industrial Medicine*. 31:113-127.

Cristy, M., Leggett, R.W., Dunning, D.E., Jr., Eckerman, K.F. (1986) "Relative Age-Specific Radiation Dose Commitment Factors for Major Radionuclides Released from Nuclear Fuel Facilities." NUREG/CR-4628 ORNL/TM-9890.

Cullen, M.R., Kayne, R.D., Robins, J.M. (1984) "Endocrine and Reproductive Dysfunction in Men Associated with Occupational Inorganic Lead Intoxication." *Archives of Environmental Health*. 39:431-440.

Dalpra, L., Tibiletti, M.G., Nocera, G., et al. (1983) "SCE Analysis in Children Exposed to Lead Emission from a Smelting Plant." *Mut Res.* 120:249-256.

Davies, D.J., Watt, J.M., Thornton, I. (1987) "Lead Levels in Birmingham Dusts and Soils." *The Science of the Total Environment.* 87(2-3):177-185.

Davies, D.J.A, Thornton, I., Watt, J.M., Culbard, E.B., Harvey, P.G., Delves, H.T., Sherlock, J.C., Smart, G.A., Thomas, J.F.A., Quinn, M.J. (1990) "Lead Intake and Blood Lead in Two-Year-Old U.K. Urban Children." *The Science of the Total Environment.* 90:13-29.

Davis, J.M., Svendsgaard, D.J. (1987) "Lead and Child Development." *Nature.* 329:297-300.

Day, J.C. (1993) *Population Projections of the United States, by Age, Sex, Race, and Hispanic Origin: 1993 to 2050.* U.S. Bureau of the Census, Current Population Reports P25-1104.

de la Burde, B., Choate, M.S., Jr. (1972) "Does Asymptomatic Lead Exposure in Children Have Latent Sequelae?" *Journal of Pediatrics.* 81:1088-1091.

de la Burde, B., Choate, M.S., Jr. (1975) "Early Asymptomatic Lead Exposure and Development at School Age." *Journal of Pediatrics.* 87:638-642.

deKort, W.L.A.M., Verschoor, M.A., Wibowo, A.A.E., et al. (1987) "Occupational Exposure to Lead and Blood Pressure: A Study of 105 Workers." *American Journal of Industrial Medicine.* 11:145-156.

Dietrich, K.N., Krafft, K.M., Bier, M., et al. (1986) "Early Effects of Fetal Lead Exposure: Neurobehavioral Findings at 6 Months." *International Journal of Biosocial and Medical Record.* 8:151-168.

Dietrich, K.N., Krafft, K.M., Bornschein, R.L., et al. (1987a) "Low-Level Fetal Lead Exposure Effect on Neurobehavioral Development in Early Infancy." *Pediatrics.* 80:721-730.

Dietrich, K.N., Krafft, K.M., Shukla, R., et al. (1987b) "The Neurobehavioral Effects of Early Lead Exposure." *Monogr Am Assoc Ment Defic.* 8:71-95.

Dietrich, K.N., Berger, O.G., Succop, P.A., Hammond, P.B., Bornschein, I. (1993) "The Developmental Consequences of Low To Moderate Prenatal and Postnatal Lead Exposure: Intellectual Attainment in the Cincinnati Lead Study Cohort Following School Entry." *Neurotoxicology and Teratology.* 15:37-44.

Drasch, G.A., Kretschmer, E., Lochner, C. (1988) "Lead and Sudden Infant Death: Investigations on Blood Samples of SID Babies." *Eur J Pediatr.* 147:79-84.

Elwood:, P.C., Davey-Smith, G., Oldham, P.D., et al. (1988) "Two Welsh Surveys of Blood Lead and Blood Pressure." *Environmental Health Perspectives.* 78:119-121.

Erenberg, G., Rinsler, S.S., Fish, B.G. (1974) "Lead Neuropathy and Sickle Cell Disease." *Pediatrics.* 54:438-441.

Ernhart, C.B., Landa, B., Schell, N.B. (1981) "Subclinical Levels of Lead and Developmental Deficit — A Multivariate Follow-up Reassessment." *Pediatrics.* 67:911-919.

Ernhart, C.B., Wolf, A.W., Kennard, M.J., et al. (1985) "Intrauterine Lead Exposure and the Status of the Neonate." In: Lekkas TD, ed. International Conference on Heavy Metals in the Environment, Athens, Greece, September. Vol. 1. Edinburgh, United Kingdom: CEP Consultants, Ltd. 35-37.

Ernhart, C.B., Wolf, A.W., Kennard, M.J., et al. (1986) "Intrauterine Exposure to Low Levels of Lead: The Status of the Neonate." *Archives of Environmental Health.* 41:287-291.

Ernhart, C.B., Morrow-Tlucak, M., Marler, M.R., et al. (1987) "Low Level Lead Exposure in the Prenatal and Early Preschool Periods: Early Preschool Development." *Neurotoxicol Teratol.* 9:259-270.

Ernhart, C.B. (1988) "Cofactors in Research on the Environmental Toxicology of Childhood: Issues and Examples from Lead Effects Studies". In: Environmental Toxicology of Childhood, University of Nebraska, Children and the Law Series.

Ernhart, C.B., Morrow-Tlucak, M., Worf, A.W., Super, D., Drotar, D. (1989) "Low Level Lead Exposure in the Prenatal and Early Preschool Periods; Intelligence Prior to School Entry." *Neurotoxicology and Teratology.* 11:161-70.

Ernhart, C.B., Green, T. (1990) "Low-Level Lead Exposure in Prenatal and Early Preschool Periods: Language Development." *Archives of Environmental Health.* 45:342-354.

Ewers, U., Stiller-Winkler, R., Idel, H. (1982) "Serum Immunoglobulin, Complement C3, and Salivary IgA Level in Lead Workers." *Environmental Research.* 29:351-357.

Factor-Litvak, P., Graziano, J.H., Kline, J.K., et al. (1991) "A Prospective Study of Birthweight and Length of Gestation in Population Surrounding a Lead Smelter in Kosovo, Yugoslavia." *Int J Epidemiol.* 20:772-728.

Fanning, D. (1988) "A Mortality Study of Lead Workers, 1926-1985." *Archives of Environmental Health.* 43:247-251.

Farfel, M.R., Chisolm, J.J., Jr. (1990) "Health and Environmental Outcomes of Traditional and Modified Practices for Abatement of Residential Lead-Based Paint." *American Journal of Public Health.* 80(10):1240-1245.

Farfel, M.R., Lim, B.S. (1995) "The Lead Paint Abatement and Repair and Maintenance Study in Baltimore." In: Lead in Paint, Soil, and Dust: Health Risks, Exposure Studies, Control Measures, Measurement Methods, and Quality Assurance, ASTM STP 1226, Michael E. Beard and S. D. Allen Iske, Eds. Philadelphia: American Society for Testing and Materials, 107-118.

Fayerweather, W.E., Karns, M.E., Nuwayhid, I.A., et al. (1991) "An Epidemiologic Study of Cancer Risk Following Exposure to Organic Lead Among the DuPont Company's Chamber Works Employees." DuPont Company, Human Resources, Epidemiology Section Medical Division, Wilmington, DE.

Fulton, M., Raab, G., Thomson, G., Laxen, D., Hunter, R., Hepburn, W. (1987) "Influence of Blood Lead on the Ability and Attainment of Children in Edinburgh." *Lancet.* 1:1221-1226.

Gant, V.A. (1938) "Lead Poisoning." *Industial Medicine.* 7:679-699.

