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Risk Analysis to Support Standards for Lead in Paint, Dust, and Soil

Supplemental Report
VOLUME I: Chapters 1 to 7

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RISK ANALYSIS TO SUPPORT STANDARDS FOR LEAD IN PAINT, DUST, AND SOIL

SUPPLEMENTAL REPORT

VOLUME I: CHAPTERS 1 TO 7

National Program Chemicals Division (7404)
Office of Pollution Prevention and Toxics
U.S. Environmental Protection Agency
Washington, DC 20460

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CONTRIBUTING ORGANIZATIONS

This report is a supplement to EPA 747-R-97-006 ("Risk Analysis to Support Standards for Lead in Paint, Dust, and Soil"). Efforts to produce this report were funded and managed by the U.S. Environmental Protection Agency. The risk analysis was conducted by Battelle Memorial Institute under contract to the U.S. Environmental Protection Agency. Each organization's responsibilities are listed below.

Battelle Memorial Institute (Battelle)

Battelle was responsible for performing the additional data analyses, literature reviews, and documentation presented in this report. Battelle was also responsible for preparing this report.

U.S. Environmental Protection Agency (EPA)

The Environmental Protection Agency was responsible for providing direction on the technical issues to be presented in this report, providing relevant information for the report, reviewing the report, contributing to the development of conclusions, and managing the peer review and publication of the report. The EPA Work Assignment Manager was Mr. Ronald Morony. The Deputy Work Assignment Managers were Mr. Brad Schultz and Mr. Dave Topping. The EPA Project Officer was Ms. Sineta Wooten.

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EXECUTIVE SUMMARY

This report is a supplement to the EPA report "Risk Analysis to Support Standards for Lead in Paint, Dust, and Soil" (USEPA, 1998a), which presented the methods and findings of a risk analysis that supported efforts by the U.S. Environmental Protection Agency (EPA) to set regulatory standards for lead levels in dust and soil and to control lead-based paint hazards within most pre-1978 housing and child-occupied facilities. These regulatory standards were mandated through §403 of the Toxic Substances Control Act (TSCA), as specified within Title X, the Residential Lead-Based Paint Hazard Reduction Act of 1992 (42 U.S.C. 4851). The §403 risk analysis provided EPA with a scientific foundation for establishing the regulatory standards. On June 3, 1998, EPA proposed a regulation to establish these standards (40 CFR Part 745); this regulation is referred to as the "§403 proposed rule."

In 1998, the Environmental Health Committee of EPA's Science Advisory Board (SAB) conducted a technical review of the §403 risk analysis. In response to this review, EPA deemed that a supplement to USEPA (1998a), referred to as the "§403 risk analysis report," was necessary to provide additional technical analyses and to clarify certain key analyses and findings presented within the report. EPA used the summaries and analyses of data considered in the original §403 risk analysis to prepare responses to the public comments on the §403 proposed rule and to prepare the final rule. Although this supplement also presents summaries and analyses of data made available to EPA since the §403 proposed rule was released, such as interim data from the National Survey of Lead and Allergens in Housing (NSLAH), this type of information is meant only to provide an alternative to the findings presented in the §403 risk analysis report. EPA has not used these new data in efforts to select the hazard standards or levels of concern that are included within the final rule. Furthermore, this supplement does not replace any parts of the original §403 risk analysis, but rather supplements this risk analysis with more detailed analyses on selected topics.

The format of this supplement follows that of the §403 risk analysis report, with separate chapters for each phase of the risk assessment (hazard identification, exposure assessment, dose-response assessment, and risk characterization) and a final chapter on risk characterization under example options for standards. The remainder of this executive summary summarizes the key issues and findings presented within each chapter of this supplement.

Hazard Identification (Chapter 2)

Additional evidence that lead-based paint hazards pose a health risk to children and the magnitude of such risk was provided in the following four areas within this supplement:

- Adverse health effects of lead exposure, with a focus on neurological effects, as observed in animal studies (to address the point raised in the SAB review that animal data can support causality effects over and above the available data on humans).

- Evidence supporting causality between lead exposure and adverse health effects.

- The association between blood-lead concentration and reduction in intelligence quotient (IQ) score.

- The role that dust particle size and the chemical composition of lead compounds in lead-contaminated dust may play in determining the extent to which lead in residential dust is bioavailable to humans.

Adverse health effects, especially neurotoxicity, observed in animal studies. Lead has been observed to have widespread neurotoxic effects, as well as behavioral and cognitive symptoms, in humans. These observations are largely consistent with the findings that have been demonstrated in controlled, dose-response studies on animals (e.g., rodents, dogs, non-human primates). Animal studies are also congruent with observations of lead exposure in humans, suggesting an increased susceptibility of the young brain to lead poisoning (Banks et al., 1997). For example, lead-induced alteration of protein kinase C (PKC) activity in the brain has been shown to correlate with poor performance in several learning tasks in animal studies, and researchers have suggested that some of the learning and memory deficits observed in children are likely to be causally related to the types of PKC activity alterations exhibited in these studies (Chen et al., 1998). In addition, animal studies have provided physiological evidence that many of the effects of lead on the differentiation of the developing nervous system, such as synaptic and dendritic development, and myelin and other nerve structure formation, have the potential to be long-lived (USEPA, 1986). Although animal models do not duplicate the human response to lead exposure, they do serve to provide strong support for expecting certain health effects to occur when humans are exposed.

Cause-effect relationship of lead exposure and adverse health effects. The combined weight of human and animal studies provides evidence that lead may be assumed to cause adverse neurological effects in young children. For example, longitudinal studies in humans (e.g., Boston, Port Pirie) have shown that disturbances occur in neurobehavioral development early in life even at low lead exposure levels. These studies have observed effects of lead exposure even after accounting for other demographic factors (e.g., socioeconomic status, maternal IQ) that could affect neurological development. While these other demographic factors tend to be highly correlated with blood-lead levels early in life, the influence that lead exposure has on blood-lead tends to increase with a child's age. This is because over time, measures of a child's lead exposure tend to change at a faster rate than the child's demographic measures, and more recent lead exposures continue to be predictive of a child's current health consequences. The §403 risk analysis assessed these adverse neurological effects in children through measuring average IQ score decrements in the population due to lead exposure.

Association between blood-lead concentration and IQ score decrement. This supplement provides additional technical justification regarding the assumptions made in the §403 risk analysis on the association between blood-lead concentration and decrement in IQ score.

While prior investigations into this relationship have considered both linear and log-linear associations, a linear relationship (with positive slope) was used in the §403 risk analysis based on the evidence taken from these investigations and the desire not to unduly over-estimate the IQ decrement associated with children having low blood-lead concentrations.

Recent studies and meta-analyses investigating the presence of a threshold in the blood-lead/IQ relationship (e.g., Schwartz, 1994) have concluded either that no non-zero threshold exists, or if one does exist, if may be very low (e.g., less than 1.0 μg/dL; Schwartz, 1993). Researchers who disagree with this conclusion have not reached a clear consensus on a value for this threshold. Several older studies that have suggested high thresholds (e.g., over 10 μg/dL) involved few, if any, children at low blood-lead levels, thereby preventing their ability to provide information on potential health effects at low levels. Other researchers have used visual inspection of data summaries to conclude that thresholds exist at relatively high levels, rather than using statistical inference techniques that would yield more scientifically defensible conclusions. As it would have been necessary to have clear, scientifically defensible evidence of a particular non-zero threshold to justify its adoption within the §403 risk analysis, and based on the findings of recent meta-analyses, the approach taken in the §403 risk analysis was to assume that no threshold exists. Nevertheless, sensitivity analyses documented in this supplement have investigated the impact of assuming a positive threshold on the risk analysis estimates.

Effect of residential dust characteristics on the bioavailability of lead in dust. It has been suggested that lead speciation and particle size may affect the bioavailability of lead in dust through their influence on solubility. Therefore, this supplement included an investigation of current knowledge about bioavailability of lead in dust and how this knowledge may impact the rulemaking process.

There is relatively little in the literature which examines the relationship between bioavailability and chemical composition specifically for household dust. Animal studies have concluded that different lead compounds are associated with different rates of absorption (e.g., metallic-lead was associated with low absorption). Otherwise, most of the investigations into the effect of lead speciation on bioavailability primarily address lead in soil. However, soil can have a considerable influence on dust-lead levels. Evidence exists that correlation between blood-lead and soil-lead levels can be influenced by both particle size and chemical composition. Generally, the smaller the particle size, the greater the absorption of lead due to more rapid dissolution in the gastrointestinal tract. Some researchers have also hypothesized that smaller particle sizes can contain higher lead concentrations.

While evidence does suggest that particle size and chemical composition can influence the level of bioavailability of lead in dust, the current information base may be inadequate to determine how such factors can reasonably be incorporated into the rulemaking effort. Furthermore, needing to characterize dust in this manner within a risk assessment would likely add to the expense of dust analyses, and dust standards that distinguish between these various characterizations could add considerable complexity to the rule.

Exposure Assessment (Chapter 3)

When using model-based procedures to estimate health risks associated with lead-based paint hazards, the §403 risk analysis used data from the 1989-1990 HUD National Survey of Lead-Based Paint in Housing to characterize lead levels in dust and soil within the nation's housing stock. As mentioned above, more recent data have been made available to EPA since the §403 risk analysis report and the proposed rule were published, and some commenters on the proposed rule suggested that EPA should use these data when available. These data include interim data (collected in 1998 and 1999) for 706 housing units from the NSLAH, an on-going national survey of lead levels in dust and soil in the nation's housing. HUD assigned interim sampling weights to these 706 surveyed units in such a way as to allow interim results that properly incorporate these sampling weights to be nationally representative of occupied housing in which children can possibly reside. Other recently-acquired data that are summarized in Chapter 3 of this supplement include additional data from the Evaluation of the HUD Lead-Based Paint Hazard Control Grant Program ("HUD Grantees") and data from the 1997 American Housing Survey.

Comparing dust-lead loadings in the HUD National Survey with those of other studies. Before dust-lead loadings measured in the HUD National Survey could be compared to dust-lead loadings from other studies, the loadings needed to be adjusted to reflect the fact that the HUD National Survey used a vacuum technique rather than a wipe technique to collect the dust samples. The §403 risk analysis included a procedure for performing this adjustment. The resulting dust-lead loadings, adjusted to represent dust samples collected using wipe techniques, tended to be lower than the wipe dust-lead loadings reported in other recent lead exposure studies (e.g., Rochester study, HUD Grantees evaluation), even when taking into account housing age category and Census region. One major exception, however, were data from the interim NSLAH, whose distribution of dust-lead loadings was lower than in the HUD National Survey for both floors and window sills. For example, household average floor dust-lead loadings based on the interim NSLAH data had a median of less than 2 $\mu g/ft^2$, compared to approximately 5.3 $\mu g/ft^2$ as estimated in the §403 risk analysis based on the HUD National Survey data. The estimated percentage of housing units with average floor dust-lead loadings that exceed 50 $\mu g/ft^2$ (i.e., the proposed floor dust-lead standard) was 6.4% based on HUD National Survey data used in the §403 risk analysis, and 0.9% based on interim data from the NSLAH.

Comparing soil-lead concentrations in the HUD National Survey with those of other studies. Data on yardwide average soil-lead concentrations from the HUD National Survey were compared with interim NSLAH data, as well as data for 22 other studies that characterized soil-lead concentrations in urban areas prior to any lead abatement. While geometric mean yardwide average soil-lead concentration was lower in the HUD National Survey relative to most of the other studies reviewed, the distributions of yardwide average soil-lead concentration were similar between the HUD National Survey and the interim NSLAH (although the estimated median was nearly 50% lower in the HUD National Survey versus the interim NSLAH for homes built prior to 1940). Regardless of which national survey is considered, the risk analysis supplement estimates that the yardwide average soil-lead concentration exceeds 2000 ppm in approximately

1.7% of housing, approximately 3.3% of housing exceeds an average soil-lead concentration of 1200 ppm, and between 11% and 12% of housing exceeds an average soil-lead concentration of 400 ppm.

Soil pica in children. Because the impact of paint pica (i.e., the purposeful ingestion of paint chips) on blood-lead concentration in the presence of deteriorated lead-based paint is not represented within other environmental exposures to lead, the §403 risk analysis accounted for paint pica as a separate factor when estimating risks. While the analysis did not consider the independent impact of soil pica (i.e., the purposeful ingestion of soil) over and above paint pica, it considered the impact of soil pica as part of the relation between soil-lead concentration and blood-lead concentration. While this supplement does not change the approach taken in the original risk analysis, it documents information obtained on the component of soil-lead exposure that may be attributable to soil pica.

Based on what has been found in the literature on studies in which paint pica and soil pica behaviors were characterized and could be separated, approximately 10% to 20% of study children appeared to exhibit soil pica behavior in the absence of paint pica. The frequency of soil pica episodes depends on many factors, including climate, access to bare soil, socioeconomic standing, age of child, and parental supervision. While estimates of the amount of soil ingested during pica episodes can vary widely among mass balance studies (i.e., from 500 to 13,000 mg/day), average daily ingestion over a year may be much lower. Because soil pica behavior tends to be episodic in nature, it is currently uncertain whether the amount of lead in soil ingested on an infrequent basis would be sufficient to elevate blood-lead concentration to unsafe levels. Again, it should be noted that the original §403 risk analysis did include an effect of soil pica in the relationship between soil-lead and blood-lead levels; the additional information presented in this supplement addresses how the effect of soil pica is separated from the effect of paint pica.

Dust-lead levels on surfaces other than floors and window sills. Comments were received on the §403 proposed rule recommending that EPA establish dust standards for surfaces other than floors and window sills. In response, EPA conducted further analysis on whether surfaces other than floors and window sills might provide significant additional information for risk assessments. EPA reviewed what has been published regarding dust-lead loadings from exterior locations, air ducts, window troughs (also known as window wells), and upholstery, with special attention given to studies that investigated the correlation between such dust-lead levels and blood-lead concentrations.

It is difficult to use data from lead exposure studies to characterize the effect of lead exposure from **exterior dust** on children's blood-lead levels, primarily because studies differ considerably in how they collect exterior dust and how they report the results of lead analysis. For example, some studies have collected both exterior dust and soil for lead analysis but report results that are combined across both media. Also, studies often differ in their sampling approaches (e.g., surface scrapings, vacuum sampling) and in the locations at which the samples are taken. Furthermore, studies frequently have not investigated the specific influence of exterior dust-lead on blood-lead level in the presence of lead in other environmental media. For these

reasons, along with the fact that lead in exterior dust can be highly correlated with soil-lead, a scientifically defensible standard for lead in exterior dust was not identified, nor was such a standard deemed essential over and above the planned standards for lead in soil and interior dust. This latter conclusion becomes more acceptable if risk assessors are made aware that lead in exterior dust can be associated with adverse health effects in children, and as a result, recommend corrective action in cases where lead is present in exterior dust and is suspected to be an important pathway of exposure (e.g., when children spend a considerable amount of time on hard surfaces immediately outside of the residence).

For **air ducts** and **upholstery,** there is insufficient data upon which to characterize the role that lead in dust from these surfaces plays in a child's total lead exposure, and therefore, to develop a hazard standard for lead on these surfaces.

Because dust from **window troughs** (i.e., window wells) has historically been sampled in lead exposure studies (along with floors and window sills), and because risk assessors sample dust from window troughs in determining clearance following a lead-based paint intervention, reports of window trough dust-lead levels are generally prevalent in the literature. However, several studies have reported that the association between window trough dust-lead and blood-lead is not statistically significant after taking into account the effects of dust-lead on floors and window sills (for which standards have been proposed under §403). For this reason, along with the likelihood that exceeding a window sill dust-lead standard will prompt a cleaning of leaded-dust from window troughs as well as sills, it does not appear that an additional standard for window troughs is necessary either to identify a home with a hazard or to guide corrective actions. Also, given the correlation in lead levels between window troughs and window sills, it is likely that if more sampling is to be done beyond a minimal risk assessment, more benefit would be obtained from sampling more windows at only the sill rather than fewer windows at both the sill and trough.

Distribution of childhood blood-lead. Information on the distribution of blood-lead concentrations in children based on data from the HUD Grantees evaluation was updated to reflect additional data collected through January, 1999. These data provided a means by which estimates based on data from Phase 2 of the Third National Health and Nutrition Examination Survey (NHANES III) (i.e., the data used in the §403 risk analysis to characterize blood-lead levels in the nation) could be evaluated. The HUD Grantees evaluation data (under venipuncture blood collection) had a geometric mean of 9.3 µg/dL for children aged 1-2 years and 8.0 µg/dL for children aged 3-5 years. In contrast, the geometric means based on data from Phase 2 of NHANES III were 3.1 µg/dL for children aged 1-2 years and 2.5 µg/dL for children aged 3-5 years. Also, 51 percent of children aged 1-2 years sampled via venipuncture methods had blood-lead concentrations at or above 10 µg/dL, compared to the estimates of 5.9% for Phase 2 of NHANES III, 53.8% for the Baltimore R&M study (pre-intervention), and 23.4% for the Rochester Lead-In-Dust study. The trend toward high blood-lead levels in the HUD Grantees evaluation reflects, among other factors, the HUD Grantees program's procedure of selecting high-risk children for monitoring. While NHANES III provides the most nationally

representative data on children's blood-lead concentration, it does not provide environmental-lead data that could be used to investigate the effect of environmental-lead exposure on blood-lead concentration. Therefore, other data sources such as the HUD Grantees evaluation must provide this information.

Regression modeling of the blood-lead concentration data suggests that the relationships between blood-lead concentration and household average dust-lead loading were relatively consistent across grantees. In particular, these relationships were similar to that observed for data from the Rochester study (i.e., the data used to develop the empirical model presented in Chapter 4 of the §403 risk analysis). This conclusion is important in that the data from the HUD Grantees evaluation reflect a much larger geographical area than the Rochester study and represent several types of exposure conditions.

This risk analysis supplement includes evidence that housing age and condition play important roles in the likelihood of a resident child having an elevated blood-lead concentration. The association between older housing and the prevalence of lead hazards has been well-documented and is accepted by many experts in residential lead exposure. The level of deterioration is an important variable in the accessibility of lead-based paint hazards to children.

Dose-Response Assessment (Chapter 4)

The objective of the dose-response assessment in the §403 risk analysis was to develop a statistical procedure to characterize the relationship between environmental-lead exposure and the resulting adverse health effects in young children. This characterization would then be used to estimate health risks at specified environmental-lead levels or over the entire population. The modeling tools used in this characterization were EPA's Integrated Exposure, Uptake, and Biokinetic (IEUBK) model and an empirical model developed especially for the §403 risk analysis from data collected in the Rochester Lead-in-Dust study. In this supplement, additional models were considered to quantify this characterization: a new model developed from epidemiological data collected from 12 lead exposure studies and made available to EPA after the §403 risk analysis was completed, and revisions to the multimedia model developed for the §403 risk analysis using the Rochester study data ("Rochester multimedia model"). In addition, this supplement provides additional detail on specific aspects of the model-based analysis employed within the §403 risk analysis, such as how post-intervention blood-lead concentration distributions are characterized and how measurement error was handled when fitting the empirical model.

HUD Model. An additional model for predicting blood-lead concentration as a function of environmental-lead levels became available after the §403 risk analysis report was published. Some commenters on the §403 proposed rule suggested that EPA use this new model. This new model is a log-linear regression model developed on behalf of the U.S. Department of Housing and Urban Development (HUD) from epidemiological data collected from 12 studies. Thus this model is referred to in this supplement as the "HUD Model." The goal of this model was to

"estimate the contribution of lead-contaminated house dust and soil to children's blood-lead levels" (Lanphear et al., 1998).

When using the HUD model to predict blood-lead concentration as a function of lead levels in various environmental media, this risk analysis supplement has noted several caveats associated with interpreting the predicted blood-lead concentration:

- Risks associated with exposure to specific environmental-lead levels (as estimated from the HUD model) are generally not comparable to population-based risks (as estimated by the IEUBK and empirical models in the §403 risk analysis).

- The prediction parameters in the HUD model are not independent. Therefore, it is not appropriate to interpret the parameter estimates in the HUD model (or in the models developed for the §403 risk analysis) in isolation.

- The HUD model has adjusted for measurement error in certain environmental-lead measures used as input. Therefore, the model assumes that these input values represent "actual" exposure levels. In contrast, the models developed for the §403 risk analysis use measured levels as input that would be reported from a risk assessment. Because the §403 standards will be compared to lead exposure measures that are subject to being measured with error, this latter approach is more relevant for rulemaking purposes.

Further discussion of the HUD model is provided in Chapters 4 and 5 of this supplement.

Risk Characterization (Chapter 5)

Health risks associated with current (i.e., baseline) lead exposures for children aged 1 to 2 years were characterized in the §403 risk analysis. Both individual risk estimates (i.e., risks associated with specific environmental-lead levels) and population-based risk estimates (i.e., average risks over the entire nation) were presented. In this supplement, additional sensitivity and uncertainty analysis associated with the baseline risk characterization was performed, where possible alternatives to various approaches taken and assumptions made in the risk characterization were identified and incorporated into the analysis, and the resulting impact on the risk estimates was evaluated.

When predictions under the Rochester multimedia model (developed in the §403 risk analysis to characterize individual risks) were compared to predictions under the HUD model, the following general findings were observed:

- At very low floor dust-lead loadings (i.e., 1-5 $\mu g/ft^2$), the HUD model and the Rochester multimedia model yield similar predictions for the geometric mean blood-lead concentration, which also results in similar predictions for the health-effect endpoints that are calculated directly from this geometric mean (e.g.,

percentage of children with blood-lead concentration at or above a specified threshold; average IQ decrement resulting from lead exposure).

- The predicted geometric mean blood-lead concentration under the HUD model ranges from 20% to nearly 60% higher than the prediction under the Rochester multimedia model as floor dust-lead loadings increase from 15 to 100 $\mu g/ft^2$ and as soil-lead concentrations decrease from 2000 ppm to 10 ppm (assuming, for the Rochester multimedia model, that window sill dust-lead loadings are at their estimated national median level). Note that for a fixed value of the geometric standard deviation (GSD) for the blood-lead distribution, the average IQ decrement in the population that is associated with lead exposure is a multiple of the geometric mean (as calculated in the §403 risk analysis). Therefore, similar differences in predictions between the two models would occur for average IQ decrement.

- If the geometric standard deviation (GSD) associated with the blood-lead distribution is fixed, then as floor dust-lead loadings increase beyond 10 $\mu g/ft^2$, the predicted percentage of children with blood-lead levels at or above 10 µg/dL increases at a much faster rate under the HUD model (at a constant soil-lead level). For example, if window sill dust-lead loading is at its estimated national median and soil-lead concentration is below 2000 ppm, the predicted percentage under the HUD model is at a minimum twice as large as the prediction under the Rochester multimedia model . This difference in predictions gets even greater as the assumed soil-lead concentration gets lower. For example, at a GSD of 1.6, a floor dust-lead loading of 100 $\mu g/ft^2$, and a soil-lead concentration of 10 ppm, the prediction is over 7 times higher for the HUD model compared to the Rochester multimedia model (13.1% versus 1.76%).

Other findings within the additional sensitivity analyses performed to support the baseline risk characterization were as follows:

- If it is assumed that a 50% across-the-board decline in blood-lead concentration has occurred relative to the distribution portrayed by data from Phase 2 of NHANES III, the estimated number of children whose blood-lead concentration was at or above 20 µg/dL declined by 95% (from 46,800 to 2,130), while the estimated number at or above 10 µg/dL was reduced by nearly 90% (from 458,000 to 46,800). The estimated average IQ decrement in the population due to lead exposure is cut in half under this assumption (from 1.06 to 0.53 points).

- Model-based baseline risk estimates were calculated for various alternative assumptions on the percentage decline in dust-lead and soil-lead levels that may have occurred in the housing stock since the HUD National Survey was conducted. Risk estimates under the empirical model seemed to be more sensitive to these changes than the estimates under the IEUBK model, and reductions in

soil-lead concentration seemed to have more of an impact on reducing these risk estimates than reductions in dust-lead levels. Baseline estimates for the percentage of children with blood-lead concentrations at or above 10 µg/dL were 45% lower under the empirical model when both dust-lead and soil-lead levels were decreased by 50%, with smaller declines occurring for less drastic total reductions in the environmental-lead levels.

- In an effort to determine whether an assumption of no threshold made in the §403 risk analysis was particularly sensitive to the risk estimates, baseline estimates of the IQ-related health effect endpoints were calculated under the assumption that specified non-zero thresholds exist in the relationship between blood-lead concentration and IQ score decrement. While the §403 risk analysis estimated an average IQ decrement of 1.06 points occurs due to lead exposure across the population of children aged 1-2 years, this average declines by approximately 44% under a assumed threshold of 2 µg/dL (0.588 points) and by 90% under a threshold of 8 µg/dL (0.103 points).

Analysis of Example Options for the §403 Standards (Chapter 6)

Prompted by public comments on the §403 proposed rule and risk analysis, this supplement included the following on methods used in the §403 rulemaking process to evaluate candidate hazard standards and levels of concern:

- Detailed information on performance characteristics analyses (also known as sensitivity/specificity analysis), used by EPA to help establish levels of concern within the §403 rule.

- Characterizing the extent to which children with elevated blood-lead concentrations reside in homes where no candidate standard is met or exceeded (i.e., children who would be "missed" by a specified set of candidate standards), as part of the candidate standards evaluation process.

- An additional investigation into the assumptions made in the risk management study on post-intervention dust-lead loading (40 µg/ft^2 on floors, 100 µg/ft^2 on window sills).

- Additional sensitivity and uncertainty analyses for the analyses performed and documented within Chapter 6 of the §403 risk analysis, including alternative assumptions on baseline and post-intervention environmental-lead levels.

Performance characteristics analysis. Performance characteristics analysis is a non-modeling approach (based on calculating conditional probabilities) to assessing how often a specified set of candidate hazard standards would "trigger" interventions in housing units within the studies in question and the extent to which these units contained a child with an elevated

blood-lead concentration (≥10 μg/dL). Data from the Rochester Lead-in-Dust study were used in these analyses. This supplement contains a detailed discussion of how to interpret performance characteristics analysis and provides additional information on analysis results that were cited in the §403 proposed rule. Furthermore, this supplement presents the results of follow-on performance characteristics analyses which EPA considered when responding to public comments and in preparing the final rule. While the analysis presented in the proposed rule was based on data for 77 housing units in the Rochester study, additional assumptions made to the soil-lead data permitted up to 184 units to be represented among the data analyzed in the follow-on performance characteristics analyses. One goal of these analyses was to identify those sets of candidate dust-lead loading standards for which the analysis estimated that no more than 5% of children living in housing units with environmental-lead levels below the standards would have elevated blood-lead concentrations.

Investigating incidence of elevated blood-lead concentrations in homes where no candidate standard is met or exceeded. As an alternative to the performance characteristics analysis, a model-based approach was developed to determine the likelihood of a child with elevated blood-lead concentration residing in a housing unit that exceeds none of a given set of candidate standards. This approach was designed to use data from the Rochester study and to yield results that would be directly comparable to those from the performance characteristics analysis.

Review of published information on post-intervention dust-lead loadings. To evaluate the performance of a given set of candidate standards in reducing population-based health risks to lead exposure, the §403 risk analysis needed to make assumptions on lead levels in dust and soil that would occur after performing interventions that would be prompted by exceeding the example standards. Assumptions made on post-intervention dust-lead loadings (40 $\mu g/ft^2$ for floors, 100 $\mu g/ft^2$ for window sills) within the §403 risk analysis were evaluated in this supplement in a detailed review of results from studies that evaluated abatement effectiveness.

In the reviewed studies, geometric mean or median floor dust-lead loadings were generally at or below 41 $\mu g/ft^2$ over periods ranging from 6 months to 6 years post-intervention, with several studies reporting levels below 21 $\mu g/ft^2$ at follow-up periods ranging from 12 months to 2 years. Of the eight grantees participating in the HUD Grantees evaluation that had post-intervention floor dust-lead loadings available at 12 months post-intervention, four had median values for these loadings that were at or below 10 $\mu g/ft^2$. Median pre-intervention floor dust-lead loadings for these four grantees ranged from 9 to 26 $\mu g/ft^2$. For post-intervention window sill dust-lead loadings, geometric means or medians ranged from 24 $\mu g/ft^2$ to 958 $\mu g/ft^2$ in the reviewed studies. Most of the study groups had geometric mean or median post-intervention window sill dust-lead loadings below 100 $\mu g/ft^2$, while a few were at or below 51 $\mu g/ft^2$.

As a result of the post-intervention dust-lead loadings review, a sensitivity analysis documented in this supplement applied the methods developed in the risk analysis under alternative post-intervention floor dust-lead loadings of 10 and 25 $\mu g/ft^2$ and post-intervention

window sill dust-lead loadings of 50 and 75 $\mu g/ft^2$. Results of this sensitivity analyses indicated that while more housing units may be assumed to achieve reductions in average dust-lead loading as a result of lowering the post-intervention dust-lead loading assumptions, the corresponding reduction in the estimated blood-lead concentration and health effect endpoints appeared to be modest, especially compared to the reduction that occurs from pre-intervention conditions.

See page I-i of Appendix I for an executive summary of an investigation on the relationship between lead levels in carpet-dust and children's blood-lead concentration and on how extending the floor dust-lead loading standard in the §403 proposed rule to include carpeted floors might impact the performance of these proposed standards.

1.0 INTRODUCTION

On June 3, 1998, the U.S. Environmental Protection Agency (EPA) proposed regulation to establish standards for lead-based paint hazards in most pre-1978 housing and child-occupied facilities (40 CFR Part 745, "Lead; Identification of Dangerous Levels of Lead; Proposed Rule"). EPA proposed these standards in accordance with Section 403 of the Toxic Substances Control Act (TSCA), as amended by Title X, the Residential Lead-Based Paint Hazard Reduction Act of 1992. The proposed standards are as follows:

- Dust-lead hazards: Household average dust-lead loadings equal to or exceeding 50 $\mu g/ft^2$ on uncarpeted floors and 250 $\mu g/ft^2$ on window sills, assuming wipe collection techniques for dust;

- Soil-lead hazards: Total lead levels equal to or exceeding 2,000 ppm based on a yard-wide average soil-lead concentration

- Hazardous lead-based paint: Lead-based paint in poor condition, defined as follows:
  - More than 10 ft^2 of deteriorated paint on exterior components with large surface areas
  - More than 2 ft^2 of deteriorated paint on interior components with large surface areas
  - Deteriorated paint consisting of more than 10% of the total surface area of exterior or interior components with small surface areas.

These standards, a focal point of the Federal lead program, identify the presence of lead-based paint hazards, defined within TSCA Section 401 as the condition of lead-based paint and the levels of lead-contaminated dust and soil that "would result" in adverse human health conditions.

To provide a scientific basis for selecting the §403 standards, EPA conducted a risk analysis to assess the health risks to young children (aged 1-2 years) from exposures to lead-based paint hazards, lead-contaminated dust, and lead-contaminated soil in the nation's housing. This risk analysis also documented EPA's approach to estimate the reduction in these risks following promulgation of the §403 standards and applied this methodology to evaluate example options for the §403 standards. Finally, the risk analysis provided estimates of the numbers of homes and children that would be affected by various example standards. EPA published this risk analysis in June, 1998, in a document hereby referred to as the "§403 risk analysis report" (USEPA, 1998a).

A period of public comment followed publication of the §403 proposed rule, extending to March, 1999. Several comments received during this period requested additional analyses and investigation. In addition, the Environmental Health Committee of EPA's Science Advisory Board (SAB) performed a review of the technical aspects of the §403 risk analysis, the §403 economic analysis, and the proposed rule. While the SAB concluded that many approaches taken

in the §403 risk analysis were technically sound and scientifically defensible, their final report provided detailed comments and recommendations for additional investigation and analysis to be considered when preparing a final rule (USEPA, 1998b).

This report is a supplement, or addendum, to the §403 risk analysis report. It contains additional information obtained since the report was published that further supports the findings and conclusions made in that report. It also contains the results of additional analyses requested by the SAB and by key public comments. This supplement does not replace any parts of the original risk analysis, but rather supplements the original risk analysis with more detailed analyses on selected topics.

Reflecting its close ties to the original §403 risk analysis report, this supplement contains the same chapters as those found in the §403 risk analysis report. These chapters represent the different components of the risk analysis: Hazard Identification, Exposure Assessment, Dose-Response Assessment, Risk Characterization, and Analysis of Example Options for the §403 Standards. The additional analyses and investigations presented in this supplement are found within the specific chapters to which their findings contribute. Each analysis or investigation is presented as an independent module within each chapter. See the chapter introductions for the contents of each chapter and the motivation for each analysis being presented. Furthermore, the reader is referred to the §403 risk analysis report for details on the risk analysis approach and findings.

1.1 PEER REVIEW

This report was reviewed independently by members of a peer review panel who, together, had considerable knowledge on the subject areas addressed in this report. The three reviewers on this panel who provided EPA with comments on this report were:

Dr. Ruth Chen, Tennessee Department of Health
Joseph Schirmer, M.S., Wisconsin Division of Health, Bureau of Public Health
Nellie K. Laughlin, Ph.D., Harlow Center for Biological Psychology, University of Wisconsin-Madison.

Two of these reviewers (Dr. Chen and Mr. Schirmer) also provided peer review comments on the §403 risk analysis report prior to its 1998 publication. Therefore, they were previously aware of the issues addressed and approaches taken in the original §403 risk analysis. The third reviewer (Dr. Laughlin), while not involved in the review of the §403 risk analysis report, was provided a copy of this latter report for reference while reviewing the present report. Dr. Laughlin was selected primarily based on her involvement in research to investigate the health effects of lead exposures in various animal species, which is addressed in Section 2.1 of this report.

EPA asked the peer reviewers to provide general comments and suggestions concerning this report. In addition, EPA suggested that the reviewers provide more detailed comments on

those sections of the document addressing specific components of the risk analysis in which the reviewer had a particular technical expertise.

The peer reviewers were appreciative in regard to the additional information being provided in this supplement report. They did provide some useful suggestions for additional additions and revisions, however, which were considered when finalizing this report. The remainder of this section discusses how the peer review comments led to report modifications.

One reviewer questioned the usefulness of measuring IQ decrements greater than 1, 2, or 3 resulting from lead exposure, which the risk analysis has included among the health effect endpoints that were estimated. This reviewer pointed out that in an individual child, the variability associated with a measured IQ score is generally greater than the decrement measures of 1, 2, or 3 being considered in the risk analysis. While this may be true, the risk analysis is targeting the decrement in IQ score that occurs on average, across an entire population of children, rather than in an individual child. The variability associated with estimating the mean IQ in a population of children is much smaller than the variability of an individual child's IQ score, thereby allowing the risk analysis to consider these types of IQ decrements. To emphasize and clarify this point to the reader, initial introductions of these health effect endpoints in this report have been revised to refer to "IQ score in the population of U.S. children."

Peer reviews of Section 2.1 of this report, which addressed the adverse health effects associated with lead exposure as observed in animal studies, resulted in considerable revision to this section. Suggestions were followed to re-format Section 2.1.2 to resemble the format of Section 2.1.3, where detailed results of specific studies were incorporated within the general discussion of observed health effects across studies. Additional published articles on specific studies that were suggested and/or provided by the reviewers were obtained and reviewed, and their relevant results were added to the discussion within Section 2.1.2. The articles suggested by the reviewers tended to focus on the effects of lead exposure on the visual and auditory systems and on social development and behavior. Requests for additional information on selected discussion items were addressed by re-reviewing the relevant references that had previously been cited and augmenting the discussion. Finally, general clarification points raised by the reviewers on the content of the section were incorporated when revising the section.

EPA has established a public record for the peer review of this report under administrative record AR-188, "Risk Analysis to Support Standards for Lead in Paint, Dust, and Soil: Peer Review." The record is available in the TSCA Nonconfidential Information Center, which is open from noon to 4 PM Eastern time Monday through Friday, except legal holidays. The TSCA Nonconfidential Information Center is located in Room NE-B607, Northeast Mall, 401 M Street SW, Washington, DC.

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2.0 HAZARD IDENTIFICATION

Chapter 2 of the §403 risk analysis report presented information on the toxicity of lead, through a discussion of how body-lead burden is measured, how lead works in the body, the resulting adverse health effects, and populations at risk. This chapter introduced the endpoints used in the risk analysis to represent the adverse health effects resulting from lead exposure and to estimate the benefits of the §403 rule. These endpoints included the likelihood of exceeding a specified blood-lead concentration threshold (10 or 20 μg/dL), the likelihood of achieving a specified IQ score decrement as a result of lead exposure (1, 2, or 3 points on average across the population), the likelihood of achieving an IQ score in the population of less than 70 points due to lead exposure, and the average IQ score decrement in the population that results from lead exposure. The blood-lead concentration thresholds were among those established by the Centers for Disease Control and Prevention (CDC) as levels of concern. The IQ-related endpoints represented measures of the neurological effects of lead exposure. The representative population upon which the §403 risk analysis focused was children aged 1-2 years, as it was considered the most appropriate age range for the estimation of health effects.

In this chapter of the supplemental report, the results of four additional investigations into hazard identification are presented:

- Section 2.1: A review of the adverse health effects of lead exposure, with a focus on neurological effects, as observed in animal studies.
- Section 2.2: Support for the causality of adverse health effects due to lead exposure.
- Section 2.3: Characterizing the relationship between blood-lead concentration and IQ score.
- Section 2.4: Documenting what is known about the role that dust particle size and the chemical composition of lead compounds in lead-contaminated dust may play in determining the extent to which lead in residential dust is bioavailable to humans.

The motivation for including each of these sections into this chapter is presented within the introduction to each section.

2.1 REVIEW OF THE ADVERSE HEALTH EFFECTS OF LEAD EXPOSURE, WITH A FOCUS ON NEUROLOGICAL EFFECTS, AS OBSERVED IN ANIMAL STUDIES

The §403 risk analysis used data from human exposure studies to characterize the relationship between environmental-lead levels and measures of the various blood-lead concentration and health effect endpoints. However, as the SAB's review of the §403 risk

analysis points out (USEPA, 1998b), causality is difficult to establish using human studies alone, due to the potential for confounding factors being present. For example, while average IQ score may differ significantly between one group of children with low blood-lead concentration and another group with elevated blood-lead concentration, the reason for this difference may not be solely due to lead exposure, but to demographic and other factors that cannot be controlled completely by the researcher. The factors left uncontrolled in the analyses of data from human exposure studies contribute to large uncertainties associated with these analyses. While ethical considerations preclude the use of humans in controlled lead-exposure experiments, a substantial amount of published literature is available on controlled lead-exposure experiments involving animals. Such studies use animals that are chosen from a homogeneous population, reared under identical conditions and randomly assigned to groups where the mode, duration, and amount of lead exposure is controlled within each group (these groups can include one or more control groups). Therefore, in a well-controlled animal study, the presence of significant differences between dose groups can be inferred to be the result of lead exposure at certain doses.

To address the SAB's recommendation to consider the findings of "... animal data, since they support human data by establishing causality, due to the absence of confounding variables, and potential mechanisms for adverse health effects" (USEPA, 1998b), this section presents the key findings of animal studies that have investigated the impact of lead exposure on adverse health effects, especially at low doses. In these studies, animals were typically exposed to lead either *in utero*, during infancy (through mother's milk/formula), during maturation or adulthood, or a combination of these life phases. The major lead-induced adverse health effects noted in humans, including neurological, neurodevelopmental, immunological, and systemic (e.g., cardiovascular, hematological, and renal effects) have been demonstrated in controlled, dose-response studies in rodents, dogs, and/or non-human primates. While this section recognizes the variety of adverse health effects, its primary focus is on the neurological effects of lead, as the §403 risk analysis has recognized that young children are most susceptible to neurological effects due to their developing central nervous systems.

A glossary of selected terms used in this section can be found in Appendix A.

2.1.1 Approach to Reporting the Findings of Animal Studies

This subsection reviews the incidence of adverse health effects associated with lead exposure, as reported in published animal studies. The emphasis of this review is on the neurological, developmental, and neurobehavioral effects of lead.

In preparing this review, it was not desired to duplicate the previous efforts of others who have prepared excellent published literature reviews that have been peer reviewed and are easily available to the general public. These include two articles cited by the SAB (USEPA, 1998b) as "important references" on animal studies data: Rice (1996) and Cory-Slechta et al. (1997). Other important review documents include USEPA (1986) and USDHHS (1999). This section frequently references the content of these and other study review documents.

To help identify any additional articles that may have been published since these key review references were prepared and published, a search of the scientific literature over the last five years was conducted. The strategy for this literature search was on the key words "lead," "effect," "exposure," "neurologic," "behavior," "development," "teratology," and "animal" as whole or root words. Upon review of abstracts identified from this literature search, articles found to be relevant to the objectives of this report were obtained and reviewed, with high priority placed on study reviews.

Certain results of recent key studies identified within the review publications and the literature search are also discussed in detail in this section. Overviews of these studies are presented in Table 2-1. Note that these selected studies represent only a subset of all studies whose results and conclusions provide important contributions to the knowledge base on adverse health effects associated with lead exposure.

Section 2.1.2 focuses on the findings of animal studies on the neurological, developmental, and neurobehavioral effects of lead. This subsection contains an overview of the physiological consequences of these effects, along with a review of general findings of studies investigating these types of health effects. In addition, certain animal studies are discussed in greater detail with summaries of their design and conclusions.

Lead has been documented to have considerably more effects on the health of humans and animals than just neurological effects. Therefore, Section 2.1.3 presents a brief overview of general health effect information and how it relates to lead exposure, as observed in animal studies. Information in this subsection is organized according to the type of health effect.

2.1.2 Neurological, Behavioral, and Developmental Health Effects

Lead has been observed to have widespread neurotoxic effects, as well as to cause behavioral and cognitive symptoms, in humans. These effects are largely consistent with results of morphological, electrophysiological, biochemical, and behavioral studies on animals. Although lead toxicity research has isolated many of the specific neurological effects of lead, it is generally considered to be a relatively indiscriminate toxin within the neurological system. This consideration is largely due to the ability of lead to disturb several fundamental biotic processes such as cellular metabolism and energy production, ion transport across membranes, and protein function. In addition, the neurological effects of lead frequently have been observed to occur as a series of interrelated events. Thus, lead poisoning is likely to cause simultaneous and interrelated disturbances in a number of processes within the nervous system. As an additional consequence, the ability to separate the direct and indirect effects of lead on the neurological system is often difficult (Banks et al. 1997).

Table 2-1. Summary of Lead Exposure Levels and Key Findings for Selected Animal Studies

Authors	Subject Species	Lead Exposure	Key Findings / Effects of Lead Exposure
Altmann et al. (1993)	Rat	0 or 750 ppm lead acetate in diet at various life stages (*in utero*, pre-weaning, and post-weaning)	Active avoidance learning and long-term hippocampal potentiation were impaired when exposure occurred prior to 16 days postnatally.
Burger et al. (1998)	Turtle	0.25, 1.0, or 2.5 mg/g lead acetate in deionized water, by injection; one-time exposure; controls received an isotonic saline solution injection	Death with high dose; dose-dependent righting response impairment with low and moderate doses.
Bushnell et al. (1977)	Monkey	control, lower-dose (targeted blood-lead of 55 µg/dL), and higher-dose (targeted blood-lead of 85 µg/dL) of lead acetate in milk formula in the first year of life	At age 18 months, visual discrimination in the higher-dose group was impaired under dim light compared both to their performance under bright light and to the other groups under various luminescence levels.
Bushnell and Bowman (1979a, b)	Monkey	control, lower-dose (targeted blood-lead of 50 µg/dL), and higher-dose (targeted blood-lead of 80 µg/dL) of lead acetate in milk formula and food in the first year of life	In the first year of life, suppressed play and increased social clinging, as well as various social delays that occur when play environment is abruptly changed (primarily occurring in animals dosed post-natally only; 1979b). At age 4 years, when a subset of the animals was further tested, diminished performance on spatial-cue reversal learning sets was observed in the higher-dose group and, to a lesser extent, in the lower-dose group (1979a).
Chen et al. (1998)	Rat	0.2% lead acetate as drinking water; exposed *in utero* and during nursing via dams, and postweaning directly; controls received 0.145% sodium acetate in drinking water	Altered protein kinase C (PKC) distribution in the hippocampus.
Cory-Slechta (1997) (review)	Rat	50 or 250 ppm lead acetate as drinking water; exposed postweaning; control groups received no lead exposure	Disruption of neurotransmitter systems (i.e., dopamine and glutamine systems); selective learning deficits (e.g., impaired repeat acquisition performance); dose-dependent alteration of fixed interval schedule-controlled response rates.