Gartside, P.S. (1988) "The Relationship of Blood Lead Levels and Blood Pressure in NHANES: II. Additional Getzcalculations." *Environmental Health Perspectives.* 78:31-34.

Gerhardsson., L., Lundstrom, N.G., Nordberg, G., et al. (1986b) "Mortality and Lead Exposure: A Retrospective Cohort Study of Swedish Smelter Workers." *British Journal of Industrial Medicine.* 43:707-712.

Glickman, L., Valciukas, J.A., Lilis, R., et al. (1984) "Occupational Lead Exposure: Effects on Saccadic Eye Movements." *International Archives of Occupational Environmental Health.* 54:115-125.

Goyer, R.A. (1993) "Lead Toxicity: Current Concerns." *Environmental Health Perspectives.* 100:177-187.

Grandjean, P., Lintrup, J. (1978) "Erythrocyte-Zn-protoporphyrin as an Indicator of Lead Exposure." *Scand J Clin Lab Invest.* 38:669-675.

Grandjean, P. (1979) "Occupational Lead Exposure in Denmark: Screening with the Haematofluorometer." *British Journal of Industrial Medicine.* 36:52-58.

Grandjean, P., Wulf, H.C., Neibuhr, E. (1983) "Sister Chromatid Exchange in Response to Variations in Occupational Lead Exposure." *Environmental Research.* 32:199-204.

Grandjean, P., Hollnagel, H., Hedegaard, L., et al. (1989) "Blood Lead-Blood Pressure Relation: Alcohol Intake and Hemoglobin as Confounders." *Am J Epidemiol.* 129:732-739.

Greene, T., Ernhart, C.B. (1991) "Prenatal and Preschool Age Lead Exposure: Relationship with Size." *Neurotoxicology and Teratology.* 13:417-427.

Griffin, T.B., Coulston, F., Wills, H. (1975b) "Biological and Clinical Effects of Continuous Exposure to Airborne Particulate Lead." *Arh Hig Toksikol.* 26:191-208. (Yugoslavian)

Gulson, B.L., Mahaffey, K.R., Mizon, K.J., Korsch, M.J., Cameron, M.A., Vimpani, G. (1995) "Contribution of Tissue Lead to Blood Lead in Adult Female Subjects Based on Stable Lead Isotope Methods." *Journal of Lab and Clinical Medicine.* 125:703.

Haas, T., Wieck, A.G., Schaller, K.H., et al. (1972) "The Usual Lead Load in New-born Infants and Their Mothers." *Zentralblatt fur Bakteriologie [B].* 155:341-349. (German)

Haenninen, H., Hernberg, S., Mantere, P., et al. (1978) "Psychological Performance of Subjects with Low Exposure to Lead." *Journal of Occupational Medicine.* 20:683-689.

Haenninen, H., Mantere, P., Hernberg, S., et al. (1979) "Subjective Symptoms in Low-Level Exposure to Lead." *Neurotoxicology.* 1:333-347.

Hammond, P.B., Bornschein, R.L., Succop, P. (1985) "Dose-effect and Dose-response Relationships of Blood Lead to Erythrocytic Protoporphyrin in Young Children." In: Bornschein, R.L., Rabinowitz, M.B., eds. The Second International Conference on Prospective Studies of Lead, Cincinnati, OH: April, 1984. *Environmental Research.* 38:187-196.

Hansen, O.N., Trillingsgaard, A., Beese, I., et al. (1989) "A Neuropsychological Study of Children with Elevated Dentine Lead Level: Assessment of the Effect of Lead in Different Socio-economic Groups." *Neurotoxicol Teratol.* 11:205-213.

Harlan, W.R. (1988) "The Relationship of Blood Lead Levels to Blood Pressure in the US Population. *Environmental Health Perspectives.* 78:9-13.

Harlan, W.R., Landis, J.R., Schmouder, R.L., et al. (1988) "Blood Lead and Blood Pressure: Relationship in the Adolescent and Adult US Population." *Journal of the American Medical Association.* 253:530-534.

Harvey, P.G., Hamlin, M.W., Kumar, R., et al. (1984) "Blood Lead, Behavior and Intelligence Test Performance in Preschool Children." *Sci Total Environ.* 40:45-60.

Harvey, P.G., Hamlin, M.W., Kumar, R,. Morgan, G., Spurgeon, A., Delves, H.T. (1988) "Relationship Between Blood Lead, Behaviour, Psychometric and Neuropsychological Test Performance in Young Children." *British Journal of Develomental Psychology.* 29:43-52.

Hatzakis, A., Kolevi, A., Katsouyanni, K., et al. (1987) "Psychometric Intelligence and Attentional Performance Deficits in Lead Exposed Children." *International Conference on Heavy Metals in the Environment.* Edinburgh, Scotland; CEP Consultants. 16:57-67.

Hatzakis, A., Kokkeni, A., Maranelias, C., Katsouyanni, K., Salaminios, F., Kalandidi, A., et al. (1989) "Psychometric Intelligence Deficits in Lead-exposed Children." In: Smith MA., Grant, I.D., Sora Al, eds. *Lead Exposure and Child Development.* London: Kluwer, 1989:211-23.

Hawk, B.A., Schroeder, S.R., Robinson, G., et al. (1986) "Relation of Lead and Social Factors to IQ of Low-SES Children: A Partial Replication." *Am J Ment Defic.* 91:178-183.

Heard, M.J., Chamberlain, A.C. (1984) "Uptake of Pb by Human Skeleton and Comparative Metabolism of Pb and Alkaline Earth Elements." *Health Physics.* 47:857-865.

Herber, R.F.M. (1980) "Estimation of Blood Lead Values from Blood Porphyrin and Urinary 5-aminolevulinic Acid Levels in Workers." *International Archives of Occupational Environmental Health.* 45:169-179.

Hernberg, S., Nikkanen, J., Mellin, G., et al. (1970) "δ-Aminolevulinic Acid Dehydrase as a Measure of Lead Exposure." *Archives of Environmental Health.* 21:140-145.

Hogstedt, C., Hane, M., Agrell, A., et al. (1983) "Neuropsychological Test Results and Symptoms Among Workers with Well-defined Long-term Exposure to Lead." *British Journal of Industrial Medicine.* 40:99-105.

Holness, D.L., Nethercott, J.R. (1988) "Acute Lead Intoxication in a Group of Demolition Workers." *Applied Industrial Hygiene.* 3:338-341.

Hu, H., Pepper, L., Goldman, R. (1991) "Effect of Repeated Occupational Exposure to Lead, Cessation of Exposure, and Chelation on Levels of Lead in Bone." *American Journal of Industrial Medicine.* 20-723-735.

Huang, X.P., Feng, Z.Y., Zhai, W.L., et al. (1988b) "Chromosomal Aberrations and Sister Chromatid Exchanges in Workers Exposed to Lead." *Biomed Environ Sci.* 1:382-387.

Hyrhorczuk, D., Rabinowitz, M., Hessel, S., Hoffman, D., Hogan, M., Mallin, K., Finch, H., Orris, P., Berman, E. (1985) "Elimination Kinetics of Blood Lead in Workers with Chronic Lead Intoxication." *American Journal of Industrial Medicine.* 8:33-42.

ICF (1995) "Regulatory Impact Analysis of the Proposed Rule on Lead-Based Paint: Requirements for Notification, Evaluation and Reduction of Lead-Based Paint Hazards in Federally-Owned Residential Property and Housing Receiving Federal Assistance." Draft report for the Office of Lead-Based Paint Abatement and Poisoning Prevention, U.S. Department of Housing and Urban Development.