Table 2-1. (cont.)

Authors	Subject Species	Lead Exposure	Key Findings / Effects of Lead Exposure
Cutler (1977)	Rat	0 or 0.1% lead acetate exposed pre-weaning, and 0 or 0.1% lead acetate exposed post-weaning in drinking water	At 8 weeks of age, duration of non-social activity was significantly greater in the exposure group versus the controls for males but not females. Across both sexes, frequency and duration of social and sexual investigation was decreased in the exposure group.
Fox et al. (1997)	Rat	0, 0.02%, or 0.20% lead acetate in diet at various life stages (*in utero*, pre-weaning, and post-weaning)	Significant association between retinal degeneration and both age and lead exposure level.
Hastings et al. (1977)	Rat	0, 0.02%, or 0.10% lead acetate in diet pre-weaning	At 60 days of age, aggressive behavior was significantly reduced in the exposed groups, but no significant differences were observed in visual discrimination tasks.
Kuhlman et al. (1997)	Rat	750 or 1000 ppm lead acetate, as feed; exposed *in utero*, *in utero* to adulthood, or postweaning to adulthood; controls received no lead exposure	Performance impairment in water maze for all rats exposed *in utero*; no impairment for rats exposed post-weaning.
Lasky et al. (1995)	Monkey	Controls or "modest" exposure to lead via lead acetate *in utero*, pre-weaning, and/or post-weaning within the first year of life	Markedly abnormal distortion product otoacoustic emissions (DPEs) for the two animals with the highest blood-lead concentrations. Otherwise, DPEs did not differ significantly between the groups, although auditory brain stem evoked response (ABR) did differ significantly.
Lilienthal et al. (1990, 1994)	Monkey	0, 350, or 600 ppm lead acetate in diet of mothers and their offspring (*in utero*, pre-weaning, and post-weaning to age 10 years)	At age 7 years, offspring in the exposed groups had significantly higher latencies in flash-evoked and brain stem auditory evoked potentials (BAEP) which increased with increasing click rates (1990). At age 12 years, offspring in the exposed groups had significantly increased amplitudes of the scotopic b-wave in electroretinogram (ERG) recordings (1994).
Mello et al. (1998)	Rat	1.0 mM lead acetate as drinking water; exposed *in utero* and during nursing via dams; control dams received deionized water only	Selective motor skill impairment (accelerated fist eye opening, startle reflex, and free-fall righting; impaired spontaneous alternation performance in maze).

Table 2-1. (cont.)

Authors	Subject Species	Lead Exposure	Key Findings / Effects of Lead Exposure
Nagymajtenyi et al. (1998)	Rat	80, 160, 320 mg/kg/day lead acetate in distilled water, by gavage; exposed pre- or post-natally; controls received same volume of distilled water by gavage	Dose dependent increase in behavioral and bioelectric aberrations (e.g., hyperactivity).
Rice and Gilbert (1990a, 1990b)	Monkey	1.5 mg/kg/day lead acetate, in capsules; exposed continuously after birth, postnatally until 400 days, or after 300 days from birth; controls received vehicle only	Nonspatial discrimination tests (1990a): group dosed continuously from birth onward exhibited greatest degree of impairment, followed by group dosed after infancy only (no impairment observed in group dosed during infancy only). Delayed alternation task (1990b): all exposed groups showed impairment to an approximately equal degree.

Research, based on both human observation and animal studies, indicates that relatively low doses of lead can adversely affect both the peripheral and central nervous systems, while high lead exposures can result in acute lead encephalopathy, and may ultimately lead to death. Lead induced damage to the brain and nervous system may be manifested as various and diverse developmental symptoms, including both behavioral and cognitive impairments.

In the following subsections, the physiological effects of lead on the brain and nervous system (Section 2.1.2.1) and subsequent effects on development and behavior (Section 2.1.2.2) are discussed. The findings of specific studies listed in Table 2-1 above are included in this discussion. In general, some caution must be taken when extrapolating the findings in these studies to humans and to other species. For example, lead neuropathy in rats is primarily characterized by demyelination of nerves, while cats, rabbits, and humans generally show damage to the axons of nerves (Davis et al., 1990). Developmental age of the brain also varies between animal species and humans. For example, at birth, the rat brain is relatively less developed and is roughly equivalent to the human brain at 5-6 months of gestation (Winneke et al., 1996). Furthermore, although rats and possibly monkeys tend to have higher tolerances for the general toxic effects of lead exposure relative to humans (i.e., they require a higher exposure level to reach an equivalent blood toxicity level), the lowest blood-lead levels at which lead-induced developmental/neurobehavioral effects have been observed in animals and in humans are reported to be similar in magnitude (Banks et al., 1997; Davis et al., 1990; USDHHS, 1999).

2.1.2.1 Physiological Effects of Lead on the Neurological System.

Overview. As lead is known to adversely affect several universal processes within biological systems, the symptoms of lead poisoning have been observed in many cell types, tissues, and organs within the neurological system.

On the cellular level, lead has been observed to cause disruption of mitochondrial function (i.e., cellular metabolism), damage to cell structural components (e.g., microtubules), and damage to glial cells and the myelin sheaths and axons of nerve cells (USEPA, 1986). Electrophysiological and biochemical processes at the cellular level may also be disrupted by lead exposure. Specific alterations of these processes reported in animal studies include impairment of synaptic events and neuron function, interference with neurotransmitter function, and protein activity inhibition (e.g., enzymes and hormones) (USEPA, 1986). Much of lead's role in cellular level dysfunctions is suggested by many researchers to be attributed to its interference with calcium-mediated processes (Banks et al., 1997). Calcium is an important ion in many biological systems and is specifically involved in neurological phenomena such as enzyme-protein activation, secondary messenger regulation of metabolic pathways, membrane potential/ion channel regulation, and neurotransmitter release. Lead ions, as they are similar to calcium ions in both size and charge, can substitute for calcium and thus competitively interfere with these types of calcium-mediated cell processes.

Disruption of cellular processes can eventually result in damage to tissues and organ systems within the neurological system. Reported effects of lead in animals at the organ and system level within the central nervous system include compromise of the blood-brain barrier, disruption of the limbic system and cerebral cortex, and damage to the cerebellum (Banks et al., 1997; USEPA, 1986). Animals exposed to lead in early post-natal life have also exhibited reductions and delays in development of various brain regions, including the hippocampus and cerebral cortex (Banks et al., 1997; USDHHS, 1999). The observed effects of lead exposure on specific organ systems and processes, as observed in animal studies, are now discussed.

Blood-brain barrier. Banks et al. (1997) reviewed animal studies that examined the effect of lead exposures on the blood-brain barrier that regulates the movement of chemical substances in and out of the brain. In studies of rats exposed to relatively high lead levels, higher concentrations of lead were observed in the barrier capillaries than were observed in the brain as a whole. USEPA (1986) reviewed studies which provided evidence that lead transport within the brain is by the same mechanisms as calcium transport. Thus, as the capacity for calcium transport (specifically, into neural cell mitochondria) is known to be much higher in the brain than other body tissues, a lead accumulation in brain capillaries is not unexpected (USEPA, 1986). Acute lead toxicity as lead accumulations in brain capillaries may disrupt barrier permeability, allow greater influxes of water, ions, and other substances, and result in swelling of the brain (encephalitis) (USEPA, 1986). Although lower lead exposure levels ($< 40 \mu g/dL$) have not been reported to result in specific damage to the barrier or in disproportionate accumulation of lead in the capillaries of the blood-brain barrier (Banks et al., 1997), developing brains have been suggested to be particularly susceptible to the transfer of even very low levels of lead from

the blood into the brain since the blood-brain barrier is not yet fully functional (Altmann et al., 1993; Cory-Slechta, 1997).

General cellular processes. Several *in vitro* studies with animal cells have provided evidence that a major site of lead interference is with cellular metabolism and energy transfer via disruption of the normal mitochondrial ion gradient (Banks et al., 1997; USEPA, 1986). In addition, mitochondria in the cerebellum and in developing brains of animals have been observed to show a greater sensitivity to lead disruption than mitochondria in other body tissues or at other ages (USEPA, 1986). This has been suggested to provide a possible explanation for one of the root causes of the greater sensitivity of the neurological system, and of the young in particular, to lead poisoning (USEPA, 1986). Impairment of mitochondrial energy production subsequently can affect many other energy-requiring cellular processes, such as protein synthesis, lipid synthesis, and membrane integrity. In animal studies, the normal development of proteins in neurons was altered in rats exposed to lead perinatally (USDHHS, 1999). Other studies reviewed by Banks et al. (1997) indicate that moderate levels of lead can interfere with microtubule formation (*in vitro* animal cell studies) and the formation of the myelin sheath in neurons in both the central and peripheral nervous systems of lead exposed rodents.

Lead can also interfere directly with calcium-mediated cell processes. This may include disruption of protein function (e.g., enzyme regulation of cell growth and differentiation), ion transport systems across membranes, and membrane potentials (Banks et al, 1997; USEPA, 1986). For example, a study performed by Chen et al. (1998) found that hippocampal protein kinase C (PKC) activity, which has been correlated with performance in several learning tasks, was altered by relatively low internal lead exposures (<30 μg/dL) in postnatal rats. More information on this study and its findings can be found in the discussion below on the limbic system and hippocampus.

Neuron and neurotransmitter function. Lead-induced disruption of cellular processes may result in neuron dysfunction. According to a body of experimental animal research reviewed by Banks et al. (1997), evidence exists that moderate to low levels of lead exposure can impair synapse formation in the hippocampus during postnatal development of rats, in the visual cortex of primates, and in the frontoparietal cortex of guinea pigs, and also appears to interfere with synaptic transmission in fetal rat hippocampal neurons by blocking postsynaptic receptors. USEPA (1986) reviewed numerous studies in which rats exhibited morphological effects such as decreased glial cell and synaptic density, and delayed maturity of synapses in neurons of the cerebral cortex with lead exposure. Several of the reviewed studies also reported abnormal development of neuron dendrites and persistent impairment of electrical activity (i.e., reduced firing rates) in the cerebellum of cats and rats exposed perinatally to low levels of lead (Banks et al., 1997). The cerebellum is responsible for the regulation and coordination of complex voluntary muscular movement, and is also implicated in cognitive attention-switching activity. The cerebral cortex is largely responsible for higher brain functions, including sensation, voluntary muscle movement, thought, reasoning, and memory. Thus, damage to neurons in these areas may explain some attention deficits, learning and memory impairments, and disturbances in

motor coordination that have been observed in behavioral studies of lead exposure (Banks et al., 1997).

Lead has also been observed to interfere with the release and uptake of neurotransmitters in various *in vitro* and *in vivo* animal studies, including the inhibition of neurotransmitter release from calcium sensitive voltage channels in snails, and alteration of synthesis and turnover rates of various neurotransmitters in different regions of the brain (hippocampus, cerebellum, hypothalamus, brainstem) in lead-exposed rats (Banks et al., 1997; USEPA, 1986; Nagymajtenyi et al., 1998). Altered activity of various neurotransmitters has been observed in studies involving rats exposed to lead prenatally and postnatally (USDHHS, 1999). In the reviewed literature, lead has generally been reported to have highly variable and non-specific effects on the various neurotransmitter systems, possibly due to its more general effect on metabolic processes (Banks et al., 1997).

Cory-Slechta (1997) conducted a review of studies linking disruptions in neurotransmitter systems (i.e., dopaminergic (DA) and glutamatergic (GLU) systems) with behavioral and cognitive impairments in lead exposed animals. The author cites previous research which indicates that the DA and GLU neurotransmitter systems are critical to various cognitive functions, and also are sensitive to lead-induced disruptions. In addition, the author reviews studies reporting non-lead related disruptions of these systems and the concurrent appearance of behavioral symptoms that are very similar to symptoms of lead toxicity. However, the author also concedes that much of the historical research linking behavioral impairments to biochemical effects is based solely on correlations and that relationships are complicated by the fact that any given behavioral symptoms may have multiple physiological mechanisms. Cory-Slechta (1997) summarized several studies conducted in her own lab on rats exposed, postweaning (i.e., at 21 days of age), to lead (0, 50 and 250 ppm lead as lead acetate) in drinking water for varying durations of time. Results of these studies indicated that the DA neurotransmitter system is vulnerable to lead-induced modifications, such as impaired regulation of DA synthesis and release. These modifications to DA system function were suspected to contribute to alterations in response control (fixed interval schedule-controlled behavior) that were observed in lead-exposed rats in the reviewed studies. Results indicated that the GLU system was also involved in lead-induced impaired learning, primarily as manifested by an increase in perseverative errors in lead-exposed rats, relative to controls. There was no evidence that the DA system was involved in learning accuracy. The author suggests that confirmation of the involvement of the GLU and DA systems in lead-induced effects, could also have implications extending beyond cognitive concerns. For example, similar patterns of neurotransmitter disruptions have been associated with schizophrenia, drug addiction, and psychosis (Cory-Slechta, 1997).

Lead-induced disruption of neuron and neurotransmitter function can result in altered bio-electric activity in the brain and nervous system. This was seen, for example, in a study by Nagymajtenyi et al. (1998) in which 120 female and 60 male rats and their 120 male offspring were administered a lead acetate solution by gavage at a concentration of either 0, 80, 160, or 320 mg/kg. There were three variations on the treatment schedule: (1) pregnant females were dosed only during the 5^{th} -15^{th} day of pregnancy; (2) pregnant females were dosed during pregnancy and

lactation; or (3) pregnant females were dosed during pregnancy and lactation, and their weaned offspring were dosed for 8 weeks. Behavioral observations of the offspring were made at 12 weeks. The study observed electrophysiological disruptions, including changes in electrocorticogram (ECoG) indices, cortical evoked potential, and slowed nerve conduction velocity, in the somatosensory area of the cerebral cortex in the rats exposed to lead both pre- and post-natally relative to controls. Electrophysiological functions showed both dose- and treatment-dependent changes, including decreased mean amplitude and increased frequency of the ECoG, and lengthened latency and duration of the evoked potentials. The observed changes in electrophysiological functioning depended on the dose and timing (i.e., age of animal at exposure) of lead administration. The authors suggested that non-invasive monitoring of electrical disturbances in the nervous system may provide a valuable and early indicator of low-level lead poisoning.

The limbic system and hippocampus. The limbic system (e.g., hippocampus) is of particular interest in lead toxicity studies as it is key in many of the processes that appear to be affected by lead poisoning, including cognition, emotion, motivation, behavior, memory, and various autonomic functions.. Some researchers have even suggested that symptomatic similarities (e.g., learning and memory impairments) between lead toxicity and other experimental limbic system disruption indicate that the limbic system is a target site for lead toxicity in the brain (Walsh and Tilson, 1984).

Reported behavioral changes that may be attributable to hippocampal damage include increased aggressiveness, seizures, inappropriate responsiveness, reversal problems, visual discrimination deficits, impaired motor coordination, and other types of learning deficiencies (Petit et al., 1983). Furthermore, the developing hippocampus may be more susceptible to functional injury by lead exposure compared to the mature hippocampus. For example, Altmann et al. (1993) conducted a study where 88 female rats were dosed with lead acetate in their diet at either 0 or 750 ppm for 50 days prior to mating through day 16 following birth of a litter, at which time 2-3 male offspring were taken from each litter and dosed under the same regimen until sacrifice. Half of the animals were tested for two-way active avoidance learning. This study observed a correlation between hippocampal disruptions and active avoidance learning deficit for rats exposed to lead during the prenatal and early postnatal stages (i.e., during hippocampal development), but not when lead exposure occurred only after 16 days postnatally.

Some studies reviewed in the literature differ on the extent to which the hippocampus is a target organ for lead. For example, relative to other regions of the brain, greater impairment of neuron function has been observed in hippocampal cells of rats exposed to lead perinatally (Banks et al., 1997). Studies reviewed by Petit et al. (1983) indicated that higher levels of lead may tend to accumulate in hippocampal cells of rat brains. However, other studies reviewed by Banks et al. (1997) did not report a preferential accumulation of lead in the hippocampus of young lead-exposed rats. For example, a 1994 study by D.V. Widzowski and D.A. Cory-Slechta (as cited in Banks et al., 1997) exposed rats postnatally to lead from dams' milk (dosed with four levels of lead from 100 to 2000 ppm lead) and measured lead levels in 12 brain regions after 7-60 days. The study found that lead tended to accumulate in similar patterns across brain regions and

exposure levels. Some researchers have hypothesized that the particular vulnerability of the hippocampus to lead poisoning may be due more to sensitivity rather than increased lead accumulation in this region (Banks et al., 1997). In addition, because the most rapid phase of development of hippocampus is known to occur postnatally (late compared to other brain regions), a particular sensitivity of the hippocampus to early-life lead exposure is plausible (Petit et al, 1983; Altmann et al., 1993).

Specific lead-associated physiological effects in the hippocampus, as reported in animal studies reviewed by Banks et al. (1997) and Petit et al. (1983), include significant reductions in size and weight of the hippocampus and reductions in hippocampal cell layer thickness with relatively high perinatal lead exposure in rats. Damage and stunting of hippocampal structural cells (glial cells/astrocytes) was observed in rats and monkeys exposed to lead prenatally and postnatally. Perinatally exposed rats also exhibited reductions in development of hippocampal neuronal dendrites and mossy fiber pathways, which are both involved in the transmission of nerve impulses (Petit et al., 1983).

Initially mentioned in the overview of general cellular processes above, Chen et al. (1998) performed a study to investigate the effects of developmental lead exposure on protein kinase C (PKC) activity in the hippocampus of rats at various postnatal ages. This study attempted to elucidate some of the physiological mechanisms of lead-induced learning deficits. In this study, lead was administered orally as 0.2% lead acetate in drinking water to pregnant and lactating female rats and then directly to their weanling pups (weaned at postnatal day 21) in drinking water. Controls received 0.145% sodium acetate in drinking water. Four to six rat pups were randomly selected for necropsy from different dams at postnatal days 7, 14, 28, and 56, and PKC activity was measured in both the membrane and cystolic fractions of the hippocampi. Results showed that lead exposure increased PKC activity in the cystolic fraction at postnatal day 56, and decreased PKC activity in the membrane fraction at postnatal day 7. The ratio of membrane to cystolic PKC activity, which is indicative of PKC distribution, decreased at postnatal days 28 and 56.

A review of studies in Chen et al. (1998) indicated that PKC activity has been associated with various brain functions (e.g., ion channel function, receptor function, and neurotransmitter release) and that alteration of hippocampal PKC, in particular, has been correlated with poor performance in several learning tasks. Therefore the authors hypothesize that the lead-induced alterations of PKC activity and distribution observed in their study may have caused functional changes in the animal brain, including modulation of ion channels, desensitization of receptors, and enhancement of neurotransmitter release. Chen et al. (1998) also suggested that some of the learning and memory deficits observed in children are likely to be causally related to the types of PKC activity alterations exhibited in this study.

The visual system. Some researchers contend that the retina serves as a good model for studying the effects of lead on the central nervous system. Because most retinal cells, like the central nervous system, develop during gestation and, in the rat, for up to two weeks postnatally, researchers have studied the effects of lead exposure on retinal cell development in post-natal

rats in order to characterize such effects for humans during early gestation and post-natal periods. Fox et al. (1997) studied the effects on retinal development of low and moderate lead exposure via mother's milk in female Long-Evans hooded rats aged 0 to 21 days (i.e., from parturition to weaning). These rats were partitioned into three dose groups (6-14 rats per group) based on the concentration of lead (0, 109, or 1090 ppm) in the lead acetate solution of drinking water that the dams were provided. At 21 days of age, lead levels in the blood and the retinas of rats in both the low and moderate dose groups averaged significantly higher than the control group, while significantly higher results were seen at 90 days of age in only the high dose level group and in only retina-lead levels. Blood-lead levels averaged 19 and 59 μg/dL in the low and high dose groups at 21 days of age compared to 1 μg/dL in the control group. Significant retinal degeneration (i.e., rod and bipolar apoptic cell death) was associated with age and lead exposure levels in this study. The higher loss rate of rods in the lead-exposed groups was associated with a loss of rhodopsin content, implying that the loss was directly due to the presence of lead. The authors concluded from these results (when considered with the results of other researchers) that the developing retina may be more sensitive to lead exposure in pre-weaned rats than the hippocampus.

Also adopting the hypothesis that the effects of lead on the central nervous system leads to adverse effects on the eye, Bushnell et al. (1977) document the findings of experiments to characterize the relationship between high food-lead exposures early in life and impaired scotopic visual function (i.e., night blindness). In their experiments, baby formula was spiked with lead acetate and given daily to six rhesus monkeys in their first year of life. Lead consumption was regulated to allow blood-lead levels to be maintained at an average of 55 μg/dL for three monkeys and 85 μg/dL for three monkeys. Four other monkeys whose formula was not spiked with lead served as a control group. Approximately 18 months after this feeding paradigm was ended, monkeys in all three groups averaged nearly normal blood-lead levels. At this time, the monkeys were administered a discrimination procedure at various light levels to test their ability to select an option which provided reinforcement (i.e., food) versus an option which did not provide reinforcement. Animals exposed to the higher levels of lead in the first year of life (i.e., the 85 μg/dL group) performed significantly worse in this procedure compared to animals in the control and lower lead groups as light levels were reduced. Various controlling factors within this experiment allowed the researchers to conclude that the primary reason for the degraded performance in the higher-dosed animals was most likely a loss of scotopic function. Thus, the researchers concluded that lead exposure early in life was associated with impaired scotopic visual performance later in life, even when blood-lead levels in these animals were allowed to return to normal levels.

In a study performed by Lilienthal et al. (1994), 15 rhesus monkeys were pre- and post-natally exposed to lead at one of three levels (0, 350, 600 mg/kg lead acetate) until they were nearly 10 years of age. At approximately 12 years of age, electroretinogram (ERG) recordings were made of each eye in these monkeys. Five animals were in the control group (average blood-lead concentration of 0.44 μg/dL), four in the lower-dosed group (average blood-lead concentration of 4.55 μg/dL), and six in the higher-dosed group (average blood-lead concentration of 8.26 μg/dL). Significant differences (at the 0.05 level) were observed across

dose groups in the amplitudes (but not latencies) of the scotopic b-wave, with increased amplitudes seen in the two lead-dosed groups, and the nature of the differences being dependent on luminescence level. The lead-induced effects were similar to those observed earlier in these animals and were similar to the effect of dopamine antagonists. This suggests that lead may be permanently affecting dopaminergic processes.

Over many years, significant visual impairment associated with lead exposure has been observed at high lead levels in rabbits, rats, and monkeys, both at the retina and visual cortex (Otto and Fox, 1993). Exposure to moderate levels of lead by rats can result in decreases in rod cells, thinning of retinal layers, reductions in the number of axons in the optic nerve, and necrosis of photoreceptors and cells in the inner retinal layer (Banks et al., 1997; USEPA, 1986). While such damage is less frequently associated with low levels of lead exposure, functional and neurochemical effects on the retinal system can be associated with low-level lead exposures in rats (i.e., blood-lead concentrations below 20 μg/dL; Otto and Fox, 1993), along with persistent decreases in visual acuity and spatial resolution (USDHHS, 1999). While considerable data exist to allow the neurotoxicity of lead to be characterized, considerably less data exist on the morphological effects of lead on the visual system, especially at low exposure levels. In addition, Otto and Fox (1993) have concluded that the effects of lead are more likely to adversely affect rod cells compared to cone cells.

Davis et al. (1990) reviewed several studies in which lead exposed rats and monkeys exhibited decreased responsiveness of neurons to visual stimuli, as assessed by parameters (i.e., visual evoked potentials) which measure nerve conduction. Lilienthal et al. (1990) found that central visual processing, as reflected in measurements of visual evoked potentials (VEP), was clearly affected in lead exposed monkeys. Monkeys were exposed to dietary lead pre- and postnatally (low lead group at 350 ppm and high lead group at 600 ppm per day), and VEP was measured in response to visual flash stimulation beginning at age seven. Results showed that amplitudes of sensory-evoked potentials were smaller and latencies were longer in lead-exposed monkeys relative to controls, even in monkeys with the lower lead exposure regime.

The study by Nagymajtenyi et al. (1998) introduced earlier in this subsection also observed disruptions in the functioning of optical nerves in prenatally and postnatally lead-exposed rats. In their study, effects of lead exposure were most often significant in the middle and high lead exposure groups, and included dose dependent changes in visual evoked potentials and slowed nerve conduction velocity, as measured in response to flash stimulation.

The auditory system. In a review of the literature (on both human and animal studies) on the effects of lead exposure on the auditory system, Otto and Fox (1993) concluded that lead exposure is more likely to adversely affect that portion of the auditory system that resides within the central nervous system (e.g., cochlear nerve) compared to more peripheral sites where sensory transduction processes occur. However, more data were deemed necessary to more definitively characterize the effects of lead on specific sites within the auditory system and how such auditory dysfunction contributes to a child's overall learning impairment that can be attributed to lead exposure.

Certain animal studies have reported an association between lead exposure and disruptions of the auditory system. For example, Lilienthal et al. (1990) observed lead-induced alterations in auditory functioning in monkeys, as reflected in measurements of brainstem auditory evoked potentials (BAEP). In their study, monkeys were exposed to dietary lead pre- and postnatally (low lead group at 350 ppm and high lead group at 600 ppm per day), and BAEP was measured in response to auditory click stimulation beginning at age seven. Results showed that BAEP latencies increased with increasing click rates in lead-exposed monkeys relative to controls, although the increase tended to be consistent in the group of monkeys with the higher lead exposure regime (600ppm). The study by Nagymajtenyi et al. (1998) (cited earlier in this subsection) reported other types of disruptions in the auditory pathway in prenatally and postnatally exposed rats, including changes of auditory evoked potential and slowed nerve conduction velocity, as measured in response to click stimulation.

To assess the long-term auditory effects of chronic lead exposure at low levels, Lasky et al. (1995) assessed auditory functioning in two groups of 11-year-old rhesus monkeys, where one group contained 11 monkeys who were exposed to lead either pre- or post-natally (within the first year of life), and the other group contained 8 monkeys were not exposed. Auditory function in these monkeys was assessed by measuring distortion product otoacoustic emissions (DPEs), auditory brain stem evoked responses (ABRs), and middle latency evoked responses (MLRs). The two animals with the highest blood-lead concentrations during their first four years had "markedly abnormal" DPEs, where this result was found not to be due to canal obstruction nor middle ear problems. Among the remaining animals, DPEs did not differ significantly between the two groups, although DPE amplitudes increased more rapidly (given the stimulus level) in the control group compared to the exposed group. The ABRs, but not the MLRs, differed significantly between the two groups of animals. The exposed animals tended to have slightly longer latency ABRs.

2.1.2.2 Behavioral and Developmental Effects of Lead. Several recent review papers have summarized previous research and advances in the area of neurotoxicological effects of lead. The findings of key studies and of study reviews by Banks et al. (1997), Cory-Slechta (1997), Davis et al. (1990), and Rice (1996), among others, are presented below. When possible, attention was focused on health effects at low levels of lead exposure or at low blood-lead levels. Many animal studies have investigated the neurobehavioral effects of lead exposure, that is, effects upon learning and performance activities. Some studies also addressed related issues such as: (1) what blood-lead levels were associated with learning and performance deficiencies in test animals; (2) whether other signs of toxicity were present; (3) what biochemical mechanisms produce toxicity; and (4) how blood levels in exposed animals correlate with specific blood-lead levels in humans at which analogous effects upon learning/IQ are observed. Many experiments utilize rats or monkeys as subjects since they respond to motivational factors, usually food rewards, and can be induced to learn certain behaviors of interest to researchers.

To synthesize the neurobehavioral effects of lead across species, Davis et al. (1990) conducted a review of rodent, primate, and human studies. The lowest levels of internal lead

exposure during early development at which neurobehavioral effects have been observed were reported to be <20 µg/dL for rodents (Cory-Slechta et al., 1985) and 15-25 µg/dL for primates (Rice, 1985). These ranges are similar to the lowest range (10-15 µg/dL) in which adverse neurobehavioral effects have been observed in children (Davis et al., 1990; Fulton et al., 1987; Silva et al., 1988; USEPA, 1999).

Rice (1996) has summarized results from both human epidemiology and animal studies of the potential behavioral and developmental effects of lead exposure as follows: "Increased distractability, inability to inhibit inappropriate responses, perseveration, and inability to change response strategy are common themes that may be extracted from both literatures." These observations are noted in the findings presented throughout this subsection.

Learning deficiencies and behavioral test performance. To investigate the association between lead exposure and learning deficiencies, Rice (1996) conducted an extensive review of animal studies, interpreted in conjunction with learning disabilities in children as reported in human epidemiological studies. In experiments with rats or monkeys, a general learning deficiency was frequently observed at high lead exposure levels. However, for monkeys exposed to low or moderate lead levels, the majority of the impairment was evident only with the performance of more complex tasks, such as non-spatial discrimination reversal tasks. This phenomenon was observed in (a) monkeys exposed to lead from birth with preweaning blood-lead levels of 50 µg/dL and adult blood-lead levels of 30 µg/dL (Rice, 1988); (b) monkeys with blood-lead levels of 30-35 µg/dL from infant formula and postweaning levels of 19-22 µg/dL (Rice and Gilbert, 1990a); and (c) monkeys with blood-lead levels of 15-25 µg/dL during infancy and steady-state levels of 11-13 µg/dL during adulthood (Rice, 1985). In these studies, lead exposure had ceased by the time performance tests were conducted.

Kuhlman et al. (1997) conducted an experiment with 10 rats reared in each of five groups. The control group received no lead exposure. The "maternal exposure" group was exposed to lead *in utero* and during lactation (750 ppm lead acetate in feed via dams), but was moved to a control diet after weaning. The permanent group was exposed to lead both *in utero* and continuously afterward into adulthood (750 ppm lead acetate in feed). Finally, there were two post-weaning groups, in which no exposure occurred until after weaning, and then pups were fed diets containing two different lead concentrations (750 or 1000 ppm lead acetate in feed). Rats were performance-tested using a water maze at about 100 days of age, which tested their ability to locate a submerged platform, and their blood-lead levels were measured at that time. Even though average blood levels in that group (1.8 µg/dL) had returned to control levels by the time of the test, a highly significant impairment in performance (e.g., longer time to find platform, longer pathway to platform) was observed in both the maternal and permanent exposure groups. The post-weaning exposure groups did not show any significant performance impairment, even though average blood-lead levels exceeded 20 µg/dL at the time of testing. The authors suggested that the results seen in the maternal group demonstrated the impact of lead upon early development.

In a study documented by Mello et al. (1998), rat pups were exposed to lead *in utero* and during nursing, using 1.0 mM lead acetate administered to dams as drinking water. Eleven litters from control females and nine litters from lead-exposed females were used, for a total of 160 pups. The pups were observed for physical development and tested for reflexes/behavior at aged 17-19 days. While lead exposure appeared to significantly accelerate the appearance of first eye opening, startle reflex, and free-fall righting, it significantly impaired spontaneous alternation performance in a maze. No explanation was given for the seemingly contradictory effects in this study, though the authors suggested that any lead-induced alterations in animal development or behavior, regardless of direction, must be considered deleterious.

Operant discrimination and reversal tasks. Rice (1996) describes experiments that were conducted with visual discrimination problems with the addition of "reverse performance" requirements, and/or the addition of irrelevant distracting signals. In the operant discrimination reversal task, the researcher changes the pattern of rewards so that previously-learned correct and incorrect responses become switched. Lead-exposed animals sometimes perform as well as controls in the original learning acquisition, but perform poorly when the change of rules requires learning a change in strategy. Also, lead-exposed animals tend to be distracted by irrelevant details more than do controls (although in some cases they may perform similar to controls in the absence of such distraction). For example, Gilbert and Rice (1987) report an experiment in which monkeys exposed to low lead levels (50 and 100 µg/kg/day) through age 10 years tended to perform poorly, relative to controls, on spatial discrimination reversal tasks with unfamiliar distracting cues, but adequately on tasks with familiar distractions. Furthermore, the lower dose group was impaired only during the tasks immediately after the introduction of the irrelevant stimuli, but not after the irrelevant stimuli became familiar. Under these dosing regimes, steady-state blood lead concentrations in these mature adult monkeys (13.1 µg/dL for the higher dose group and 10.9 µg/dL for the lower group) approximate levels typical for humans in industrialized environments (Gilbert and Rice, 1987).

Bushnell and Bowman (1979a) reported diminished performance on spatial discrimination reversal tasks by adult monkeys (4 years of age) that were exposed to dietary lead in the first year of life (either 0.287 mg/kg or 0.880 mg/kg per day as lead acetate in formula). This finding was observed despite average blood-lead levels in each group being essentially normal at the time of testing.

Impairments in learning and performance have also been noted in experiments with lead-exposed rats. Cory-Slechta (1997) found that selective learning deficits were present after lead exposures of 50 and 250 ppm (as lead acetate in drinking water) with resulting blood-lead levels as low as 20-25 µg/dL. Lead-exposed rats performed as well as controls during the performance component of the experiment (i.e., the correct sequence of responses remained constant across trials), but less accurately during the repeat acquisition component (the correct sequence changed in an unpredictable way with each new set of trials). Rats also displayed perseverative behavior, pressing the same lever repeatedly, even though the experiment precluded this pattern of response from generating a food reward. The author discusses the research attempting to elucidate some of the biochemical mechanisms underlying these results, including evidence

which strongly suggests the existence of a link between learning impairments and lead-induced disruptions of neurotransmitters, particularly those of the glutamine system.

Cory-Slechta (1997) also reviewed the reported effects of lead on fixed interval (FI) test performance (delayed response operant schedule of food reinforcement). The FI test requires the animal to bar-press only at specific minimum time intervals before a food reward can be provided. Studies conducted by Cory-Slechta showed that rats exposed to low doses of lead (25-300 ppm with postweaning exposures) tended to respond more rapidly than did controls, even though this behavior resulted in withheld rewards. In contrast, animals exposed to higher doses (500 ppm and above) showed decreased rates of FI responding, at least initially. It is hypothesized that the increased response rates may actually be a form of perseverative behavior. The author notes that, in other studies, lead-induced interference with the dopamine system has been suggested as a possible mechanism for perseverative behavior. Also of significance, Cory-Slechta reports that dose-dependent patterns in FI performance, like those in her own study, have consistently been observed across a wide range studies and methodological conditions, including species (i.e., reported effects described in rats, monkeys, sheep, pigeons, and mice) and developmental period (i.e., prenatal, postnatal, postweaning, adult, old adult) during which lead exposure occurs. Thus, evidence in the literature strongly suggests that changes in FI schedule-controlled behavior seem to be one of the most reliable parameters, relative to other measures, for assessing the behavioral effects of experimental lead exposures (Cory-Slechta, 1997).

Rice and Gilbert (1990a, 1990b) conducted a series of behavioral impairment studies on 52 infant monkeys. Shortly after birth monkeys were assigned in equal numbers to one of four feeding groups: (1) a control diet; (2) a lead-containing diet continuously after birth; (3) a lead-containing diet from birth until age 400 days, followed by a control diet; and (4) a control diet from birth until age 300 days, followed by a lead-containing diet. Lead was administered orally in gelatin capsules as lead acetate in 0.05 M sodium carbonate, equivalent to 1.5 mg/kg/day. Feeding regimes were maintained up to and through the time of behavioral testing, which occurred from about 5 through 7 years of age. When monkeys were 5 to 6 years old, they were tested on a series of nonspatial discrimination reversal tasks, including form, form with irrelevant color cues, color with irrelevant form cues, and alternating form and color (Rice and Gilbert, 1990a). Based on this series of tests, the group dosed continuously from birth exhibited the greatest degree of impairment, followed by the group dosed after infancy only. The group dosed during infancy only did not exhibit significant impairment on these tasks. The results of this study provided evidence that while exposure to lead after infancy can produce impairment, this effect is exacerbated if the animal was also exposed during infancy. Approximately one year later, the same group of monkeys were tested on a spatial delayed alternation task in which they were required to alternate responses between two push buttons, with an increasing delay in response time (Rice and Gilbert, 1990b). In this series of tests, all three exposure groups showed performance impairment relative to controls and were impaired to an approximately equal degree, exhibiting "perseverative behavior" and an inability to suppress inappropriate responses during test delay intervals. The authors conclude that that there is not an early critical period for lead-induced impairment on spatial delayed alternation tasks, and that lead exposure only during

infancy (i.e., < 400 days) can result in impairment comparable to exposure that continues beyond infancy (Rice and Gilbert, 1990b).

General neurobehavioral effects. A review of animal studies which evaluated the persistence of lead-induced effects on cognitive development was conducted by Tong (1998). Neurobehavioral toxicity was reported to persist for up to 10 years of age in monkeys exposed to low levels of lead and was strongly suggested in some studies to be an irreversible neurotoxin. The physiological evidence of lead disruption of the developing brain (e.g., neuron and synapse formation), and the fact that intracellular lead may not be removable from neural cells, also supports the plausibility of enduring, and possibly irreversible, deficits in neurobehavioral function with lead exposure.

Burger et al. (1998) conducted an experiment on 48 slider turtle hatchlings which were randomly assigned in equal numbers to a control group and three lead exposure groups. The exposed groups experienced a single injection (intramuscularly) of one of three doses of lead acetate (0.25, 1.0, or 2.5 mg/g). No survivors remained in the high dose group by 120 days of age. Behavioral observations, growth (weight and length), and survival data were taken both prior to and at various times after lead injection (i.e., weekly during the first month, and at 4 weeks, 4 months, and 6 months post-exposure). The primary behavioral measure was a righting response (i.e., latency before attempting righting and time required to turn over), as the animals naturally tended to right themselves whenever placed on their backs, and other behavioral measures were not reliably exhibited in this species. By the age of 6 months, righting response adjusted for body weight was significantly impaired by lead dosing. Both the low and medium dose groups performed significantly worse than did controls, and the medium dose group performed significantly worse than did the low dose group, indicating a dose-response effect. The range of lead doses used in the study had such a marked impact on survival that "the threshold for behavioral effects is on the same order of magnitude as the LD_{50}" (i.e., the lowest lead dose at which only 50% of animals would survive). The authors noted that although these results suggest hatchling turtles are vulnerable to lead exposure, behavioral effects due to lead may be very subtle in the field for this species. In addition, when effects do become evident, the levels of lead in turtles may be dangerously close to the threshold for lethality.

USEPA (1986) contains an extensive review of experimental studies using rats exposed to lead. A variety of behavioral and physiological responses affected by lead neurotoxicity, as well as some social interactions in rats, were examined. Although it was generally not possible in the review to standardize results across all studies due to different dose levels and administration modes being utilized across studies at different life stages in the animals, adverse effects were often observed in rats with blood-lead levels around 30 µg/dL, and some effects upon learning were detected even when maximum blood-lead levels were below 20 µg/dL. Many studies (e.g., Angell and Weiss, 1982; Cory-Slechta and Thompson, 1979; Geist et al., 1985) have observed greater behavioral and physiological effects in rats exposed after weaning or during maturation than in those exposed prenatally or during infancy. Generally lead-exposed rats have been observed to acquire performance skills on discrimination tests more slowly than controls, and to commit more errors.

Social behavior and development. While lead exposure has been linked with the incidence of certain behavioral difficulties in children (e.g., irritation, aggressiveness) since the 1940's, more recent studies have investigated whether such a link exists at low-level lead exposures. One such study investigated the development of social behavior in monkeys and has been documented in Bushnell and Bowman (1979b). In this study, 21 newborn monkeys of both sexes were randomized into three groups of 7 animals each, where the control group received no added lead to their diet, while lead was spiked into the diet of animals in the lower and higher dosed groups over a one-year period in order to achieve targeted average blood-lead concentrations of 50 and 80 μg/dL, respectively. Social tests were administered to the animals once per week for 30-39 weeks, either alone, in the company of the other animals in its group, or with animals of the same age from each group. The frequency and duration of "rough-and-tumble play" was reduced in the lead-exposed groups compared to the control group ($p < 0.05$), while the duration of "contact cling" was significantly higher in these groups. The researchers suggested that although lead-exposed monkeys did occasionally respond to others' prompts to play, they were less likely to initiate such invitations to others (although their tests did not differentiate between initiating and responding to such play). The lead-exposed monkeys were also observed to be less adaptable to a sudden change in their environment for social interaction (which occurred at approximately 21 weeks of age in this study) compared to control animals.

Bushnell and Bowman (1979b) followed up this experiment with one in which 16 monkeys were placed into four groups of four animals each, where one group served as a control, one was dosed with dietary lead over a one-year period to achieve an average blood-lead concentration of 80 μg/dL ("chronic"), one was dosed with lead for only two weeks at two months of age ("pulse"), and one was dosed under both "chronic" and "pulse" regimens. Animals were tested in a play cage until week 40 (when interactive play behaviors have typically been developed), when they were moved to a play room. This study found that animals exposed chronically to lead had reduced frequency and duration of "rough-and-tumble play," the development of "initiated social explore," and the frequency of approach, compared to the control and "pulse" groups. This suggested that reduced desire to play is associated with chronic lead exposure during the period of social development. Meanwhile, animals in the "pulse" group did show significant behavioral effects when the play environment was changed at week 40, suggesting a latent effect associated with an acute exposure early in life and which occurred upon introducing a new and potentially intimidating environment. Furthermore, such effects were less apparent among monkeys exposed to lead only *in utero*.

In a study testing electric shock-elicited aggression (Hastings et al., 1977), lead-exposed animals (through post-natal lead exposure in mother's milk, where the dams were provided drinking water at concentrations of either 0.02% or 0.20%) showed significantly less aggressive behavior than did controls. This finding was the opposite of what the authors expected at the time. At 60 days of age (i.e., the time of testing), the lead-exposed animals averaged blood-lead levels of 5-9 μg/dL, and exposure levels were generally low in the study. Other tests performed in this study (e.g., wheel-running, visual discrimination task with reversal) did not see significant differences across exposure groups.

A study (Cutler, 1977) which investigated gender differences with respect to rodent behavior found that lead-exposed male mice showed significantly reduced levels of aggressive behavior (even though body weights and overall activity levels were not affected) compared to controls. In addition, both sexes showed significantly increased social/sexual investigative behavior in the lead-exposed group compared to controls. Total activity and the occurrence of tremors or other jerky movements did not differ significantly between the exposure and control groups.

As cited in Section 2.1.2.1 above, the study performed by Nagymajtenyi et al. (1998) observed an increase in bio-electric aberrations in the brains of rats exposed to lead pre- and post-natally compared to control rats. In addition, behavioral aberrations were observed in this study among the lead-exposed rats. Results showed that lead dosing during pregnancy was related to a significant, dose-dependent increase in hyperactive behavior (e.g., as measured by higher ambulation rates and increased grooming) in the offspring. The authors conclude that low-level lead exposure during prenatal and postnatal development can interfere with normal development and bioelectric functioning within the nervous system and is associated with behavioral changes. In addition, the authors suggest that the functional and behavioral effects of lead, which occur without other overt signs of lead toxicity, are much more harmful than has been previously supposed.

2.1.3 General Health Effects

Although the health effects of lead are diverse, and in general depend on the duration and degree of lead exposure, they are all thought to originate from lead's ability to interfere with fundamental biochemical processes (i.e., mitochondrial energy production, calcium-mediated processes, and protein function). All of the major types of health effects of lead that have been observed in humans, including hematological, neurodevelopmental, immunological, cardiovascular, and renal effects, have been demonstrated in controlled, dose-response studies in rodents, dogs, and/or non-human primates. Although conclusive evidence for carcinogenicity is still lacking in human studies, several animal studies have indicated that there is an association between high levels of oral exposure to several lead compounds and renal tumors in various species (USDHHS, 1999). The U.S. Department of Health and Human Services has determined that lead acetate and lead phosphate may reasonably be expected to be capable of causing cancer, based on sufficient evidence from animal studies (USDHHS, 1999).

While the focus of this report is on the neurobehavioral effects of lead exposure, any type of health effect that is demonstrated to have resulted from lead exposure in animal studies could be of interest, especially at low lead doses. Therefore, an overview of experimental animal studies which have investigated the general physical health effects that result from lead exposure is presented below. This overview will provide only brief statements of findings across animal

studies. Much of the information presented here has previously been cited in USDHHS (1999)[1] and USEPA (1986).

2.1.3.1 Death. At high levels of exposure, lead is known to cause death in humans following severe lead encephalopathy, and has been suggested to be a causative agent in sudden infant death syndrome (USDHHS, 1999). The data are minimal, however, regarding high exposures to lead and death in animals. Increased mortality has been observed in studies with rats and mice given lead in food or drinking water, although in some cases mortality did not occur in a dose-related manner (USDHHS, 1999).

2.1.3.2 Systemic Effects.

Hematological Effects. There have been numerous studies in animals demonstrating the adverse effects of lead on heme (hematin) biosynthesis, which can in turn affect many organ systems. In acute and intermediate-duration studies, the activities of several enzymes involved in the heme biosynthetic pathway have been observed to be altered by administration of lead to rats. Adverse hematological effects have also been observed in rats and dogs in longer term, lower dose studies (USDHHS, 1999).

Renal Effects. The effects of lead on the renal system have been documented in animal studies involving rats, dogs, monkeys, and rabbits. Symptoms of renal insufficiency, including both transient and irreversible kidney lesions, tubular dysfunction, and increased excretion of amino-acids and nitrogen compounds in urine, have been observed at high lead exposures in these studies (USDHHS, 1999; USEPA, 1986).

Cardiovascular Effects. Exposure to lead has been associated with adverse cardiovascular effects in studies with laboratory animals. While older animal studies concluded that hypertension was clearly associated with extremely high doses of lead, the direct effects of lead on blood pressure and the secondary effects (i.e., hypertension as a result of renal damage) of lead were difficult to separate (USEPA, 1986). However, USDHHS (1999) includes a review of several more recent chronic-duration experiments in rats and found that lower lead exposures (i.e., levels that were otherwise non-toxic) were associated with sustained increases in blood pressure as compared to controls. Other adverse cardiovascular effects, such as structural and functional changes relative to controls (e.g., degeneration of the myocardium and aorta), have also been observed in rats following lead ingestion (USDHHS, 1999).

Hepatic Effects. Indications of possible lead toxicity to the hepatic system, as suggested by increased liver weight, morphological changes, and changes in liver enzyme activity, have been observed in animal studies involving mice, rats, dogs, and baboons (USDHHS, 1999; USEPA, 1986). In general, adverse effects of lead on the liver in animal studies were observed at high exposure levels.

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[1] A 1998 draft of this document was also used in obtaining information for this section.

Respiratory Effects. There is limited evidence from inhalation studies that prolonged exposure to lead may cause respiratory system irritation in mice. Data suggest that lung irritation, if significant, is largely dependent on the duration of exposure (USDHHS, 1999).

Other Systemic Effects. Lead, at relatively high exposure levels, has also been associated in experimental animal studies with other various systemic effects. These include general impairment of cellular function (via interference with membranes, calcium ion transport, and mitochondrial respiration), long-term visual system deficits in animals exposed during post-natal development, weight loss, impairment of vitamin D metabolism, and endocrine disruption leading to a decrease in thyroid function (USDHHS, 1999; USEPA, 1986).

2.1.3.3 Immunological Effects. The literature provides good evidence that lead can have an immunosuppressive effect on animals at relatively low doses. Although additional research is required to elucidate the exact mechanisms of action, the macrophage is postulated to be the primary immune system target cell for lead (USEPA, 1986). Studies reviewed in USEPA (1986) and USDHHS (1999) showed that in rats, mice, and rabbits, both oral and airborne lead exposures contributed to increased susceptibility to bacterial and viral infections, decreased antibody counts, decreased cell-mediated immunity, depressed lymphocyte function, and suppressed macrophage-dependent immune response, as compared to control animals. In many of the studies cited, lead-induced immunosuppression occurred at low exposure levels that otherwise induced no overt symptoms of systemic lead toxicity, such as increased blood pressure or weight loss.

2.1.3.4 Reproductive and Genotoxic Effects. The observed effects of lead on the reproductive system are mixed across animal studies. Adverse effects observed included decreased pregnancy rates, decreased male fertility, damage to ovaries and testes, and altered pubertal progression, all presumably by interference with hormone production. High doses of lead have also been associated with fetal stunting and fetotoxicity. Lead was not observed to be teratogenic in limited rodent studies, except when lead was administered via injection (USDHHS, 1999). In various animal studies on the genotoxic effects of lead, chronic exposures to lead produced either slight or no significant increases in chromosomal aberrations in mice or monkeys, except in one study (a 1977 study by Deknudt and associates, as cited in USDHHS, 1999) in which monkeys were given a calcium-deficient diet (USDHHS, 1999).

2.1.3.5 Carcinogenicity. Most animal studies conducted to investigate the carcinogenicity of lead compounds have considered only one or two doses, preventing detailed dose-effect characterizations from being performed (USDHHS, 1999). Available data generally suggest that lead acetate and lead phosphate can be carcinogenic following ingestion by laboratory animals, and that the most common tumor is renal. There are currently no animal data on the carcinogenicity of lead by inhalation or dermal exposure.

In its review of animal studies performed over a 30-year period, USDHHS (1999) has reported that high levels of oral exposure (approximately 25-100 mg/kg/day) to lead acetate and

lead phosphate have been shown to increase the incidence of renal tumors in rats and mice (USDHHS, 1999). However, due to the extremely high cumulative doses of lead used in these studies, and the uncertainties regarding mechanisms by which lead induces tumors in the rat kidney (i.e., by acting on species-specific or trans-species proteins), extrapolation to low-level exposure in humans is difficult. Therefore, observations of lead-induced kidney tumors in rats may not be relevant to humans (USDHHS, 1999). The carcinogenicity of lead at lower doses in animals is yet to be determined.

2.1.4 Conclusions from Animal Studies Investigation

Lead has been observed to have widespread neurotoxic effects, as well as behavioral and cognitive symptoms, in humans. These observations are largely consistent with the findings of morphological, electrophysiological, and biochemical studies on animals. Animal studies are also congruent with observations of lead exposure in humans, suggesting an increased susceptibility of the young brain to lead poisoning (Banks et al., 1997). In addition, animal studies have provided physiological evidence that many of the effects of lead on the differentiation of the developing nervous system, such as synaptic and dendritic development, and myelin and other nerve structure formation, have the potential to be long-lived effects (USEPA, 1986). Although animal models do not duplicate the human response to lead exposure, they do serve to provide strong support for expecting certain health effects to occur when humans are exposed. In addition, animal studies allow for more specific determination of dose-response relationships. Single-route exposure and dose-response data are generally not available for humans. Animal studies provide an opportunity to elucidate some of the physiological mechanisms of lead toxicity, and thus they are a valuable tool for assessing the potential risks of lead exposure for human health.

A large resource of published literature also exists in which animal experiments were conducted to investigate the neurobehavioral effects caused by lead dosing. Most of these used monkeys or rats in experiments to assess the effects of lead dosing upon learning and performance activities and were conducted using low to moderate doses of lead. In many studies using monkeys, no other overt signs of toxicity (e.g., weight loss) were observed even when learning impairments were observed.

Several experiments have demonstrated that lead-dosed animals perform similarly to controls on some simple tasks (e.g., visual discrimination of simple shapes), but perform worse than controls on more complex tasks such as reverse discrimination tasks (where the animals have to change a previously-learned strategy) or fixed-interval/delayed response tests. Perseverative behavior is a recurring phenomenon in the latter type of test, whereby the animal is unable to inhibit inappropriate, repetitious movements despite having rewards withheld as a consequence.

Experiments to compare the severity of impairments in animals dosed only during early development (i.e., prior to birth or during infancy) versus those dosed only during maturation did

not yield consistent results. Generalizations about dosing schedules could not be made across all studies or across species, as to which groups of animals faired worse.

There are general similarities in studies and across species, however, with regards to the lowest levels of internal lead exposure during early development at which neurobehavioral effects are commonly observed. For example, the review by Davis et al. (1990) of earlier research findings concluded that internal lead exposures associated with neurobehavioral effects were reported to be as low as <20 μg/dL for rodents, <15 μg/dL for primates, and 10-15 μg/dL for children. Although direct comparisons of blood-lead levels are generally not considered to be accurate across species, evidence that rodents and primates may actually tolerate higher exposures to lead before reaching a given blood-lead level, suggests that exposures and blood-lead levels of concern would even lower in humans than in animals (Davis et al., 1990).

The published literature varied in the amount of detail given to explaining experimental designs used. For example, it was often implied but not always explicitly stated that subjects were randomized to exposure groups according to some statistical criteria, and that the experimenters observing the performance tests were blind to the dosing status of subjects. Articles also provided varying levels of detail on experimental procedures, such as the amount of lead provided through food or water. Blood-lead levels at the time of performance testing were usually measured and reported.

The choice of response endpoint was clearly of particular importance in the design of performance tests. For example, as noted in Burger et al. (1998), behavioral tests need to include procedures in which the given species (absent of lead exposure) will readily participate, otherwise there is a risk that the response endpoint will not be sensitive enough to allow the researcher to detect significant differences between dosed and control animals. Also, lead exposure may delay or accelerate the physical development of some reflexes, so peak performance may occur at different times in different exposure groups. Therefore, the time at which statistical comparisons are made may produce seemingly paradoxical results. In particular, any apparent non-monotonic dose-response relationships must be interpreted with caution.

Despite the aforementioned caveats, animal studies do provide an opportunity to elucidate some of the physiological mechanisms of lead toxicity, as well as generate single-route exposure and dose data that are generally not available in humans. Although animal models cannot claim to duplicate the human response to lead exposure, the resulting ability to control confounding factors permits these models to succeed in providing substantial evidence that supports the existence of a causal relationship between low level lead exposure and neurological impairment, especially in the young.

2.2 SUPPORT FOR THE CAUSALITY OF ADVERSE HEALTH EFFECTS DUE TO LEAD EXPOSURE

Chapter 2 of the §403 risk analysis report documented previously-published information on adverse health effects associated with lead exposure to humans. Subsequent chapters characterized how environmental-lead exposure impacts selected blood-lead concentration and health effect endpoints in children. Specifically, IQ-based health endpoints were selected to represent the neurological effects associated with lead exposure.

A concern, particularly with the IQ-based health endpoints, is whether lead can be assumed to cause the adverse health effects. For ethical reasons, the controlled lead exposure studies necessary to test this hypothesis can not be performed on humans. However, animal studies can be used to supplement the evidence of human studies in this area. To this end, Section 2.1 of this document presented key findings of animal studies that investigated the impact of lead exposure on adverse health effects. This section examines whether the combined evidence of human and animal studies suggests that lead exposure causes neurological damage that can be measured through intelligence testing.

2.2.1 Principles of Causality

On the issue of causality, Needleman & Gatsonis (1990) make the following assertions and present the following principles of causality (citing Kenny, 1979, as a reference):

> *"Epidemiologic studies cannot, by themselves, establish causal relationships. Causality is not subject to empirical proof, whether in the field or in the laboratory. Given that direct demonstration of proof of a low-dose lead effect in a naturalistic setting is not achievable, epidemiologists rely on canons that, if satisfied, permit the conservative drawing of causal inferences. They are (1) time precedence of the putative cause, (2) biologic plausibility, (3) nonspuriousness, and (4) consistency."*

Needleman & Gatsonis (1990) also introduce a fifth principle, biologic gradient, which is part of biologic plausibility. Needleman (1998) indicates that these principles originate from investigations of whether tobacco use causes cancer and attributes them to the British statistician Sir Austin Bradford Hill. Together, these principles can be applied to the findings of existing studies (both human and animal studies) to imply that lead exposure causes neurological damage that may be measured through IQ score decrements.

The following paragraphs discuss the five principles of causality relative to the evidence in human and animal studies.

Time precedence. Time precedence means that the proposed cause must exist before the proposed effect occurs. That is, a child must become exposed to lead before neurological deficit is observed.

In cross-sectional studies in humans, it is impossible to establish the time precedence of lead exposure and neurological deficit. Longitudinal studies in humans, however, have shown that disturbances in early neurobehavioral development occur even at low lead exposure levels in early life. For example, in Boston, 4 to 8 point differences in performance on the Bayley Mental Development Index were reported at 6, 12, 18, and 24 months, after adjusting for other covariates, when children with low prenatal blood-lead levels (mean of 1.9 μg/dL) were compared to children with modestly elevated prenatal blood-lead levels (mean of 14.6 μg/dL) (Bellinger et al., 1985a, 1985b, 1986a, 1986b, 1987a; 1991; 1992). Additional detail on this and other longitudinal studies is presented in Section 2.2.2 below and Section 2.3.1 of the §403 risk analysis report.

Controlled animal studies further support the time precedence of lead exposure. For example, groups of monkeys exposed to lead either continuously from birth or only after infancy showed learning impairment on a series of non-spatial discrimination reversal tasks, relative to a control group that were not exposed to lead (Rice and Gilbert 1990a). These lead-exposed monkeys, along with a group exposed only during infancy, also showed performance impairment relative to controls in a spatial-delayed alternation task at age 6-7 years. Rats exposed to lead *in utero* and during lactation displayed a highly significant impairment in performance on a water maze test at 100 days of age, relative to controls, even though the average blood-lead level had declined to 1.8 μg/dL by this time (Kuhlman et al., 1997). Additional animal research described in Section 2.1 also supports the conclusion that lead exposure precedes the neurological deficit.

Biologic plausibility. Biologic plausibility means that the causal relationship between exposure and adverse health effects must be consistent with known biological function.

While investigation of the mechanisms of lead toxicity remains the subject of active research, it is known that lead can interfere with cell function by competing with essential minerals, such as calcium and zinc, for binding sites on membranes and proteins. Lead binding to membranes or transport proteins can inhibit or alter ion transport across the membrane or within the cell. In the brain, lead can substitute for calcium and zinc in ion transport events at the synapse (i.e., the junction where the axon of one neuron terminates with the dendrite of another neuron, through which nerve impulses must travel to move from one nerve cell to another). The normally-developing brain appears to delete synapses that are unused and to keep and strengthen synapses that are used. Goldstein (1990, 1992) suggested that lead may disrupt, or delay, the development of synapses and that, perhaps, the resulting connections in the brain are "poorly chosen," leading to functional impairment. Silbergeld (1991) found that exposure of fetal animals to lead affects both regional growth and synaptogenesis, with synaptogenesis being the more sensitive. Although some of these conclusions are speculative, it is biologically plausible that exposure to lead causes neurological damage.

Biologic gradient. Biologic gradient means that a dose-response relationship must be present (i.e., increased doses of lead should cause increased impairment).

It is well known that high-level exposure to lead produces encephalopathy in children, starting at blood-lead levels of 80 to 100 µg/dL. At lower exposure levels, IQ decrements, fine motor dysfunction, and disturbances in neurobehavioral development have been related to varying levels of lead exposure. These effects are summarized in Section 2.3.1 of the §403 risk analysis report. As summarized in Section 4.4 and Appendix D2 of the §403 risk analysis report, many studies have focused on estimating the magnitude of this dose-response relationship.

Nonspuriousness. Nonspuriousness means that confounding factors associated with adverse health effects must be ruled out.

Studies that investigate the relationship between children's IQ and blood-lead concentration have adjusted for other demographic factors that may affect neurological development, such as Home Observation for Measurement of the Environment (HOME) score, maternal IQ, and socioeconomic status. These studies are summarized in Appendix D2 (Tables D2-1 and D2-2) of the §403 risk analysis report. In many of these studies, the level of lead exposure remains a highly-significant factor after adjusting for these potential confounding factors. Because the method of adjusting for confounding factors in human exposure studies does not necessarily remove all confounding, however, animal research is required to supplement human subject research. Animal studies minimize confounding by administering lead in controlled doses to randomly-selected subjects that are genetically similar and are otherwise treated similarly. Controlled animal studies described in Section 2.1 of this document support the conclusion that lead exposure precedes neurological deficit. In addition, these studies demonstrate that the effects are nonspurious by establishing study designs that include control groups and that attempt to control for confounding factors.

Consistency. Consistency, also known as coherence, requires that the phenomenon be demonstrated in different studies under similar, but not identical conditions.

Human studies investigating the relationship between blood-lead concentration and IQ scores have generally associated IQ decrements with increases in blood-lead concentration in various populations around the world, as summarized in Appendix D2 (Tables D2-1 and D2-2) of the §403 risk analysis report. The magnitude of the estimated IQ decrement varied from study to study, as may be expected, and the relationship was not always statistically significant. However, the relationship was consistently negative (i.e., increased blood-lead concentration was associated with IQ decrement in more than ten studies). Furthermore, a loss of approximately 2-3 IQ points was associated with an increase in blood-lead concentration from 10 to 20 µg/dL in several of these studies.

2.2.2 Causality As Addressed in Longitudinal Studies

The consistency principle of causality implies that causality can not be concluded from the findings of a single human monitoring study. In turn, the time precedence principle indicates that repeated data collection over time for study subjects within longitudinal studies provides an important component of an investigation into the specific role of lead exposure as the cause of

certain adverse health effects. Therefore, the findings of multiple longitudinal studies on the association between lead exposure and diminished performance on cognitive function or intelligence testing can be an important contribution to the argument of causality (while the ability to conclude causality exclusively from such findings remains very limited). The level of importance of the contribution increases when consistent findings are observed across studies (and across different cohorts having different demographic characteristics and lead exposure potential).

This section provides some key findings from two longitudinal studies (Boston and Port Pirie) that can be used in evaluating the hypothesis of causality. This is not meant to be an exhaustive presentation of all results (significant or otherwise) across all longitudinal studies, but instead is a presentation of only key findings from selected studies. For example, results from other longitudinal studies that monitored lead exposure (e.g., Cleveland, Cincinnati, Sydney, Yugoslavia) have not been reviewed. In addition, the potential for reverse causality (i.e., children with lower levels of intelligence are more prone to elevated blood-lead concentrations) is not addressed.

The Boston Prospective Study (Bellinger et al., 1985a, 1985b, 1986a, 1986b, 1987a; 1991; 1992)

The Boston prospective study considered infants born at the Brigham and Women's Hospital in Boston, MA, from August, 1979, to April, 1981. Cord blood-lead levels were measured for 9489 infants (97% of all available infants), and those whose cord blood-lead levels were within one of the following three categories were considered for the study: < 3 µg/dL (low), 6-7 µg/dL (mid), and ≥ 10 µg/dL (high). These categories represented the 10^{th}, 50^{th}, and 90^{th} percentiles of the cord blood-lead distribution observed in the first three months of the study. A total of 249 infants in these categories were enrolled in the study (85 in the low category having mean 1.5 µg/dL, 88 in the mid category having mean 6.5 µg/dL, and 76 in the high category having mean 14.6 µg/dL).

The cohort was considered to be of high socioeconomic standing (e.g., 87% of the enrolled infants were white; 92% were considered to come from intact families). While such a cohort tends to have a lower likelihood of lead exposure than more disadvantaged children (e.g., those in poor, inner-city neighborhoods), thereby restricting the ability to generalize results to a wide population, the low occurrence of certain demographic conditions that are typically highly correlated with lead exposure gives this study a greater opportunity to isolate the effect of lead exposure (especially at low levels) on cognitive function.

Post-natal blood-lead concentrations were measured on the study cohort at ages 6, 12, 18, 24, and 57 months, and at age 10 years. Capillary blood was obtained through 24 months of age, and venous blood was obtained at ages 57 months and 10 years. Cognitive function was measured by the Mental Development Index (MDI) of the Bayley Scales of Infant Development at 6, 12, 18, and 24 months of age, by the McCarthy Scales of Children's Abilities at age 57 months, and by the Wechsler Intelligence Scale for Children-Revised (WISC-R) and Battery

Composite scores on the Kaufman Test of Educational Achievement -Brief Form (K-TEA) at age 10 years.

Investigations on the association between lead exposure (as measured by cord-lead or blood-lead concentration) and intellectual functioning within the study cohort were performed at various time points during the study. The extent of association was measured by multiple regression modeling, adjusting for parameters that represent potential confounding factors. This resulted in the following key findings:

1. At ages 6, 12, 18, and 24 months, children in the high cord-blood group had significantly lower MDI scores relative to each of the other two groups (4.8 points lower than the low group and 3.8 points lower than the mid group) at the 0.05 level. Furthermore, the MDI scores at these ages were not significantly associated with post-natal blood-lead concentrations measured up to that time (Bellinger et al., 1987). The level of association increased upon adjusting for potential confounders within the multiple regression equation.[2]

2. By age 57 months, the association between cord-blood grouping and cognitive function diminished considerably from what was observed at earlier ages and was no longer statistically significant, except in the instance where only children with blood-lead concentrations at or above 10 µg/dL at age 57 months was considered. However, a significant (inverse) association was observed between blood-lead concentration at 24 months of age and score on the McCarthy scales at 57 months, upon adjusting for potential confounders[3] (Bellinger et al., 1991). The perceptual-performance subscale of the McCarthy scales, which measures visual-spatial and visual-motor integration skills, was especially sensitive to post-natal lead exposure.

3. The association between increased blood-lead concentration at 24 months of age and observed deficits in full-scale and verbal IQ scores was statistically significant at age 10 years, even after adjusting for confounding variables[4] (p=0.007 for full-scale IQ, 0.004 for verbal IQ, but only 0.091 for performance IQ). Blood-lead concentrations at other ages, however, were not significantly associated with such deficits at the 0.05 level.

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[2] Mother's age, mother's race (white vs. nonwhite), mother's IQ (as measured by the Peabody Picture vocabulary test), mother's education level, # of years mother smoked, # alcoholic drinks per week by mother in the third trimester of pregnancy, Hollingshead Four-Factor Index measure of family social class, quality of care-giving environment, child's sex, child's birth weight, child's gestational age, child's birth order.

[3] Family social class, maternal IQ, marital status, preschool attendance, HOME score, # hours per week of "out-of-home" care, # family residence changes, recent medication use, # adults in household, gender, race, birth weight, birth order.

[4] HOME score at 10 years, total HOME score at 57 months, child stress, maternal age, race, maternal IQ, socioeconomic status, sex, birth order, maternal marital status, and # family residence changes prior to 57 months.

Finding #1 shows that despite a child's lead exposure during the first two years, cognitive function during this period is more significantly associated with pre-natal exposure, which is generally less prone to behavioral and environmental confounding than is direct, post-natal exposure. As a child ages, as seen in Findings #2 and #3, post-natal lead exposure (especially its peak at approximately two years of age) becomes more dominant than pre-natal exposure in its association with the child's performance on intelligence tests. This suggests that the effect of cumulative post-natal exposure through approximately age 2 years eventually outweighs pre-natal exposure. In particular, children can eventually see reduced performance on intelligence testing if their post-natal lead exposure becomes significant, despite their pre-natal lead exposure. Furthermore, a child's post-natal lead exposure tends to change at a faster rate than other demographic variables that are highly correlated with blood-lead levels early in life, with the more recent lead exposures being predictive of a child's current health consequences. These findings support the hypothesis of causality, especially in the relatively homogeneous and highly-privileged cohort considered in this study.

Typically, in environments having high lead levels (e.g., inner-cities, smelter/mining communities), correlations between blood-lead concentrations measured at different ages are so highly correlated that it is difficult to separate out the age effects. However, the lower age-to-age correlations observed in the Boston study (resulting from relatively low lead exposure potential) allowed for investigating age-specific vulnerabilities within this study.

The Port Pirie Cohort Study (Baghurst et al., 1992; Tong et al., 1996; Burns et al., 1999)

This study consisted of 723 subjects born in the lead smelting community of Port Pirie, Australia (and surrounding rural communities) from May, 1979, to May, 1982. Cord-blood was obtained and analyzed for lead (geometric mean = 8.3 μg/dL). In addition, capillary blood samples were collected at ages 6 and 15 months and annually from ages 2 through 7 years. A venous blood sample was collected at age 11-13 years. From the measured blood-lead levels, average lifetime blood-lead concentration was calculated for each child using trapezoidal integration. Geometric mean blood-lead concentrations increased to 21.2 μg/dL by age 2, then declined to 7.9 μg/dL by age 11-13, when 375 children remained in the cohort. Children from more advantaged backgrounds were more likely to remain in the cohort through age 11-13 years than children from disadvantaged backgrounds.

Measures of developmental status were made at ages 2 years (Bayley scales), 4 years (McCarthy scales), and at 7 and 11-13 years (Wechsler intelligence scale). In addition, emotional and behavioral problems were assessed by their mothers using a Child Behavior Checklist, and other demographic parameters were measured via questionnaire.

The key findings within this longitudinal study on how lead exposure is associated with decreased developmental status measures were as follows:

1. The inverse relationships between IQ at age 7 years and average lifetime blood-lead concentration measured at 15 months and at 2, 3, and 4 years were statistically significant at the 0.05 level after adjusting for potential confounding factors[5]. This result held for both full-scale and verbal IQ measures, but not performance IQ.

2. Despite blood-lead concentration declining after age 2 or 3 years, the association with cognitive development continued into later childhood. Furthermore, lifetime blood-lead concentration was significantly associated with childhood emotional and behavioral problems (after adjusting for such confounding factors as HOME score, maternal psychopathology, and child's IQ) at ages 11-13 years.

Finding #1 was consistent with the Boston prospective study in that no significant relationship was observed between IQ and pre-natal blood-lead concentration at approximately age 7 years, while the relationship was significant when considering blood-lead concentration at 2 years of age.

2.2.3 Conclusions on Causality

The combined weight of human and animal studies provide evidence, consistent with the principles of causality presented in Section 2.2.1, that lead may be assumed to cause adverse neurological effects in young children. In particular, longitudinal studies in humans have shown that disturbances occur in neurobehavioral development early in life even at low lead exposure levels. These studies have observed effects of lead exposure even after accounting for other demographic factors (e.g., socioeconomic status, parents' IQ) that could affect neurological development. While these other demographic factors tend to be highly correlated with blood-lead levels early in life, the influence that post-natal lead exposure has on blood-lead tends to increase with a child's age. This is because over time, measures of a child's lead exposure tend to change at a faster rate than the child's demographic measures, and more recent lead exposures continue to be predictive of a child's current health consequences. For the §403 risk analysis, adverse neurological effects are assessed through IQ score decrements. Reasons for selecting IQ score decrement as a health effect endpoint are presented in Section 2.5.2 of the §403 risk analysis report.

2.3 THE ASSOCIATION BETWEEN BLOOD-LEAD CONCENTRATION AND IQ SCORE

In its risk characterization, the §403 risk analysis used IQ score decrement associated with lead exposure as the basis for measures of neurological effects. As the risk analysis used

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[5] Sex, parents' level of education, maternal age at delivery, parents' smoking habits, socioeconomic status, quality of home environment, maternal IQ, birth weight, birth order, feeding method (bottle, breast, both), duration of breast feeding, whether the child's natural parents were living together.

blood-lead concentration as its primary measure of body lead burden to quantify environmental-lead exposure, it was necessary to determine IQ score as a function of blood-lead concentration and to characterize the extent to which a change in IQ score occurs when blood-lead concentration changes within a child. The following assumptions were made in this analysis on the association between blood-lead concentration and IQ score for the representative population of 1-2 year old children:

- The relationship between blood-lead concentration and IQ score decrement was assumed to be linear.

- The risk characterization assumed a loss of 0.257 IQ points per 1 μg/dL increase in blood-lead concentration (with alternatives of 0.185 and 0.323 considered in sensitivity analyses).

- No threshold was assumed in this relationship (i.e., no blood-lead concentration exists below which a relationship between blood-lead concentration and IQ score is not apparent), although selected non-zero thresholds have been assumed in sensitivity analyses presented in Sections 5.1.5 and 6.4.2 of this report.

While the §403 risk analysis report discusses the basis for making these assumptions (e.g., see Section 4.4 and Appendix D2), this section presents additional information that is necessary to judge the correctness and accuracy of the assumptions. Section 2.3.1 addresses the linearity and slope assumptions, while Section 2.3.2 addresses the threshold assumption.

2.3.1 Linearity and Slope Assumptions

How was such an assumption made? As researchers have used primarily linear and log-linear models to characterize the relationship between blood-lead concentration and IQ scores , these two types of models were considered for use in the risk analysis. The log-linear model predicts IQ score as a linear function of log-transformed blood-lead concentration (plus other important confounding variables, such as maternal IQ and HOME score), while the linear model does not take a log transformation of the blood-lead concentration. The scientific community does not appear to have reached a consensus on which form is more appropriate. For example, the meta-analysis in Schwartz (1994) included three studies that employed log-linear models and four studies that employed linear models.

To obtain a single measure of the relationship that would be comparable across studies, despite the different model forms used, Schwartz (1994) used the change in IQ score associated with a doubling of blood-lead concentrations from 10 to 20 μg/dL. The meta-analysis (Schwartz, 1994) yielded an estimated decrease of 2.57 IQ points for an increase in blood-lead concentration from 10 to 20 μg/dL. This was the slope estimate used in the §403 risk analysis.

Using the measure discussed in the previous paragraph, Schwartz (1994) provides some evidence that the log-linear relationship may be more appropriate than the linear relationship. In

an analysis to investigate the presence of a threshold (see Section 2.3.2), Schwartz (1994) estimated an IQ point decrement of 3.23 IQ points for the three studies with mean blood-lead concentrations below 15 μg/dL, compared to a 2.32 IQ decrement for the four studies with mean blood-lead concentrations at or above 15 μg/dL. Thus, if anything, a trend toward greater IQ loss associated with lower blood-lead concentrations was observed. This result is consistent with a log-linear relationship.

Despite this evidence, a linear relationship was applied in the §403 risk analysis. The assumption of a linear model reduces the likelihood of overestimating the number of children with low blood-lead concentrations at risk, or who may benefit from actions taken in response to the §403 standards. See Section 4.2.1 and Appendix D2 of the §403 risk analysis report for additional information.

Additional information: Tables D2-1 and D2-2 in Appendix D2 of the §403 risk analysis report summarize a total of 18 studies that report the relationship between children's blood-lead concentration and IQ. Each of these studies was used in at least one of the meta-analysis studies reviewed in Appendix D2 of the §403 risk analysis report. Table 2-2 provides a subset of the information previously reported in Tables D2-1 and D2-2 of the §403 risk analysis report for these studies and also reports the type of model (linear or log-linear) which each study used to predict IQ as a function of blood-lead concentration.

A few of the studies included in Table 2-2 used a log-linear model rather than a linear model to characterize the effect of blood-lead concentration on IQ. However, the overall evidence that these studies provide regarding a log-linear relationship was more limited than the existing evidence on a linear relationship. Furthermore, if EPA had adopted a log-linear model approach, the risk analysis would have estimated that blood-lead concentration had a greater impact on IQ at lower levels than at higher levels. This would have resulted in a greater possibility that the risk analysis would have overestimated benefits at lower levels, compared to underestimating benefits. For these reasons, EPA felt that a linear model was the better approach over a log-linear model. However, the §403 risk analysis did include a sensitivity analysis which considered the effects of a steeper slope in the linear model, in order to evaluate the possibility of underestimating the relationship between blood-lead concentration and IQ.

A recent article by Marais and Wecker (1998) has suggested that researchers who have characterized IQ as a function of blood-lead concentration using linear regression techniques have often reported biased estimates for the effect of blood-lead concentration on IQ for one or both of the following reasons:

- by not having all four of the following predictor variables in the model: blood-lead concentration, mother's intelligence, father's intelligence, and socioeconomic status.

- by not taking into account measurement error in these predictor variables.

Table 2-2. Summary of Key Findings from Studies that Investigate the Relationship Between Blood-Lead Concentration and IQ Score

Study	Type of Study	Location	N	PbB Mean (SD) (µg/dL)	IQ Score Mean (SD)	Association Between IQ and Blood-Lead Levels		
						Change in IQ as PbB increases from 10-20 µg/dL	P-Value	Model Form
Hatzakis et al. (1987)	Prospective	Lavrion, Greece	509	23.7 (9.2)		-2.7	<0.001	linear
Hatzakis et al. (1989)	Prospective	Lavrion, Greece	509	23.7 (9.2)	87.7 (14.8)	-2.7	<0.001	linear
Bellinger et al. (1991)	Prospective	Boston, MA	150	6.4 (4.1)	115.5 (14.5)	-1.6	0.23	log-linear
Bellinger et al. (1992)	Prospective	Boston, MA	147	6.5 (4.9)	119.1 (14.8)	-5.8	0.007	linear
Baghurst et al. (1992)	Prospective	Port Pirie, Australia	494	20	104.7	-3.3	0.04	log-linear
Ernhart et al. (1989)	Prospective	Cleveland, OH	212	16.7 (6.45)	87.5 (16.6)	-1.1	<0.01	linear
Cooney et al. (1991)	Prospective	Sidney, Australia	175	14.2		+0.4		linear
Schroeder et al. (1985)	Prospective	Wake County, NC	104		Range = 45-140	-2.0	<0.01	linear
Hawk et al. (1986)	Replication of Schroeder Study	Lenoir & New Hanover counties, NC	75	20.9 (9.7)	Range = 59-118	-2.6	<0.05	linear
Dietrich et al. (1993)	Prospective	Cincinnati, OH	231	15.2 (11.3)	86.9 (11.3)	-1.3	<0.10	linear
Yule et al. (1981)	Pilot Study	London, England	166	13.52 (4.13)	98.21 (13.44)	-5.6	0.084	log-linear
Lansdown et al. (1986)	Replication of Yule Study	London, England	166	12.75 (3.07)	105.24 (14.2)	+1.5	0.63	log-linear
Winneke et al. (1990)	Multi-Center, Cross - Sectional Study	Bucharest	301	GM = 18.9 (1.3)			<0.1	linear
		Budapest	254	GM = 18.2 (1.7)			<0.1	linear
		Moden	216	GM = 11.0 (1.3)			<0.1	linear
		Sofia	142	GM = 18.2 (1.6)			<0.1	linear
		Dusseldorf	109	GM = 8.3 (1.4)	116		<0.1	linear
		Dusseldorf	109	7.4 (1.3)			<0.1	linear
Silva (1988)	Cross - Sectional	Dunedin, New Zealand	579	11.1 (4.91)	108.9 (15.12)	-1.5		log-linear
Harvey et al. (1988)	Cross - Sectional	Birmingham, England	177	12.3 (0.2)	105.9 (10.6)			linear
Wang et al. (1989)	Cross - Sectional	Shanghai, China	157	21.1 (10.11)	89	-9		linear
Winneke et al. (1985a)	Cross - Sectional	Nordenham, Germany	122	8.2 (1.4)	120.2 (10.3)		<0.1	linear
Fulton et al. (1987)	Cross - Sectional	Edinburgh, Scotland	501	GM = 11.5	112 (13.4)	-2.6	0.003	log-linear

PbB = blood-lead concentration (µg/dL); SD = standard deviation; GM = geometric mean

By analyzing data from four case studies, the authors imply that the bias overestimates the effect, thereby making it likely that the researcher would declare that blood-lead concentration (especially at low levels) has a significant effect on IQ, when in reality, such an effect is insignificant. The authors show how to arrive at an estimate of the blood-lead effect that is not subject to this bias; this estimate is a function of the correlations among the four predictor variables and the measurement variability associated with these variables. The article prompted two responses that were published simultaneously with the article, both of which challenged the article's conclusions.

Despite their claims, the findings in Marais and Wecker (1998) have not resulted in any change to the approach taken by the §403 risk analysis to characterize the relationship between blood-lead concentration and children's IQ for the following reasons:

- Sensitivity analyses performed by other researchers[6] and documented in a response published with the article by Marais and Wecker (1998) have shown that the approach to obtaining an "unbiased" estimate for the effect of blood-lead on IQ is highly sensitive to the values of the estimates for the correlations and variability among the four predictor variables that are input to the calculation, thereby implying that input values that do not represent the target population can lead to a highly inaccurate estimate for this effect under this approach.

- The authors have not shown that any overestimation associated with this bias is always significantly large enough to warrant concern or will result in an incorrect declaration that blood-lead has a significant effect on IQ.

- The meta-analysis documented in Schwartz (1994), which the §403 risk analysis used to characterize the relationship between blood-lead concentration and IQ, utilizes the estimated blood-lead effects on IQ that were reported by seven studies, six of which estimated these effects after taking into account both parental IQ and socioeconomic status (i.e., the predictor variables of most concern to Marais and Wecker). Furthermore, the §403 risk analysis considered not only the outcome of this meta-analysis, but also the sensitivity analyses associated with this analysis, when investigating the effect of deviation from the meta-analysis outcome on the risk analysis results.

- The need to adjust for measurement error in the predictor variables is not relevant to the §403 risk analysis, as the goal is to predict how a measured blood-lead concentration (after adjusting for the measured values of other potentially important variables) is associated with IQ.

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[6] Waternaux, C., Petkova, E., and DuMouchel, W. "Comment: Problems with Using Auxiliary Information to Correct for Omitted Variables When Estimating the Effect of Lead on IQ." *Journal of the American Statistical Association*. 93:505-513.

The issue of whether a linear model is appropriate over the entire range of blood-lead concentration must address the presence of a threshold in the relationship. This is discussed further in the next section.

2.3.2 Threshold Assumption

Despite the claims of some researchers on the presence of a threshold in the blood-lead/IQ relationship, the majority of findings across studies and in meta-analyses have failed to find sufficient evidence of a non-zero threshold. Furthermore, when claims of a non-zero threshold were made, the value of this threshold (when suggested) differed considerably across these claims. Therefore, the approach taken in the §403 risk analysis was to assume that no threshold exists (although risk calculations assuming certain non-zero threshold values have been included in sensitivity analyses found in Sections 5.1.5 and 6.4.2 of this document).

In Section 3.3 of USEPA (1998b), the SAB concurred that "available data have not identified a clear threshold," and, therefore, "the assumption of no threshold for lead effects on IQ score is both defensible and appropriate statistically." However, it was desired to document the technical justifications for this assumption more thoroughly. Furthermore, the investigation into the presence of a threshold could be addressed by evaluating whether the dose-response function is linear across the entire range of blood-lead concentration.

How was such an assumption made? The assumption of no threshold made in the §403 risk analysis was based on the findings of Schwartz (1994), who noted that the presence of a threshold would result in a decline in the estimated slope associated with blood-lead concentration as the range of blood-lead concentrations declined across studies. However, as mentioned in Section 2.3.1 above, a larger effect size was observed in the four studies with mean blood-lead levels of 15 µg/dL or lower (-0.323 ± 0.126) compared to the other three studies (-0.232 ± 0.040). This observed trend toward higher slopes at lower concentrations discounted the likelihood of a threshold.

Also, Schwartz (1994) examined data from the Boston prospective lead study (discussed in Section 2.2.2 above) specifically to investigate the presence of a threshold. This study was selected as it had the lowest mean blood-lead concentration at two years of age (6.5 µg/dL; n=133) of the studies considered in the meta-analysis, thereby allowing thresholds at low blood-lead levels to be identified if present. In addition, the study cohort's high socioeconomic (SES) standing may have limited the likelihood of certain confounding, and the Boston study coordinators found relatively weak association between blood-lead concentration at two years of age and various sociodemographic characteristics and psychosocial environment parameters (Bellinger et al., 1986; Bellinger et al., 1992).

Schwartz's examination of the Boston study data involved fitting two separate regression curves to the same set of covariates: one using IQ score (at 10 years) as the dependent variable, and the other using blood-lead concentration (at 2 years) as the dependent variable. The covariates included age, race, stress and HOME scores, maternal IQ, educational level and

occupational status for each parent, mother's time working out of the house, marital status, gestational age, birth weight, mother's use of alcohol during pregnancy, otitis media history, birth order, and SES. Then, a nonparametric smoothed curve (LOESS) was used to characterize the residuals from the IQ score regression as a function of the residuals from the blood-lead regression. The residuals were used in this curve-fitting exercise as they represent blood-lead and IQ score measures after any effects of the above covariates have been removed. The LOESS technique allowed for nonlinear curve fits, such as those that would result if a threshold was present. The curve fit suggested that IQ score decrement was associated with declines in blood-lead concentration even when blood-lead levels were below 5 μg/dL, supporting the hypothesis that a blood-lead threshold on IQ score decrement was essentially not present.

In Schwartz (1993), this nonparametric smoothing approach was performed on McCarthy index data collected at age 57 months and blood-lead concentration data collected at 24 months, as recorded in the Boston prospective lead study (Bellinger et al., 1991). Again, after adjusting for potential confounding variables, a definite relationship was observed even at levels below 10 μg/dL, with no evidence of a threshold (Schwartz, 1993). To allow any potential threshold to be identified, a piecewise-linear regression model was fitted to these data which allowed the relationship to resemble a "hockey-stick" (i.e., the fit resembled two lines of different slopes that meet at some point representing the potential threshold, with the line below the threshold having nearly a zero slope, and the line above the threshold having a larger, positive slope). This model fit suggested that any potential threshold would be less than 0.0001 μg/dL (Schwartz, 1993).

The meta-analysis by Pocock et al. (1994) involving 26 studies concluded that no single study has collected a sufficient amount of information to make definitive statements on the presence of a threshold, and contradictory results (due to chance) on the presence of a threshold can be observed for different studies. Thus, the analysis did not have enough evidence to reject the hypothesis that no threshold exists.

Identifying a threshold. If a statistical hypothesis is used to determine the presence of a threshold, the test should take the following form:

Null hypothesis: No threshold exists (i.e., the "threshold" is at 0 μg/dL)
Alternative hypothesis: A non-zero threshold exists.

If a statistical test is used as a scientific basis for making a decision, one either rejects the null hypothesis or fails to reject it. One never says that the null hypothesis is "true." Therefore, given a set of data and the statistical methods being applied, one either rejects the hypothesis that no threshold exists or cannot reject it.

The statistical method used to test the above hypotheses can also vary from study to study. In general, the method is applied as part of an investigation into a dose-response relationship between blood-lead concentration and IQ. Two examples of statistical approaches are as follows:

- Several studies (e.g., Dietrich et al., 1993; Hatzakis et al., 1989) investigated dose-response by placing the study cohort into from 5 to 10 groups according to blood-lead concentration, determining the predicted IQ score associated with the mean blood-lead concentration in each group (using some pre-determined regression model), calculating confidence intervals associated with the prediction, and determining how the groups differ in their predictions (as well as any patterns among the groups).

- Another approach focuses on attempting to fit the piecewise-linear "hockey-stick" regression model discussed above that predicts IQ score as a function of blood-lead concentration (and other confounding variables), where the fitted line has a different (larger, positive) slope once blood-lead concentration achieves a certain level, which is interpreted as a threshold value.

Problems associated with suggesting that a threshold exists: Determining whether a threshold exists in the relationship between blood-lead concentration and IQ score is problematic due to the difficulties in accurately characterizing the blood-lead/IQ relationship and the inability to generalize findings across studies and to the nation as a whole. Major sources of these difficulties include the following:

- Different protocols for measuring IQ and different IQ measures (e.g., performance IQ, verbal IQ, full-scale IQ measures are all associated with the Wechsler protocol) are used in different studies.

- Study designs differ, as do the methods used to make inferences from the data.

- Children's IQ can be difficult to measure and can be more variable than adult IQ.

- Outcomes are often highly dependent on the given set of confounding variables being considered. This set differs from one study to the next. Furthermore, when multiple studies consider the same confounding variables, these variables are often measured differently, using different protocols, from study to study.

- Different ages of children and different ranges of blood-lead concentration are found across studies.