Janin, Y., Couinaud, C., Stone, A., et al. (1985) "The 'Lead-Induced Colic' Syndrome in Lead Intoxication." *Surg Ann.* 17:287-307.

Johnson, N.E., Renuta, K. (1979) "Diets and Lead Blood Levels of Children Who Practice Pica." *Environmental Research.* 18:869-376.

Kang, H.K., Infante, P.F., Carra, J.S. (1980) "Occupational Lead Exposure and Cancer (Letter)." *Science.* 207:935-936.

Kehoe, R.A. (1961) "The Metabolism of Lead in Man in Health and Disease: Present Hygienic Problems Relating to the Absorption of Lead: The Harben Lectures, 1960." *J R Inst Public Health Hyg.* 24:177-203.

Khera, A.K., Wibberley, D.G., Edwards, K.W., et al. (1980b) "Cadmium and Lead Levels in Blood and Urine in a Series of Cardiovascular and Normotensive Patients." *International Journal of Environmental Studies.* 14:309-312.

Kimbrough, R.D., LeVois, M., Webb, D.R. (1994) "Management of Children with Slightly Elevated Blood Lead Levels." *Pediatrics.* 93(2):188-191.

Kirkby, H., Gyntelberg, F. (1985) "Blood Pressure and Other Cardiovascular Risk Factors of Long-term Exposure to Lead." *Scand J Work Environ Health.* 11:15-19.

Koo, W.W.R., Succop, P.A., Bornschein, R.L., et al. (1991) "Serum Vitamin D Metabolites and Bone Mineralization in Young Children with Chronic Low to Moderate Lead Exposure." *Pediatrics.* 87:680-687.

Kosmider, S., Petelenz, T. (1962) "Electrocardiographic Changes in Elderly Patients with Chronic Professional Lead Poisoning." *Pol Arch Med Wewn.* 32:437-442. (Polish)

Kotok, D. (1972) "Development of Children with Elevated Blood Levels: A Controlled Study." *Journal of Pediatrics.* 80:57-61.

Kotok, D., Kotok, R., Heriot, T. (1977) "Cognitive Evaluation of Children with Elevated Blood Lead Levels." *Am J Dis Child.* 131:791-793.

Kuhnert, P.M., Erhard, P., Kuhnert, B.R. (1977) "Lead and δ-aminoluvulinic Acid Dehydratase in RBC's of Urban Mothers and Fetuses." *Environmental Research.* 14:73-80.

Kumar, S., Jain, S., Aggarwal, C.S., et al. (1987) "Encephalopathy Due to Inorganic Lead Exposure in an Adult." *Jpn J Med.* 26:253-254.

Lancranjan, I., Popescu, H.I., Gavanescu, O., et al. (1975) "Reproductive Ability of Workmen Occupationally Exposed to Lead." *Archives of Environmental Health.* 30:396-401.

Landis, J.R., Flegal, K.M. (1988) "A Generalized Mantel-Haenszel Analysis of the Regression of Blood Pressure on Blood Lead Using NHANES II Data." *Environmental Health Perspectives.* 78:35-41.

Landrigan, P.J., Gehlbach, S.H., Rosenblum, B.F., Shoults, J.M., Candelaria, R.M., Barthel, W.F., Liddle, J.A., Smrek, A.L., Staehling, N.W., Sanders, J.F. (1975). "Epidemic Lead Absorption Near an Ore Smelter: The Role of Particulate Lead." *New England Journal of Medicine.* 292(3):123-129.

Landrigan, P.J., Baker, E.L., Jr., Feldman, R.G., et al. (1976) "Increased Lead Absorption with Anemia and Slowed Nerve Conduction in Children Near a Lead Smelter." *Journal of Pediatrics.* 89:904-910.

Lanphear, B.P., Emond, M., Jacobs, D.E., Weitzman, M., Tanner, M., Winter, N.L., Yakir, B., Eberly, S. (1995) "A Side-by-Side Comparison of Dust Collection Methods for Sampling Lead-Contaminated House Dust." *Environmental Research.* 68(2):114-123.

Lansdown, R., Yule, W., Urbanowicz, M.A., et al. (1986) "The Relationship Between Blood Lead Concentrations, Intelligence, Attainment and Behavior in a School Population: The Second London Study." *International Archives of Occupational Environmental Health.* 57:225-235.

Lauwers, M.C., Hauspie, R.C., Susanne, C., et al. (1986) "Comparison of Biometric Data of Children with High and Low Levels of Lead in the Blood." *Am J Phys Anthropol.* 69:107-116.

Lauwerys, R., Buchet, J-P, Roels, H.A., et al. (1974) "Relationship Between Urinary δ-aminolevulinic Acid Excretion and the Inhibition of Red Cell δ-aminolevulinate Dehydratase by Lead." *Clin Toxicol.* 7:383-388.

Lauwerys, R., Buchet, J-P, Roels, H.A., et al. (1978) "Placental Transfer of Lead, Mercury, Cadmium, and Carbon Monoxide in Women: I. Comparison of the Frequency Distributions of the Biological Indices in Maternal and Umbilical Cord Blood." *Environmental Research.* 15:278-289.

Leal-Garza, C., Moates, De Oca R, Cerda-Flores, R.M., et al. (1986) "Frequency of Sister-Chromatid Exchanges (SCE) in Lead Exposed Workers." *Arch Invest Med.* 17:267-276.

Leggett, R.W., Eckerman, K.F., and Williams, L.R. (1982) "Strontium-90 in Bone: A Case Study in Age-Dependent Dosimetric Modeling." *Health Physics*. 43(3):307-322.

Lewis and Clark County Health Department, Montana Department of Health and Environmental Sciences, Centers for Disease Control, U.S. Department of Health and Human Services, and U.S. EPA (1986) "East Helena, Montana: Child Lead Study, Summer 1983." Final Report, July 1986.

Lilis, R., Gavrilescu, N., Nestorescu, B., et al. (1968) "Nephropathy in Chronic Lead Poisoning." *British Journal of Industrial Medicine.* 25:196-202.

Lilis, R., Eisinger, J., Blumberg, W., et al. (1978) "Hemoglobin, Serum Iron, and Zinc Protoporphyrin in Lead-exposed Workers." *Environmental Health Perspectives.* 25:97-102.

Lyngbye, T., Hansen, O.N., Grandjean, P. (1987) "The Influence of Environmental Factors on Physical Growth in School Age: A Study of Low Level Lead Exposure." In: Lindberg S.E., Hutchinson, T.C., eds. International Conference on Heavy Metals in the Environment, Vol. 2, New Orleans, LA, September. Edinburgh, UK: CEP Consultants, Ltd. 210-212.

Mahaffey, K.R. (1977) "Quantities of Lead Producing Health Effects in Humans: Sources and Bioavailability." *Environmental Health Perspectives.* 19:285-295.

Mahaffey, K.R., Rosen, J.F., Chesney, R.W., et al. (1982) "Association Between Age, Blood Lead Concentration, and Serum 1,25-dihydroxycholecalciferol Levels in Children." *Am J Clin Nutr.* 35:1327-1331.

Mahaffey, K.R., Annest, J.L. (1986) "Association of Erythrocyte Protoporphyrin with Blood Lead Level and Iron Status in the Second National Health and Nutrition Examination Survey, 1976-1980." *Environmental Research.* 41-327-338.

Mahaffey, K.R. (1994) "Analysis of Epidemiological Data on Association between Blood Lead and Lead in Soil and Dust for OPPTS/EPA." February 1994.