A non-zero threshold would result in reduced estimates of the likelihood of adverse health effects, as children with blood-lead concentrations below the threshold would no longer be labeled as experiencing an exposure-related IQ decrement. The level of reduction would depend on how large the value of the threshold is. Thus, if a decision on a non-zero threshold was made in error, the incidences of adverse health effects would be underestimated. The impact that a non-zero threshold has on reducing the risk estimates calculated in this risk analysis is addressed in sensitivity analyses presented in Sections 5.1.5 and 6.4.2 of this document.

Examples of Possible Non-zero Thresholds Concluded from Study Findings. Relatively high thresholds (e.g., 10 µg/dL or above) have been suggested in some older studies conducted 10 or 15 years ago. However, some of the higher suggested thresholds appear to have lost their legitimacy as they are higher than the levels for which more recent studies have observed some type of health effect. For those older studies that did not report a possible threshold, many involved children with a range of blood-lead concentrations that would be considered high by today's standards. Thus, the findings from these studies cannot be used to determine whether thresholds exist at lower lead levels (i.e., levels below the observed ranges). A study's design must allow for a sufficiently large range of blood-lead concentrations, and in particular, cover a sufficient range of lower-lead levels (i.e., below 5 or 10 µg/dL), to ensure that any threshold value would occur within the observed range.

Some researchers attempting to prove the existence of a threshold have reviewed results of applying the first statistical approach above (i.e., making predictions within groups of the cohort), but have made conclusions based upon simple plots of the results rather than by citing the outcome of statistical comparisons. For example, Kaufman (1996) has concluded that threshold effects may exist at about 20 µg/dL from data presented in Dietrich et al. (1993), at from 10-15 µg/dL from data presented in Bellinger et al. (1992), and at from 25-35 µg/dL in Hatzakis et al. (1989). However, each conclusion was based on visually interpreting selected figures within these articles rather than on the results of controlled statistical hypothesis tests. Furthermore, the following must also be considered when interpreting these conclusions:

- Dietrich et al. (1993): From this prospective study conducted in Cincinnati, OH, Kaufman (1996) cites the authors' presentation of predicted Wechsler Scale Performance IQ (PIQ) for four groups of children approximately 6.5 years of age, where the groups are determined by lifetime mean blood-lead concentration (i.e., average blood-lead concentration measured at 3-month intervals from age 3 to 60 months, and at ages 66 and 72 months). The predicted PIQ score was lower in the group with the highest lifetime mean blood-lead concentration (>20 µg/dL) compared to the other groups. However, the authors caution against interpreting this finding as evidence of a threshold effect, as most children in this group had one or more individual measurements above 30 µg/dL. Furthermore, in his meta-analysis, Schwartz (1994) considered a different relationship cited by the authors: full-scale IQ as a function of average blood-lead concentration through age 3 years (and other covariates, including HOME score and material IQ). This relationship, cited in Appendix D2 of the §403 risk analysis report, is more relevant to the representative population in the risk analysis.

- Bellinger et al. (1992): From this prospective study conducted in Boston, MA, Kaufman (1996) cites the authors' presentation of predicted WISC-R full-scale IQ and K-TEA Battery Composite scores for four groups of 10-year old children, where the groups are determined by blood-lead concentration at 24 months of age (PbB_{24}). Kaufman (1996) indicates that differences were apparent only between the two lowest and the two highest groups, suggesting an apparent threshold

between them (i.e., 10-15 μg/dL). However, little difference among any of the four groups would have been identified if the assertion was based on confidence intervals associated with the predictions, rather than standard errors for the individual groups. Meanwhile, the regression model used to predict IQ from PbB_{24} indicated a highly significant linear trend (p=0.007) across the entire range of observed values of PbB_{24}. This trend was present even among the three groups having the lowest blood-lead concentrations, suggesting that the trend is in fact present at the lower range of the observed concentrations (i.e., below 15 μg/dL).

- Hatzakis et al. (1989): From this study conducted in Greece within a city in which lead mining and smelting occurred, Kaufman (1996) cites how the authors present predicted full-scale IQ for primary school-aged children grouped by blood-lead concentration. Blood-lead concentrations in this study were high: the average blood-lead concentration in this study was 23.7 μg/dL, no child had blood-lead concentration below 7 μg/dL, and more than 90% of the children exceeded 10 μg/dL. The mean predicted IQ in the first two groups (≤14.9 μg/dL, 15-24.9 μg/dL) appeared to be statistically equivalent, then steadily declined for the remaining three groups, suggesting the presence of a threshold around 25 μg/dL. However, many other more recent studies (including animal studies) have observed neurological and developmental effects at lower blood-lead concentrations, making the concept of a threshold at 25 μg/dL highly unlikely. In fact, the linear regression model developed in this study (which included 17 covariates) had a highly significant slope for blood-lead concentration ($p < 0.001$) across the entire range of data in this study, even though more than 50% of the data occurred from 7-25 μg/dL. Furthermore, it is unclear if different conclusions would have been made if the groups of children were defined differently.

These examples illustrate the complexities associated with characterizing the dose-response relationship and the ability to conclude that a threshold exists in this relationship.

The findings of a study by Fulton et al. (1987), which was included in the Schwartz (1994) meta-analysis, appear to discount the high threshold level suggested by the findings of Hatzakis et al. (1989). This study was conducted on 501 children aged 6-9 years in Scotland. The predicted BASC score was calculated for ten groups of children determined by log-transformed blood-lead concentration. No evidence of a threshold was found among these data, and the estimated slope associated with log-transformed blood-lead concentration was significant (p=0.003). These findings were observed despite having only 10 study children with blood-lead concentrations exceeding 25 μg/dL.

Concluding Possible Thresholds for Tooth-Lead Concentration: Some studies (e.g., Bellinger and Needleman, 1983; Rabinowitz et al., 1992) have observed the potential for thresholds in tooth-lead concentration when relating tooth-lead concentration to IQ score. Based on their investigation of the relation between tooth-lead concentration and IQ score, Rabinowitz et al. (1992) suggested that a threshold for blood-lead concentration exists at approximately 8

µg/dL. Their investigation was centered around their 1989-1990 study of 764 children in grades 1-3 in Taiwan (an average age of 6.7 years). In this study, teeth shed by these children were analyzed for lead. In addition, the children were administered Raven's Colored Progressive Matrices (CPM) test, the score of which is considered a measure of IQ (average=25, SD=5.7). A model was developed which found CPM test score to be highly correlated with selected non-lead predictors (parental education level, sex, grade level, and whether or not the child is ambidextrous). The difference between the model-predicted and observed test scores for a child (the "CPM score deficit") was interpreted as a measure of the change in the test score that results from lead exposure.

Each of the 380 children for which CPM score deficit could be calculated was placed into one of two groups according to whether or not their tooth-lead level (µg/g) exceeded a specified value. Then, a Mann-Whitney test was performed to determine whether the mean CPM score deficit differed significantly between the two groups. This was done for a series of grouping values for tooth-lead, from 2 to 6 µg/g. Significant differences between the two groups ($p < 0.05$) were seen at grouping levels of tooth-lead at 3.5 µg/g or above, but not at 3 µg/g or below. Therefore, the authors concluded that a tooth-lead threshold for intelligence deficit existed at approximately 3.25 µg/g. Finally, the authors relate this tooth-lead threshold value to blood-lead by applying a modeled relationship between tooth-lead and blood-lead levels (formulated from data for 88 Boston children aged 57 months), and concluding that a tooth-lead threshold of 3.25 µg/g corresponded roughly to a blood-lead threshold of 8 µg/dL (± 2 µg/dL).

While the cohort was considered to have low tooth-lead concentrations, the following must be considered when interpreting the above conclusion on the presence of a threshold and its relevance to the §403 risk analysis:

- A non-zero threshold existing for tooth-lead concentration does not necessarily imply that one exists for blood-lead concentration, as tooth-lead may impact children's health in a different way from blood-lead.

- When noting the lack of significant difference between two groups defined by whether or not tooth-lead exceeds a given threshold when this threshold gets low enough, it is uncertain of the extent to which the lack of significance is actually due to reduced power to detect differences as the sample size in the non-exceedance group declines with the threshold being considered.

- It is uncertain whether the model to predict blood-lead based on tooth-lead from the Boston study, which was used to obtain the blood-lead threshold estimate of 8 µg/dL, can be applied directly to the findings of the Taiwan study without needing to consider certain statistical issues. For example, measurement error associated with tooth-lead levels may differ between the Boston and Taiwan studies.

- Rabinowitz et al. (1992) state that, when attempting to fit a "hockey-stick" regression model to the data, "this data shows no change in the slope (or intercept)

of the lines across any trial threshold." While this statement appears to support the hypothesis that no tooth-lead threshold exists, the authors do not provide any further information on the outcome of this model-based analysis.

2.3.3 Verifying the Results of Schwartz (1994)

Schwartz (1994) applied a random effects modeling approach suggested by DerSimonian and Laird (1986), a highly-regarded reference on meta-analysis. As a result, it was considered among the best encountered by the §403 risk analysis, and therefore, was an important contributor to how the §403 risk analysis characterized the relationship between blood-lead concentration and IQ score in children. For this reason, the meta-analysis findings were verified as part of the §403 risk analysis. Using either the weighted noniterative method or the weighted maximum likelihood method suggested by DerSimonian and Laird (1986), the §403 risk analysis obtained the same finding as Schwartz (1994): that a decrease of 0.257 (± 0.041) IQ points was associated with an increase in blood-lead concentration of 1.0 µg/dL within the range of 10-20 µg/dL. In addition, it was noted that heterogeneity of variance among the seven studies considered by Schwartz (1994) was not significant, and the random effects model gave the same results as a fixed effects model.

2.4 IMPACT OF CERTAIN RESIDENTIAL DUST CHARACTERISTICS ON DUST-LEAD EXPOSURE

The bioavailability of lead can be an important factor in determining the toxic effects of lead exposure to children within a specific environment. Because lead is found in a variety of chemical and physical forms depending on its source, the bioavailability of lead has been studied as a function of chemical make-up (i.e., the particular form of lead present) and particle size in various environmental matrices (e.g., dusts and soils, mining wastes). Generally, the literature concludes that the bioavailability of lead can depend on, among other things, the particular lead species present (which varies depending on the source of lead), the size of the lead-containing particles, the matrix incorporating the lead species, and the types of nutrients or other compounds ingested with the lead (Freeman et al., 1992; USEPA, 1994). It has been suggested that lead speciation and particle size may affect the bioavailability of lead through their influence on solubility (USEPA, 1994). For example, lead bioavailability appears to be lower in mining areas relative to urban and smelter areas. Some authors (e.g., Rieuwerts and Farago, 1995; Davis et al., 1995; Freeman et al., 1992) have suggested that this difference may be, in part, explained by variations in chemical form (dissolutions rates) and particle size. Studies have also shown that correlations between soil-lead and blood-lead levels are influenced by particle size and composition of the lead compounds (USDHHS, 1992).

The purposes of this section are:

1. To present a brief review of the some of the available literature which specifically examines the bioavailability of lead in dust, as a function of particle size and chemical composition of the dust.

2. To determine if there is evidence which warrants the consideration of particle size and lead speciation, as related to lead bioavailability in dust, in the §403 rulemaking; and, if warranted, determine if there is sufficient information available in the literature to allow for a thorough consideration.

3. If relevant, to identify significant information gaps and potential issues that may warrant further research.

2.4.1 Review of Literature: Effects of Chemical Composition on Lead Bioavailability in Dust

There is substantial evidence in the scientific literature that the particular chemical species, as well as the matrix (e.g., mineralogy, organic matter content) within which the lead compound is found, are important in determining lead bioavailability (USDHHS, 1999; USEPA, 1994). Many of the studies in the literature have been based on comparisons of relatively simple lead compounds in controlled animal feeding studies or have focused on lead bioavailability in urban and mining-associated soils. With respect to household dust in particular, there is relatively little in the literature which specifically examines the relationship between bioavailability and chemical composition.

The literature does recognize, however, that the composition of interior dust is substantially influenced by soil and exterior dust (Diemel et al., 1981; USEPA, 1994). For example, USEPA (1994) characterizes total lead in household dust as being comprised of soil-lead, air-lead, lead from outside sources (e.g., workplace, school), and lead from household paint. As a default value to the IEUBK exposure model, EPA has set the ratio of household dust-lead concentration to soil-lead concentration at 0.70, which was considered appropriate for neighborhoods or residences where loose particles of surface soil are readily transported into the house (USEPA, 1994). Thus, soil particles have the potential to be a significant contributor to lead levels in household dust.

The following sections will provide a brief review of scientific findings related to the general physical and chemical principles of lead and how they relate to bioavailability differences in controlled environments and in soil studies. Because the literature recognizes that the composition of interior dust is influenced by soil and exterior dust, discussion of these general bioavailability factors will, in the absence of more dust-specific data, serve as a starting point in understanding the relationship between lead bioavailability in household dust and chemical composition and particle size.

2.4.1.1 Research on Lead Bioavailability in Controlled Animal Studies. The relative bioavailabilities of simple lead compounds have been studied under controlled conditions in animal studies. For example, Barltrop and Meek (1975) (as cited in USDHHS, 1992) compared the absorptions of 12 different lead compounds in rats by measuring the kidney contents following oral exposure. They found that the absorption of metallic-lead (particle size 180-250

μm) was the lowest of the lead compounds tested. Data also suggested that the absorption of lead sulfide (particle size <50 μm) was significantly less than the oral bioavailability of other lead salts (oxide, acetate). Lead carbonate had the highest absorption, which was suggested to be due to its high solubility in gastric juice.

Dieter et al. (1993) also found differing blood-lead, bone-lead and kidney-lead levels in rats fed different lead compounds, indicating variability in bioavailability. For example, maximum blood-lead levels were higher (80 μg/dl) in rats fed lead acetate and lead oxide, in comparison to rats fed lead sulfide and a lead ore concentrate. Similar differences were observed in bone-lead and kidney-lead levels between the rats receiving the more soluble (e.g., lead acetate and oxide) and less soluble (e.g., lead sulfide and ore) lead compounds.

2.4.1.2 Research on Lead Bioavailability in Soils. The literature also suggests that the soil matrix itself can be an important factor in determining the bioavailability of lead. For example, Freeman et al. (1992) found that tissue-lead concentrations were lower in rats fed lead-contaminated mining waste soils from Butte, Montana, as compared to rats fed comparable doses of soluble lead acetate. It was suggested that the inherent chemical properties of soil-adsorption sites and the alteration of lead-bearing solids (e.g., encapsulation processes which inhibit dissolution) may reduce the bioavailability of soil-lead, as compared to lead ingested without soil. In general, the fate and bioavailability of lead in soils are affected by the species of lead incorporated into the soil, the degree of absorption at mineral interfaces, precipitation of solid phases, and the formation of relatively stable complexes/chelates with organic matter, as well as other complex soil matrix factors such as pH (USDHHS, 1999; McKinney, 1993; Freeman et al., 1992). This has been suggested to be largely due to the influence of these factors on solubility, although it is important to note that solubility is but one factor in the bioavailability of lead to humans or animals (USEPA, 1994).

In a study of lead bioavailability in soil, Laperche et al. (1997) found that apatite (calcium fluoride phosphate) amendments to a lead-contaminated soil lowered the bioavailability of soil lead (as determined by plant uptake) by inducing the formation of geochemically-stable lead phosphate compounds. Similarly, in a study of an old mining village with elevated lead levels in both garden soils and house dust, Cotter-Howells (1994) identified the predominance of lead phosphate compounds (of limited bioavailability) as probable explanation of why blood-lead levels in the village were not elevated in an otherwise contaminated area. In a study of mining-associated soils in Butte, Montana, Davis et al. (1995) suggested that the predominance of lead sulfide/sulfate and oxide/phosphates in soil and mine waste samples might provide an explanation for the limited lead bioavailability that was observed when the Butte soils were fed to rats in a previous study (Freeman et al., 1992).

Urban area soils are typically contaminated with alkyl lead species originating from combustion of leaded gasoline; lead halides (chlorides and bromides) from auto exhaust particulates; or lead carbonate, chromate, and octoate (as chips, flakes, and dusts) from exterior and interior lead-based paint (USEPA, 1994). Lead halides in soils are quickly transformed to

(or associated with) oxides or sulfates (USEPA, 1986 as cited in USEPA, 1994). In many lead-mining districts, the predominant form of lead is galena or lead sulfide (USDHHS, 1992).

2.4.2 Review of Literature: Effects of Particle Size on Lead Bioavailability in Dust

Data in the literature are limited with specific regards to how particle size of lead-contaminated house dust influences the bioavailability of lead in the dust. However, studies have been conducted to examine the general relationship in soils between particle size and bioavailability, as well as particle size and lead concentration. For example, Barltrop and Meek (1979) found that the bioavailability of lead in the intestinal tract of rats fed metallic-lead of various particle sizes increased fivefold as particle size decreased from 197 microns to 6 microns. Particle size, due to kinetic limitations that control dissolution rates in the gastrointestinal tract, has also been hypothesized to contribute to the lower bioavailability of lead observed in mining waste soils relative to urban and smelter soils (Davis et al., 1995). In general, the smaller the particle size, the greater the absorption of lead due to more rapid dissolution (small particles have higher surface area to mass) in the gastrointestinal tract (Freeman et al., 1992).

Que Hee et al. (1985) found that when lead concentrations were measured in dust samples categorized by size fraction, lead concentration was generally independent of the particle size. However, most of the dust particle mass (about 75%), and thus most of the lead (about 77%), was present in the <149 μm size fraction. Lead concentration in smaller particle size ranges may possibly maximize intestinal absorption, and thus increase bioavailability (USDHHS, 1992). Duggan and Inskip (1985) performed an extensive literature review on the variation of lead concentration with particle size and reported that higher lead concentrations are usually found in the smaller-sized fractions of soil and dust. As reported by the Agency for Toxic Substances and Disease Registry, numerous studies have also observed the lead content of soil, street dust, city dust, and house dust to increase with decreasing particle size (USDHHS, 1992).

2.4.3 Information Gaps, Issues and Conclusions

Although the literature is generally lacking in data which directly address the bioavailability of lead in household dust as a function of chemical composition and particle size, by recognizing that interior dust composition can be greatly influenced by outside soil, it can reasonably be expected that the factors which affect lead bioavailability in soils will also influence the bioavailability of lead in household dust. Therefore, based on general knowledge of the bioavailability of simple lead compounds and studies of lead compounds in soil matrices, evidence suggests that particle size and chemical composition have the potential to significantly affect lead bioavailability in dust. Nonetheless, the current information base which specifically addresses particle size and chemical composition of dust as factors in lead bioavailability may be inadequate to determine how such factors can reasonably be incorporated into the rulemaking effort. Furthermore, needing to characterize dust by particle size and lead by chemical speciation within a risk assessment will likely add to the expense of dust analyses, and dust standards that

distinguish between these various characterizations could add considerable complexity to the rule.

Specific uncertainties that remain concerning bioavailability of lead in household dust include the following: physical and chemical properties that may be unique to dust versus soil; whether the effect of lead speciation in dust is significant enough to affect dust standards for lead; distributions of lead across particle sizes found in household dust (e.g., whether dust is enriched with the smaller size fraction relative to outside soil) and whether particle size differences are significant enough to affect standards; and possibly variances in exposure mechanisms that may occur across particle sizes.

The need for further research in these and related areas has been supported by several authors. For example, Freeman et al. (1992), based on comparisons of mining waste soils and other soil types in reviewed studies, emphasized the importance of evaluating the soil mineralogy and lead species present when predicting bioavailability values for lead in soils. In addition, USEPA (1994) in the *Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children*, notes that adequate characterization of lead contaminated media, for the purpose of estimating bioavailability, should include assessment of physical and chemical parameters, such as particle size and media solubility.

3.0 EXPOSURE ASSESSMENT

According to Chapter 3 of the §403 risk analysis report, the goal of the exposure assessment was to document the important sources of lead in the environment, to document the major pathways by which children are exposed to lead, to characterize the current (baseline) 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.

In particular, Chapter 3 introduced those data sources used to characterize environmental-lead levels in the nation's housing stock and presented summaries of household average lead levels in dust and soil as reported in these studies. The U.S. Department of Housing and Urban Development (HUD)'s National Survey of Lead-Based Paint in Housing ("HUD National Survey", Section 3.3.1.1 of the §403 risk analysis report) was selected as the data source for characterizing baseline environmental-lead levels in the nation's housing stock. Pre-intervention data from other selected studies, such as the Rochester Lead-in-Dust study and the ongoing Evaluation of HUD Lead-Based Paint Hazard Control Grant Program ("HUD Grantees") were also summarized in Section 3.3.1 of the §403 risk analysis report to provide supporting information on environmental-lead levels and to obtain information on the relationship between these levels and blood-lead concentration in children.

Since the §403 risk analysis report was published, additional data on environmental-lead levels in the nation's housing stock have been made available to EPA. These data include interim data from the National Survey of Lead and Allergens in Housing, and additional data from the HUD Grantees evaluation. In addition, updated data from the U.S. Census Bureau are available on numbers of young children associated with the various types of lead exposures found in the national housing stock. Some comments on the §403 proposed rule suggested that EPA use these additional data when available. Therefore, EPA has investigated these new data to document additional, more recent information on lead levels in the nation's housing stock and, when available, blood-lead levels in children exposed to these lead levels. For example, it was of interest to document more recent information on the distribution of lead levels in dust deposited on interior uncarpeted floors and window sills (i.e., the surfaces included in the proposed §403 standards), as well as on other types of surfaces (e.g., exterior surfaces, window troughs) to help evaluate their potential contribution to overall lead exposure at a residence. It was also of interest to characterize the national distribution of residential soil-lead levels and percentages of the housing stock whose soil-lead levels exceed specified thresholds. Therefore, this chapter provides additional information on lead exposure within the following sections:

- Section 3.1: Information on the National Survey of Lead and Allergens in Housing (NSLAH), a national survey begun in 1997 of lead levels in dust and soil in U.S. residential housing.

- Section 3.2: Comparison of the HUD National Survey data summaries for dust-lead loading and soil-lead concentration with summaries from other lead exposure studies, including interim data (for 706 households) from the NSLAH and pre-

intervention data from the HUD Grantees evaluation that have been revised and augmented since the §403 risk analysis report was published).

- Section 3.3: Information on the prevalence of soil pica tendencies in young children and how such tendencies may occur over and above paint pica tendencies.

- Section 3.4: Updated information on numbers of children in the nation's housing stock, using interim data from the NSLAH.

- Section 3.5: Distribution of dust-lead levels on surfaces other than uncarpeted floors and window sills.

- Section 3.6: Revised summaries of pre-intervention blood-lead concentration based on updated data from the HUD Grantees evaluation.

3.1 THE NATIONAL SURVEY OF LEAD AND ALLERGENS IN HOUSING

The National Survey of Lead and Allergens in Housing (NSLAH) is a currently-ongoing survey sponsored by the U.S. Department of Housing and Urban Development (HUD) and the National Institute of Environmental Health Sciences (NIEHS) to assess the lead and allergen burden in that portion of the regularly-occupied U.S. housing stock that can potentially include young children among its residents. In particular, the survey is assessing lead burden by characterizing levels of lead-contaminated dust, lead-based paint, and lead-contaminated soil in housing and residential areas. HUD initiated this survey in 1997 and has been approved by the Office of Management and Budget to collect information through April 2001 for up to 1000 housing units.

The NSLAH provides a more recent nationally-representative characterization of environmental-lead levels in the U.S. housing stock than the 1989-1990 HUD National Survey and involves sampling in considerably more housing units. In addition, dust samples in the NSLAH are collected using wipe techniques (i.e., the technique assumed in the §403 rule) rather than the Blue Nozzle vacuum method used in the older survey, and the NSLAH did not restrict the sampling frame to only housing built prior to 1980. Therefore, the information collected in the NSLAH is very important for the §403 risk analysis to consider. However, the survey's scheduled completion date and the expected date for finalizing the survey's database do not fall within the time frame necessary to complete the risk analysis. Therefore, in order to utilize data from the NSLAH, the risk analysis could only consider data collected up to an interim point in the survey.

Interim NSLAH data for 706 housing units, collected from 1998-1999, were made available to the §403 risk analysis in August, 1999. This is a preliminary subset of the survey's final database that will represent an expected 825 housing units. To allow the data for these 706

units to be considered a nationally-representative characterization of lead levels in the housing stock, the interim database included sampling weights assigned to each unit based on its set of selection probabilities within each stage of the multi-staged sampling design and adjusted for nonresponse. These are interim sampling weights as they were generated by only considering the 706 units represented in the interim database. As the final sampling weights to be assigned at the end of the survey will reflect all housing units in the survey, and as there is a potential for additional correction of the existing data before the survey database is finalized, any analysis results based on the interim database of 706 housing units will likely differ from those to be based on the final database.

Table 3-1 contains key design specifications and approaches of the NSLAH, such as the types of rooms in which dust samples were collected and paint-lead levels were measured, the approach to taking soil samples, and laboratory analytical methods. Also included for comparison purposes in Table 3-1 are the design specifications and approaches taken in the older HUD National Survey. Note that in both surveys, dust samples were taken from the same types of surfaces (floors, window sills, and window troughs, also known as window wells) and analyzed under similar methods, and soil sampling occurred in the same areas of the yard. The method for analyzing soil samples was changed from ICP-AES in the older survey to FAA in the NSLAH due to the need to reduce detection limits associated with the method. Specific focus was made in the NSLAH to ensure that rooms in which children frequently reside are more dominantly represented in the sampling design.

Various types of data are being collected from housing units participating in the NSLAH. Household questionnaire data are collected at two time points: at the initial contact with the household during recruitment (to screen for eligibility and to perform an inventory on interior rooms) and during an interview with residents during the study (to obtain information on the building, household, and residents). Allergen dust levels are measured by collecting and analyzing vacuum dust samples. Lead levels in the unit are characterized through the following types of measures:

- Dust samples: Dust-lead loadings ($\mu g/ft^2$, assuming wipe collection techniques) for floors, window sills, and window troughs (also known as window wells)
- Soil samples: Soil-lead concentrations ($\mu g/g$) at entryway, dripline, and mid-yard
- Lead on painted surfaces: X-ray fluorescence (XRF) measurements (mg/cm^2)

To determine the numbers of housing units represented by the interim NSLAH sampling weights within certain housing categories and how these numbers compare with estimates made in the §403 risk analysis and by the U.S. Census Bureau, Tables 3-2 and 3-3 provide estimated numbers of occupied housing within specified housing age categories and the four Census regions, respectively. These totals are presented based on data from the NSLAH as well as from the following additional surveys/analyses:

Table 3-1. Differences in Approaches and Outcomes Between the HUD National Survey of Lead-Based Paint in Housing and the HUD National Survey of Lead and Allergens in Housing

Area	HUD National Survey of Lead-Based Paint in Housing[1]	HUD National Survey of Lead and Allergens in Housing[1]
Types/numbers of housing units selected for the survey and whose data were available to the §403 risk analysis	284 housing units selected from privately-owned, year-round occupied housing in the 48 conterminous states built prior to 1980 and having the potential for containing children. Institutional and group (i.e., housing units with at least 10 unrelated persons) housing were excluded from consideration for the survey.	Interim data for 706 housing units selected from year-round occupied housing in the 50 states and the District of Columbia having the potential for containing children were provided to EPA on August 13, 1999 (out of an expected 825 housing units in the survey). The sample represents 67 of the planned 75 primary sampling units (PSUs). Institutional and group (i.e., housing units with at least 10 unrelated persons) housing were excluded from consideration for the survey.
Breakdown of selected units by year built	Pre-1940: 27% 1940-1959: 31% 1960-1979: 42% Post-1979: 0%	Pre-1940: 18% 1940-1959: 23% 1960-1977: 31% Post-1977: 28% (Percentages are relative to the 640 units with housing age information from either the recruitment or resident questionnaire.)
Dates of environmental sampling	November 1989 to March 1990	August 1998 to February 1999 (according to dates specified in the survey's interim database -- sampling in a small number of units may have occurred earlier in 1998)

Table 3-1. (cont.)

Area	HUD National Survey of Lead-Based Paint in Housing[1]	HUD National Survey of Lead and Allergens in Housing[1]
Selecting rooms for environmental sampling	Telephone household interview provided information on rooms. One room was selected for sampling in each of the following strata: • Wet room -- rooms containing plumbing (e.g., kitchen, bathroom, laundry room, utility room) • Dry room -- all rooms not classified as wet rooms • Main entryway (floor dust samples only)	Room Inventory Form from the Screening/Recruiting Questionnaire was used to obtain information on rooms. One room was randomly selected for sampling in each of the following four strata: • Kitchen • Common living area (e.g., living room, den, family room) • Bedroom in which one or more children aged 17 years or younger regularly slept, or any regularly-occupied bedroom if no such children lived in the unit (occasionally, two such bedrooms were selected) • Other random room among the remaining rooms in the housing unit. (Note: Two rooms were randomly selected from this stratum if the stratum contained at least six rooms. Adult bedrooms were included if a child's bedroom was available for selection in the bedroom stratum.) In addition to the selected rooms, floor dust samples from the main entryway were collected.
Method to assigning sampling weights	Weights reflect the various stages of sampling and were designed to sum to the approximately 77 million pre-1980 homes then in the occupied housing stock. The weights were stratified to control for the number of housing units with children (13.9 million) and without children. Total of the sampling weights within a given census region equaled the estimated number of units with children under age 7 years in the census region.	Interim weights reflect the various stages of sampling and were designed to sum to the estimated 89 million housing units in the occupied housing stock that do not exclude children.
Method for taking dust samples for lead analysis	Blue Nozzle vacuum (a few wipe samples were also collected)	Wipes, collected in accordance with ASTM E1728-95, *Practice for the field determination of settled dust samples using wipe sampling methods for lead determination by atomic absorption spectrometry techniques.*
Number and location of floor-dust samples per room	One sample from each selected room (location not dictated in the protocol)	One sample from each selected room, generally taken from the largest open area.

Table 3-1. (cont.)

Area	HUD National Survey of Lead-Based Paint in Housing[1]	HUD National Survey of Lead and Allergens in Housing[1]
Window sill/trough dust sampling approach	A window was selected within each selected room according to some ranking scheme. Sampling was performed from both the sill and trough of the selected window until "enough" dust was collected or until the entire sill or trough was vacuumed.	Entire sill and trough sampled from a random window in the selected room.
Number and location of sill and trough dust samples per room	One sample from each of the sill and trough of the selected window in the selected wet room and dry room	One sample from the sill and one sample from the trough of the selected window in each selected room
Method of analyzing dust samples	GFAA (with SW-846 digestion method)	FAA (Digestion method: modification of SW-846 Method 3050 or ASTM ES 36-94 -- hot-plate digestions utilizing nitric/perchloric acid and H_2O_2) Method must be that used in proficiency testing within the Environmental Lead Laboratory Accreditation Program (ELLAP)
Soil sampling approach	One composite sample of 3 core samples (the latter two taken within 20 inches of the first), each taken at a depth of 10 cm, was collected at each of the following locations: entryway, drip-line, and remote area (i.e., an area halfway between the unit and its property boundary, or within 25 feet of the unit).	Samples were collected from bare soil when possible. If no bare soil existed, soil samples were collected from covered surfaces if possible. Two sides of the unit were selected for soil sampling: the side containing the major entryway (Wall 1) and a second, randomly-selected side (Wall 2). Samples were collected from the top 0.5 inches of soil at the following three locations: • Main entry – a single sample from Wall 1 • Foundation/drip-line – one sample from each of Walls 1 and 2, each sample being a composite of 3 core samples taken within 3 feet of the foundation • Mid-yard area – one sample from each of Walls 1 and 2, each sample being a composite of up to 4 core samples taken midway between the drip-line and the closer of the boundary line or another building on the property. Soil samples were collected in accordance with core sampling procedures based on ASTM E1727-95 (described in the HUD *Guidelines* and in EPA's *Residential Sampling for Lead: Protocols for Leaded Dust and Soil Sampling*)

Table 3-1. (cont.)

Area	HUD National Survey of Lead-Based Paint in Housing[1]	HUD National Survey of Lead and Allergens in Housing[1]
Method of analyzing soil samples	ICP-AES (with SW-846 digestion method)	ICP-AES Digestion method: modification of SW-846 Method 3050 or ASTM ES 36-94 (hot-plate digestions utilizing nitric acid and/or HCl/H_2O_2), or SW-846 Method 3051 (microwave nitric acid digestion) Method must be that used in proficiency testing within the Environmental Lead Laboratory Accreditation Program (ELLAP)
Handling dust-lead and soil-lead measurements below the detection limit	As log-transformed lead amounts are reported in the database, only positive measurements are represented. No indication is given as to when data may have been truncated due to being below detection limits.	The final results as reported by the instrument are recorded in the database (i.e., not-detected results are not censored), along with detection limits.
Method for taking paint-lead measurements	MAP-3 XRF instrument (single 60-second "spectrum reading" measurement using a 40 millicurie cobalt source). Measurements were adjusted to statistically correct for measurement bias.	XRF (Niton XL-309 running software version 5.1)
Approach to selecting interior painted components for paint-lead measurements	Painted surfaces were categorized into the following four strata: • Walls/ceilings/floors • Metal substrate • Non-metal substrate • Other surfaces Five painted components were selected randomly for testing in each of the selected wet and dry rooms, one from each stratum along with a fifth selected randomly from among all strata. In addition, up to two purposive measurements were taken from paint anywhere in the unit that may be suspected to contain lead.	A list of 25 possible interior components was developed and included: • All four major walls • Ceiling • Floor • Window system components • Doors and doorways • Trim • Porches All components present in a given room were tested.

Table 3-1. (cont.)

Area	HUD National Survey of Lead-Based Paint in Housing[1]	HUD National Survey of Lead and Allergens in Housing[1]
Approach to selecting exterior painted components for paint-lead measurements	Painted surfaces were categorized into the following four strata: • Wall (randomly-selected) • Metal substrate within the selected wall • Non-metal substrate within the selected wall • Other surfaces within the selected wall Five painted components were selected randomly for testing from the side of the unit containing the selected wall, one from each stratum along with a fifth selected *randomly from among all strata. In addition, up to two* purposive measurements were taken from paint anywhere on the exterior of the unit that may be suspected to contain lead.	A list of 25 possible exterior components was developed and included: • Siding • Window system components • Doors and doorways • Trim • Porches All components present on the sampled wall were tested.

[1] Information reflects only that part of the survey whose data and information were used in the §403 risk analysis.

Table 3-2. Estimated Number of Occupied Housing Units in the U.S. Housing Stock Within Year-Built Categories, According to Four Recent Surveys and/or Analyses

Year in Which the Unit Was Built	Number of Units in the National Housing Stock (and Percentage of Total Units), as Estimated by the ...				# Units Surveyed	
	1995 American Housing Survey[1]	§403 Risk Analysis[2]	1997 American Housing Survey[3]	National Survey of Lead and Allergens in Housing (NSLAH)[4]	1989-90 HUD National Survey	NSLAH (interim)
Prior to 1940	19,308,000 (20%)	19,676,000 (20%)	19,441,000 (20%)	14,412,000 (18%)	77	114
1940-1959	19,885,000 (20%)	19,718,000 (20%)	19,797,000 (20%)	16,886,000 (21%)	87	145
1960-1979 (1960-1977 for NSLAH)	35,300,000 (36%)	34,985,000 (35%)	34,884,000 (35%)	25,688,000 (32%)	120	201
After 1979 (After 1977 for NSLAH)	23,201,000 (24%)	24,893,000 (25%)	25,367,000 (25%)	24,076,000 (30%)	--	180
Not specified[5]	--	--	--	8,089,000	--	66
Total	97,693,000	99,272,000	99,487,000	89,151,000	284	706

[1] Estimates represent only year-round occupied housing in the 1995 national housing stock and were obtained from information within Table 1A-1 of "American Housing Survey for the United States in 1995" (Current Housing Reports H150/95RV, published by the Bureau of the Census and HUD's Office of Policy Development and Research). This national survey was conducted on about 55,000 surveyed units from August 1995 through February 1996. An updated report reflecting the 1997 American Housing Survey data has not yet been published.

[2] Estimates were obtained from Table 3-5 of the §403 risk analysis report. Estimates are based on data from the 1989-90 HUD National Survey of Lead-Based Paint in Housing, augmented by other Census information in order to represent the 1997 housing stock (see the §403 risk analysis report for details).

[3] Estimates were obtained from Table 2-1 of U.S. Census Bureau (1999). The estimates represent total year-round occupied units in the 1997 national housing stock.

[4] This survey, conducted from 1998-1999, characterized only occupied housing in which a young child could reside. Information in this table is based on an interim dataset for 706 surveyed housing units. Year-built information was determined from responses given in the survey's resident questionnaire. If no year-built information was available from the resident questionnaire, any year-built information provided from the recruitment questionnaire (when available) was used. Note differences in how year-built categories were defined in this survey. The specified percentages are relative to the total minus the number of units represented by surveyed units with no year-built information specified (i.e., 89,151,000 - 8,089,000 - 81,062,000).

[5] Total sampling weights for surveyed units where either no housing age information was provided, or responses of "Don't Know" or "Not Ascertained" were given, on both the resident and recruitment surveys.

Table 3-3. Estimated Number of Occupied Housing Units in the U.S. Housing Stock Within Each Census Region, According to Four Recent Surveys and/or Analyses

Census Region	Number of Units in the National Housing Stock (and Percentage of Total Units), as Estimated by the ...				# Units Surveyed	
	1995 American Housing Survey[1]	§403 Risk Analysis[2]	1997 American Housing Survey[3]	National Survey of Lead and Allergens in Housing (NSLAH)[4]	1989-90 HUD National Survey	NSLAH (interim)
Northeast	19,200,000 (20%)	15,878,000 (16%)	19,484,000 (20%)	14,977,000 (17%)	53	109
Midwest	23,662,000 (24%)	22,313,000 (22%)	23,951,000 (24%)	22,202,000 (25%)	69	150
South	34,236,000 (35%)	41,733,000 (42%)	34,808,000 (35%)	32,519,000 (36%)	116	265
West	20,596,000 (21%)	19,348,000 (19%)	21,245,000 (21%)	19,453,000 (22%)	46	182
Total	97,693,000	99,272,000	99,487,000	89,151,000	284	706

[1] Estimates represent only year-round occupied housing in the 1995 national housing stock and were obtained from information within Table 1A-1 of "American Housing Survey for the United States in 1995" (Current Housing Reports H150/95RV, published by the Bureau of the Census and HUD's Office of Policy Development and Research). This national survey was conducted on about 55,000 surveyed units from August 1995 through February 1996. An updated report reflecting the 1997 American Housing Survey data has not yet been published.

[2] Estimates were obtained from Table 3-5 of the §403 risk analysis report. Estimates are based on data from the 1989-90 HUD National Survey of Lead-Based Paint in Housing, augmented by other Census information in order to represent the 1997 housing stock (see the §403 risk analysis report for details).

[3] Estimates were obtained from Table 2-1 of U.S. Census Bureau (1999). The estimates represent total year-round occupied units in the 1997 national housing stock.

[4] This survey, conducted from 1998-1999, characterized only occupied housing in which a young child could reside. Information in this table is based on an interim dataset for 706 surveyed housing units.

- the 1995 American Housing Survey (i.e., the last survey in which estimates of these totals were published in documents issued by the Census Bureau)

- the §403 risk analysis (which characterized the 1997 housing stock by revising the sampling weights from the 1989-90 HUD National Survey)

- the 1997 American Housing Survey (based on information obtained from the HUD web-site)

As noted in these tables, the sum of the interim sampling weights in the NSLAH (89,151,000) is over ten million units lower than the corresponding sums from the §403 risk analysis and the 1997 American Housing Survey. It is possible that this difference is due to the NSLAH's exclusion of housing that forbids resident children (i.e., adult-only housing), while the §403 risk analysis and the 1997 American Housing Survey results reflect the entire regularly-occupied housing stock.

When reviewing the sum of sampling weights by housing age category (Table 3-2), the interim NSLAH data represent a slightly smaller percentage of pre-1940 housing compared to the other surveys. The housing age categories for the NSLAH differ slightly from the categories in which the other surveys are represented and what was used in the 1989-90 HUD National Survey. Approximately eight million housing units in the U.S. housing stock are represented by 66 units in the interim NSLAH dataset that do not have a housing age specified. The final two columns of Table 3-2 present numbers of surveyed units by housing age category in both HUD surveys.

The percentages of housing units within Census regions (Table 3-3) are similar between the interim NSLAH and the 1997 American Housing Survey except for the Northeast, where the percentage in the interim NSLAH was lower than in the 1997 American Housing Survey. Differences relative to the 1997 American Housing Survey were even greater for the §403 risk analysis, where the adjustments made to the sampling weights in the 1989-90 HUD National Survey to represent the 1997 housing stock did not take into account Census region.

Summaries of the interim NSLAH data and comparison to the 1989-90 HUD National Survey data summaries (cited in the §403 risk analysis report) are provided in the next section.

3.2 COMPARISON OF ENVIRONMENTAL-LEAD LEVELS IN THE HUD NATIONAL SURVEY WITH THOSE OF OTHER KEY STUDIES

As discussed in Sections 3.2 and 3.3 of the §403 risk analysis report, the risk analysis used data from the HUD National Survey to represent baseline (pre-§403) environmental-lead levels (paint, dust, soil) in the nation's housing stock. To help evaluate how accurate this representation may be and how environmental-lead levels may have changed since the HUD National Survey was conducted (1989-1990), the survey data were compared with data from other environmental field studies that were conducted more recently and that measured

environmental-lead levels in a large number of housing units. This section also summarizes how housing selection, sample collection techniques, laboratory testing practices, and the distribution of environmental-lead levels reported in the HUD National Survey differ from those in these other studies.

The studies whose dust-lead and soil-lead data were used in the comparisons in this section included the ongoing National Survey of Lead and Allergens in Housing (NSLAH, introduced in Section 3.1 above), the Baltimore Repair & Maintenance (R&M) Study, the Rochester Lead-in-Dust Study, and the various portions of the ongoing HUD Grantees evaluation (design information and data for the latter three studies were summarized in Section 3.2.2 of the §403 risk analysis report). These studies were conducted since 1993 in locations within the United States where a specific point source of lead was not necessarily present. The latter three studies provided the §403 risk analysis with the most useful and available data on the relationship between environmental-lead levels (paint, dust, and soil) and childhood blood-lead concentration. In particular, dust samples in these studies were collected from floors and window sills using either a wipe technique or a method whose resulting dust-lead loadings could be converted to wipe-equivalent loadings using methods such as those documented in Section 4.3 of the §403 risk analysis report. Data summaries for the HUD Grantees evaluation were updated from the §403 risk analysis report summaries to reflect data collected through February, 1999.

The risk associated with elevated soil-lead concentrations and intervention practices designed to alleviate that risk are more frequently debated in the scientific literature than are the risk from and the intervention practices targeting elevated dust-lead loadings. As a result, this section supplements the comparison of the HUD National Survey's characterization of soil-lead concentrations to the interim NSLAH and the aforementioned three recent studies with the results of other relevant studies.

Boxplots were used in this section to summarize household average dust-lead and soil-lead levels graphically. A boxplot, also known as a box-whisker plot, portrays the distribution visually by using a box to represent data falling within the 25th and 75th percentiles and using different graphical symbols for the remaining data values according to their distance from the box. The following features are included within the boxplots presented in this section:

- A horizontal line within the box corresponds to the median.
- A dot within the box corresponds to the geometric mean.
- The bottom and top edges of the box correspond to the 25th and 75th percentiles, respectively.
- Central vertical lines ("whiskers") extend to 1.5 interquartile ranges (IQR, equal to the difference between the 75th and 25th percentiles on a log scale) of the box. However, if the data extend to less than 1.5 IQRs of the box, the whiskers extend only as far as the data exist.
- Open circles represent data values that exceed 1.5 IQRs but no more than 3 IQRs from the box.
- Asterisks represent data values that exceed 3.0 IQRs from the box.

The boxplots were plotted on a logarithmic scale to improve the readability of the data distributions, due to the tendency of the data to be skewed toward the lower end of these distributions. Selected information portrayed within the boxplots have also been included within tables of descriptive statistics presented throughout this section.

Dust-lead loading data comparisons are provided in Section 3.2.1, while soil-lead concentration data are addressed in Section 3.2.2.

3.2.1 Characterizing Dust-Lead Loadings on Floors and Window Sills

Household area-weighted average dust-lead loadings (assuming wipe techniques) as calculated in the §403 risk analysis were the basis for the comparisons made in this section. This average, calculated for each building component sampled for dust (i.e., floor, window sill), represented a single dust-lead measure for the component within a housing unit and was calculated by weighting each dust sample's result by the area that was sampled.

While the household average dust-lead loadings assumed wipe collection techniques, the dust collection device differed among the studies:

- HUD National Survey: Blue Nozzle vacuum
- Baltimore R&M study: BRM vacuum
- NSLAH, Rochester study, and the HUD Grantees evaluation: wipes.

To obtain wipe-equivalent dust-lead loadings for samples taken in the HUD National Survey and the Baltimore R&M study, the reported loadings were entered into the conversion equations presented in Sections 4.3.1 (Blue Nozzle vacuum to wipe) and 4.3.2 (BRM vacuum to wipe) of the §403 risk analysis report. Note that dust-lead loadings for samples collected by other collection methods in these studies were not included in determining the area-weighted averages.[7]

In the §403 risk analysis, the household averages were calculated on wipe-equivalent sample loadings associated with the 284 units in the HUD National Survey, with imputed averages assigned to those units having no available data (Section 3.3.1.1 of the §403 risk analysis report). When characterizing the distribution of these averages across units, the §403 risk analysis weighted each unit by its 1997 sample weight as calculated for the §403 risk analysis (Appendix C1 of the §403 risk analysis report), and each unit built between 1960 and 1979 and without lead-based paint also represented post-1979 housing (Section 3.3.1.5 of the §403 risk analysis report). The resulting data distribution was used in the §403 risk analysis to characterize the distribution of average dust-lead loadings in the nation's housing stock.

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[7] The HUD National Survey database included a few wipe dust-lead loadings that were used as reported in determining household area-weighted averages.

3.2.1.1. Data Summaries for the §403 Risk Analysis Versus the Interim NSLAH. Descriptive statistics of household average dust-lead loadings for floors and window sills as calculated in the §403 risk analysis using the HUD National Survey data are presented in this subsection as they compare with the same statistics calculated on interim data for 706 housing units in the NSLAH. Note that these statistics reflect the sampling weights used in the §403 risk analysis and the interim NSLAH sample weights, thereby allowing these summaries to be nationally representative of the 1997 housing stock.

The interim NSLAH summaries include imputed average dust-lead loading data values which are assigned to households when no such data are available for a given surface (floors, window sills). Assigning an imputed dust-lead loading average to a household that has no dust-lead loading data ensures that it (and its corresponding sampling weight representing a given portion of the national housing stock) is represented in the risk analysis. The method used to impute data closely follows the method used in the §403 risk analysis for housing units in the HUD National Survey; this method is detailed in Appendix C. This appendix also gives the imputed data values and how they were assigned to housing units. Summaries of the interim NSLAH dust-lead loading data with imputed data excluded are found in Appendix D1.

When using the interim NSLAH data to calculate a household's average dust-lead loading for floors or window sills, five different approaches were considered for handling individual sample results that fell below the instrument's detection limit. These five approaches, which include censoring the not-detected results, are presented in Appendix D1. The data summaries that exclude imputed data values, found in Appendix D1, were performed and presented for each of these five approaches. Of these five approaches, two were specifically identified as most likely to be applied in the supplemental risk analysis involving the interim NSLAH data:

- making no adjustment to not-detected data values, and
- replacing not-detected data values with one-half of the detection limit.

The first approach eliminates potential bias that can be introduced when an adjustment is made to a reported data value, but it also permits a household's average to be zero or below, preventing the data from being used as input to the empirical model within the §403 risk analysis. (As the survey's analytical method adjusted for potential analytical bias by subtracting a specified amount from a given sample result, reported results of less than zero were possible. Such results were included in the survey database used in this analysis.) The second approach prevents this problem from occurring and represents the best estimate of a sample's actual lead amount value when the analytical result is only known to fall somewhere between zero and the instrument's detection limit. Interim NSLAH data summaries under both approaches are presented in this section to illustrate the impact that any one approach has on the characterized distribution.

National comparisons

Tables 3-4 and 3-5 present descriptive statistics of average household dust-lead loadings for floors and window sills, respectively, for the 1997 national housing stock. These summaries

Table 3-4. Descriptive Statistics of Area-Weighted Average Floor Wipe Dust-Lead Loadings for Households, As Reported in the §403 Risk Analysis Versus the Interim NSLAH Data

Study	How Not-Detected and Negative Data were Handled	Area-Weighted Average Floor Dust-Lead Loading ($\mu g/ft^2$)[1]								
		# Surveyed Units with Positive Averages	Arith-metic Mean	Geo-metric Mean[2]	Geo-metric Std. Dev.[2]	Minimum	25th Percen-tile	Median	75th Percen-tile	Maximum
§403 Risk Analysis (HUD Natl. Survey)		284	16.5	6.27	3.49	0.508	2.65	5.32	12.2	375
Interim NSLAH[3]	No adjustment	633	10.4	1.22	4.57	-1.23	0.300	1.05	2.30	5940
	Replaced by LOD/2	706	10.8	1.82	2.78	0.750	0.950	1.31	2.46	5950

[1] All statistics are calculated by weighting each household by its sampling weight.
[2] Only household averages greater than zero are used to calculate this value (data for all units with floor dust-lead data are used to calculate the remaining statistics).
[3] Summaries include imputed data for households having no floor wipe dust-lead loading data. The method for imputation is presented in Appendix C.

Table 3-5. Descriptive Statistics of Area-Weighted Average Window Sill Wipe Dust-Lead Loadings for Households, As Reported in the §403 Risk Analysis Versus the Interim NSLAH Data

Study	How Not-Detected and Negative Data were Handled	Area-Weighted Average Window Sill Dust-Lead Loading ($\mu g/ft^2$)[1]								
		# Surveyed Units with Positive Averages	Arith-metic Mean	Geo-metric Mean[2]	Geo-metric Std. Dev.[2]	Minimum	25th Percen-tile	Median	75th Percen-tile	Maximum
§403 Risk Analysis (HUD Natl. Survey)		284	550	23.0	15.8	0.0118	4.35	19.5	198	43700
Interim NSLAH[3]	No adjustment	690	137	14.5	7.83	-9.43	2.90	12.8	51.3	11100
	Replaced by LOD/2	706	137	15.8	6.57	0.445	3.35	13.6	51.0	11100

[1] All statistics are calculated by weighting each household by its sampling weight.
[2] Only household averages greater than zero are used to calculate this value (data for all units with window sill dust-lead data are used to calculate the remaining statistics).
[3] Summaries include imputed data for households having no window sill wipe dust-lead loading data. The method for imputation is presented in Appendix C.

imply that the average dust-lead loadings for both floors and window sills based on the interim NSLAH data are considerably lower than that reported in the §403 risk analysis (based on the HUD National Survey after converting to wipe-equivalent loadings). For example, the median floor dust-lead loading is less than 2 $\mu g/ft^2$ based on the interim NSLAH data compared to 5.3 $\mu g/ft^2$ from the §403 risk analysis, and the median window sill dust-lead loading is less than 12 $\mu g/ft^2$ based on the interim NSLAH data compared to nearly 20 $\mu g/ft^2$ from the §403 risk analysis.

Median detection limits for dust-lead loadings in the interim NSLAH were 1.5 $\mu g/ft^2$ for floors and 3.6 $\mu g/ft^2$ for window sills. When considering all dust samples in the interim NSLAH that had lead amounts reported, approximately two-thirds of the floor dust-lead samples and one-third of the window sill dust-lead samples had results below the detection limit.

Boxplots of the data distributions presented in Tables 3-4 and 3-5 are found in Figures 3-1 and 3-2, respectively. Appendix D1 contains these tabular summaries and boxplots after excluding imputed data values.

In addition to these data summaries that are based solely on the observed data and the sampling weights, it was desired to characterize the national distribution of household average floor dust-lead loading in such a way that the percentage of housing where this average exceeds a specified threshold could be estimated. This was done for both the HUD National Survey and interim NSLAH data by assuming that these data originate from a lognormal distribution. Then, the fitted distributions and corresponding estimated exceedance percentages were compared between the two surveys. These results are presented in Section 3.2.1.3 below.

Comparisons by housing age category

While the summaries in Tables 3-4 and 3-5 represent the entire nation, Tables 3-6 and 3-7 present descriptive statistics according to the housing age category scheme defined in Table 3-2 above. Considerable declines in the geometric means and medians from the §403 risk analysis to the interim NSLAH data were observed in all four age categories.

Boxplots of the data distributions presented in Tables 3-6 and 3-7 are found in Figures 3-3 and 3-4, respectively. Appendix D1 contains these tabular summaries and boxplots after excluding imputed data values.

Comparisons by Census region

Tables 3-8 and 3-9 present descriptive statistics according to Census region. Declines in the geometric means and medians were observed from the §403 risk analysis to the interim NSLAH data for all regions but the West region, where very slight increases in these estimates were observed. The greatest declines were observed in the Northeast and Midwest.

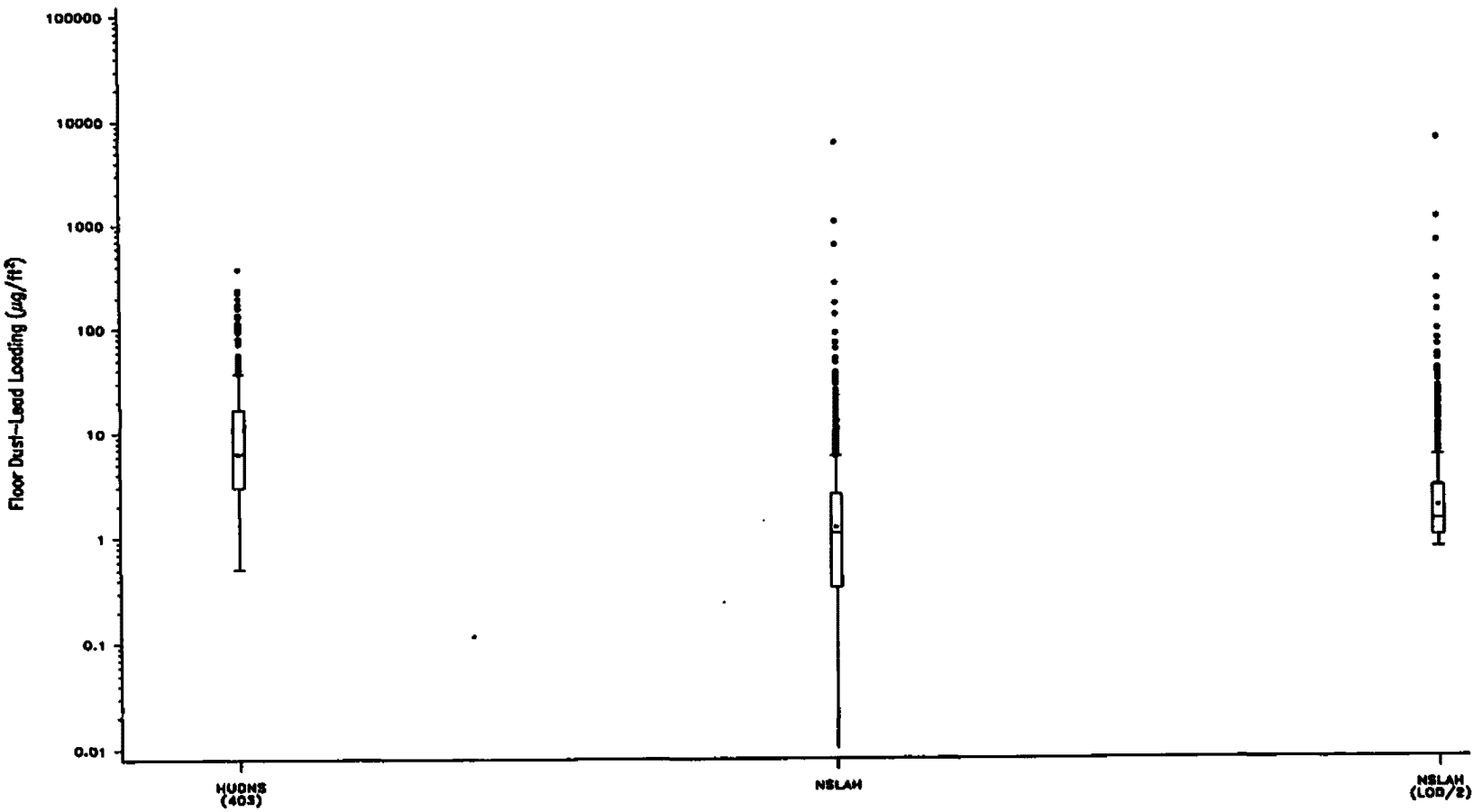

Figure 3-1. Boxplots of Area-Weighted Average Floor Wipe Dust-Lead Loadings (µg/ft²) As Observed in the §403 Risk Analysis (Using HUD National Survey Data) and in the Interim NSLAH (under 2 approaches to handling not-detected values)

(Note: Dust-lead loadings from the HUD National Survey have been converted to wipe-equivalents in the §403 risk analysis using the methods documented in the §403 risk analysis report. Boxplots include imputed household averages but not negative averages. See text for definitions of labels along the horizontal axis.)

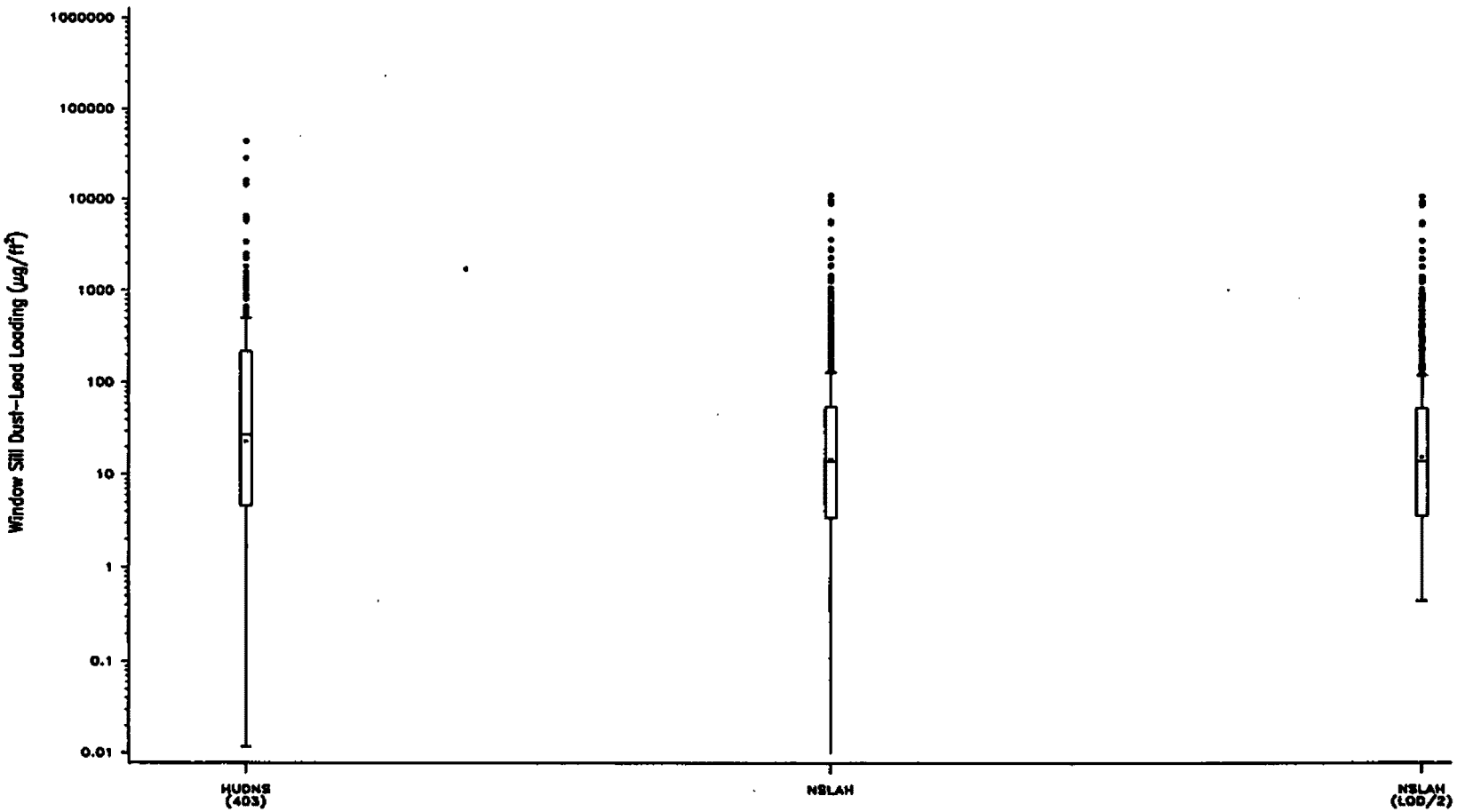

Figure 3-2. Boxplots of Area-Weighted Average Window Sill Wipe Dust-Lead Loadings (µg/ft²) As Observed in the §403 Risk Analysis (Using HUD National Survey Data) and in the Interim NSLAH (under 2 approaches to handling not-detected values)

(Note: Dust-lead loadings from the HUD National Survey have been converted to wipe-equivalents in the §403 risk analysis using the methods documented in the §403 risk analysis report. Boxplots include imputed household averages but not negative averages. See text for definitions of labels along the horizontal axis.)

Table 3-6. Descriptive Statistics of Area-Weighted Average Floor Wipe Dust-Lead Loadings for Households, Presented by Housing Age Category, As Reported in the §403 Risk Analysis Versus the Interim NSLAH Data

Study	How Not-Detected and Negative Data were Handled	Area-Weighted Average Floor Dust-Lead Loading ($\mu g/ft^2$)[1]								
		# Surveyed Units with Positive Averages	Arith-metic Mean	Geo-metric Mean[2]	Geo-metric Std. Dev.[2]	Minimum	25th Percen-tile	Median	75th Percen-tile	Maximum
Units Built Prior to 1940										
§403 Risk Analysis (HUD Natl. Survey)		77	47.9	22.6	3.63	0.991	8.84	17.7	79.7	375
Interim NSLAH[3]	No adjustment	111	36.9	3.74	4.53	-0.600	1.30	2.42	9.50	5940
	Replaced by LOD/2	114	37.0	4.00	3.97	0.750	1.45	2.71	9.50	5950
Units Built from 1940 - 1959										
§403 Risk Analysis (HUD Natl. Survey)		87	18.1	8.74	3.34	0.508	4.07	7.81	22.4	171
Interim NSLAH[3]	No adjustment	134	4.11	1.90	3.57	-0.720	0.719	1.80	4.00	71.0
	Replaced by LOD/2	145	4.38	2.31	2.64	0.750	1.05	1.99	4.00	71.0
Units Built from 1960-1977 (1960 - 1979 for the §403 risk analysis)										
§403 Risk Analysis (HUD Natl. Survey)		120	6.74	4.14	2.45	0.657	2.25	3.62	7.59	106
Interim NSLAH[3]	No adjustment	176	1.50	0.912	3.47	-0.733	0.236	0.900	1.68	28.5
	Replaced by LOD/2	201	1.96	1.46	1.92	0.750	0.900	1.20	1.92	28.8
Units Built After 1977 (after 1979 for the §403 risk analysis)										
§403 Risk Analysis (HUD Natl. Survey)		28	4.16	3.14	2.06	1.06	1.76	2.84	5.66	12.9
Interim NSLAH[3]	No adjustment	151	1.20	0.545	3.35	-1.05	0.146	0.400	1.08	265
	Replaced by LOD/2	180	1.71	1.14	1.72	0.750	0.750	1.00	1.35	265
NSLAH Units with Unspecified Year-Built Indicator										
Interim NSLAH[3]	No adjustment	61	31.7	1.37	6.64	-1.23	0.300	1.24	2.72	1040
	Replaced by LOD/2	66	32.1	2.20	3.92	0.750	1.00	1.40	2.56	1040

[1] All statistics are calculated by weighting each household by its sampling weight.

[2] Only household averages greater than zero are used to calculate this value (data for all units with floor dust-lead data are used to calculate the remaining statistics).

[3] Summaries include imputed data for households having no floor wipe dust-lead loading data. The method for imputation is presented in Appendix C.

Table 3-7. Descriptive Statistics of Area-Weighted Average Window Sill Wipe Dust-Lead Loadings for Households, Presented by Housing Age Category, As Reported in the §403 Risk Analysis Versus the Interim NSLAH Data

Study	How Not-Detected and Negative Data were Handled	Area-Weighted Average Window Sill Dust-Lead Loading ($\mu g/ft^2$)[1]								
		# Surveyed Units with Positive Averages	Arithmetic Mean	Geometric Mean[2]	Geometric Std. Dev.[2]	Minimum	25th Percentile	Median	75th Percentile	Maximum
Units Built Prior to 1940										
§403 Risk Analysis (HUD Natl. Survey)		77	2060	168	16.7	0.0155	35.6	198	1220	43700
Interim NSLAH[3]	No adjustment	113	400	77.5	6.59	-0.152	21.2	79.8	294	11100
	Replaced by LOD/2	114	400	76.8	6.44	1.03	21.2	79.8	294	11100
Units Built from 1940 - 1959										
§403 Risk Analysis (HUD Natl. Survey)		87	285	22.0	10.7	0.0118	6.47	19.1	107	16100
Interim NSLAH[3]	No adjustment	144	129	24.5	6.80	-1.73	6.35	23.0	88.6	3630
	Replaced by LOD/2	145	129	26.1	5.97	0.923	6.58	22.0	88.6	3630
Units Built from 1960-1977 (1960 - 1979 for the §403 risk analysis)										
§403 Risk Analysis (HUD Natl. Survey)		120	184	16.2	14.6	0.0164	2.05	16.6	217	5790
Interim NSLAH[3]	No adjustment	195	36.6	10.7	4.71	-2.32	2.89	9.40	29.0	1390
	Replaced by LOD/2	201	36.9	11.3	4.18	1.02	3.17	9.54	29.3	1390
Units Built After 1977 (after 1979 for the §403 risk analysis)										
§403 Risk Analysis (HUD Natl. Survey)		28	83.0	8.17	9.94	0.0164	2.58	8.11	57.8	1590
Interim NSLAH[3]	No adjustment	174	15.6	3.56	5.27	-9.43	0.916	3.19	10.3	426
	Replaced by LOD/2	180	16.0	4.57	3.79	0.445	1.72	3.67	9.99	427
NSLAH Units with Unspecified Year-Built Indicator										
Interim NSLAH[3]	No adjustment	64	367	39.8	7.32	-0.629	18.6	36.4	118	9030
	Replaced by LOD/2	66	367	40.2	6.72	0.720	18.8	36.4	118	9030

[1] All statistics are calculated by weighting each household by its sampling weight.

[2] Only household averages greater than zero are used to calculate this value (data for all units with window sill dust-lead data are used to calculate the remaining statistics).

[3] Summaries include imputed data for households having no window sill wipe dust-lead loading data. The method for imputation is presented in Appendix C.

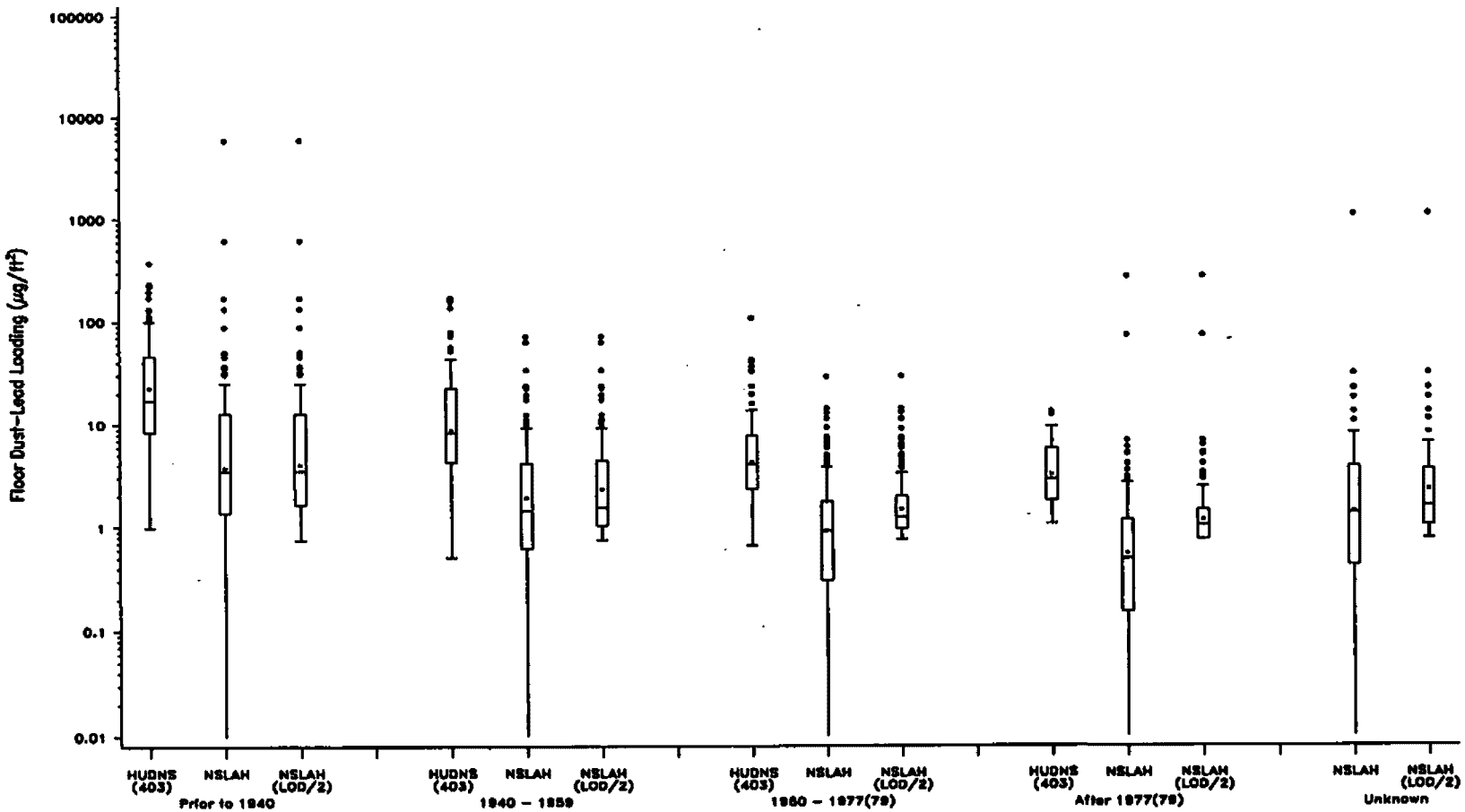

Figure 3-3. Boxplots of Area-Weighted Average Floor Wipe Dust-Lead Loadings (µg/ft²), by Housing Age Category, As Observed in the §403 Risk Analysis (Using HUD National Survey Data) and in the Interim NSLAH (under 2 approaches to handling not-detected values)

(Note: Dust-lead loadings from the HUD National Survey have been converted to wipe-equivalents in the §403 risk analysis using the methods documented in the §403 risk analysis report. Boxplots include imputed household averages but not negative averages. See text for definitions of labels along the horizontal axis.)

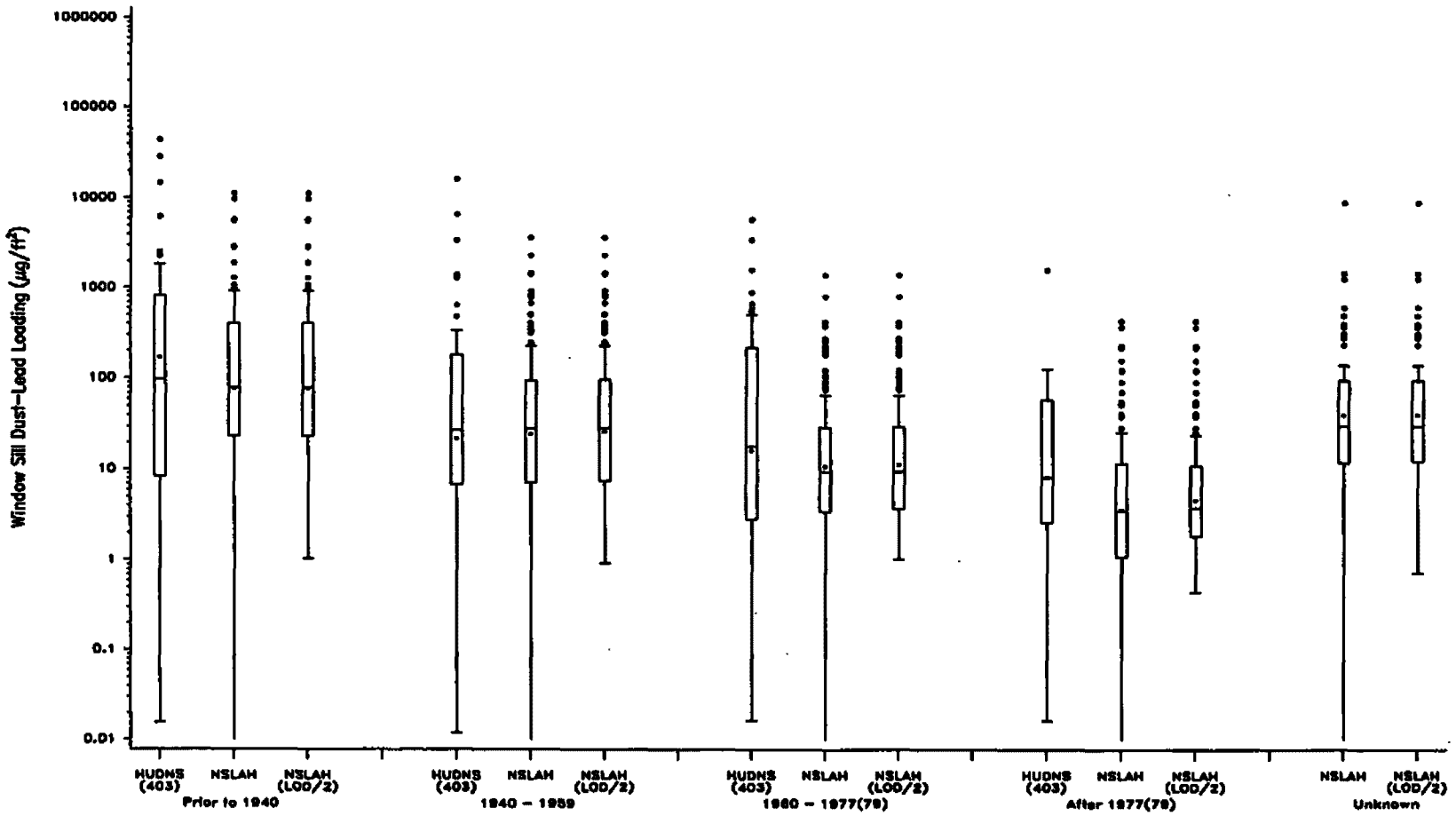

Figure 3-4. Boxplots of Area-Weighted Average Window Sill Wipe Dust-Lead Loadings (µg/ft²), by Housing Age Category, As Observed in the §403 Risk Analysis (Using HUD National Survey Data) and in the Interim NSLAH (under 2 approaches to handling not-detected values)

(Note: Dust-lead loadings from the HUD National Survey have been converted to wipe-equivalents in the §403 risk analysis using the methods documented in the §403 risk analysis report. Boxplots include imputed household averages but not negative averages. See text for definitions of labels along the horizontal axis.)

Table 3-8. Descriptive Statistics of Area-Weighted Average Floor Wipe Dust-Lead Loadings for Households, Presented by Census Region, As Reported in the §403 Risk Analysis Versus the Interim NSLAH Data

Study	How Not-Detected and Negative Data were Handled	Area-Weighted Average Floor Dust-Lead Loading ($\mu g/ft^2$)[1]								
		# Surveyed Units with Positive Averages	Arithmetic Mean	Geometric Mean[2]	Geometric Std. Dev.[2]	Minimum	25th Percentile	Median	75th Percentile	Maximum
Northeast										
§403 Risk Analysis (HUD Natl. Survey)		53	35.6	14.9	3.95	0.632	4.79	11.0	76.3	375
Interim NSLAH[3]	No adjustment	103	10.0	2.28	4.42	-0.620	0.800	1.90	6.00	617
	Replaced by LOD/2	109	10.3	2.90	3.15	0.750	1.20	2.13	6.00	617
Midwest										
§403 Risk Analysis (HUD Natl. Survey)		73	14.7	6.32	3.26	0.508	2.83	6.32	11.0	173
Interim NSLAH[3]	No adjustment	136	14.7	1.34	5.81	-0.733	0.283	1.20	2.48	1040
	Replaced by LOD/2	150	15.0	2.04	3.39	0.750	0.760	1.29	3.25	1040
South										
§403 Risk Analysis (HUD Natl. Survey)		134	13.3	5.01	3.28	0.735	2.00	3.89	10.0	236
Interim NSLAH[3]	No adjustment	235	2.65	0.981	3.94	-1.05	0.254	0.940	1.76	265
	Replaced by LOD/2	265	3.07	1.55	2.25	0.750	0.970	1.21	1.94	265
West										
§403 Risk Analysis (HUD Natl. Survey)		52	9.81	4.97	2.75	1.06	2.65	4.01	8.43	197
Interim NSLAH[3]	No adjustment	159	18.7	0.949	3.66	-1.23	0.255	0.800	1.67	5940
	Replaced by LOD/2	182	19.1	1.46	2.31	0.750	0.800	1.20	1.88	5950

[1] All statistics are calculated by weighting each household by its sampling weight.

[2] Only household averages greater than zero are used to calculate this value (data for all units with floor dust-lead data are used to calculate the remaining statistics).

[3] Summaries include imputed data for households having no floor wipe dust-lead loading data. The method for imputation is presented in Appendix C.

Table 3-9. Descriptive Statistics of Area-Weighted Average Window Sill Wipe Dust-Lead Loadings for Households, Presented by Census Region, As Reported in the §403 Risk Analysis Versus the Interim NSLAH Data

Study	How Not-Detected and Negative Data were Handled	Area-Weighted Average Window Sill Dust-Lead Loading ($\mu g/ft^2$)[1]								
		# Surveyed Units with Positive Averages	Arith-metic Mean	Geo-metric Mean[2]	Geo-metric Std. Dev.[2]	Minimum	25th Percen-tile	Median	75th Percen-tile	Maximum
Northeast										
§403 Risk Analysis (HUD Natl. Survey)		53	1440	92.2	16.1	0.0155	15.3	173	335	14600
Interim NSLAH[3]	No adjustment	107	172	21.5	8.01	-1.89	5.94	16.0	89.5	5530
	Replaced by LOD/2	109	172	22.6	7.06	0.578	5.94	16.0	90.0	5530
Midwest										
§403 Risk Analysis (HUD Natl. Survey)		73	564	48.5	13.2	0.0706	7.76	83.0	309	43700
Interim NSLAH[3]	No adjustment	145	218	21.0	7.25	-2.32	4.00	16.6	60.1	9630
	Replaced by LOD/2	150	218	21.6	6.49	1.12	4.75	16.4	60.1	9630
South										
§403 Risk Analysis (HUD Natl. Survey)		134	432	19.6	12.4	0.118	4.60	15.0	127	28400
Interim NSLAH[3]	No adjustment	259	115	13.8	8.11	-9.43	2.88	12.8	53.8	11100
	Replaced by LOD/2	265	116	15.6	6.42	0.646	3.06	13.9	53.8	11100
West										
§403 Risk Analysis (HUD Natl. Survey)		52	62.2	4.45	12.7	0.0118	1.68	5.40	28.0	1400
Interim NSLAH[3]	No adjustment	179	54.3	7.73	6.65	-0.115	2.07	7.54	29.0	3630
	Replaced by LOD/2	182	54.4	8.72	5.59	0.445	2.30	7.76	29.3	3630

[1] All statistics are calculated by weighting each household by its sampling weight.

[2] Only household averages greater than zero are used to calculate this value (data for all units with window sill dust-lead data are used to calculate the remaining statistics).

[3] Summaries include imputed data for households having no window sill wipe dust-lead loading data. The method for imputation is presented in Appendix C.

Boxplots of the data distributions presented in Tables 3-8 and 3-9 are found in Figures 3-5 and 3-6, respectively. Appendix D1 contains these tabular summaries and boxplots after excluding imputed data values.

Comparisons by combination of housing age and Census region

Tables 3-10a and 3-10b present descriptive statistics for household average floor dust-lead loadings according to the 16 combinations of Census region and housing age category. Table 3-10a considers no adjustment to the interim NSLAH data when not-detected results were observed, while Table 3-10b summarizes data where not-detected data were replaced by one-half of the detection limit. Tables 3-11a and 3-11b present the same descriptive statistics for household average window sill dust-lead loadings. As the central tendency of the dust-lead loading data was of primary interest to compare across the different combinations, these tables only contain estimates of the arithmetic and geometric means, geometric standard deviation (GSD), and median. Appendix D1 contains these tabular summaries after excluding imputed data values.

Due to the small number of housing units within certain combinations, caution is warranted when making inferences based on the numbers in these tables.

3.2.1.2. Data Summaries for the §403 Risk Analysis Versus Three Other Studies. This subsection provides descriptive statistics of household average dust-lead loadings for floors and window sills for the HUD National Survey (both as collected and as used in the §403 risk analysis), comparing these summaries to those for the three studies identified in the introduction to this section that provided the most useful and available information to the §403 risk analysis on the relationship between environmental-lead levels and childhood blood-lead concentration: the Baltimore R&M study, the Rochester Lead-in-Dust study, and the ongoing HUD Grantees evaluation (data collected through February 1999).

Summaries of the reported dust-lead loadings in the HUD National Survey and the Baltimore R&M study were performed on wipe-equivalent dust-lead loadings using conversion methods presented in the §403 risk analysis report. In addition, the household averages based on HUD National Survey data were summarized in two different ways: by ignoring the sample weights assigned to the surveyed housing units and any imputed data for households with missing data, and by handling the data as used in the §403 risk analysis (described earlier in this section).

Because the HUD Grantees program emphasizes local control of the individual programs, each grantee participating in the HUD Grantee evaluation is responsible for designing and implementing lead-hazard reduction approaches applicable to its specific needs and objectives. These responsibilities include the recruitment methods, enrollment criteria, and intervention strategies. However, to enable comparison of results from the various approaches, grantees participating in the evaluation follow the same sampling protocols and use standard data collection forms developed specifically for this evaluation. Table 3-4 of the §403 risk analysis

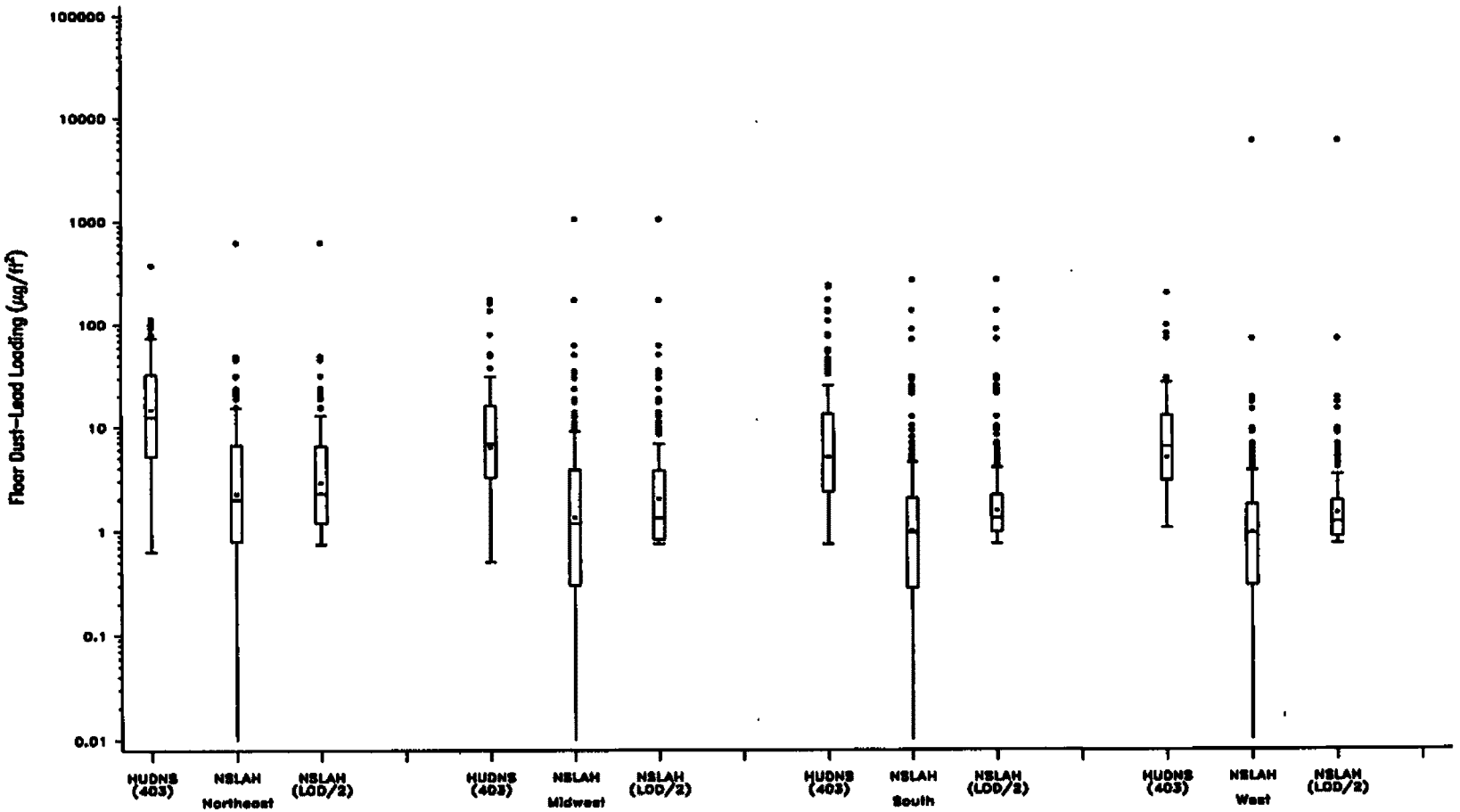

Figure 3-5. Boxplots of Area-Weighted Average Floor Wipe Dust-Lead Loadings (µg/ft²), by Census Region, As Observed in the §403 Risk Analysis (Using HUD National Survey Data) and in the Interim NSLAH (under 2 approaches to handling not-detected values)

(Note: Dust-lead loadings from the HUD National Survey have been converted to wipe-equivalents in the §403 risk analysis using the methods documented in the §403 risk analysis report. Boxplots include imputed household averages but not negative averages. See text for definitions of labels along the horizontal axis.)