Maki-Paakkanen, J., Sorsa, M., Vainio, H. (1981) "Chromosome Aberrations and Sister Chromatid Exchanges in Lead-Exposed Workers." *Hereditas.* 94:269-275.

Malcolm, D., Barnett, H.A.R. (1982) "A Mortality Study of Lead Workers: 1925-76." *British Journal of Industrial Medicine.* 39:404-410.

Mantere, P., Haenninen, H., Hernberg, S. (1982) "Subclinical Neurotoxic Lead Effects: Two-Year Follow-up Studies with Psychological Test Methods." *Neurobehav Toxicol Teratol.* 4:725-727.

Maranelli, G., Apostoli, P. (1987) "Assessment of Renal Function in Lead Poisoned Workers." *Occup Environ Chem Hazards.* 344-348.

Marcus, A.H. (1985) "Multicompartment Kinetics Models for Lead. I. Bone Diffusion Models for Long-term Retention." *Environmental Research.* 36:441-458.

Marcus, A.H., Schwartz, J. (1987) "Dose-response Curves for Erythrocyte Protoporphyrin Vs Blood Lead: Effects of Iron Status." *Environmental Research.* 44:221-227.

Marcus, A.H., Elias, R.W. (1994) "Estimates of Soil and Dust Lead Slope Factors for U. S. Children, Ages 12 to 84 Months, Based on Consistent Analyses of Studies Since 1980." January 1994. DO NOT CITE OR QUOTE.

Marino, P.E., Franzblau, A., Lilis, R., et al. (1989) "Acute Lead Poisoning in Construction Workers: The Failure of Current Protective Standards." *Archives of Environmental Health.* 44:140-145.

Markowitz, M.E., Bijur, P.E., Ruff, H.A., Rosen, J.F. (1993) "Effects of Calcium Disodium Versenate ($Cana_2edta$) Chelation in Moderate Childhood Lead Poisoning." *Pediatrics.* 92(2):265-271 .

Matte, T.D., Figueroa, J.P., Burr, G., et al. (1989) "Lead Exposure Among Lead-acid Battery Workers in Jamaica." *American Journal of Industrial Medicine.* 16:167-177.

McBride, W.G., Black, B.P., English, B.J. (1982) "Blood Lead Levels and Behavior of 400 Preschool Children." *Med J Aust.* 10:2(1):26-29.

McMichael, A.J., Vimpani, G.V., Robertson, E.F., et al. (1986) "The Port Pirie Cohort Study: Maternal Blood Lead and Pregnancy Outcome." *J Epidemiol Community.* 40:18-25.

McMichael, A.J., Baghurst, P.A., Wigg, N.R., et al. (1988) "Port Pirie Cohort Study: Environmental Exposure to Lead and Children's Abilities at the Age of Four Years." *New England Journal of Medicine.* 319:468-476.

Menton, R.G., Burgoon, D.A., Marcus, A.H. (1995) "Pathways of Lead Contamination for the Brugham and Women's Hospital Longitudinal Lead Study." In: Lead in Paint, Soil, and Dust: Health Risks, Exposure Studies, Control Measures, Measurement Methods, and Quality Assurance, ASTM STP 1226, Michael E. Beard and S. D. Allen Iske, Eds. Philadelphia: American Society for Testing and Materials, 92-106.

Meredith, P.A., Moore, M.R., Campbell, B.C., et al. (1978) "δ-aminolaevulinic Acid Metabolism in Normal and Lead-exposed Humans." *Toxicology.* 9:1-9.

Michaels, D., Zoloth, S.R., Stern, F.B. (1991) "Does Low-level Lead Exposure Increase Risk of Death?: A Mortality Study of Newspaper Printers." *Int J Epidemiol.* 20:978-983.

Milburn, H., Mitran, E., Crockford, G.W. (1976) "An Investigation of Lead Workers for Subclinical Effects of Lead Using Three Performance Tests." *Ann Occup Hyg.* 19:239-249.

Moore, M.R., Goldberg, A., Pocock, S.J., et al. (1982) "Some Studies of Maternal and Infant Lead Exposure in Glasgow." *Scott Med J.* 27:113-122.

Muijser, H., Hoogendijk, E.M., Hoosma, J., et al. (1987) "Lead Exposure During Demolition of a Steel Structure Coated with Lead-based Paints. II. Reversible Changes in the Conduction Velocity of the Motor Nerves in Transiently Exposed Workers." *Scand J Work Environ Health.* 13:56-61.

Murphy, M.J., Graziano, J.H., Popovac, D., et al. (1990) "Past Pregnancy Outcomes Among Women Living in the Vicinity of a Lead Smelter in Kosovo, Yugoslavia." *American Journal of Public Health.* 80:33-35.

National Academy of Sciences. (1972) "Lead: Airborne Lead in Perspective: Biologic Effects of Atmospheric Pollutants." Washington, DC: National Academy of Sciences. 71-177, 281-313.

Needleman, H.L., Gunnoe, C., Leviton, A., et al. (1979) "Deficits in Psychologic and Classroom Performance of Children with Elevated Dentine Lead Levels." *New England Journal of Medicine.* 300:689-695.

Needleman, H.L., Rabinowitz, M., Leviton, A., et al. (1984) "The Relationship Between Prenatal Exposure to Lead and Congenital Anomalies." *Journal of the American Medical Association.* 251:2956-2959.

Needleman, H.L., Geiger, S.K., Frank, R. (1985) "Lead and IQ Scores: A Reanalysis (letter)." *Science.* 227:701-704.

Needleman, H., Gatsonis, C. (1990) "Low-Level Lead Exposure and the IQ of Children." *Journal of the American Medical Association.* 263:673-678.

Needleman, H.L., Schell, A., Bellinger, D., Leviton, A., Allred, E.N. (1990) "The Long-Term Effects of Exposure to Low Doses of Lead in Childhood: An 11-Year Follow-up Report." *The New England Journal of Medicine.* 322(2):83-88.

Neri, L.C., Hewitt, D., Orser, B. (1988) "Blood Lead and Blood Pressure: Analysis of Cross-sectional and Longitudinal Data from Canada." *Environmental Health Perspectives.* 78:123-126.

Neter, J. and Wasserman, W. (1974) "Applied Linear Statistical Models." Homewood, IL: Richard D. Irwin, Inc.

Nilsson, U., Attewell, R., Christoffersson, J.O., Schutz, A., Ahlgren, S., Skerfving, S., Mattson, S. (1991) "Kinetics of Lead in Bone and Blood After End of Occupational Exposure." *Pharmacology and Toxicology.* 69:477-484.

Nordberg, G.R., Mahaffey, K.R., and Fowler, B.A. (1991) "Introduction and Summary." International Workshop on Lead in Bone: Implications for Dosimetry and Toxicology. *Environmental Health Perspectives.* 91:3-7.

Nordenson, I., Beckman, G., Beckman, L., et al. (1978) "Occupational and Environmental Risks In and Around a Smelter in Northern Sweden: IV. Chromosomal Aberrations in Workers Exposed to Lead." *Hereditas.* 88:263-267.

Nordstrom, S., Beckman, L., Nordenson, I. (1979) "Occupational and Environmental Risks in and Around a Smelter in Northern Sweden: V. Spontaneous Abortion Among Female Employees and Decreased Birth Weight in Their Offspring." *Hereditas.* 90:291-296.

Nye, L.J.J. (1929) "An Investigation of the Extraordinary Incidence of Chronic Nephritis in Young People in Queensland." *Med J Aust.* 2:145-159.

Ong, C.N., Endo, G., Chia, K.S., et al. (1987) "Evaluation of Renal Function in Workers with Low Blood Lead Levels." In: Fao V., Emmett, E.A., Maroni, M., et al., eds. *Occupational and Environmental Chemical Hazards.* Chichester: Ellis Horwood Limited, 327-333.

O'Riordan, M.L., Evans, H.J. (1974) "Absence of Significant Chromosome Damage in Males Occupationally Exposed to Lead." *Nature.* 247:50-53.

Paglia, D.E., Valentine, W.N., Dahlgren, J.G. (1975) "Effects of Low-level Lead Exposure on Pyrimidine 5'-nucleotidase and Other Erythrocyte Enzymes: Possible Role of Pyrimidine 5'-nucleotidase in the Pathogenesis of Lead-induced Anemia." *J Clin Invest.* 56:1164-1169.

Paglia, D.E., Valentine, W.N., Fink, K. (1977) "Lead Poisoning: Further Observations on Erythrocyte Pyrimidine-nucleotidase Deficiency and Intracellular Accumulation of Pyrimidine Nucleotides." *J Clin Invest.* 60:1362-1366.

Pagliuca, A., Mufti, G.J., Baldwin, D., et al. (1990) "Lead Poisoning: Clinical, Biochemical, and Hematological Aspects of a Recent Outbreak." *J Clin Path.* 43:277-281.

Panhandle District Health Department, Idaho Department of Health and Welfare, Centers for Disease Control, and U.S. EPA. (1986) "Kellogg Revisited - 1983: Childhood Blood Lead and Environmental Status Report." Final Report of the U.S. Public Health Service, July 1986.

Parkinson, D.K., Ryan, C., Bormet, J., et al. (1986) "A Psychiatric Epidemiologic Study of Occupational Lead Exposure." *Am J Epidemiol.* 123:261-269.

Parkinson, D.K., Hodgson, M.J., Bromet, E.J., et al. (1987) "Occupational Lead Exposure and Blood Pressure." *British Journal of Industrial Medicine.* 44:744-748.

Pasternak, G., Becker, C.E., Lash, A., et al. (1989) "Cross-sectional Neurotoxicology Study of Lead-exposed Cohort." *Clin Toxicol.* 27:37-51.

Piomelli, S., Seaman, C., Zullow, D., et al. (1982) "Threshold for Lead Damage to Heme Synthesis in Urban Children." *Proc Natl Acad Sci.* 7:3335-3339.

Pirkle, J.L., Schwartz, J., Landis, J.R., et al. (1985) "The Relationship Between Blood Lead Levels and Blood Pressure and its Cardiovascular Risk Implications." *Am J Epidemiol.* 121:246-258.

Pirkle, J.L., Brody, D.J., Gunter, E.W., Kramer, R.A., Paschal, D.C., Flegal, K.M., Matte, T.D. (1994) "The Decline in Blood Lead Levels in the United States: The National Health and Nutrition Examination Surveys (NHANES)." *Journal of the American Medical Association.* 272(4):284-291.

Pocock, S.J., Shaper, A.G., Ashby, D., et al. (1985) "Blood Lead and Blood Pressure in Middle-aged Men." In: Lekkas, T.D., ed. International Conference on Heavy Metals in the Environment, Vol. 1, Athens, Greece, Sepember. Edinburgh, United Kingdom: CEP Consultants, Ltd., 303-305.

Pocock, S.J., Shaper, A.G., Ashby, D., et al. (1988) "The Relationship Between Blood Lead, Blood Pressure, Stroke, and Heart Attacks in Middle-aged British Men." *Environmental Health Perspectives.* 78:23-30.

Pocock, S.J., Ashby, D., Smith, M.A. (1989) "Lead Exposure and Children's Intellectual Performance: The Institute of Child Health/Southampton Study." In: Smith, M., Grant, L.D., Sors, A., eds. Lead Exposure and Child Development: An International Assessment. Lancaster, UK: Kluwer Academic Publishers.

Pocock, S.J., Smith, M., Baghurst, P. (1994) "Environmental Lead and Children's Intelligence: A Systematic Review of the Epidemiological Evidence." *BMJ.* 309:1189-1197.

Pollock, C.A., Ibels, L.S. (1986) "Lead Intoxication in Paint Removal Workers on the Sidney Harbour Bridge." *Med J Aust.* 145:635-639.

Popcock, S.J., Shaper, A.G., Ashby, D., et al. (1984) "Blood Lead Concentration, Blood Pressure, and Renal Function." *Br Med J.* 289:872-874.

Pueschel, S.M., Kopito, L., Schwachman, H. (1972) "Children with an Increased Lead Burden: A Screening and Follow-up Study." *Journal of the American Medical Association.* 222:462-466.

Que Hee, S.S., Peace, B., Clark, S., Boyle, J.R., Bornschein, R.L., Hammond, P.B. (1985) "Evolution of Efficient Methods to Sample Lead Sources, Such as House Dust and Hand Dust, in the Homes of Children." *Environmental Research* 38:77-95.

Rabinowitz, M.B., Wetherill, G.W., Kopple, J.D. (1973) "Lead Metabolism in the Normal Human: Stable Isotope Studies." *Science.* 182:725-727.

Rabinowitz, M.B., Wetherill, G.W., Kopple, J.D. (1976) "Kinetic Analysis of Lead Metabolism in Healthy Humans." *Journal of Clinical Investigation.* 58:260-270.

Rabinowitz, M.B., and Needleman, H.L. (1982) "Temporal Trends in the Lead Concentrations of Umbilical Cord Blood." *Science.* 216:1429-1430.

Rabinowitz, M.B., and Needleman, H.L. (1984a) "Environmental, Demographic, and Medical Factors Related to Cord Blood Lead Levels." *Biological Trace Element Research.* 6:57-67.

Rabinowitz, M.B., Needleman, H.L., Leviton, A. (1984b) "Variability of Blood Lead During Normal Infancy." *Archives of Environmental Health.* 39:74-77

Rabinowitz, M., Leviton, A., Needleman, H., Bellinger, D., Waternaux, C. (1985a). "Environmental Correlates of Infant Blood Lead Levels in Boston." *Environmental Research.* 38:96-107.

Rabinowitz, M., Leviton, A., Bellinger, D. (1985b) "Home Refinishing, Lead Paint, and Infant Blood Lead Levels." *American Journal of Public Health.* 75(4):403-404.

Rabinowitz, M.B., Leviton, A., Needleman, H. (1986) "Occurrence of Elevated Protoporphyrin Levels in Relation to Lead Burden in Infants." *Environmental Research.* 39:253-257.

Rabinowitz, M.B. (1991) "Toxicokinetics of Bone Lead." *Environmental Health Perspectives.* 91:33-37.

Reigart, J.R., Graber, C.D. (1976) "Evaluation of the Humoral Immine Response of Children with Low Level Lead Exposure." *Bull Environ Contam Toxicol.* 16:112-117.

Robinson, G.S., Baumann, S., Kleinbaum, D., et al. (1985) "Effects of Low to Moderate Lead Exposure on Brainstem Auditory Evoked Potentials in Children: Environmental Health Document 3." Copenhagen, Denmark: World Health Organization Regional Office for Europe. 177-182.

Rodamilans, M., Osaba, M.J., To-Figueras, J., et al. (1988) "Lead Toxicity on Endocrine Testicular Function in an Occupationally Exposed Population." *Hum Toxicol.* 7:125-128.