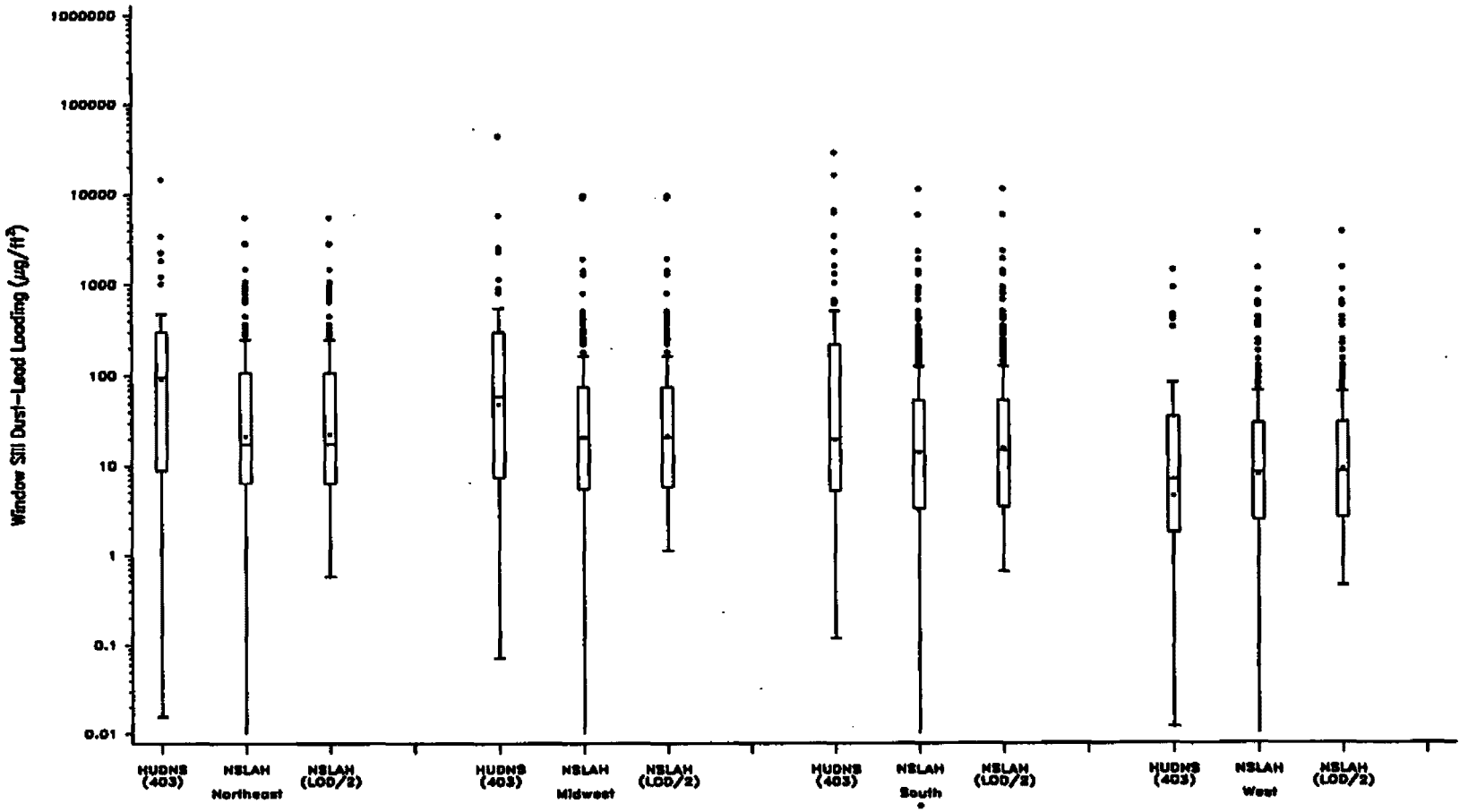

Figure 3-6. Boxplots of Area-Weighted Average Window Sill Wipe Dust-Lead Loadings (µg/ft²), by Census Region, As Observed in the §403 Risk Analysis (Using HUD National Survey Data) and in the Interim NSLAH (under 2 approaches to handling not-detected values)

(Note: Dust-lead loadings from the HUD National Survey have been converted to wipe-equivalents in the §403 risk analysis using the methods documented in the §403 risk analysis report. Boxplots include imputed household averages but not negative averages. See text for definitions of labels along the horizontal axis.)

Table 3-10a. Descriptive Statistics of Area-Weighted Average Floor Wipe Dust-Lead Loadings for Households, Presented by Housing Age and Census Region, As Reported in the §403 Risk Analysis Versus the Interim NSLAH Data Where No Adjustments Were Made to Not-Detected Results

Census Region	Study	Housing Age Category	Area-Weighted Average Floor Dust-Lead Loading ($\mu g/ft^2$)				
			# Surveyed Units	Arithmetic Mean	Geometric Mean	Geometric Std. Dev.	Median
Northeast	§403 Risk Anal.	Prior to 1940	26	63.5	36.5	3.39	76.3
	Interim NSLAH		41	23.7	5.02	4.31	4.20
	§403 Risk Anal.	1940 - 1959	17	13.2	8.84	2.54	7.81
	Interim NSLAH		21	3.75	2.37	3.36	2.38
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	10	7.00	4.73	2.23	4.76
	Interim NSLAH		19	3.34	1.72	3.76	1.46
	Interim NSLAH	After 1977	15	1.12	0.714	2.78	0.867
Midwest	§403 Risk Anal.	Prior to 1940	19	31.3	14.7	3.01	8.94
	Interim NSLAH		33	8.49	2.62	4.47	2.16
	§403 Risk Anal.	1940 - 1959	21	15.8	6.69	3.95	5.79
	Interim NSLAH		35	5.48	2.05	4.16	1.59
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	29	6.33	4.58	2.35	4.44
	Interim NSLAH		32	1.52	0.737	4.77	1.12
	§403 Risk Anal.	After 1977 (1979 for §403)	4	3.32	2.77	1.83	2.80
	Interim NSLAH		25	0.913	0.545	3.86	0.320
South	§403 Risk Anal.	Prior to 1940	19	50.7	20.8	4.01	19.0
	Interim NSLAH		26	11.0	3.66	3.93	2.74
	§403 Risk Anal.	1940 - 1959	33	25.4	10.3	3.91	10.0
	Interim NSLAH		42	3.66	1.63	3.40	1.77
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	64	8.06	4.13	2.74	3.39
	Interim NSLAH		71	1.16	0.825	3.04	0.880
	§403 Risk Anal.	After 1977 (1979 for §403)	18	4.19	3.16	2.05	2.84
	Interim NSLAH		72	1.04	0.549	3.12	0.480
West	§403 Risk Anal.	Prior to 1940	13	34.9	16.2	3.51	17.2
	Interim NSLAH		11	264	3.84	6.17	2.30
	§403 Risk Anal.	1940 - 1959	16	14.6	9.04	2.46	7.47
	Interim NSLAH		36	2.86	1.70	2.92	1.36
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	17	4.50	3.53	2.03	3.35
	Interim NSLAH		54	1.16	0.949	2.42	0.990
	§403 Risk Anal.	After 1977 (1979 for §403)	6	4.60	3.36	2.21	3.00
	Interim NSLAH		39	1.75	0.454	3.67	0.270

Note: Summaries include imputed data for households having no floor wipe dust-lead loading data. The method for imputation is presented in Appendix C.

Table 3-10b. Descriptive Statistics of Area-Weighted Average Floor Wipe Dust-Lead Loadings for Households, Presented by Housing Age and Census Region, As Reported in the §403 Risk Analysis Versus the Interim NSLAH Data Where Not-Detected Results Were Replaced by LOD/2

Census Region	Study	Housing Age Category	Area-Weighted Average Floor Dust-Lead Loading ($\mu g/ft^2$)				
			# Surveyed Units	Arithmetic Mean	Geometric Mean	Geometric Std. Dev.	Median
Northeast	§403 Risk Anal.	Prior to 1940	26	63.5	36.5	3.39	76.3
	Interim NSLAH		41	23.8	5.47	3.91	4.35
	§403 Risk Anal.	1940 - 1959	17	13.2	8.84	2.54	7.81
	Interim NSLAH		23	4.03	2.86	2.23	2.40
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	10	7.00	4.73	2.23	4.76
	Interim NSLAH		21	3.58	2.16	2.60	1.68
	Interim NSLAH	After 1977	16	1.68	1.43	1.72	1.29
Midwest	§403 Risk Anal.	Prior to 1940	19	31.3	14.7	3.01	8.94
	Interim NSLAH		36	8.79	2.88	3.41	2.19
	§403 Risk Anal.	1940 - 1959	21	15.8	6.69	3.95	5.79
	Interim NSLAH		36	5.80	2.57	3.20	1.53
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	29	6.33	4.58	2.35	4.44
	Interim NSLAH		37	2.00	1.50	2.03	1.20
	§403 Risk Anal.	After 1977 (1979 for §403)	4	3.32	2.77	1.83	2.80
	Interim NSLAH		30	1.31	1.09	1.67	0.938
South	§403 Risk Anal.	Prior to 1940	19	50.7	20.8	4.01	19.0
	Interim NSLAH		26	11.1	3.87	3.76	2.70
	§403 Risk Anal.	1940 - 1959	33	25.4	10.3	3.91	10.0
	Interim NSLAH		48	3.94	1.99	2.35	1.54
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	64	8.06	4.13	2.74	3.39
	Interim NSLAH		81	1.67	1.31	1.73	1.18
	§403 Risk Anal.	After 1977 (1979 for §403)	18	4.19	3.16	2.05	2.84
	Interim NSLAH		84	1.54	1.13	1.57	1.06
West	§403 Risk Anal.	Prior to 1940	13	34.9	16.2	3.51	17.2
	Interim NSLAH		11	264	4.03	5.91	2.19
	§403 Risk Anal.	1940 - 1959	16	14.6	9.04	2.46	7.47
	Interim NSLAH		38	3.07	1.99	2.34	1.52
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	17	4.50	3.53	2.03	3.35
	Interim NSLAH		62	1.62	1.40	1.65	1.38
	§403 Risk Anal.	After 1977 (1979 for §403)	6	4.60	3.36	2.21	3.00
	Interim NSLAH		50	2.34	1.07	1.95	0.900

Note: Summaries include imputed data for households having no floor wipe dust-lead loading data. The method for imputation is presented in Appendix C.

Table 3-11a. Descriptive Statistics of Area-Weighted Average Window Sill Wipe Dust-Lead Loadings for Households, Presented by Housing Age and Census Region, As Reported in the §403 Risk Analysis Versus the Interim NSLAH Data Where No Adjustments Were Made to Not-Detected Results

Census Region	Study	Housing Age Category	Area-Weighted Average Window Sill Dust-Lead Loading ($\mu g/ft^2$)				
			# Surveyed Units	Arithmetic Mean	Geometric Mean	Geometric Std. Dev.	Median
Northeast	§403 Risk Anal.	Prior to 1940	26	2700	265	15.8	176
	Interim NSLAH		40	396	99.4	6.33	91.7
	§403 Risk Anal.	1940 - 1959	17	98.5	32.6	5.55	50.7
	Interim NSLAH		23	62.7	20.1	4.31	18.5
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	10	499	38.9	20.8	217
	Interim NSLAH		20	13.9	7.88	2.67	6.49
	Interim NSLAH	After 1977	16	18.3	3.28	5.69	2.06
Midwest	§403 Risk Anal.	Prior to 1940	19	1660	435	5.79	542
	Interim NSLAH		36	361	72.5	6.15	67.3
	§403 Risk Anal.	1940 - 1959	21	98.2	17.7	11.6	17.4
	Interim NSLAH		35	103	20.0	6.33	17.1
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	29	223	20.9	11.6	48.3
	Interim NSLAH		33	27.9	9.94	4.75	9.54
	§403 Risk Anal.	After 1977 (1979 for §403)	4	62.5	27.5	6.78	83.0
	Interim NSLAH		30	21.0	6.57	3.64	5.86
South	§403 Risk Anal.	Prior to 1940	19	2450	64.0	23.1	24.4
	Interim NSLAH		26	600	112	5.87	115
	§403 Risk Anal.	1940 - 1959	33	657	38.9	9.93	26.2
	Interim NSLAH		48	160	30.7	8.58	32.0
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	64	149	24.0	12.6	32.0
	Interim NSLAH		80	55.4	14.3	5.44	15.4
	§403 Risk Anal.	After 1977 (1979 for §403)	18	112	9.09	8.60	7.58
	Interim NSLAH		80	18.2	3.93	6.00	3.89
West	§403 Risk Anal.	Prior to 1940	13	125	11.5	14.7	7.05
	Interim NSLAH		11	47.6	14.2	5.17	17.1
	§403 Risk Anal.	1940 - 1959	16	107	7.35	13.2	6.96
	Interim NSLAH		38	186	29.0	7.21	33.8
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	17	58.7	3.83	11.5	4.35
	Interim NSLAH		62	26.1	8.34	4.19	7.51
	§403 Risk Anal.	After 1977 (1979 for §403)	6	9.66	2.65	11.6	5.94
	Interim NSLAH		48	5.64	1.99	4.08	1.63

Note: Summaries include imputed data for households having no window sill wipe dust-lead loading data. The method for imputation is presented in Appendix C.

Table 3-11b. Descriptive Statistics of Area-Weighted Average Window Sill Wipe Dust-Lead Loadings for Households, Presented by Housing Age and Census Region, As Reported in the §403 Risk Analysis Versus the Interim NSLAH Data Where Not-Detected Results Were Replaced by LOD/2

Census Region	Study	Housing Age Category	Area-Weighted Average Window Sill Dust-Lead Loading ($\mu g/ft^2$)				
			# Surveyed Units	Arithmetic Mean	Geometric Mean	Geometric Std. Dev.	Median
Northeast	§403 Risk Anal.	Prior to 1940	26	2700	265	15.8	176
	Interim NSLAH		41	396	90.1	6.91	91.7
	§403 Risk Anal.	1940 - 1959	17	98.5	32.6	5.55	50.7
	Interim NSLAH		23	62.7	19.6	4.49	18.9
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	10	499	38.9	20.8	217
	Interim NSLAH		21	14.7	8.39	2.55	7.37
	Interim NSLAH	After 1977	16	18.6	4.80	3.80	3.73
Midwest	§403 Risk Anal.	Prior to 1940	19	1660	435	5.79	542
	Interim NSLAH		36	361	75.7	5.65	67.3
	§403 Risk Anal.	1940 - 1959	21	98.2	17.7	11.6	17.4
	Interim NSLAH		36	103	20.9	5.49	17.6
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	29	223	20.9	11.6	48.3
	Interim NSLAH		37	28.4	10.3	3.81	9.54
	§403 Risk Anal.	After 1977 (1979 for §403)	4	62.5	27.5	6.78	83.0
	Interim NSLAH		30	21.4	7.01	3.54	6.20
South	§403 Risk Anal.	Prior to 1940	19	2450	64.0	23.1	24.4
	Interim NSLAH		26	600	112	5.86	115
	§403 Risk Anal.	1940 - 1959	33	657	38.9	9.93	26.2
	Interim NSLAH		48	160	35.5	6.78	32.0
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	64	149	24.0	12.6	32.0
	Interim NSLAH		81	55.7	15.3	4.88	15.8
	§403 Risk Anal.	After 1977 (1979 for §403)	18	112	9.09	8.60	7.58
	Interim NSLAH		84	18.8	5.21	3.86	4.00
West	§403 Risk Anal.	Prior to 1940	13	125	11.5	14.7	7.05
	Interim NSLAH		11	47.8	15.9	4.23	17.2
	§403 Risk Anal.	1940 - 1959	16	107	7.35	13.2	6.96
	Interim NSLAH		38	186	30.6	6.51	33.8
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	17	58.7	3.83	11.5	4.35
	Interim NSLAH		62	26.0	8.77	3.88	7.51
	§403 Risk Anal.	After 1977 (1979 for §403)	6	9.66	2.65	11.6	5.94
	Interim NSLAH		50	5.77	2.57	3.14	1.85

Note: Summaries include imputed data for households having no window sill wipe dust-lead loading data. The method for imputation is presented in Appendix C.

report documented the differences between grantees in their enrollment/recruitment criteria. As a result, the HUD Grantees data summaries in this subsection are presented by grantee.

Overall data summaries

Figures 3-7 and 3-8 present boxplots of the area-weighted household average dust-lead loadings for floors and window sills, respectively. Each of these two figures contains a boxplot for each study, along with separate boxplots for each grantee in the HUD Grantees evaluation[8]. Each figure also includes three boxplots associated with the HUD National Survey data:

- "HUDNS (U)" summarizes the data without regard to sampling weights
- "HUDNS (403)" summarizes the data as used in the §403 risk analysis (e.g., using sampling weights reflecting the 1997 housing stock; incorporating imputed data assigned to housing units with missing data)
- "HUDNS (OW)" summarizes the data weighted according to the original weights assigned in the survey.

Tables 3-12 and 3-13 present values of the statistics presented in the boxplots (geometric mean, minimum, median, maximum, 25th and 75th percentiles), along with other important information not explicitly observable from the boxplots (number of houses whose data enter into these statistics, geometric standard deviation) that is necessary when comparing distributions across studies. The GSD reported for the overall HUD Grantees evaluation is the exponentiation of the square root of the weighted average of log-transformed variances for the different grantees, where the weights correspond to the numbers of units with data.

Comparisons by housing age category

Figures 3-9 and 3-10 contain boxplots on pre-1980 housing data (floors and window sills, respectively) from the HUD National Survey, Baltimore R&M, and Rochester studies, and pre-1978 data from the HUD Grantees evaluation (data combined across grantees) according to three housing age categories (pre-1940, 1940-1959, 1960-1977/79). As in the overall summaries above, the HUD National Survey data are presented within three boxplots for each age category. Caution is warranted when interpreting results in these figures for the Rochester study, as the actual age of certain houses may be older than what was specified in the Rochester study database (see Section 3.3.1.3 of the §403 risk analysis report). Also for this reason, and since the other studies surveyed few, if any, post-1979 homes, boxplots were not created for homes built after 1979. Boxplots for non-control houses in the Baltimore R&M study, all of which were built prior to 1941, are also included in these figures and are displayed in the "pre-1940" category.

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[8] "Alam"=Alameda County; "Balt"=Baltimore; "Bos"=Boston; "CA"=California; "Cle"=Cleveland; "MA"=Massachusetts; "MN"=Minnesota; "NJ"=New Jersey; "RI"=Rhode Island; "WI"=Wisconsin; "Milw"=Milwaukee; "Chic"=Chicago; "NYC"=New York City; "VT"=Vermont.

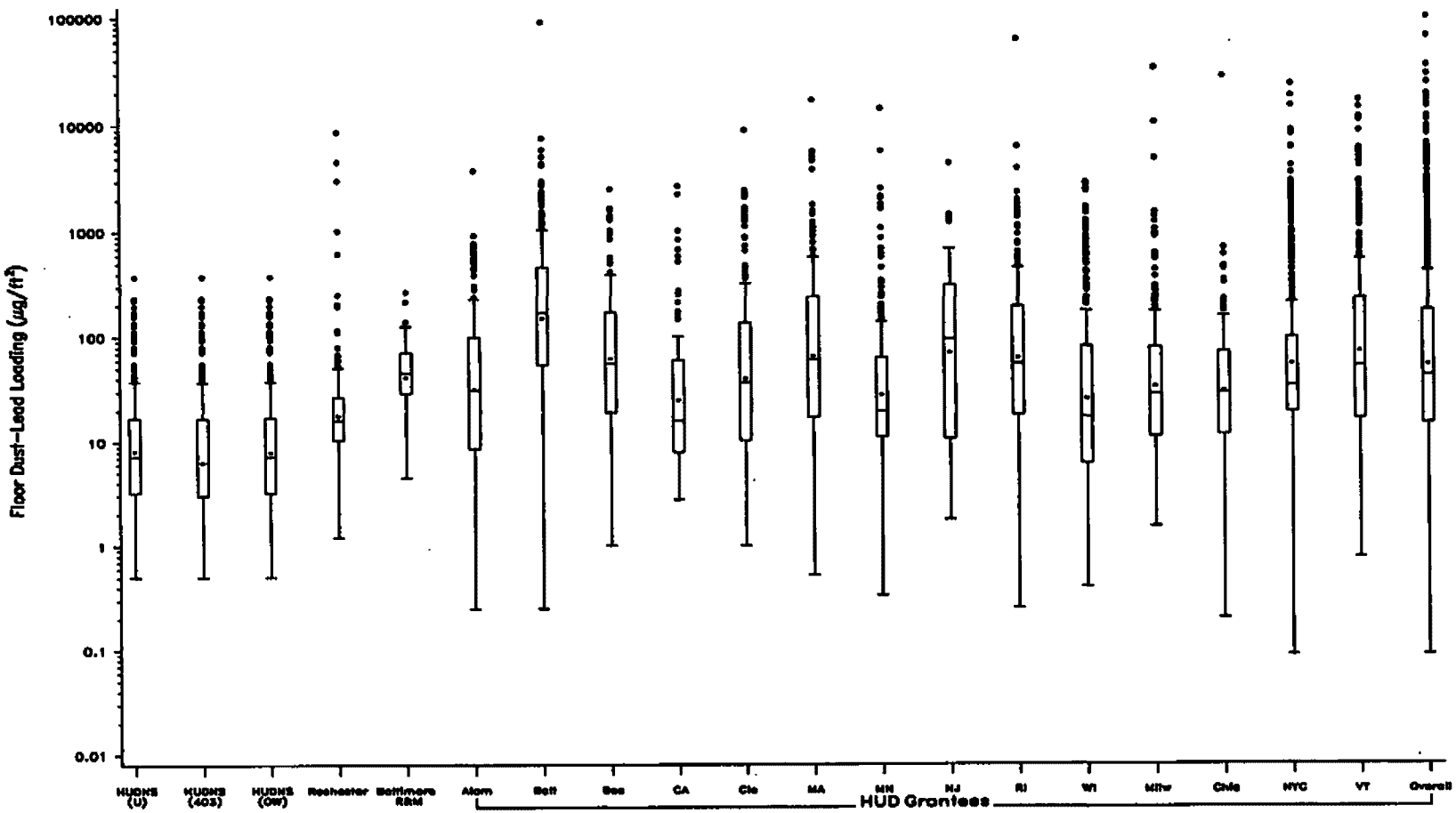

Figure 3-7. Boxplots of Area-Weighted Average Pre-Intervention Floor Wipe Dust-Lead Loadings (µg/ft²) for Houses in the HUD National Survey, Baltimore R&M Study, Rochester Lead-in-Dust Study, and Grantees Within the HUD Grantees Evaluation

(Note: Dust-lead loadings from the HUD National Survey and Baltimore R&M study have been converted from vacuum to wipe-equivalents using the methods documented in the §403 risk analysis report. See text for definitions of labels along the horizontal axis.)

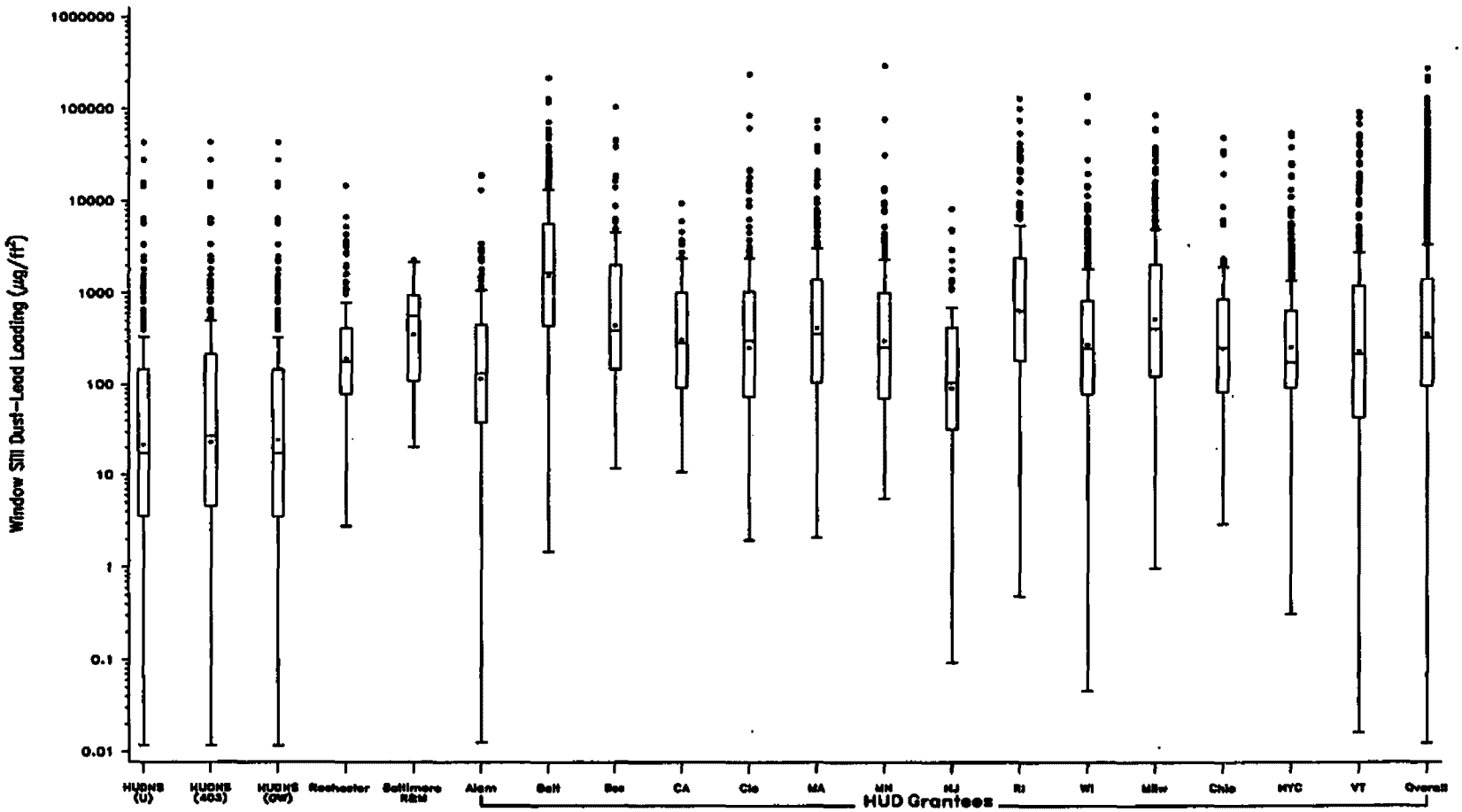

Figure 3-8. Boxplots of Area-Weighted Average Pre-Intervention Window Sill Wipe Dust-Lead Loadings (µg/ft²) for Houses in the HUD National Survey, Baltimore R&M Study, Rochester Lead-in-Dust Study, and Grantees Within the HUD Grantees Evaluation

(Note: Dust-lead loadings from the HUD National Survey and Baltimore R&M study have been converted from vacuum to wipe-equivalents using the methods documented in the §403 risk analysis report. See text for definitions of labels along the horizontal axis.)

Table 3-12. Descriptive Statistics of Area-Weighted Average Pre-Intervention Floor Wipe Dust-Lead Loadings for Households, As Reported in the §403 Risk Analysis, the HUD National Survey, and Other Studies

Study	Approach/ Grantee	Area-Weighted Average Pre-Intervention Floor Dust-Lead Loading ($\mu g/ft^2$)								
		# Units with Data	Arith-metic Mean	Geo-metric Mean	Geo-metric Std. Dev.	Minimum	25th Percen-tile	Median	75th Percen-tile	Maximum
HUD National Survey	unweighted[1]	281	21.0	8.19	3.66	0.508	3.23	7.27	17.3	375
	orig. weights[2]	281	21.0	7.97	3.70	0.508	3.17	6.94	17.0	375
	§403 RA[3]	284	16.5	6.27	3.49	0.508	2.65	5.32	12.2	375
Rochester Lead-in-Dust		205	110	17.7	3.20	1.21	10.4	16.1	26.6	8660
Baltimore R&M[4]		90	54.3	40.9	2.27	4.48	29.1	45.2	70.4	266
HUD Grantees	Alameda Co.	168	127	31.4	5.78	0.250	8.59	31.0	98.0	3730
	Baltimore	402	642	149	5.48	0.250	53.2	167	456	89100
	Boston	114	205	61.3	4.79	1.00	18.8	55.3	170	2490
	California	90	130	24.6	4.89	2.75	7.95	15.6	59.3	2650
	Cleveland	190	232	39.4	6.51	1.00	10.3	36.4	134	8800
	Massachusetts	229	408	64.4	6.47	0.521	17.0	59.8	234	16600
	Minnesota	212	202	27.3	5.14	0.333	10.9	19.2	62.4	13800
	New Jersey	45	308	68.2	6.71	1.75	10.5	93.4	298	4250
	Rhode Island	203	530	60.6	5.85	0.250	17.7	54.0	187	59200
	Wisconsin	236	172	24.8	6.92	0.400	5.99	16.9	79.1	2780
	Milwaukee	291	247	32.2	4.61	1.50	11.0	27.5	76.3	31900
	Chicago	158	234	29.4	4.40	0.200	11.5	28.2	69.2	26400
	New York City	399	462	52.6	5.90	0.0880	18.5	32.9	94.4	22200
	Vermont	354	515	67.9	6.70	0.750	15.8	49.9	219	15600
	All Grantees	3091	366	50.1	5.76	0.0880	14.3	40.2	165	89100

[1] Area-weighted average dust-lead loadings, as reported in the HUD National Survey but converted to wipe-equivalent loadings, are summarized without weighting by sample weights.
[2] Area-weighted average dust-lead loadings, as reported in the HUD National Survey but converted to wipe-equivalent loadings, are summarized by weighting each average by the original sample weights assigned in the survey.
[3] Area-weighted average dust-lead loadings, as calculated in Chapter 3 of the §403 risk analysis, are summarized by weighting each average to reflect the 1997 U.S. housing stock and imputing averages for units with missing data.
[4] BRM dust-lead loadings are converted to wipe-equivalent loadings prior to summary in this table.

Table 3-13. Descriptive Statistics of Area-Weighted Average Pre-Intervention Window Sill Wipe Dust-Lead Loadings for Households, As Reported in the §403 Risk Analysis, the HUD National Survey, and Other Studies

Study	Approach/ Grantee	Area-Weighted Average Pre-Intervention Window Sill Dust-Lead Loading ($\mu g/ft^2$)								
		# Units with Data	Arith-metic Mean	Geo-metric Mean	Geo-metric Std. Dev.	Minimum	25th Percen-tile	Median	75th Percen-tile	Maximum
HUD National Survey	*unweighted*[1]	245	678	21.7	15.4	0.0118	3.57	17.6	149	43700
	orig. weights[2]	245	721	24.9	17.9	0.0118	5.22	36.3	217	43700
	§403 RA[3]	312	550	23.0	15.8	0.0118	4.35	19.5	198	43700
Rochester Lead-in-Dust		196	558	196	3.96	2.83	80.6	183	416	14900
Baltimore R&M[4]		90	627	356	3.55	20.6	112	576	960	2330
HUD Grantees	Alameda Co.	178	677	118	9.14	0.0016	37.7	134	464	19700
	Baltimore	402	6690	1560	7.39	<0.0001	444	1690	5800	220000
	Boston	95	4090	452	9.87	0.0053	135	385	2040	106000
	California	81	909	316	4.60	11.0	94.2	293	1030	9630
	Cleveland	185	4050	259	16.2	<0.0001	72.8	288	949	241000
	Massachusetts	206	2990	425	7.13	2.15	108	369	1420	76100
	Minnesota	193	3160	308	6.17	5.66	72.6	262	1030	300000
	New Jersey	51	758	93.7	27.8	<0.0001	32.8	104	435	8450
	Rhode Island	192	4930	659	11.9	<0.0001	186	666	2450	132000
	Wisconsin	234	2790	279	8.44	0.0008	80.7	256	845	142000
	Milwaukee	271	3520	536	6.89	1.00	127	424	2110	88000
	Chicago	146	1600	260	5.71	3.02	86.7	267	877	50500
	New York City	382	1580	267	5.58	0.320	97.0	183	670	57100
	Vermont	318	3740	246	14.6	<0.0001	45.0	227	1260	98100
	All Grantees	2934	3360	380	8.68	<0.0001	102	343	1490	300000

[1] Area-weighted average dust-lead loadings, as reported in the HUD National Survey but converted to wipe-equivalent loadings, are summarized without weighting by sample weights.

[2] Area-weighted average dust-lead loadings, as reported in the HUD National Survey but converted to wipe-equivalent loadings, are summarized by weighting each average by the original sample weights assigned in the survey.

[3] *Area-weighted average dust-lead loadings, as calculated in Chapter 3 of the §403 risk analysis, are summarized by* weighting each average to reflect the 1997 U.S. housing stock and imputing averages for units with missing data.

[4] BRM dust-lead loadings are converted to wipe-equivalent loadings prior to summary in this table.

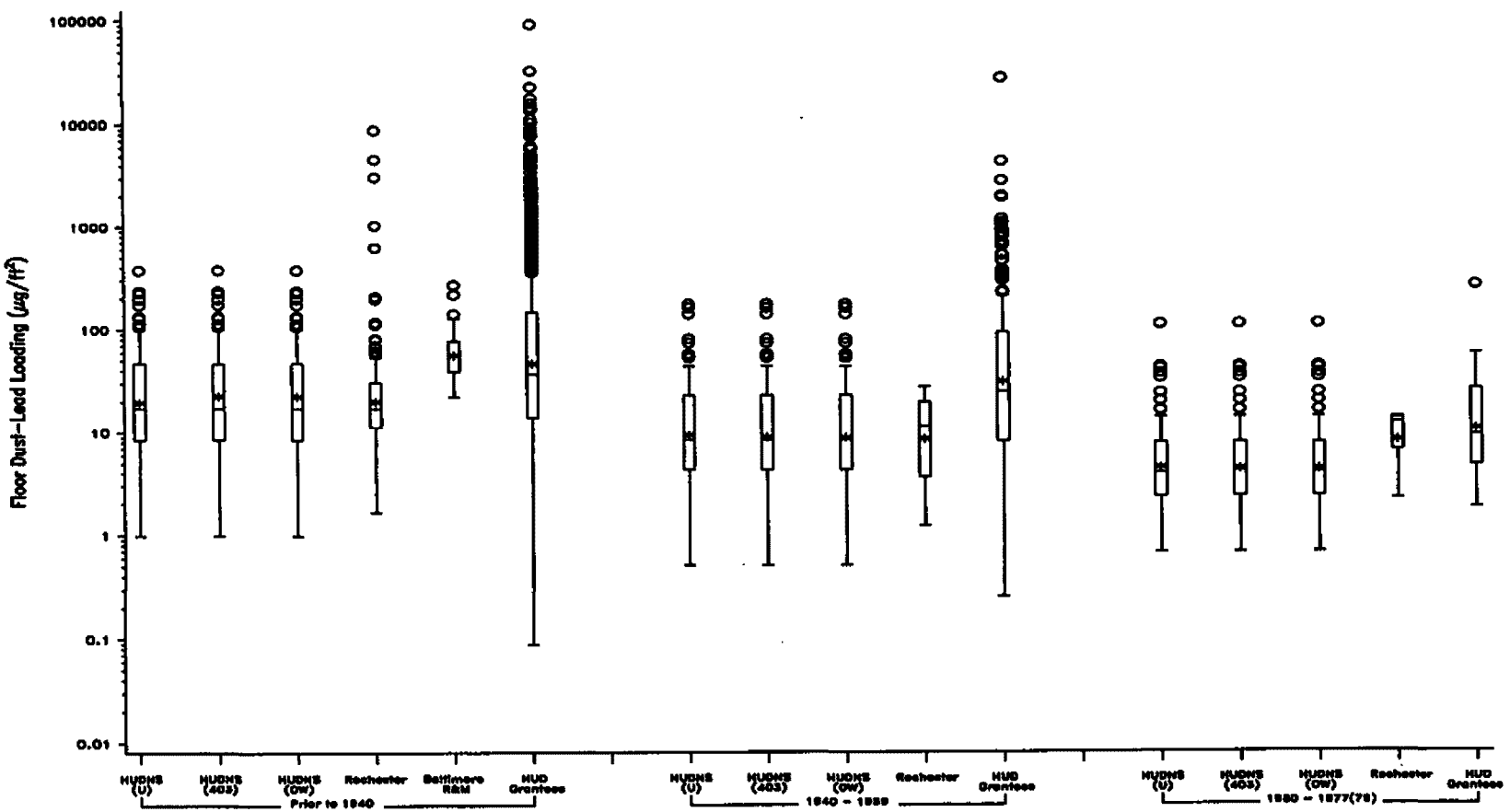

Figure 3-9. Boxplots of Area-Weighted Average Pre-Intervention Floor Wipe Dust-Lead Loadings (µg/ft²) for Houses in the HUD National Survey, Baltimore R&M Study, Rochester Lead-in-Dust Study, and HUD Grantees Evaluation, by Age of House Category (pre-1979 only)

(Note: Dust-lead loadings from the HUD National Survey and Baltimore R&M study have been converted from vacuum to wipe-equivalents using the methods documented in the §403 Risk Analysis report. See text for definitions of labels along the horizontal axis. Caution must be taken when categorizing houses in the Rochester study by age of house.)

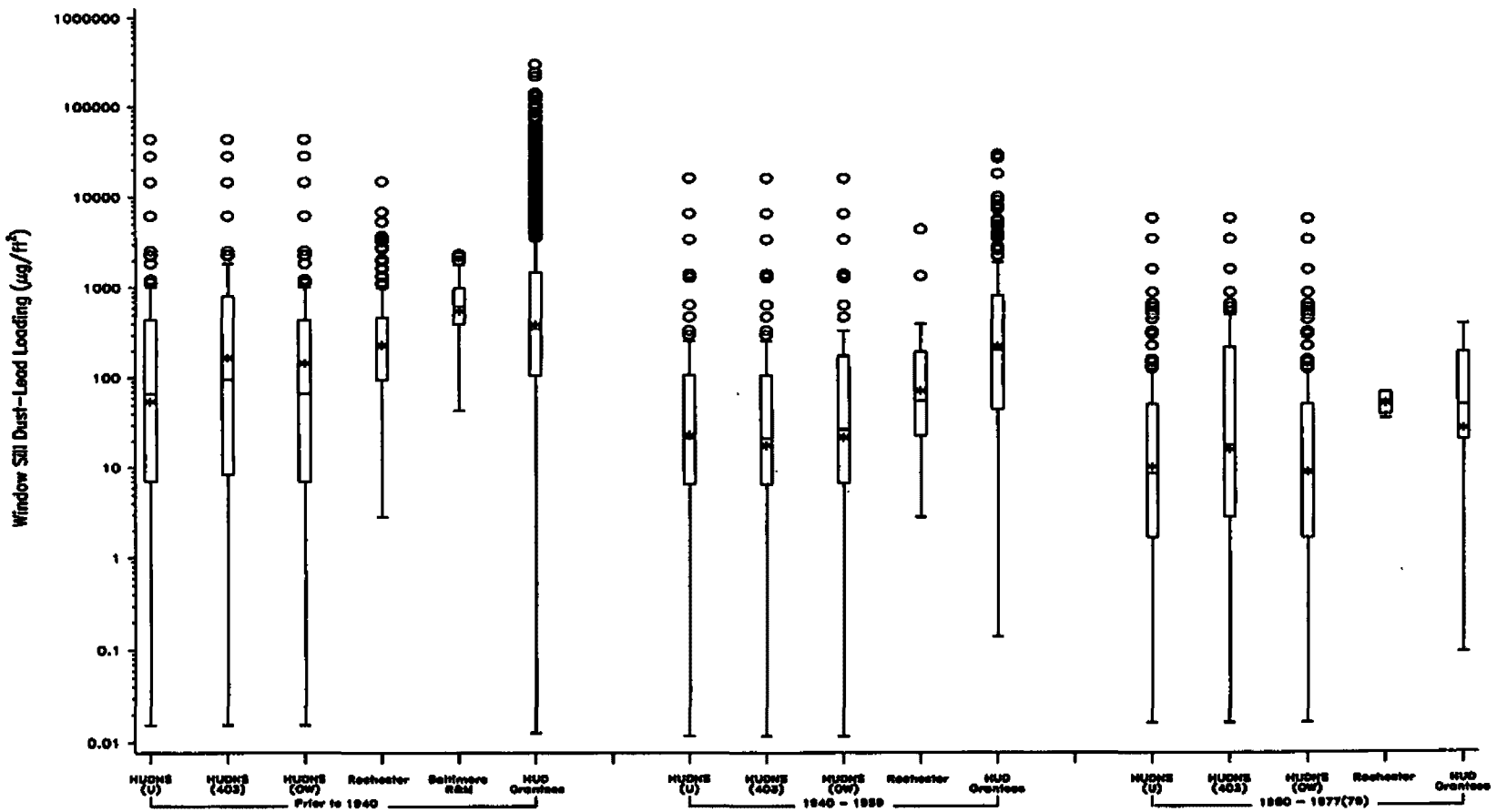

Figure 3-10. Boxplots of Area-Weighted Average Pre-Intervention Window Sill Wipe Dust-Lead Loadings (µg/ft²) for Houses in the HUD National Survey, Baltimore R&M Study, Rochester Lead-in-Dust Study, and HUD Grantees Evaluation, by Age of House Category (pre-1979 only)

(Note: Dust-lead loadings from the HUD National Survey and Baltimore R&M study have been converted from vacuum to wipe-equivalents using the methods documented in the §403 Risk Analysis report. See text for definitions of labels along the horizontal axis. Caution must be taken when categorizing houses in the Rochester study by age of house.)

Values of the statistics entering into the boxplots in Figures 3-9 and 3-10 are included within Tables 3-14 and 3-15. While not included in the figures, these tables include summary statistics for homes labeled as post-1979 (although the Rochester study units may not have actually been built in this time period, as mentioned in the previous paragraph). The post-1979 results labeled as "HUD National Survey (§403 RA)" represent surveyed homes built from 1960-1979 that contain no lead-based paint (Section 3.3.1.5 of the §403 risk analysis report).

3.2.1.3 Calculating National Exceedance Percentages for Household Average Floor Dust-Lead Loading. With respect to the national summaries of household average floor dust-lead loading presented in Section 3.2.1.1 above, it was desired to estimate the percentage of housing with average floor dust-lead loadings at or above specified thresholds (i.e., "exceedance percentage"), with separate estimates originating from data for each of the two national surveys (i.e., HUD National Survey and the interim NSLAH). This was done by fitting a lognormal distribution to the household average floor dust-lead loadings summarized in Section 3.2.1.1 and calculating the exceedance percentages based on this distribution.[9] If the household averages from the two surveys could each be considered a sample from their respective fitted lognormal distributions, with the probability of selection for the sample determined by the sampling weights, then the estimates based on these fitted distributions would be considered representative of actual percentages for the nation. The fitted lognormal distributions and the resulting exceedance percentage estimates are now presented for both surveys.

For both surveys, normal probability plots prepared on the log-transformed average floor dust-lead loadings indicated that a lognormal distribution did not adequately represent data in the upper tails of the distribution (i.e., typically the upper quartile). This was because the fitted distribution was heavily influenced by the considerable amount of data at the lower end of the distribution. Because it was necessary in this exercise to characterize the upper tail of the distribution as accurately as possible (due to calculating exceedance percentages from the distribution), the actual values of the data at the lower end of the distribution did not need to influence the fitted distribution to the extent that they were. Under these considerations, the procedure to fit a lognormal distribution was as follows:

- For values of P from 5 to 50 (in multiples of 5), the value of the log-transformed average floor dust-lead loading (call this value X) was identified for which P% of the (weighted) data fell below.

- For each value of P, log-transformed data values falling below the value X were considered to be *censored* at X. That is, rather than using these actual log-transformed data values, the procedure assumed that each of these values was somewhere at or below X.

[9] For the interim NSLAH, household averages calculated from data where no adjustment was made when below detection limits were used in this exercise.