Roels, H.A., Lauwerys, R.R., Buchet, J-P, et al. (1975) "Response of Free Erythrocyte Porphyrin and Urinary-δ-aminolevulinic Acid in Men and Women Moderately Exposed to Lead." *Int Arch Arbeitsmed.* 34:97-108.

Roels, H.A., Buchet, J-P, Lauwerys, R., et al. (1976) "Impact of Air Pollution by Lead on the Heme Biosynthetic Pathway in School-age Children." *Archives of Environmental Health.* 31:310-316.

Roels, H.A., Balis-Jacques, M.N., Buchet, J-P, et al. (1979) "The Influence of Sex and Chelation Therapy on Erythrocyte Protoporphyrin and Urinary-δ-aminolevulinic Acid in Lead-exposed Workers." *Journal of Occupational Medicine.* 21:527-539.

Roels, H.A., Lauwerys, R. (1987) "Evaluation of Dose-effect and Dose-response Relationships for Lead Exposure in Different Belgian Population Groups (Fetus, Child, Adult Men and Women)." *Trace Elements in Medicine.* 4:80-87.

Rosen, J.F., Zarate-Salvador, C., Trinidad, E.E. (1974) "Plasma Lead Levels in Normal and Lead-intoxicated Children." *Journal of Pediatrics.* 84:45-48.

Rosen, J.F., Chesney, R.W., Hamstra, A.J., et al. (1980) "Reduction in 1,25-dihydroxyvitamine D in Children with Increased Lead Absorption."*New England Journal of Medicine.* 302:1128-1131.

Rosen, I., Wildt, K., Gullberg, B., et al. (1983) "Neurophysiological Effects of Lead Exposure." *Scand J Work Environ Health.* 9:431-441.

Rothenberg, S.J., Schnaas, L., Cansino-Ortiz, S., et al. (1989) "Neurobehavioral Deficits After Low Level Lead Exposure in Neonates: The Mexico City Pilot Study." *Neurotoxicol Teratol.* 11:85-93.

Rummo, J.H., Routh, D.K., Rummo, N.J., et al. (1979) "Behavioral and Neurological Effects of Symptomatic and Asymptomatic Lead Exposure in Children." *Archives of Environmental Health.* 34:120-125.

Rust, S.W., Burgoon, D.A. (1993) "Development of Health-Based Standards for Lead in Residential Environments." December 1993.

Rust, S.W., Kumar, P., Burgoon, D.A., Schultz, B. (1996) "Influence of Bone-lead Stores on the Observed Effectiveness of Lead Hazard Intervention." In press.

Ryan, C.M., Morrow., L., Parkinson, D., et al. (1987) "Low Level Lead Exposure and Neuropsychological Functioning in Blue Collar Males." *Int J Neurosci.* 36:29-39.

Sachs, H.K., Moel, D.I. (1989) "Height and Weight Following Lead Poisoning in Childhood." *American Journal of Diseases and Children.* 143:820-822.

Saenger, P., Markowitz, M.E., Rosen, J.F. (1984) "Depressed Excretion of 6β-hydroxycortisol in Lead-toxic Children." *J Clin Endocrinol Metab.* 58:363-367.

Schmid, E., Bauchinger, M., Pietruck, S., et al. (1972) "Cytogenic Action of Lead in Human Peripheral Lymphocytes *In Vitro* and *In Vivo*." *Mut Res.* 16:401-406. (German)

Schneitzer, L., Osborn, H.H., Bierman, A., et al. (1990) "Lead Poisoning in Adults from Renovation of a Older Home." *Ann Emerg Med.* 19:415-420.

Schroeder, H.A., Tipton, I.H. (1968) "The Human Body Burden of Lead." *Archives of Environmental Health.* 17:965-977.

Schroeder, S.R., Hawk, B., Otto, D., Mushak, P., Hicks, R.E. (1985) "Separating the Effects of Lead and Social Factors on IQ." *Environmental Research.* 91:178-183.

Schroeder, S.R., Hawk, B. (1987) "Psycho-social Factors, Lead Exposure and IQ." *Monogr Am Assoc Ment Defic.* 8:97-137.

Schutz, A., Skerfving, S., Ranstam, J., Christoffersson, J. (1987) "Kinetics of Lead in Blood after the End of Occupational Exposure." *Scand. J. Work Environ. Health.* 13:221-231.

Schwartz, J., Otto, D.A. (1987) "Blood Lead, Hearing Thresholds, and Neurobehavioral Development in Children and Youth." *Archives of Environmental Health.* 42:153-160.

Schwartz, J. (1988) "The Relationship Between Blood Lead and Blood Pressure in the NHANES II Survey." *Environmental Health Perspectives.* 78:15-22.

Schwartz, J., Landrigan, P.J., Feldman, R.G., et al. (1988) "Threshold Effect in Lead-induced Peripheral Neuropathy." *Journal of Pediatrics.* 112:12-17.

Schwartz, J., Landrigan, P.J., Baker, E.L., Jr. (1990) "Lead-induced Anemia: Dose-response Relationships and Evidence for a Threshold." *American Journal of Public Health.* 80:165-168.

Schwartz, J. (1993) "Beyond LOEL's, p Values, and Vote Counting: Methods for Looking at the Shapes and Strengths of Association." *Neurotoxicology and Teratology.* 14(2-3):237-246.

Schwartz, J. (1994) "Low-Level Lead Exposure and Children's IQ: A Meta-Analysis and Search for a Threshold." *Environmental Research.* 65:42-55.

Secchi, G.C., Erba, L., Cambiaghi, G. (1974) "δ-aminolevulinic Acid Dehydratase Activity of Erythrocytes and Liver Tissue in Man: Relationship to Lead Exposure." *Archives of Environmental Health.* 28:130-132.

Selander, S., Cramer, K. (1970) "Interrelationships Between Lead in Blood, Lead in Urine, and ALA in Urine During Lead Work." *British Journal of Industrial Medicine.* 27:28-39.

Seppalainen, A.M., Hernberg, S., Vesanto, R., et al. (1983) "Early Neurotoxic Effects of Occupational Lead Exposure: A Prospective Study." *Neurotoxicology.* 4:181-192.

Seto, D.S.Y., Freeman, J.M. (1964) "Lead Neuropathy in Childhood." *Am J Dis Child.* 107:337-342.

Shaheen, S. (1984) "Neuromaturation and Behavior Development: The Case of Childhood Lead Poisoning." *Dev. Psych.* 20:542-550.

Shukla., R. Bornschein, R.L., Dietrich, K.N., et al. (1987) "Effects of Fetal and Early Postnatal Lead Exposure on Child's Growth in Stature — The Cincinnati Lead Study." In: Lindberg, S.E. Hutchinson, T.C., eds. International Conference on Heavy Metals in the Environment, Vol. 1. New Orleans, LA, September. Edinburgh, UK: CEP Consultants, Ltd., 210-212.

Shukla, R., Bornschein, R.L., Dietrich, K.N., et al. (1989) "Fetal and Infant Lead Exposure: Effects on Growth in Stature." *Pediatrics.* 84:604-612.

Siegel, M., Forsyth, B., Siegel, L., et al. (1989) "The Effect of Lead on Thryoid Function in Children." *Environmental Research.* 49:190-196.

Silva, P.A., Hughes, P., Williams, S., Faed, J.M. (1988) "Blood Lead, Intelligence, Reading Attainment, and Behaviour in Eleven Year Old Children in Dunedin, New Zealand." *Journal of Child Psychology and Psychiatry.* 29:43-52.

Silver, W., Rodriguez-Torres, R. (1968) "Electrocardiographic Studies in Children with Lead Poisoning." *Pediatrics.* 41:1124-1127.

Smith, M., Delves, T., Lansdown, R., et al. (1983) "The Effects of Lead Exposure on Urban Children: The Institute of Child Health/Southampton Study." *Dev Med Child Neurol. 25 (suppl 47).*

Smith, F.L., II, Rathmell, T.K., Marcil, G.E. (1938) "The Early Diagnosis of Acute and Latent Plumbism." *Am J Clin Pathol.* 8:471-508.

Smith, D.R., Osterloh, J.D., Flegal, A.R. (1996) "Use of Endogenous Stable Lead Isotopes to Determine Release of Lead from the Skeleton." *Environmental Health Perspectives.* 104(1):60-66.

Spivey, G.H., Baloh, R.W., Brown, C.P., et al. (1980) "Subclinical Effects of Chronic Increased Lead Absorption--A Prospective Study: III. Neurologic Findings at Follow-up Examintion." *Journal of Occupational Medicine.* 22:607-612.

Staes, C., Matte, T., Copley, G., Flanders, D., Binder, S. (1994) "Retrospective Study of the Impact of Lead-based Paint Hazard Remediation on Children's Blood Lead Levels in St. Louis, Missouri." *American Journal of Epidemiology.* 139(10):1016-1026.

Staessen, J., Yeoman, W.B., Fletcher, A.E., et al. (1990) "Blood Lead Concentration, Renal Function, and Blood Pressure in London Civil Servants." *British Journal of Industrial Medicine.* 47:442-447.

Staessen, J., Sartor, F., Roels, H., et al. (1991) "The Association Between Blood Pressure, Calcium and Other Divalent Cations: A Population Study." *Journal of Human Hypertension.* 5:485-494.

Stark, A.D., Meigs, J.W., Fitch, R.A., DeLouise, E.R. (1978) "Family Operational Cofactors in the Epidemiology of Childhood Lead Poisoning." *Archives of Environmental Health.* 33:222-226.

Stark, A.D., Quah, R.F., Meigs, J.W., DeLouise, E.R. (1982) "The Relationship of Environmental Lead to Blood Lead Levels in Children." *Environmental Research.* 27:372-383.

Stollery, B.T., Banks, H.A., Broadbent, D.E., et al. (1989) "Cognitive Functioning in Lead Workers." *British Journal of Industrial Medicine.* 46:698-707.

Strauss, W., Buxton, B., Rust, S., Boyd, H. (1996) "Statistical Evaluation of the Relationship Between Blood-Lead and Dust-Lead Based on Data from the Rochester Lead-in-Dust Study." February 1996.

Strauss, W., Rust, S., Boyd, H. (1996) "Statistical Evaluation of the Relationship Between Blood-Lead and Dust-Lead Based on Pre-Intervention Data from the Baltimore Repair and Maintainence Study." February 1996.

Stuik, E.J. (1974) "Biological Response of Male and Female Volunteers to Inorganic Lead." *Int Arch Arbeitsmed.* 33:83-97.

Thornton I, Davies D.J., Watt J.M., Quinn M.J. (1990) "Lead Exposure in Young Children From Dust and Soil in the United Kingdom." *Environmental Health Perspectives.* 89:55-60.

Tola, S., Hernberg, S., Asp, S., et al. (1973) "Parameters Indicative of Absorption and Biological Effect in New Lead Exposure: A Prospective Study." *British Journal of Industrial Medicine.* 30:134-141.

Triebig, G., Weltle, D., Vanentin, H. (1984) "Investigations on Neurotoxicity of Chemical Substances at the Workplace: V. Determination of the Motor and Sensory Nerve Conduction Velocity in Persons Occupationally Exposed to Lead." *International Archives of Occupational Environmental Health.* 53:189-204.

Tuppurainen, M., Wagar, G., Kurppa, K. (1988) "Thyroid Function as Assessed by Routine Laboratory Tests of Workers with Long-term Lead Exposure." *Scand J Work Environ Health.* 14:175-180.

U.S. Department of Health and Human Services (1992) "Sample Design: Third National Health and Nutrition Examination Survey." DHHS Publication No. (PHS) 92-1887.

U.S. Department of Health and Human Services (1994) "Plan and Operation of the Third National Health and Nutrition Examination Survey." 1988-94 DHHS Publication No. (PHS) 94-1308.

U.S. Department of Housing and Urban Development, Office of Policy Development and Research. (1990) "Comprehensive and Workable Plan for the Abatement of Lead-Based Paint in Privately Owned Housing," Report to Congress, December 1990.

U.S. Department of Housing and Urban Development (1995a) "The Relation of Lead-Contaminated House Dust and Blood Lead Levels Among Urban Children." Volumes I and II. Final Report to the U.S. Department of Housing and Urdan Development Grant from The University of Rochester School of Medicine, Rochester, New York, and The National Center for Lead-Safe Housing, Columbia Maryland.

U.S. Department of Housing and Urban Development (1995b) "Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing." Office of Lead-Based Paint Abatement and Poisoning Prevention.

U.S. Environmental Protection Agency (1986) Air Quality Criteria for Lead. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office. EPA 600/8-83-028F.

U.S. Environmental Protection Agency (1990a) "Air Quality Criteria for Lead: Supplement to the 1986 Addendum." Office of Research and Development, Washington, D.C. EPA/600/8-89/049F.

U.S. Environmental Protection Agency (1990b). "National Air Quality and Emissions Trends Report, 1988." Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency. EPA 450/4-90-002.

U.S. Environmental Protection Agency (1992) "Boston Lead-in-Soil/Lead Free Kids Demonstration Project." Final Report, July 1992.

U.S. Environmental Protection Agency (1993) "Cincinnati Soil Lead Abatement Demonstration Project." Final Report, March 1993.

U.S. Environmental Protection Agency (1994a) "Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children." Washington, DC. EPA/540/R-93/081, PB93-963510.

U.S. Environmental Protection Agency (1994b) "Validation Strategy for the Integrated Exposure Uptake Biokinetic Model for Lead in Children." EPA/540/R-94/039.

U.S. Environmental Protection Agency (1994c) "Descriptive Statistics From the Initial Sampling Campaign of the Lead Paint Abatement and Repair and Maintenance Study in Baltimore." Draft Final Report to the U.S. Environmental Protection Agency from the Kennedy Krieger Institute.

U.S. Environmental Protection Agency (1994d) "Reducing Lead Hazards When Remodeling Your Home." Office of Pollution Prevention and Toxics, U.S. Environmental Protection Agency. EPA 747-R-94-002, April 1994.

U.S. Environmental Protection Agency (1995a) "Report on the National Survey of Lead-Based Paint in Housing: Base Report." Office of Pollution Prevention and Toxics, U.S. Environmental Protection Agency, EPA 747-R95-003, April 1995.

U.S. Environmental Protection Agency (1995b) "Review of Studies Addressing Lead Abatement Effectiveness." Office of Pollution Prevention and Toxics, U.S. Environmental Protection Agency. EPA 747-R-95-006, July 1995.

U.S. Environmental Protection Agency (1995c) "Sampling House Dust for Lead: Basic Concepts and Literature Review." Office of Pollution Prevention and Toxics, U.S. Environmental Protection Agency. EPA 747-R-95-007, September 1995.