Table 3-14. Descriptive Statistics of Area-Weighted Average Pre-Intervention Floor Wipe Dust-Lead Loadings for Households, Presented by Housing Age Category, As Reported in the §403 Risk Analysis, the HUD National Survey, and Other Studies

Study	Approach/ Grantee	Area-Weighted Household Average Pre-Intervention Floor Dust-Lead Loading ($\mu g/ft^2$)								
		# Units with Data	Arith-metic Mean	Geo-metric Mean	Geo-metric Std. Dev.	Minimum	25th Percen-tile	Median	75th Percen-tile	Maximum
Houses Built Prior to 1940										
HUD National Survey	unweighted[1]	76	43.9	19.5	3.68	0.991	8.45	17.1	47.1	375
	orig. weights[2]	76	47.9	22.4	3.65	0.991	8.84	17.7	79.7	375
	§403 RA[3]	77	47.9	22.6	3.63	0.991	8.84	17.7	79.7	375
Baltimore R&M[4]		74	63.6	55.5	1.65	22.0	38.7	54.3	76.0	266
Rochester Lead-in-Dust		172	127	19.8	3.18	1.66	11.3	16.9	30.0	8660
HUD Grantees	Alameda Co.	138	118	30.6	5.40	0.250	10.3	31.0	97.7	3730
	Baltimore	345	672	153	5.10	1.00	54.6	164	456	89100
	Boston	71	222	55.4	5.11	1.00	16.0	35.0	151	2490
	California	35	269	48.4	6.75	2.75	8.38	35.0	250	2650
	Cleveland	173	209	34.7	6.30	1.00	9.50	31.0	121	8800
	Massachusetts	146	147	31.7	5.03	0.521	11.9	26.5	83.1	4540
	Minnesota	182	171	21.3	4.72	0.333	10.0	16.8	40.0	13800
	New Jersey	26	511	215	4.19	10.5	134	239	513	4250
	Rhode Island	123	197	44.2	4.68	2.00	16.4	38.7	106	6050
	Wisconsin	214	183	28.4	6.77	0.400	7.26	18.5	99.5	2780
	Milwaukee	262	254	30.7	4.42	1.50	11.0	26.3	71.0	31900
	Chicago	144	60.7	25.6	3.92	0.200	10.7	25.4	62.4	668
	New York City	375	470	50.0	5.93	0.0880	18.1	31.4	84.4	22200
	Vermont	288	478	63.7	6.81	0.750	15.8	49.0	197	15500
	All Grantees	2522	328	45.9	5.45	0.0880	13.7	36.1	145	89100
Houses Built From 1940 - 1959										
HUD National Survey	unweighted[1]	87	19.8	9.20	3.53	0.508	4.20	8.32	22.5	171
	orig. weights[2]	87	18.1	8.74	3.34	0.508	4.07	7.81	22.4	171
	§403 RA[3]	87	18.1	8.74	3.34	0.508	4.07	7.81	22.4	171
Rochester Lead-in-Dust[5]		19	11.8	8.36	2.61	1.21	3.54	11.1	19.2	26.9
HUD Grantees	Alameda Co.	19	153	32.1	7.15	2.00	5.75	17.0	157	909
	Baltimore	43	494	120	9.13	0.250	39.5	197	648	4170
	Boston	4	57.3	26.6	4.46	5.00	10.0	27.0	105	170
	California	51	41.7	15.4	3.29	2.75	6.25	10.1	33.3	825
	Massachusetts	5	55.5	46.5	1.93	22.5	30.0	39.8	70.3	115
	Minnesota	1	149	149	--	149	149	149	149	149

Table 3-14. (cont.)

Study	Approach/ Grantee	Area-Weighted Household Average Pre-Intervention Floor Dust-Lead Loading (µg/ft²)								
		# Units with Data	Arith-metic Mean	Geo-metric Mean	Geo-metric Std. Dev.	Minimum	25th Percen-tile	Median	75th Percen-tile	Maximum
Houses Built From 1940 - 1959 (cont.)										
HUD Grantees	Rhode Island	34	81.3	27.3	5.47	0.250	7.60	36.6	77.3	617
	Wisconsin	15	87.0	7.22	6.99	0.800	1.60	5.72	17.1	1050
	Milwaukee	5	14.0	6.78	4.14	1.50	2.25	4.88	22.5	38.8
	Chicago	5	5300	102	23.8	16.4	17.8	19.2	75.8	26400
	Vermont	31	38.4	26.4	2.23	8.00	15.0	17.8	45.5	219
	All Grantees	213	276	30.1	5.39	0.250	8.00	24.3	89.3	26400
Houses Built From 1960 - 1979 (1960 - 1977 for HUD Grantees)										
HUD National Survey	unweighted[1]	118	7.14	4.30	2.50	0.657	2.26	3.85	7.59	106
	orig. weights[2]	118	6.74	4.11	2.46	0.657	2.25	3.62	7.59	106
	§403 RA[3]	120	6.74	4.14	2.45	0.657	2.25	3.62	7.59	106
Rochester Lead-in-Dust[5]		4	9.65	7.84	2.40	2.13	6.38	11.6	12.9	13.2
HUD Grantees	Boston	1	18.8	18.8	--	18.8	18.8	18.8	18.8	18.8
	Cleveland	1	9.25	9.25	--	9.25	9.25	9.25	9.25	9.25
	New Jersey	16	32.6	13.6	3.70	1.75	6.58	10.0	34.6	245
	Wisconsin	6	4.42	4.01	1.61	2.40	2.50	3.84	5.93	8.02
	All Grantees	24	24.0	10.0	3.14	1.75	4.45	8.88	24.6	245
Houses Built After 1979 (After 1977 for HUD Grantees)										
HUD National Survey (§403 RA)[3]		28	4.16	3.14	2.06	1.06	1.76	2.84	5.66	12.9
Baltimore R&M[4]		16	10.9	9.97	1.55	4.48	7.13	10.5	14.7	17.4
Rochester Lead-in-Dust[5]		10	37.2	15.0	3.34	3.48	5.57	16.8	21.2	250
HUD Grantees	Minnesota	1	32.4	32.4	--	32.4	32.4	32.4	32.4	32.4
	Rhode Island	3	984	838	2.00	440	440	763	1750	1750
	All Grantees	4	746	372	2.00	32.4	236	602	1260	1750

[1] Area-weighted average dust-lead loadings, as reported in the HUD National Survey but converted to wipe-equivalent loadings, are summarized without weighting by sample weights.

[2] Area-weighted average dust-lead loadings, as reported in the HUD National Survey but converted to wipe-equivalent loadings, are summarized by weighting each average by the original sample weights assigned in the survey.

[3] Area-weighted average dust-lead loadings, as calculated in Chapter 3 of the §403 risk analysis, are summarized by weighting each average to reflect the 1997 U.S. housing stock and imputing averages for units with missing data.

[4] BRM dust-lead loadings are converted to wipe-equivalent loadings prior to summary in this table.

[5] Some houses in this housing age category may belong to an earlier age category, as some houses may have actually been built earlier than the year specified within the study's database.

Table 3-15. Descriptive Statistics of Area-Weighted Average Pre-Intervention Window Sill Wipe Dust-Lead Loadings for Households, Presented by Housing Age Category, As Reported in the §403 Risk Analysis, the HUD National Survey, and Other Studies

Study	Approach/ Grantee	Area-Weighted Household Average Pre-Intervention Window Sill Dust-Lead Loading ($\mu g/ft^2$)								
		# Units with Data	Arithmetic Mean	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum
Houses Built Prior to 1940										
HUD National Survey	unweighted[1]	71	1610	54.7	19.6	0.0155	7.05	67.1	442	43700
	orig. weights[2]	71	2060	146	16.8	0.0155	35.6	198	1220	43700
	§403 RA[3]	77	2060	168	16.7	0.0155	35.6	198	1220	43700
Baltimore R&M[4]		74	751	555	2.41	44.0	399	628	989	2330
Rochester Lead-in-Dust		164	613	234	3.67	2.85	95.3	223	475	14900
HUD Grantees	Alameda Co.	148	767	138	9.34	0.0016	44.7	164	566	19700
	Baltimore	347	7070	1600	7.69	<0.0001	451	1690	6140	220000
	Boston	71	5150	577	7.89	14.0	135	425	2410	106000
	California	35	1530	506	5.70	28.0	159	524	2440	9630
	Cleveland	172	4120	233	16.9	<0.0001	63.8	270	876	241000
	Massachusetts	146	1770	322	5.81	2.60	93.8	296	1090	63400
	Minnesota	177	3320	282	6.30	5.66	71.0	190	945	300000
	New Jersey	26	1080	328	5.08	21.3	99.6	276	1170	8450
	Rhode Island	123	5780	816	6.49	12.0	192	709	2500	132000
	Wisconsin	211	3020	294	8.91	0.0008	81.4	258	1090	142000
	Milwaukee	261	3610	543	6.89	1.00	127	413	2110	88000
	Chicago	140	1630	259	5.78	3.02	85.0	267	852	50500
	New York City	368	1530	258	5.51	0.320	95.6	175	543	57100
	Vermont	269	3860	272	15.9	<0.0001	72.0	275	1340	98100
	All Grantees	2494	3480	391	8.22	<0.0001	106	351	1470	300000
Houses Built From 1940 - 1959										
HUD National Survey	unweighted[1]	79	430	23.1	11.4	0.0118	6.47	21.7	107	16100
	orig. weights[2]	79	285	17.9	10.5	0.0118	6.47	19.1	107	16100
	§403 RA[3]	87	285	22.0	10.7	0.0118	6.47	19.1	107	16100
Rochester Lead-in-Dust[5]		18	399	72.0	6.16	2.83	23.0	56.0	194	4390
HUD Grantees	Alameda Co.	20	152	47.7	8.04	0.140	14.5	71.1	260	580
	Baltimore	43	4310	1330	5.39	33.0	256	1600	4820	29400
	Boston	4	382	150	5.20	39.4	39.6	160	724	1170
	California	42	395	203	3.41	11.0	89.9	190	565	1850
	Massachusetts	4	142	59.7	8.20	2.79	47.9	123	237	321
	Minnesota	1	289	289	--	289	289	289	289	289

Table 3-15. (cont.)

Study	Approach/ Grantee	Area-Weighted Household Average Pre-Intervention Window Sill Dust-Lead Loading ($\mu g/ft^2$)								
		# Units with Data	Arith-metic Mean	Geo-metric Mean	Geo-metric Std. Dev.	Minimum	25th Percen-tile	Median	75th Percen-tile	Maximum
Houses Built From 1940 - 1959 (cont.)										
HUD Grantees	Rhode Island	34	1520	416	7.53	0.500	144	617	1120	9970
	Wisconsin	16	497	148	4.24	24.0	47.4	105	338	4750
	Milwaukee	6	552	140	7.09	18.0	28.8	123	797	2220
	Chicago	5	835	449	3.84	111	120	521	1170	2250
	Vermont	30	52.4	40.4	2.08	7.00	31.0	45.0	45.0	212
	All Grantees	205	1350	222	4.94	0.140	45.0	205	814	29400
Houses Built From 1960 - 1979 (1960 - 1977 for HUD Grantees)										
HUD National Survey	unweighted[1]	95	190	10.3	13.3	0.0164	1.68	8.69	51.3	5790
	orig. weights[2]	95	184	9.10	14.5	0.0164	2.05	16.6	217	5790
	§403 RA[3]	120	184	16.2	14.6	0.0164	2.05	16.6	217	5790
Rochester Lead-in-Dust[5]		4	54.4	52.3	1.38	36.2	40.0	55.2	68.7	70.7
HUD Grantees	Boston	1	289	289	–	289	289	289	289	289
	Cleveland	1	409	409	–	409	409	409	409	409
	New Jersey	20	59.8	12.9	63.1	<0.0001	17.8	29.6	72.7	333
	Wisconsin	6	209	153	2.90	21.0	105	240	289	359
	All Grantees	28	112	27.8	39.3	<0.0001	20.9	44.4	179	409
Houses Built After 1979 (After 1977 for HUD Grantees)										
HUD National Survey (§403 RA)[3]		28	83.0	8.17	9.94	0.0164	2.58	8.11	57.8	1590
Baltimore R&M[4]		16	50.8	45.6	1.65	20.6	27.1	52.6	66.5	85.9
Rochester Lead-in-Dust[5]		10	134	113	1.95	26.9	75.7	125	159	320
HUD Grantees	Minnesota	1	2350	2350	–	2350	2350	2350	2350	2350
	Rhode Island	1	816	816	–	816	816	816	816	816
	All Grantees	2	1580	1390	–	816	816	1580	2350	2350

[1] Area-weighted average dust-lead loadings, as reported in the HUD National Survey but converted to wipe-equivalent loadings, are summarized without weighting by sample weights.
[2] Area-weighted average dust-lead loadings, as reported in the HUD National Survey but converted to wipe-equivalent loadings, are summarized by weighting each average by the original sample weights assigned in the survey.
[3] Area-weighted average dust-lead loadings, as calculated in Chapter 3 of the §403 risk analysis, are summarized by weighting each average to reflect the 1997 U.S. housing stock and imputing averages for units with missing data.
[4] BRM dust-lead loadings are converted to wipe-equivalent loadings prior to summary in this table.
[5] Some houses in this housing age category may belong to an earlier age category, as some houses may have actually been built earlier than the year specified within the study's database.

- For each value of P, a normal distribution was fitted to the log-transformed data, taking into account the censoring of the lower P% of the data and the sample weights, using the LIFEREG procedure in the SAS® System.

- The value of P (and its corresponding cut-off X) was identified that resulted in the best fit for normality in the upper tail of the distribution (based on review of normal probability plots). The exceedance percentages were estimated based on this final distribution, using normal probability theory.

This procedure was applied separately to HUD National Survey data and interim data from the NSLAH. Exceedance percentages were estimated for each of the following floor dust-lead loading thresholds: 5, 10, 20, 30, 40, and 50 $\mu g/ft^2$.

Figure 3-11 contains the fitted distributions based on the HUD National Survey data (top plot) and the interim NSLAH data (bottom plot). (The top plot is labeled "Section 403 risk analysis" as it reflects sample weights adjusted for the 1997 housing stock and dust-lead loadings converted to wipe-equivalents, both done within the §403 risk analysis.) Each plot contains a bar chart of the observed data, onto which the fitted lognormal distribution curve is superimposed. Note that the same floor dust-lead loading (horizontal) axis is used for both plots, so that the two plots can be directly compared. As can be noted in this figure (and which was seen in the summaries in Section 3.2.1.1), the distribution based on the interim NSLAH data covers a considerably lower range compared to the distribution based on the HUD National Survey data used in the §403 risk analysis. Thus, the estimated exceedance percentages for each of the six thresholds, also annotated within each plot, are considerably lower based on the interim NSLAH data, especially as the threshold increases.

Each estimated exceedance percentage within Figure 3-11 is accompanied by an approximate 95% confidence interval on the number of homes in the U.S. housing stock that exceeds the threshold. These intervals were calculated based on the estimated total number of housing units in the housing stock, as determined by the sum of the sampling weights for the given survey (which is specified within each plot).

In Figure 3-11, the distribution based on the HUD National Survey data used in the §403 risk analysis was determined by censoring data values below 3.81 $\mu g/ft^2$ (i.e., the bottom 40 percent of the data, taking into account the sample weights). The distribution based on the interim NSLAH data was determined by censoring data values below 0.2025 ug/ft^2, which corresponds to the bottom 20 percent of the observed weighted distribution, including negative values.

For both surveys, the estimated exceedance percentages specified within Figure 3-11 for household average floor dust-lead loading, based on the fitted lognormal distribution, are also included within Table 3-16 (columns 2 and 4) for the same six thresholds. Also included in Table 3-16 (columns 3 and 5) are estimated exceedance percentages that were determined solely by the proportion of total sampling weights in the survey that corresponded to surveyed units

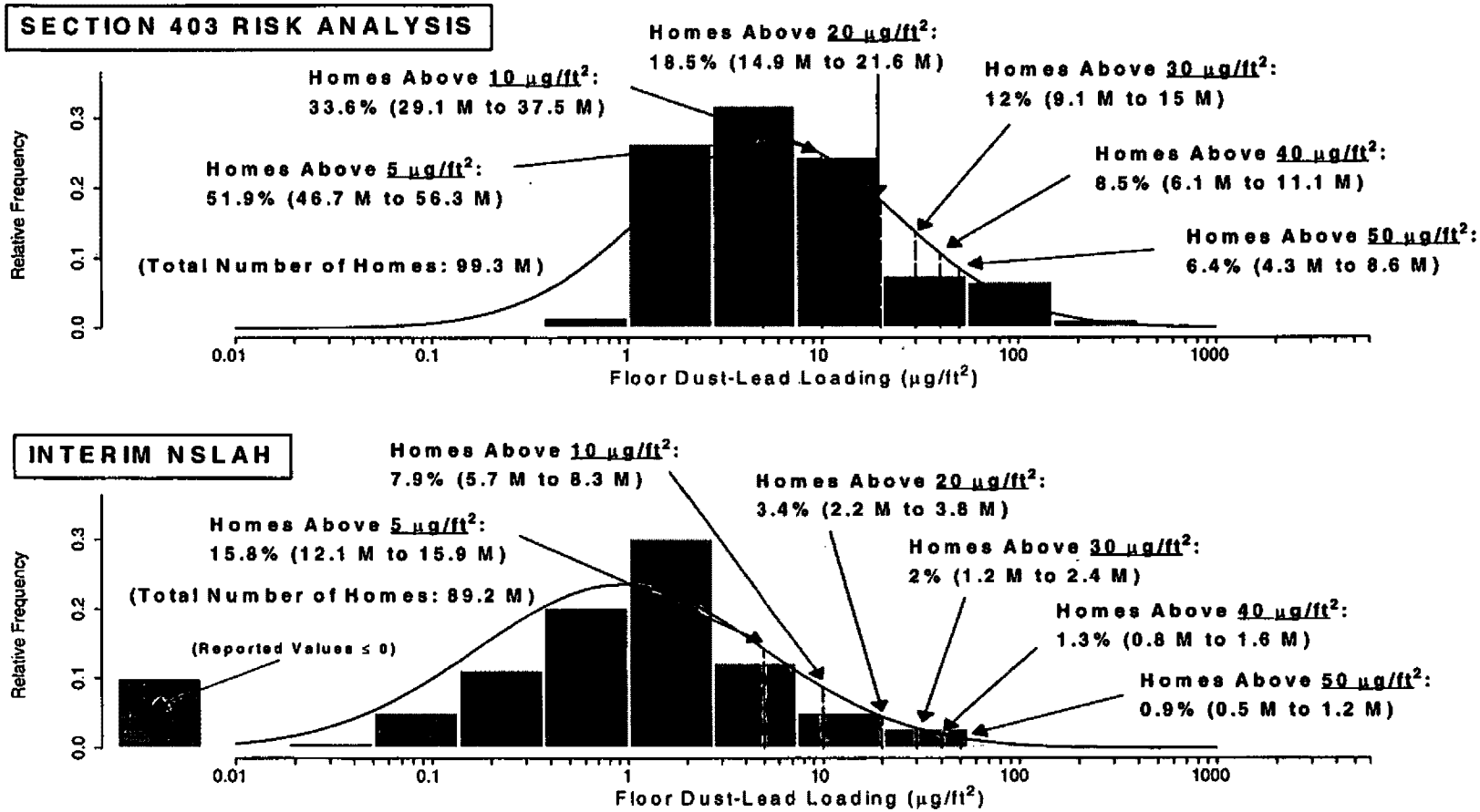

Figure 3-11. Estimated Distribution of Household Average Floor Dust-Lead Loading in the Nation's Housing Stock, and Corresponding Estimates of the Percentage of Homes Exceeding Specified Thresholds (with 95% Confidence Intervals on the Corresponding Number of Homes, in Millions), Based on Data from the HUD National Survey (top plot) and the Interim NSLAH (bottom plot)

Note: The estimated exceedance percentages are calculated based on the fitted distribution (solid curve).

Table 3-16. Estimated Percentages of 1997 U.S. Housing Exceeding Specified Thresholds of Household Average Dust-Lead Loading

Dust-Lead Loading Threshold ($\mu g/ft^2$)	§403 Risk Analysis – Based on Data from the HUD National Survey (n = 284)		Data from the Interim NSLAH (n = 706)	
	Based on the Fitted Lognormal Distribution (i.e., the curve in Figure 3-11)	Based on the Weighted Observed Data (i.e., the bar chart in Figure 3-11)	Based on the Fitted Lognormal Distribution (i.e., the curve in Figure 3-11)	Based on the Weighted Observed Data (i.e., the bar chart in Figure 3-11)
5	51.9%	51.1%	15.8%	13.2%
10	33.6%	30.6%	7.9%	7.2%
20	18.5%	15.6%	3.4%	4.0%
30	12.0%	11.9%	2.0%	2.0%
40	8.5%	9.6%	1.3%	1.4%
50	6.4%	8.3%	0.9%	1.2%

Note: Data are imputed for those surveyed units with missing data prior to calculating the above statistics (3 observations in the HUD National Survey and 9 observations in the interim NSLAH had imputed data). The estimates based on the weighted observed data are simple weighted percentiles that do not originate from a fitted distribution.

whose household average floor dust-lead loadings exceeded the given threshold (i.e., information from the bar charts within Figure 3-11). These results are included to evaluate the similarity between the lognormal-based estimates and those generated from an approach that uses only the observed data without an underlying distribution assumption. As Table 3-16 shows, the lognormal-based estimates are slightly lower for the lower thresholds and slightly higher for the higher thresholds, while the two approaches yield nearly equivalent estimates at the threshold of 30 $\mu g/ft^2$. It should be noted that the lognormal-based estimates for the exceedance percentages (which were also portrayed in Figure 3-11) should be used when making inferences on the nation's housing stock.

3.2.1.4 Interpreting the Observed Differences with Other Studies. In order to make proper interpretations from the results portrayed in this subsection, in particular why differences exist between the studies, one must be aware of how the housing selection procedure and sample collection and analysis procedures differ between the studies and can contribute to the differences observed in the boxplots and tables. For the studies highlighted in the §403 risk analysis report, this information was summarized in Tables 3-3a through 3-3f of that report. Some of the differences among these studies that may contribute to differences in the reported data are as follows:

- All non-control housing units in the Baltimore R&M study, approximately 88 percent of units selected in the HUD Grantees evaluation, and at least 84 percent of the Rochester study units were built prior to 1941. In contrast, only 27 percent of the housing units in the HUD National Survey were built prior to 1940.

- The neighborhoods surveyed within the Baltimore R&M study and HUD Grantees evaluation had a high prevalence of homes with lead-based paint hazards, along with a history of children with elevated blood-lead concentrations and/or considered at high-risk for lead poisoning.

- The HUD National Survey targeted occupied permanent housing throughout the 48 contiguous states. The units were selected via a statistically-based sampling design to represent the national housing stock built prior to 1980. Excluding two grantees from California, the HUD Grantees (as well as the Rochester and Baltimore R&M studies) sampled housing from the Northeast and Midwest census regions. Approximately two out of every five homes sampled in the HUD National Survey were from the South.

- For the HUD Grantees evaluation, 28 percent of the homes were single-family buildings (16 percent were single-family detached, and 12 percent were single-family attached, or rowhouses). All homes in the R&M intervention group within the Baltimore R&M study were urban rowhouses (single-family attached). Eighty percent of the homes in the HUD National Survey were single-family dwellings.

- From 8 percent to 67 percent of the dwelling units for any one Grantee were vacant prior to sampling. Out of the 5,265 dwelling units in total that were enrolled as of January 1999, 1524 units were vacant prior to pre-intervention sampling. Overall vacancy rate was 29 percent for the Evaluation. On the other hand, the HUD National Survey contained dwelling units which were permanent and occupied, with the potential for containing children.

- The dates of environmental sampling were 11/89-3/90 for the HUD National Survey, 12/93-1/99 for the HUD Grantees evaluation (pre-intervention), 8/93-11/93 for the Rochester study, and 3/93-11/94 for the Baltimore R&M study. Therefore, the HUD National Survey performed sampling roughly three years before each of the other studies and during the late fall and winter months.

Section 3.1 discussed differences in approaches and methods between the HUD National Survey and the NSLAH that could impact observed differences in the reported data.

3.2.1.5 Conclusions of the Dust-Lead Data Comparisons. The following conclusions could be made upon review of the dust-lead loading summaries within Tables 3-4 through 3-16 and Figures 3-1 through 3-11:

- For both floors and window sills, the interim NSLAH data are considerably lower than that reported in the §403 risk analysis (and based on the HUD National Survey data), as well as for all other sources of data available to the risk analysis. Household average floor dust-lead loadings had a median of less than 2.0 $\mu g/ft^2$

across the interim NSLAH data, while household average window sill dust-lead loadings had a median of approximately 12.0 $\mu g/ft^2$. Approximately two-thirds of the floor-dust samples and one-third of the window sill-dust samples had lead measurements below the detection limit in the interim NSLAH. Further investigation is necessary to determine the reasons for such low dust-lead loadings in the interim NSLAH.

- Compared to the other lead exposure studies whose data were considered in the §403 risk analysis (e.g., Rochester study, Baltimore R&M study, HUD Grantees evaluation), geometric mean dust-lead loadings tended to be lower in the HUD National Survey. However, all of these studies had similar ranges of observed dust-lead loading data. This suggests that 1) the conversions to wipe-equivalent dust-lead loadings performed on the HUD National Survey data in the §403 risk analysis did not lead to extreme adjustments overall, and 2) there is not sufficient evidence that data from the HUD National Survey are higher than what is representative of the 1997 housing stock simply because it was performed some years earlier.

- The importance of housing age is evident in the summaries within the four housing age categories. Older housing is more likely to contain higher average dust-lead loadings compared to newer housing. However, within an age category, the summaries were quite consistent across studies (with the exception of the interim NSLAH).

- The percentage of housing units with average floor dust-lead loadings that exceed 50 $\mu g/ft^2$ (i.e., the proposed floor dust-lead standard) was 6.4% based on data used in the §403 risk analysis, and 0.9% based on interim data from the NSLAH.

3.2.2 Characterizing Soil-Lead Concentrations

This subsection summarizes observed soil-lead concentrations in the HUD National Survey and how these data were used to characterize soil-lead levels in the §403 risk analysis, and compares these summaries with summaries of the interim NSLAH data (Section 3.1), as well as data for 22 other studies that characterized soil-lead concentrations in urban areas prior to any lead abatement. These 22 studies include the three recent studies included in the dust-lead data summaries of the previous section (Baltimore R&M study, Rochester study, and HUD Grantees evaluation) and other studies dating to the early 1970s (e.g., Omaha, Charleston). Sampling and laboratory protocols for the 22 additional studies are summarized in Table 3-17. The soil-lead data summaries for these 22 studies were either calculated directly from the available data set or culled from the published scientific literature.

Household mass-weighted average soil-lead concentration for a specific portion of the yard was the basis for the comparisons made in this section. This average was calculated by weighting the result for each soil sample taken at that location by the sample's mass. If this

Table 3-17. Information on Soil Sampling and Analysis Protocols for Studies Whose Soil-Lead Data Were Compared to Results from the §403 Risk Analysis and the HUD National Survey

Study [Reference]	Soil Sampling and Analysis Details	Soil-Lead Parameter(s) Used in This Section for Comparison to HUD National Survey
Baltimore R&M (USEPA, 1996c)	1993-94. Three 0.5" core samples per composite, taken from randomly determined areas along the dripline using a 6" stainless steel recovery probe and collected into a polysterene liner. Samples were sieved and homogenized and digested using SW 846-3015 and SW 846-3051. GFAA (SW 846-7421) laboratory analysis method. Only data for occupied units were used.	Soil-lead concentration for each composite sample (one composite sample per housing unit, taken from the dripline).
Baltimore Urban Garden Soil (Mielke et al., 1983)	1982. Samples were from garden soil in random locations within a 30-mile radius of downtown Baltimore. Samples were air-dried and sieved with a 2mm stainless steel mesh screen and digested in nitric acid. Extracts were filtered and analyzed using a Varian atomic absorption spectrophotometer with deuterium background correction.	Soil-lead concentration for each collected sample.
3-City (Baltimore, Boston, Cincinnati) (USEPA, 1996a) (Also known as the Urban Soil Lead Abatement Demonstration Project)	Only round 1 (pre-abatement) measurements were used. Baltimore/Boston: Only results for the top 2 cm of a 15 cm core sample were considered. Cincinnati: Soil samples were collected within neighborhoods, as well as within the yards of surveyed housing units.	Baltimore: Yard-wide average for a unit, equal to the unweighted arithmetic average of the unit's average dripline, average mid-yard and average boundary soil-lead concentrations within a property (set to missing if any of these measurements were missing). The location averages were also summarized. Boston: Yard-wide average for a unit, equal to the average across all samples associated with that unit. Cincinnati: Yard-wide average for a unit, equal to the unweighted arithmetic average of average building and average play area soil-lead concentrations for the unit (set to missing if either of these measurements are missing). These and other location averages were also summarized.
Boston Brigham and Women (Rabinowitz et al., 1985)	3 samples were collected one meter apart and at least 3 meters from any road structure (preference given to obvious play areas). These samples were composited prior to analysis. Soil sampling occurred twice: when the resident child of interest was 18 and 24 months of age. Laboratory analysis method was atomic absorption spectrophotometry (AAS).	Soil-lead concentration for each unit, equal to the unweighted arithmetic average of soil-lead concentrations for composite samples taken at the 18 and 24 month visits.
CAP Study (USEPA, 1996b)	1990. Soil samples taken from Denver units that were abated in 1989 during the HUD Abatement Demonstration Study. Samples were collected from the dripline, entryway, and remote areas of the yard with a soil recovery probe (1" diameter liner and 12" core sampler). At each location, a composite sample consisted of 3 cores, each 0.5" in depth. The sample preparation method was EPA SW846 Method 3050 (included use of nitric acid and hydrogen peroxide for digestion). The laboratory analysis method was ICP-AES.	Soil-lead concentration for the dripline, entryway, and remote areas of the yard. (One composite sample per location per unit.)

Table 3-17 (cont.)

Study (Reference)	Soil Sampling and Analysis Details	Soil-Lead Parameter(s) Used in This Section for Comparison to HUD National Survey
California (Sutton et al., 1995)	1987-91. Older units in Oakland, Los Angeles, and Sacramento. Composite soil samples (of 4 subsamples) were collected at each of the front, side, and rear yards. In addition, units in Oakland and Los Angeles had a composite soil sample collected from a secondary structure (e.g., garage) and a single sample collected from rain drains. All but rain drain samples were composites. Samples were < 1" in depth and were collected using a trowel (visible paint chips removed first). The laboratory analysis method was AAS.	Soil-lead concentration for each collected sample (from 3 to 5 per unit).
Cincinnati Longitudinal (Bornschein et al., 1985a; 1986; Que Hee et al., 1985)	1980-87. Surface scrapings rather than soil cores were taken. Laboratory analysis method was AAS. Enrolled expectant mothers residing in areas with a history of child residents with elevated blood-lead concentrations.	Not determined[1].
Cincinnati Roadside (Tong, 1990)	1990. Samples were collected near highways, boulevards, and cul-de-sacs in two neighborhoods (not industrial areas nor poor neighborhoods with deteriorated housing) within the Greater Cincinnati Metropolitan District. Samples were from a depth of 0-5cm and were analyzed using a Leeman plasma spectrophotometer with background correction.	Not determined[1].
Charleston (Galke et al., 1975)	1973. Soil samples taken from a child's primary play area. Laboratory analysis method was AAS.	Not determined[1].
Corpus Christi (Harrison, 1987)	1984. Samples were collected from parks, schools, and roadside embankments within the city limits of Corpus Christi, Texas from vegetated, non-sandy soil. The top 2 cm of soil was sampled with a Teflon knife. The laboratory analysis method was AAS.	Soil-lead concentration for each collected sample.
I-880 (Alameda County) (Teichmean et al., 1993)	1990. Samples were collected from homes, parks, playgrounds, and public housing developments within one mile east or west of I-880. The top 0.50" to 0.75" of soil was sampled. The laboratory analysis method was AAS.	Soil-lead concentration for each collected sample.
HUD Abatement Demonstration Study (USHUD, 1991)	Dripline samples were taken from 1 to 3 feet from an exterior wall and were composites of 5 subsamples. Soil sampling (and compositing) occurred twice: prior to and following lead-based paint abatements performed in this demonstration. The laboratory analysis method was AAS.	Soil-lead concentration for each dripline composite sample (one composite sample per housing unit collected at pre-intervention, and one sample per unit collected at post-intervention)
HUD Grantees Evaluation (USHUD, 1998)	1994-97. Pre-Intervention phase only. From 5-10 core samples were taken at 0.5-1" depths at a given location and composited. Locations were the dripline (samples taken from all sides of the unit, 2' from foundation and 2' from each other) and play areas (samples collected along x-shaped grids at least 1' from each other).	Yard-wide average for a unit, equal to the unweighted arithmetic average of dripline and play area soil-lead concentrations within a unit (set to missing if either of these measurements were missing).
Maine Urban (Krueger et al., 1989)	1988. Samples collected from units at least 30 years of age and from parks/playgrounds in Portland, Maine. A single composite sample, consisting of 4 cores taken 2' from the foundation, was associated with each housing unit. Laboratory analysis method was AAS.	Soil-lead concentration for each composite sample (housing units) and each sample collected from parks/playgrounds.

Table 3-17 (cont.)

Study [Reference]	Soil Sampling and Analysis Details	Soil-Lead Parameter(s) Used in This Section for Comparison to HUD National Survey
Milwaukee (Pendleton)	Soil samples collected from perimeter and play areas at each housing unit.	Yard-wide average for a unit, equal to the unweighted arithmetic average of perimeter and play area soil-lead concentrations (set to missing if either of these measurements are missing).
Minneapolis Clean-Up (Mielke et al., 1992)	Only pre-cleanup data were considered. Deep scrape samples were taken at a depth of 2.5 cm, air-dried and sieved with a 2 mm stainless steel mesh screen, and digested in nitric acid. Extracts were filtered and analyzed using a Varian atomic absorption spectrophotometer with deuterium background correction.	Not determined[1].
Minnesota (Schmitt et al., 1988; Mielke et al., 1989)	1986-87. Only results for St. Paul and Minneapolis were considered (except results labeled "Whole Study" also included Duluth, Rochester, St. Cloud and rural areas). Foundation samples were taken within 1.5 m of building. Yard samples (front, side, and back) were taken at the midpoint of the yard and at least 1.5 m from the foundation. Street samples were taken within 1.5 m of a curb. Samples were from the top 2 cm of soil. The laboratory analysis method was ICP-AES.	Soil-lead concentration for each collected sample.
New Orleans (Mielke, 1995; 1993)	1983. Samples taken from residential neighborhoods within 283 census tracts in the New Orleans metropolitan area. Foundation samples were taken within 1 m of a house. Streetside samples were taken from within 1 m of a street. Open area samples were from vacant lots or parks. The laboratory analysis method was AAS with deuterium background correction.	Soil-lead concentration for each collected sample.
New Haven, Connecticut (Stark et al., 1982)	1974-77. Samples (5-10 g) collected from homes of children who lived at the same address for at least one year. Only the top 0.5" of soil was analyzed.	Not determined[1].
Omaha (Angle et al., 1979)	1971-77. Soil core samples (2" depth) self-selected from halfway between the building and lot line on four sides of the selected units.	Yard-wide average for a unit, equal to the arithmetic average soil-lead concentration across all collected samples at the unit.
Rochester Lead-In-Dust (USHUD, 1995a; Lanphear et al., 1996a)	1993. Two composite samples, one from the dripline (12 samples per composite) and one from play areas (8-10 samples per composite). Core samples were taken at a depth of 0.5". Composites were mixed and sieved into fine and coarse fractions and analyzed separately. Digestion method was SW 846-3050, and the laboratory analysis method was FAA (method 239.1). Total soil-lead concentrations were computed as 0.25*Fine Soil Fraction + 0.75*Coarse Soil Fraction (see Appendix E).	Yard-wide average for a unit (for both total soil and fine soil only), equal to the unweighted arithmetic average of the unit's dripline and play area soil-lead concentrations (set to missing if either of these measurements was missing). The soil-lead concentration for the dripline sample at each unit was also summarized (for both total soil and fine soil only).
Washington, DC (Elhelu et al., 1995)	Housing units were randomly selected from each of the 8 wards of Washington, DC. Soil samples were collected from unpaved front yards approximately 1 m from the unit and at a depth of 15cm. Average dwelling distance from the road was 4.5 m. Fine soil samples were analyzed with a Perkin Elmer 2100 Atomic Absorption Spectrophotometer, with one result associated with each surveyed unit.	Soil-lead concentration for each collected sample (i.e., each housing unit).

[1] Most likely soil-lead concentration for each collected sample.

average could not be calculated for a given study due to insufficient data, then alternative statistics were calculated. For example, if mass weights were not available, the arithmetic average soil-lead concentration was instead calculated.

When possible, a yard-wide average soil-lead concentration was calculated in a manner that attempted to be consistent with the §403 risk analysis. This involved taking a weighted arithmetic average of the soil-lead concentrations reported at the dripline, unit entryway, and remote areas of the yard, with remote concentrations weighted twice as much as the dripline and entryway concentrations. (When only one of the dripline or entryway concentrations was available at a housing unit, the yard-wide average was the unweighted arithmetic average of that one concentration and the remote soil-lead concentration.) Thus, the yard-wide average was essentially an arithmetic average of two measures: the average soil-lead level at the dripline and unit entryway (i.e., "near" the housing unit) and the soil-lead level at a remote area of the yard (i.e., "far" from the housing unit). It was assumed that "play areas" represented remote areas of the yard. Imputed data values replaced missing values for a housing unit in the §403 risk analysis summaries, where imputation methods discussed in Section 3.3.1.1 of the §403 risk analysis report were used.

3.2.2.1 Data Summaries for the §403 Risk Analysis Versus the Interim NSLAH. Descriptive statistics of yard-wide average soil-lead concentrations as calculated in the §403 risk analysis using the HUD National Survey data are presented in this subsection as they compare with the same statistics calculated on interim data for 706 housing units in the NSLAH. Note that these statistics reflect the sampling weights used in the §403 risk analysis and the interim NSLAH sample weights, thereby allowing these summaries to be nationally representative of the 1997 housing stock. In addition, the interim NSLAH summaries do not include any data that may have been imputed within the revised §403 risk analysis when missing data for key parameters were encountered for a housing unit.

As in the dust-lead loading summaries (Section 3.2.1.1), the interim NSLAH summaries include imputed values of yard-wide average soil-lead concentration for those housing units having no reported soil-lead concentration data. As discussed in Appendix C, the imputation method involved imputing values for average dripline/entryway soil-lead concentration and for average mid-yard soil-lead concentration, then averaging these two imputed values together. If data existed for one of the two locations but not the other, the yard-wide average for that unit equaled the average soil-lead concentration at the location represented by the available data. Appendix C also gives the imputed data values and how they were assigned to housing units. Summaries of the interim yard-wide average soil-lead concentration data from the interim NSLAH excluding any imputed data can be found in Appendix D2.

Also, in the same manner as the dust-lead loading summaries (Section 3.2.1.1), Appendix D2 presents soil-lead concentration summaries for the interim NSLAH under five different approaches (including data censoring) to handling sample results that were below the detection limit. The summaries in this subsection were calculated under two of these approaches:

- making no adjustment to not-detected data values
- replacing not-detected data values with one-half of the detection limit.

These two approaches, the same two used in the dust-lead loading data summaries in Section 3.2.1.1, were included together in the summary tables to illustrate the impact that any one approach has on the characterized distribution of yard-wide average soil-lead concentration.

National comparisons

Table 3-18 presents descriptive statistics of yard-wide average soil-lead concentrations for the 1997 national housing stock. These results indicate that only a slight downward shift in the distribution of soil-lead concentrations was observed from the §403 risk analysis to the interim NSLAH data. (e.g., a decline in the geometric mean from 62 µg/g to approximately 53 µg/g). This decline was much smaller than that observed for dust-lead loadings.

Boxplots of the data distributions presented in Table 3-18 are found in Figure 3-12. When not-detected data in the NSLAH were replaced by one-half of the detection limit, the observed distribution of yard-wide average soil-lead concentration appears similar to what was characterized in the §403 risk analysis. Appendix D2 contains tabular summaries and boxplots after excluding imputed data values.

Table 3-18. Descriptive Statistics of Yard-Wide Average Soil-Lead Concentrations for Households, As Reported in the §403 Risk Analysis Versus the Interim NSLAH Data

Study	How Not-Detected and Negative Data were Handled	Yard-Wide Average Soil-Lead Concentration (µg/g)[1]								
		# Surveyed Units with Positive Averages	Arith-metic Mean	Geo-metric Mean[2]	Geo-metric Std. Dev.[2]	Minimum	25th Percen-tile	Median	75th Percen-tile	Maximum
§403 Risk Analysis (HUD Natl. Survey)		284	235	61.9	4.46	4.63	21.3	49.2	142	7030
Interim NSLAH[3]	No adjustment	689	200	53.0	5.09	0.00	16.6	41.8	158	9270
	Replaced by LOD/2	706	200	52.6	4.73	4.62	16.8	41.4	158	9270

[1] All statistics are calculated by weighting each household by its sampling weight.
[2] Only household averages greater than zero are used to calculate this value (data for all units with soil-lead data are used to calculate the remaining statistics).
[3] Summaries include imputed data for households having no soil-lead concentration data. The method for imputation is presented in Appendix C.
Note: The yard-wide average for a household is the average of the following two statistics: 1) the average of the mid-yard sample results, and 2) the average of results for the dripline and entryway samples.

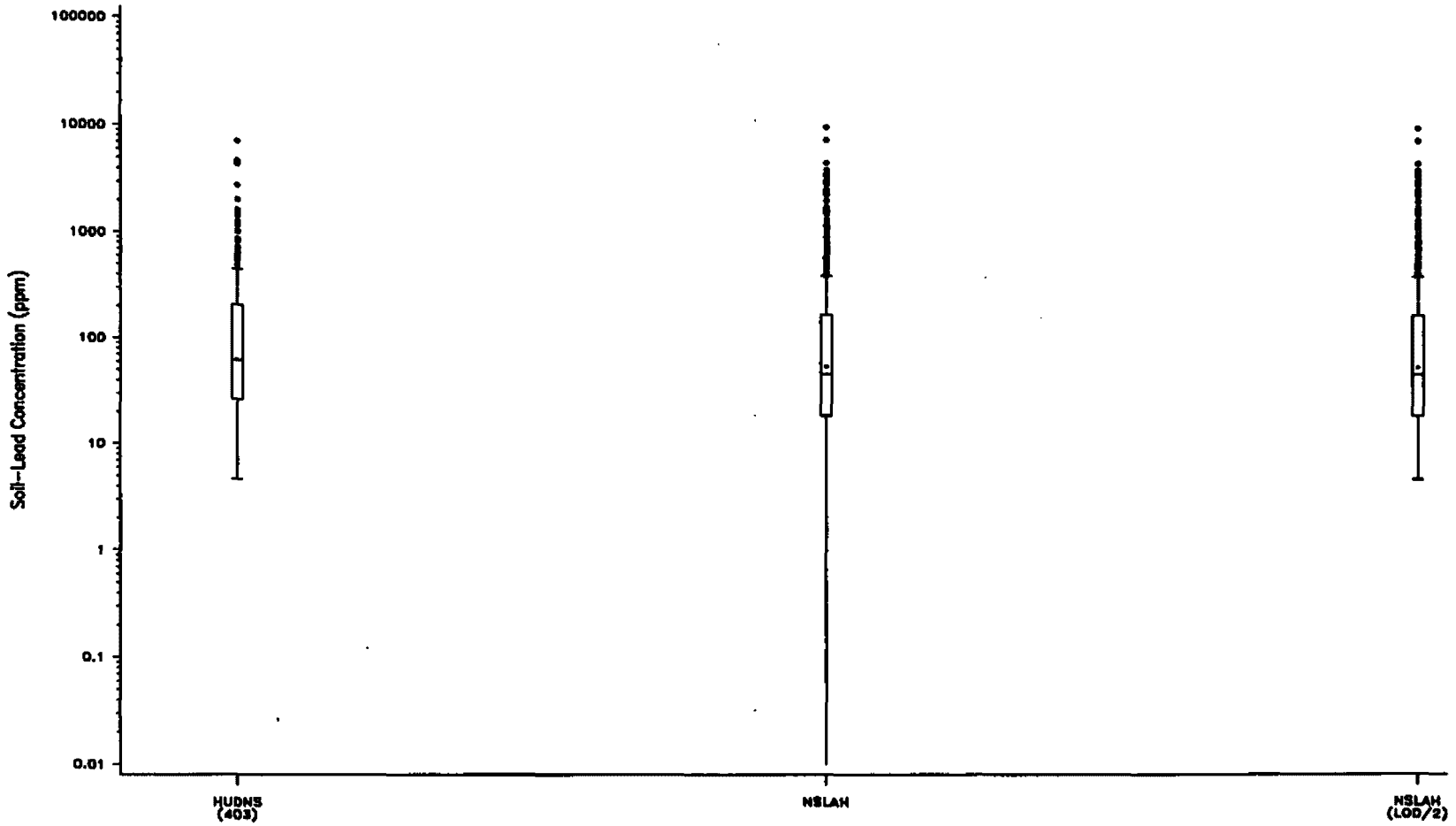

Figure 3-12. Boxplots of Yard-Wide Average Soil-Lead Concentrations (µg/g) As Observed in the §403 Risk Analysis (Using HUD National Survey Data) and in the Interim NSLAH (under 2 approaches to handling not-detected values)

(Note: Boxplots include imputed data but not negative or zero values.)