U.S. Environmental Protection Agency (1995d) "Laboratory Evaluation of Dust and Dust Lead Recoveries for Samplers and Vacuum Cleaners: Vol. I and II." Office of Pollution Prevention and Toxics, U.S. Environmental Protection Agency. EPA 747-R-95-004A, EPA 747-R-95-004B, March 1995.

U.S. Environmental Protection Agency (1995e) "Technical Support Document: Parameters and Equations Used in the Integrated Exposure Uptake Biokinetic Model for Lead in Children (v 0.99D)." EPA 540/R-94/040.

U.S. Environmental Protection Agency (1995f) "Comprehensive Abatement Performance Study, Volume I: Summary Report." Final Report prepared by Battelle to the Office of Pollution Prevention and Toxics, U.S. Environmental Protection Agency, EPA Technical Report No. 230-R-94-013a.

U.S. Environmental Protection Agency (1995g) "Urban Soil Lead Abatement Demonstration Project EPA Integrated Report." EPA/600/R-95/139, September 1995.

U.S. Environmental Protection Agency (1995h) "Guidance on Identification of Lead-Based Paint Hazards." Notice. Federal Register, pp 47248-47257, September 11, 1995.

U.S. Environmental Protection Agency (1996) "Urban Soil Lead Abatement Demonstration Project, Volume I: Integrated Report." Office of Research and Development, U.S. Environmental Protection Agency. EPA 600-P-93-001aF, April 1996.

Valciukas, J.A., Lilis, R., Eisinger, J., et al. (1978) "Behavioral Indicators of Lead Neurotoxicity: Results of a Clinical Field Survey." *International Archives of Occupational Environmental Health.* 41:217-236.

Vershoor, M., Wibowo, A., Herber, R., et al. (1987) "Influence of Occupational Low-level Lead Exposure on Renal Parameters." *American Journal of Industrial Medicine.* 12:341-351.

Vimpani, G.V., Wigg, N.R., Robertson, E.F., et al. (1985) "The Port Pirie Cohort Study: Blood Lead Concentration and Childhood Developmental Assessment". Presented at: Lead Environmental Health: Current Issues, May, Duke University, Durham, NC.

Vimpani, G.V., Baghurst, P.A., Wigg, N.R., et al. (1989) "The Port Pirie Cohort Study — Cumulative Lead Exposure and Neurodevelopmental Status at Age 2 Years: Do HOME Scores and Maternal IQ Reduce Apparent Effects of Lead on Bayley Mental Scores?" In: Smith, M., Grant., L.D., Sors, A., eds. Lead Exposure and Child Development: An International Assessment. Lancaster, UK: *Kluwer Academic Press.*

Wada, O., Yano, Y, Ono, T., et al. (1973) "The Diagnosis of Different Degrees of Lead Absorption in Special References to Choice and Evaluation of Various Parameters Indicative of an Increased Lead Absorption." *Industrial Health.* 11:55-67.

Wallsten, T.S., Whitfield, R.G. (1986) "Assessing the Risks to Young Children of Three Effects Associated with Elevated Blood-lead Levels." Report by Argonne National Laboratory. Report No. ANL/AA-32. Sponsored by the U.S. EPA Office of Air Quality Planning and Standards.

Wang, L., Xu S.E., Zhang, G.D, Wang, W.Y. (1989) "Study of Lead Absorption and its Effect on Children's Development." *Biomedical and Environmental Sciences.* 2:325-330.

Ward, N.I., Watson, R., Bryce-Smith, D. (1987) "Placental Element Levels in Relation to Fetal Development for Obstetrically Normal Births: A Study of 37 Elements: Evidence for the Effects of Cadmium, Lead, and Zinc on Fetal Growth and for Smoking as a Source of Cadmium." *Int J Biosoc Res.* 9:63-81.

Wedeen, R.P., Mallik, D.K., Batuman, V. (1979) "Detection and Treatment of Occupational Lead Nephropathy." *Archives of Internal Medicine.* 139:53-57.

Wedeen, R.P. (1988) "Bone Lead, Hypertension, and Lead Nephropathy." *Environmental Health Perspectives.* 78:57-60.

Weiss, S.T., Munoz, A., Stein, A., et al. (1986) "The Relationship of Blood Lead to Blood Pressure in a Longitudinal Study of Working Men." *Am J Epidemiol.* 123:800-808.

Weiss, S.T., Munoz, A., Stein, A., et al. (1988) "The Relationship of Blood Lead to Systolic Blood Pressure in a Longitudinal Study of Policemen." *Environmental Health Perspectives.* 78:53-56.

Weitzman, M., Aschengrau, A., Bellinger, D., Jones, R., Hamlin, J.S., Beiser, A. (1993) "Lead-Contaminated Soil Abatement and Urban Children's Blood Lead Levels." *Journal of the American Medical Association.* 269(13):1647-1654.

Wibberley, D.G., Khera, A.K., Edwards, J.H., et al. (1977) "Lead Levels in Human Placentae from Normal and Malformed Births." *J Med Genet.* 14:339-345.

Wigg, N.R., Vimpani, G.V., McMichael, A.J., et al. (1988) "Port Pirie Cohort Study: Childhood Blood Lead and Neuropsychological Development at Age Two Years." *J Epidemiol Community Health.* 42:213-219.

Wildt, K., Eliasson, R., Berlin, M. (1983) "Effects of Occupational Exposure to Lead on Sperm and Semen." In: Clarkson, T.W., Nordberg, G.F., Sager, P.R., eds. Reproductive and Developmental Toxicity of Metals. Proceedings of a Joint Meeting, Rochester, NY, May 1982. New York, NY: *Plenum Press.* 279-300.

Wilhelm, M., Lombeck, I., Hafner, D., et al. (1989) "Hair Lead Levels in Young Children from the F.R.G." *J Trace Elements and Electrolytes in Health and Disease,* 3:165-170.

Williamson, A.M., Teo, R.K.C. (1986) "Neurobehavioral Effects of Occupational Exposure to Lead." *British Journal of Industrial Medicine.* 43:374-380.

Winneke, G., Beginn, U., Ewert, T., et al. (1985) "Comparing the Effects of Perinatal and Later Childhood Lead Exposure on Neurophysiological Outcome." *Environmental Research.* 38:155-167.

Winneke, G., Brockhaus, A., Collet, W., et al. (1985) "Predictive Value of Different Markers of Lead-exposure for Neuropsychological Performance." In: Lekkas, T.D., ed. International Conference on Heavy Metals in the Environment, Athens, Greece, September, Vol. 1. Edinburgh, United Kingdom: CEP Consultants, Ltd., 44-47.

Wolf, A.W., Ernhart, C.B., White, C.S. (1985) "Intrauterine Lead Exposure and Early Development." In: Lekkas T.D., ed. International Conference: Heavy Metals in the Environment, Athens, Greece, September, Vol. 2. Edinburgh, United Kingdom: CEP Consultants, Ltd. 153-155.

Wrenn, M.E., Cohen, M., Rosen, J.C., Eisenbud, M., Blanchard, R.L. (1972) "In-vivo Measurements of Lead-210 in Man. Assessment of Radioactive Contamination in Man." *IAEA.* 129-146.

Yule, W., Lansdown, R., Millar, I., Urbanowiez, M. (1981) "The Relationship Between Blood Lead Concentration, Intelligence, and Attainment in a School Population: a Pilot Study." *Dev Med Child Neuro.* 23:567-576.

Zimmerman-Tansella, C., Campara, P., D'Andrea, F., et al. (1983) "Psychological and Physical Complaints of Subjects with Low Exposure to Lead." *Hum Toxicol.* 2:651-623.