The detection limit for soil-lead concentrations in the interim NSLAH ranged from 7.2 to 12.4 μg/g, with a mean (and median) of 9.9 μg/g. Of those soil samples in the interim NSLAH with soil-lead concentrations reported, approximately 22% (covering approximately 38% of housing units reporting soil-lead concentrations) had soil results below the detection limit.

In addition to these data summaries that are based solely on the observed data and the sampling weights, it was desired to characterize the national distribution of yardwide average soil-lead concentration in such a way that the percentage of housing where this average exceeds a specified threshold could be estimated. Like what was done in Section 3.2.1.3 above for floor dust-lead loading, this was done for both the HUD National Survey and interim NSLAH data by assuming that these data originate from a lognormal distribution. Then, the fitted distributions and corresponding estimated exceedance percentages were compared between the two surveys. These results are presented in Section 3.2.2.4 below.

Comparisons by housing age category

The distribution of yard-wide average soil-lead concentrations is portrayed for each study according to housing age category in Table 3-19. The importance of housing age on yard-wide average soil-lead concentration is seen in both surveys, as the geometric mean and median concentrations tend to increase with the age of house. The method to handling not-detected values in the interim NSLAH dataset affected the data summaries only slightly, if at all.

Boxplots associated with the data distributions portrayed in Table 3-19 are found in Figure 3-13. Appendix D2 contains tabular summaries and boxplots after excluding imputed data values.

Comparisons by Census region

The distribution of yard-wide average soil-lead concentrations is portrayed for each study according to Census region in Table 3-20. Geometric mean estimates declined from the §403 risk analysis to the interim NSLAH data for each Census region, but the magnitude of the declines were typically small. Observed median values increased from the §403 risk analysis to the interim NSLAH data for the Midwest and West, but these increases were likely due to random chance. No changes from the §403 risk analysis in the pattern of the yard-wide soil-lead concentration distributions across Census regions were observed, with the Northeast continuing to be associated with somewhat higher concentrations compared to the others (although the ranges of observed soil-lead concentrations are comparable across all Census regions).

Boxplots associated with the data portrayed in Table 3-20 are found in Figure 3-14. Appendix D2 contains tabular summaries and boxplots after excluding imputed data values.

Table 3-19. Descriptive Statistics of Yard-Wide Average Soil-Lead Concentration for Households, Presented by Housing Age Category, As Reported in the §403 Risk Analysis Versus the Interim NSLAH Data

Study	How Not-Detected and Negative Data were Handled	Yard-Wide Average Soil-Lead Concentration (µg/g)[1]								
		# Surveyed Units with Positive Averages	Arith-metic Mean	Geo-metric Mean[2]	Geo-metric Std. Dev.[2]	Minimum	25th Percen-tile	Median	75th Percen-tile	Maximum
Units Built Prior to 1940										
§403 Risk Analysis (HUD Natl. Survey)		77	761	463	3.09	17.4	259	569	1030	4620
Interim NSLAH[3]	No adjustment	114	646	297	3.56	12.8	135	294	711	9270
	Replaced by LOD/2	114	646	297	3.56	10.8	135	294	711	9270
Units Built from 1940 - 1959										
§403 Risk Analysis (HUD Natl. Survey)		87	287	92.6	3.15	5.40	44.3	77.3	162	7030
Interim NSLAH[3]	No adjustment	145	264	112	3.43	1.65	45.2	110	273	4340
	Replaced by LOD/2	145	264	114	3.33	4.62	45.2	110	273	4340
Units Built from 1960-1977 (1960 - 1979 for the §403 risk analysis)										
§403 Risk Analysis (HUD Natl. Survey)		120	55.0	32.8	2.56	4.63	19.7	29.7	61.6	996
Interim NSLAH[3]	No adjustment	198	76.7	31.8	3.65	0.00	14.0	29.4	58.3	1120
	Replaced by LOD/2	201	77.2	33.3	3.24	4.83	14.7	29.4	58.3	1120
Units Built After 1977 (after 1979 for the §403 risk analysis)										
§403 Risk Analysis (HUD Natl. Survey)		28	31.3	22.4	2.31	5.35	13.6	21.2	45.0	97.4
Interim NSLAH[3]	No adjustment	168	27.4	15.7	3.19	0.00	6.07	16.0	28.7	474
	Replaced by LOD/2	180	28.2	16.2	2.65	4.65	6.34	14.9	28.7	475
NSLAH Units with Unspecified Year-Built Indicator										
Interim NSLAH[3]	No adjustment	64	175	72.9	4.15	0.00	22.3	63.8	211	2290
	Replaced by LOD/2	66	175	68.9	4.13	4.74	22.4	64.4	211	2290

[1] All statistics are calculated by weighting each household by its sampling weight.

[2] Only household averages greater than zero are used to calculate this value (data for all units with soil-lead data are used to calculate the remaining statistics).

[3] Summaries include imputed data for households having no soil-lead concentration data. The method for imputation is presented in Appendix C.

Note: The yard-wide average for a household is the average of the following two statistics: 1) the average of the mid-yard sample results, and 2) the average of results for the dripline and entryway samples.

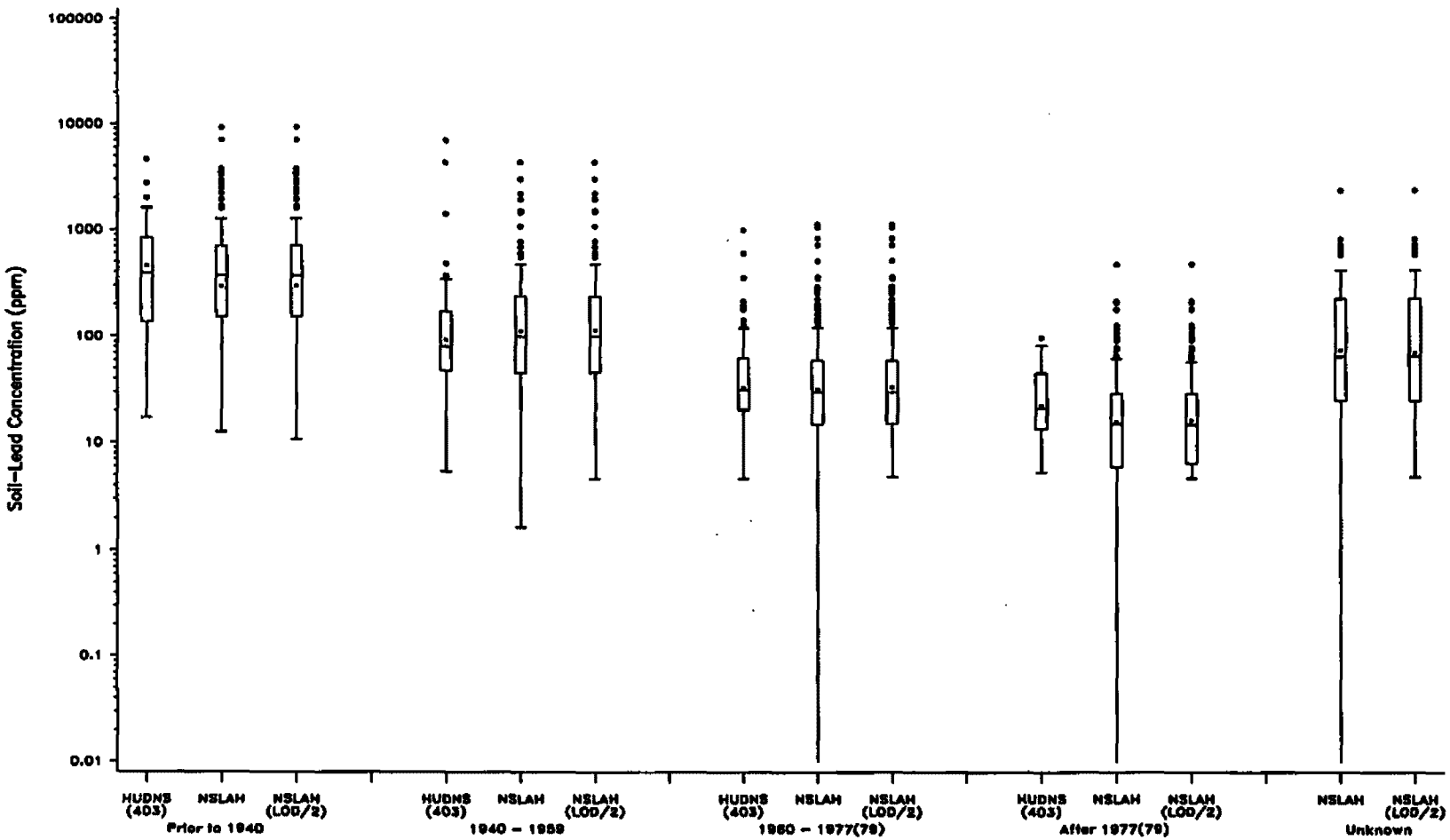

Figure 3-13. Boxplots of Yard-Wide Average Soil-Lead Concentration (µg/g), by Housing Age Category, As Observed in the §403 Risk Analysis (Using HUD National Survey Data) and in the Interim NSLAH (under 2 approaches to handling not-detected values)

(Note: Boxplots include imputed data but not negative or zero values.)

Table 3-20. Descriptive Statistics of Yard-Wide Average Soil-Lead Concentration for Households, Presented by Census Region, As Reported in the §403 Risk Analysis Versus the Interim NSLAH Data

Study	How Not-Detected and Negative Data were Handled	Yard-Wide Average Soil-Lead Concentration (μg/g)[1]								
		# Surveyed Units with Positive Averages	Arith-metic Mean	Geo-metric Mean[2]	Geo-metric Std. Dev.[2]	Minimum	25th Percen-tile	Median	75th Percen-tile	Maximum
Northeast										
§403 Risk Analysis (HUD Natl. Survey)		53	437	206	3.58	14.8	60.1	279	569	4320
Interim NSLAH[3]	No adjustment	109	423	160	4.24	3.92	52.3	176	396	3460
	Replaced by LOD/2	109	423	162	4.16	6.24	52.9	176	396	3460
Midwest										
§403 Risk Analysis (HUD Natl. Survey)		73	404	81.4	6.33	4.63	19.7	51.6	264	2750
Interim NSLAH[3]	No adjustment	149	220	65.5	4.97	0.00	22.1	63.2	206	7070
	Replaced by LOD/2	150	220	65.8	4.71	4.90	22.1	63.2	206	7070
South										
§403 Risk Analysis (HUD Natl. Survey)		134	125	44.5	2.94	5.22	22.6	40.8	79.3	7030
Interim NSLAH[3]	No adjustment	258	162	37.3	4.62	0.00	11.9	27.6	79.2	9270
	Replaced by LOD/2	265	163	36.4	4.38	4.65	13.1	27.9	79.2	9270
West										
§403 Risk Analysis (HUD Natl. Survey)		52	112	34.4	3.92	4.79	14.2	27.2	61.6	2020
Interim NSLAH[3]	No adjustment	173	68.2	30.5	4.36	0.00	12.5	29.4	77.5	776
	Replaced by LOD/2	182	69.0	31.7	3.55	4.62	12.8	29.4	79.3	776

[1] All statistics are calculated by weighting each household by its sampling weight.

[2] Only household averages greater than zero are used to calculate this value (data for all units with soil-lead data are used to calculate the remaining statistics).

[3] Summaries include imputed data for households having no soil-lead concentration data. The method for imputation is presented in Appendix C.

Note: The yard-wide average for a household is the average of the following two statistics: 1) the average of the mid-yard sample results, and 2) the average of results for the dripline and entryway samples.

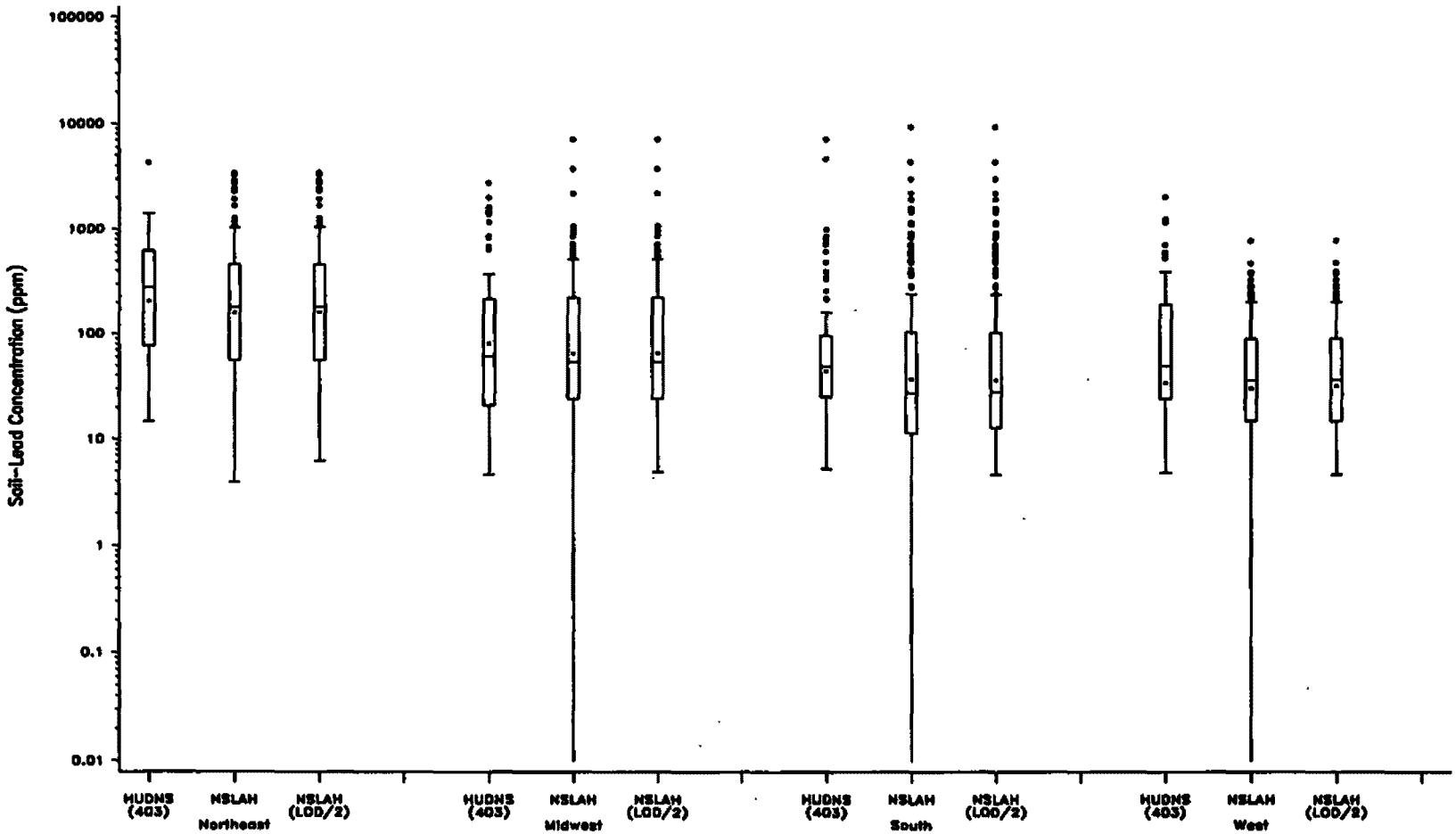

Figure 3-14. Boxplots of Yard-Wide Average Soil-Lead Concentration (µg/g), by Census Region, As Observed in the §403 Risk Analysis (Using HUD National Survey Data) and in the Interim NSLAH (under 2 approaches to handling not-detected values)

(Note: Boxplots include imputed data but not negative or zero values.)

Comparisons by combination of housing age and Census region

Tables 3-21a and 3-21b present descriptive statistics for yard-wide average soil-lead concentration according to the 16 combinations of Census region and housing age category. Table 3-21a reflects the data when no adjustment to not-detected results, while not-detected results are replaced by one-half of the detection limit prior to performing the summaries in Table 3-21b. As the central tendency of the soil-lead concentrations was of primary interest to compare across the different combinations, these tables only contain estimates of the arithmetic and geometric means, geometric standard deviation, and median.

Due to the small numbers of housing units entering into each summary within Tables 3-21a and 3-21b, caution must be taken when making inferences from the results portrayed in these tables. Appendix D2 contains these tabular summaries after excluding imputed data values.

3.2.2.2 Data Summaries for the §403 Risk Analysis Versus Other Studies. This subsection presents data summaries for the 22 studies in Table 3-17 that characterized soil-lead concentrations in urban areas and how these summaries compare to that for the HUD National Survey and to the distribution of yard-wide average soil-lead concentration characterized in the §403 risk analysis. The soil-lead concentration parameters that are summarized in this subsection were specified for each study in Table 3-17.

The 22 studies whose data are considered in this subsection include the three recent studies included in the dust-lead data summaries in Section 3.2.1: Baltimore R&M study (pre-intervention), Rochester Lead-in-Dust study, and HUD Grantees evaluation (pre-intervention data available through 1/99). Figure 3-15 contains boxplots of household average soil-lead concentration for these three studies and the HUD National Survey ("HUDNS"). These boxplots represent yard-wide averages in all cases except the Baltimore R&M study, where only dripline soil samples were collected. Separate boxplots are included for each grantee in the HUD Grantees evaluation[10].

As in Figures 3-7 through 3-10, the left-most three boxplots in Figure 3-15 represent yard-wide average soil-lead concentration data from the HUD National Survey:

- "HUDNS (U)" summarizes the data without regard to sampling weights
- "HUDNS (403)" summarizes the data as used in the §403 risk analysis (e.g., using sampling weights reflecting the 1997 housing stock; incorporating imputed data assigned to housing units with missing data)

[10] "Alam"=Alameda County; "Balt"=Baltimore; "Bos"=Boston; "CA"=California; "Cle"=Cleveland; "MA"=Massachusetts; "MN"=Minnesota; "NJ"=New Jersey; "RI"=Rhode Island; "WI"=Wisconsin; "Milw"=Milwaukee; "Chic"=Chicago; "NYC"=New York City; "VT"=Vermont.

Table 3-21a. Descriptive Statistics of Yard-Wide Average Soil-Lead Concentrations for Households, Presented by Housing Age and Census Region, As Reported in the §403 Risk Analysis Versus the Interim NSLAH Data Where No Adjustments Were Made to Not-Detected Results

Census Region	Study[2]	Housing Age Category	Yard-Wide Average Soil-Lead Concentration[1] (µg/g)				
			# Surveyed Units	Arithmetic Mean	Geometric Mean[3]	Geometric Std. Dev.[3]	Median
Northeast	§403 Risk Anal.	Prior to 1940	26	542	491	1.57	444
	Interim NSLAH		41	877	499	3.22	569
	§403 Risk Anal.	1940 - 1959	17	573	136	4.40	60.1
	Interim NSLAH		23	290	199	2.24	273
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	10	79.1	60.7	2.15	69.7
	Interim NSLAH		21	132	65.5	2.95	50.9
	Interim NSLAH	After 1977	16	57.8	40.0	2.63	38.8
Midwest	§403 Risk Anal.	Prior to 1940	19	1310	941	2.68	1390
	Interim NSLAH		36	498	224	3.34	238
	§403 Risk Anal.	1940 - 1959	21	127	92.6	2.41	123
	Interim NSLAH		36	236	110	3.14	82.0
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	29	42.7	27.1	2.32	23.4
	Interim NSLAH		37	93.8	38.3	3.34	34.6
	§403 Risk Anal.	After 1977 (1979 for §403)	4	13.0	11.5	1.66	12.4
	Interim NSLAH		29	34.1	12.9	3.92	9.36
South	§403 Risk Anal.	Prior to 1940	19	417	174	3.68	159
	Interim NSLAH		26	684	278	3.74	186
	§403 Risk Anal.	1940 - 1959	33	327	83.1	3.27	81.0
	Interim NSLAH		48	364	96.6	4.40	77.9
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	64	54.6	36.5	2.30	34.7
	Interim NSLAH		79	68.7	26.9	3.60	26.1
	§403 Risk Anal.	After 1977 (1979 for §403)	18	38.5	29.7	2.11	25.0
	Interim NSLAH		81	22.2	15.7	2.45	15.0
West	§403 Risk Anal.	Prior to 1940	13	594	295	3.76	394
	Interim NSLAH		11	155	122	2.23	158
	§403 Risk Anal.	1940 - 1959	16	96.8	72.1	2.19	60.4
	Interim NSLAH		38	143	86.9	3.08	90.3
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	17	56.2	23.8	3.02	20.0
	Interim NSLAH		61	47.4	24.7	3.81	26.9
	§403 Risk Anal.	After 1977 (1979 for §403)	6	21.7	15.0	2.34	13.6
	Interim NSLAH		42	17.3	10.6	3.54	9.53

[1] All statistics are calculated by weighting each household by its sampling weight.

[2] Summaries include imputed data for households having no soil-lead concentration data. The method for imputation is presented in Appendix C.

[3] Only household averages greater than zero are used to calculate this value (data for all units with soil-lead data are used to calculate the remaining statistics).

Note: The yard-wide average for a household is the average of the following two statistics: 1) the average of the mid-yard sample results, and 2) the average of results for the dripline and entryway samples.

Table 3-21b. Descriptive Statistics of Yard-Wide Average Soil-Lead Concentrations for Households, Presented by Housing Age and Census Region, As Reported in the §403 Risk Analysis Versus the Interim NSLAH Data Where Not-Detected Results Were Replaced by LOD/2

Census Region	Study[2]	Housing Age Category	Yard-Wide Average Soil-Lead Concentration[1] (µg/g)				
			# Surveyed Units	Arithmetic Mean	Geometric Mean	Geometric Std. Dev.	Median
Northeast	§403 Risk Anal.	Prior to 1940	26	542	491	1.57	444
	Interim NSLAH		41	877	497	3.26	569
	§403 Risk Anal.	1940 - 1959	17	573	136	4.40	60.1
	Interim NSLAH		23	290	199	2.24	273
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	10	79.1	60.7	2.15	69.7
	Interim NSLAH		21	132	65.4	2.96	50.9
	Interim NSLAH	After 1977	16	58.1	42.0	2.36	38.8
Midwest	§403 Risk Anal.	Prior to 1940	19	1310	941	2.68	1390
	Interim NSLAH		36	498	224	3.34	238
	§403 Risk Anal.	1940 - 1959	21	127	92.6	2.41	123
	Interim NSLAH		36	236	111	3.11	82.0
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	29	42.7	27.1	2.32	23.4
	Interim NSLAH		37	94.1	39.0	3.27	34.6
	§403 Risk Anal.	After 1977 (1979 for §403)	4	13.0	11.5	1.66	12.4
	Interim NSLAH		30	34.7	14.0	3.06	9.67
South	§403 Risk Anal.	Prior to 1940	19	417	174	3.68	159
	Interim NSLAH		26	684	278	3.74	186
	§403 Risk Anal.	1940 - 1959	33	327	83.1	3.27	81.0
	Interim NSLAH		48	364	97.7	4.34	77.9
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	64	54.6	36.5	2.30	34.7
	Interim NSLAH		81	69.4	27.8	3.24	26.1
	§403 Risk Anal.	After 1977 (1979 for §403)	18	38.5	29.7	2.11	25.0
	Interim NSLAH		84	22.7	15.4	2.29	14.7
West	§403 Risk Anal.	Prior to 1940	13	594	295	3.76	394
	Interim NSLAH		11	155	122	2.21	158
	§403 Risk Anal.	1940 - 1959	16	96.8	72.1	2.19	60.4
	Interim NSLAH		38	143	89.8	2.78	90.3
	§403 Risk Anal.	1960 -1977 (1960-79 for §403)	17	56.2	23.8	3.02	20.0
	Interim NSLAH		62	48.0	27.8	2.91	26.9
	§403 Risk Anal.	After 1977 (1979 for §403)	6	21.7	15.0	2.34	13.6
	Interim NSLAH		50	18.9	12.1	2.42	11.2

[1] All statistics are calculated by weighting each household by its sampling weight.

[2] Summaries include imputed data for households having no soil-lead concentration data. The method for imputation is presented in Appendix C.

Note: The yard-wide average for a household is the average of the following two statistics: 1) the average of the mid-yard sample results, and 2) the average of results for the dripline and entryway samples.

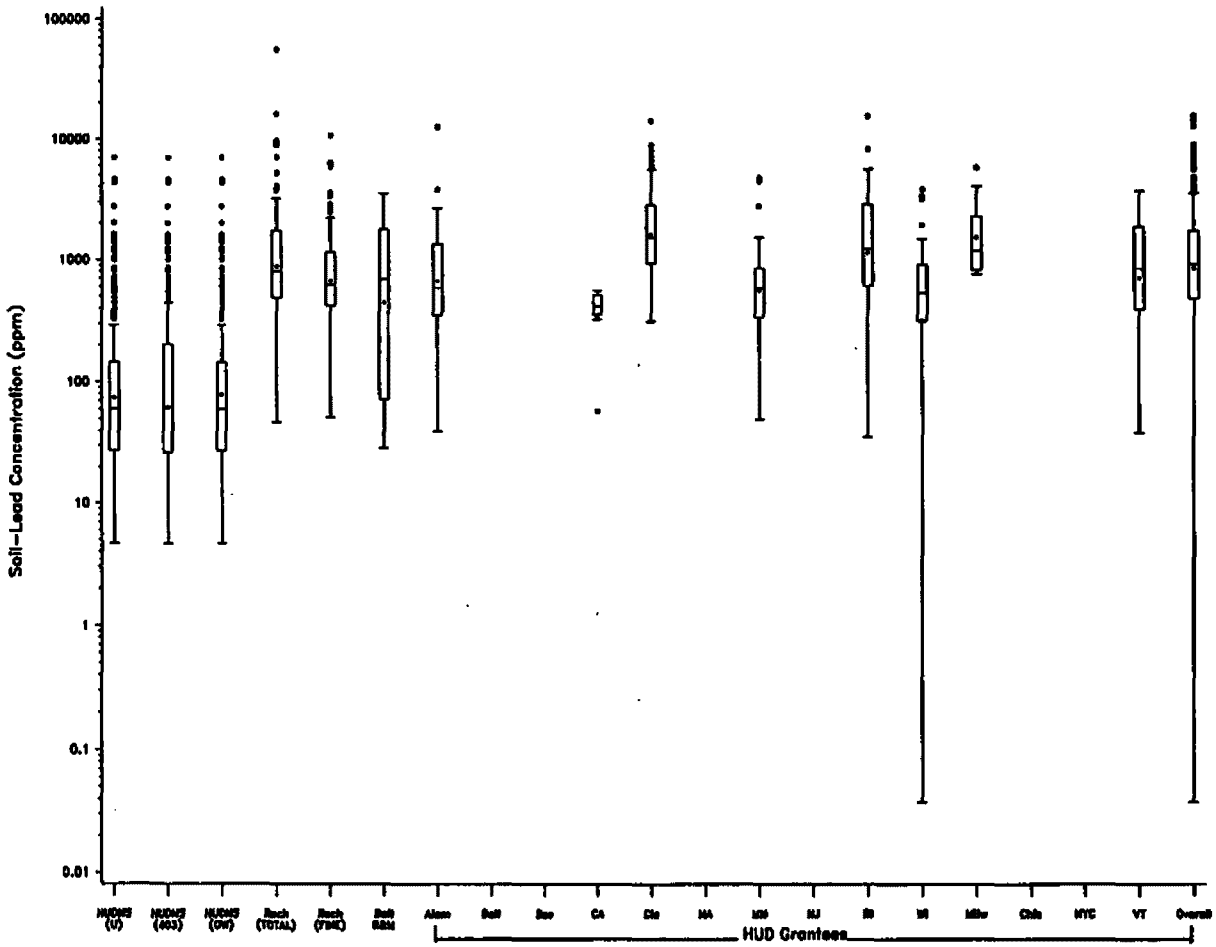

Figure 3-15. Boxplots of Household Average Soil-Lead Concentrations (μg/g) for Houses in the HUD National Survey, Baltimore R&M Study, Rochester Lead-in-Dust Study, and Grantees Within the HUD Grantees Evaluation

(Note: Household averages represent yard-wide averages except for the Baltimore R&M study, where only dripline soil samples were collected. See text for definitions of labels along the horizontal axis.)

- "HUDNS (OW)" summarizes the data weighted according to the original weights assigned in the survey.

As soil samples were sieved into fine and coarse fractions in the Rochester study, Figure 3-15 includes two boxplots for the Rochester soil-lead data. The boxplot labeled "Rochester (FINE)" summarizes household average soil-lead concentration considering only the fine-sieved fraction of the collected soil samples. The boxplot labeled "Rochester (TOTAL)" summarizes estimated household average soil-lead concentration assuming the total soil sample was analyzed. Total soil-lead concentration for each sample was estimated as the average of the reported concentrations for the fine and coarse fractions of the sample, with the coarse fraction result weighted three times that of the fine sample result (see Appendix E for the derivation of this estimate using data from the Milwaukee study). Estimating soil-lead concentration in the total soil sample was intended to allow soil-lead data from the Rochester study to be more comparable to data from the other studies in which no sieve-fractions were calculated.

Figure 3-15 shows that while the ranges of average soil-lead concentrations among the study households tended to overlap from study to study, the distributions based upon the HUD National Survey data (including the §403 risk analysis) tended to be shifted lower than for the other studies.

Figure 3-16 contains a graphical presentation of how the distribution of household average soil-lead concentration in other selected studies listed in Table 3-17 compare with the distributions based upon the HUD National Survey data (i.e., the same three distributions portrayed in the boxplots labeled "HUDNS" in Figure 3-15). The studies selected for Figure 3-15 were among those in which an average soil-lead concentration for a particular area could be determined. As only summary statistics for many of the studies in Table 3-17 were available from the references or prior literature reviews, boxplots like those in Figure 3-15 could not be created for these other studies. Instead, specific descriptive statistics (when cited in the references) are plotted in Figure 3-16 for each study by using plotting symbols that indicate the type of statistic. These statistics, with their plotting symbols following in parentheses, are the minimum (MIN), 25th percentile (25th), median (50th), 75th percentile (75th), maximum (MAX), and geometric mean (GM) soil-lead concentrations. In studies where the arithmetic mean is specified instead of the geometric mean, the arithmetic mean (AVE) was plotted. The vertical dashed line in Figure 3-16 separates results based on the HUD National Survey data from the results for the other studies.

Yard-wide average soil-lead concentration (or an average that is not specific to a given location) were available or could be calculated within eight of these studies (one being the 3-Cities study, which consisted of three sub-studies). Table 3-22a presents values of descriptive statistics (e.g., geometric mean, minimum, maximum, selected percentiles) for yard-wide average soil-lead concentration within these studies. This table also includes the estimated number of averages represented in the descriptive statistics that exceed a given soil-lead concentration threshold (400, 1200, 2000, and 5000 μg/g). The following features can be found within this table:

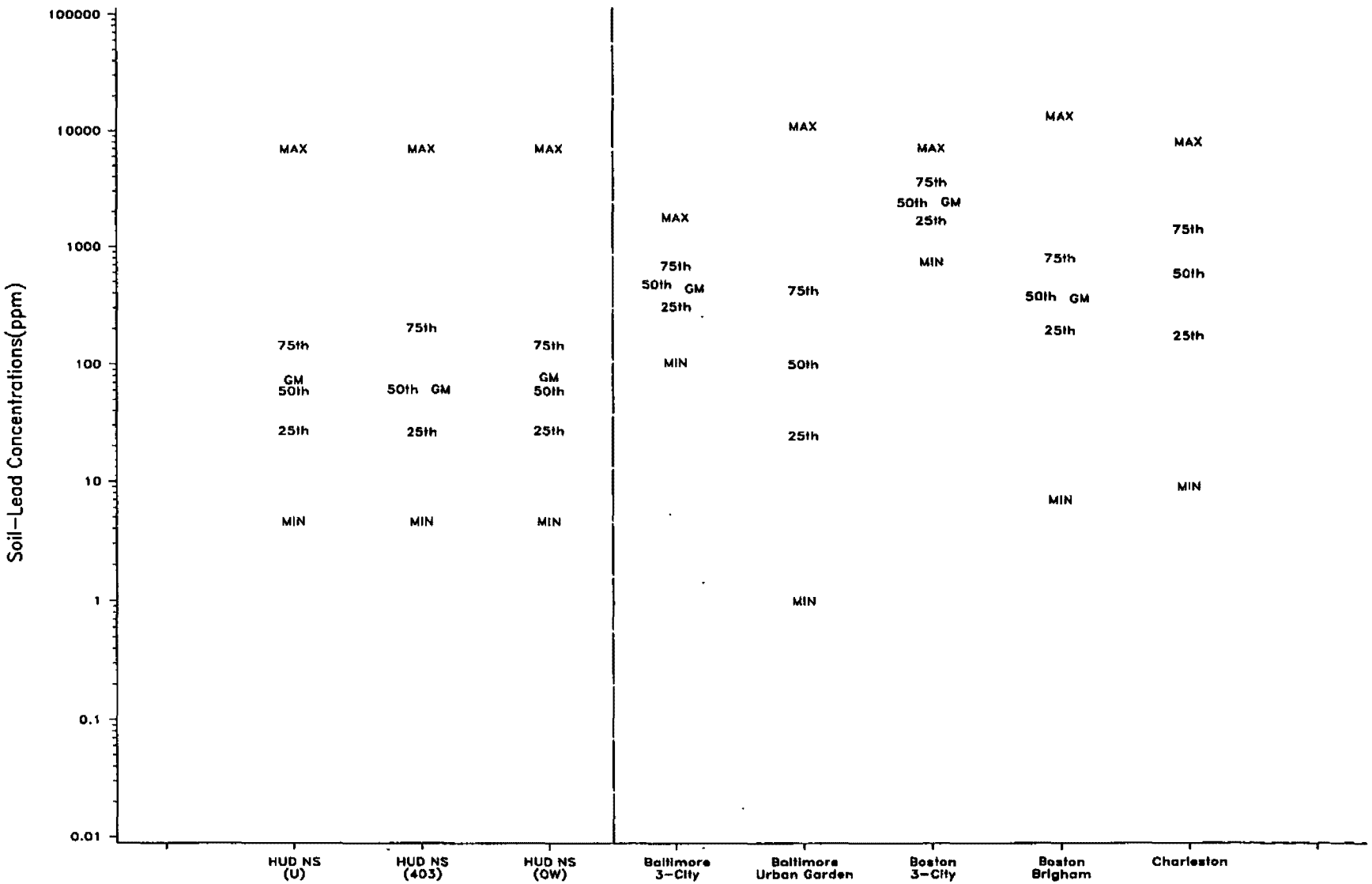

Figure 3-16. Summary Statistics of Average Household Soil-Lead Concentrations (μg/g) for Selected Studies as Compared to Summaries Based on Data from the HUD National Survey

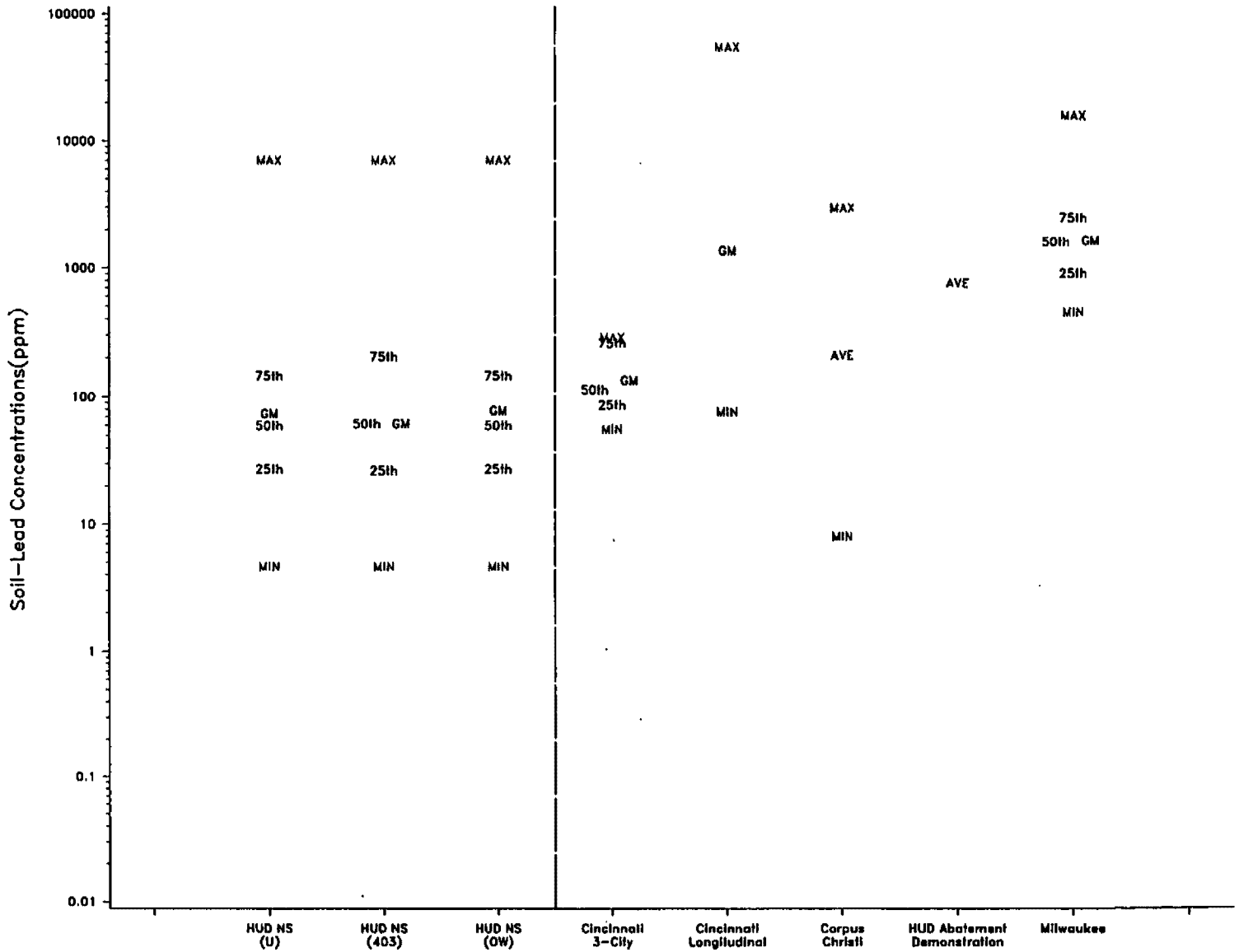

Figure 3-16. (cont.)

Table 3-22a. Descriptive Statistics of Yard-Wide Average Soil-Lead Concentrations, According to Study and Within Specific Subsets of the Sampled Housing Within a Study

Study	Subset of Units or Type of Soil/cover	Yard-Wide Average Soil-Lead Concentration (µg/g)								Percentage of Homes with Yard-Wide Average Soil-Lead Concentration ≥			
		N	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
HUD National Survey (unweighted) [1]	All Units	250	74.0	4.0	4.6	27.2	59.9	145.1	7025	12.0%	4.8%	2.4%	0.4%
	Big City/ Metro	67	80.6	3.3	5.4	35.3	70.9	159.7	1463	10.4%	4.5%	0.0%	0.0%
	Big City/ Suburb	60	72.0	3.6	4.8	30.4	60.6	171.0	2019	10.0%	1.7%	1.7%	0.0%
	Small City/ Metro	42	112.0	6.2	5.2	26.0	93.9	585.6	7025	26.2%	11.9%	7.1%	2.4%
	Small City/ Suburb	24	65.1	3.6	6.7	28.6	50.8	118.4	4318	8.3%	4.2%	4.2%	0.0%
	Non-Metro	57	53.5	3.8	4.6	21.7	45.2	126.2	2002	7.0%	3.5%	1.8%	0.0%
	City	109	91.5	4.3	5.2	32.0	77.3	213.7	7025	16.5%	7.3%	2.8%	0.9%
	Non-City	141	62.8	3.7	4.6	25.9	53.8	135.9	4318	8.5%	2.8%	2.1%	0.0%
	Northeast	31	144.5	3.7	14.8	53.8	115.2	357.6	4318	22.6%	6.5%	3.2%	0.0%
	Midwest	63	75.0	4.6	4.6	21.7	58.9	162.3	2752	12.7%	9.5%	3.2%	0.0%
	South	111	60.6	3.5	5.2	26.3	52.7	115.1	7025	7.2%	1.8%	1.8%	0.9%
	West	45	75.0	4.4	4.8	25.9	60.4	197.9	2019	15.6%	4.4%	2.2%	0.0%
HUD National Survey ($403 RA) [2]	All Units	284	61.9	4.5	4.6	26.5	61.8	203.6	7025	13.2%	4.7%	2.5%	0.2%
	Big City/ Metro	96	87.5	4.2	5.4	43.0	94.5	313.3	1463	17.9%	2.5%	0.0%	0.0%
	Big City/ Suburb	73	56.2	3.8	4.8	28.6	61.1	161.8	2019	10.2%	0.7%	0.7%	0.0%
	Small City/ Metro	50	77.9	5.5	5.2	24.5	83.3	391.9	7025	19.4%	11.7%	6.5%	1.3%
	Small City/ Suburb	28	46.2	3.4	6.7	26.0	49.2	92.9	4318	4.9%	3.8%	3.8%	0.0%
	Non-Metro	65	46.9	4.9	4.6	17.7	35.9	105.0	2002	11.0%	6.8%	3.4%	0.0%
	City	146	83.3	4.7	5.2	34.6	88.7	313.3	7025	18.5%	6.4%	2.7%	0.5%
	Non-City	166	50.5	4.2	4.6	23.4	51.9	123.3	4318	9.5%	3.6%	2.3%	0.0%
	Northeast	53	205.8	3.6	14.8	77.3	278.9	627.9	4318	34.8%	4.4%	2.6%	0.0%
	Midwest	73	81.4	6.3	4.6	21.1	61.1	216.7	2752	21.2%	14.9%	6.9%	0.0%
	South	134	44.5	2.9	5.2	25.4	49.2	97.4	7025	3.8%	0.8%	0.8%	0.5%
	West	52	34.4	3.9	4.8	24.3	49.7	191.8	2019	6.3%	1.9%	1.0%	0.0%

Table 3-22a. (cont.)

Study	Subset of Units or Type of Soil/cover	Yard-Wide Average Soil-Lead Concentration (µg/g)								Percentage of Homes with Yard-Wide Average Soil-Lead Concentration ≥			
		N	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
HUD National Survey (original weights)[3]	All Units	250	78.1	4.5	4.6	27.2	59.9	145.1	7025	15.3%	6.8%	3.7%	0.3%
	Big City/ Metro	67	74.0	3.3	5.4	35.3	70.9	159.7	1463	12.6%	2.3%	0.0%	0.0%
	Big City/ Suburb	60	69.1	3.9	4.8	30.4	60.6	171.0	2019	11.3%	1.1%	1.1%	0.0%
	Small City/ Metro	42	121.9	6.5	5.2	26.0	93.9	585.6	7025	29.1%	18.2%	10.1%	1.9%
	Small City/ Suburb	24	60.7	3.6	6.7	28.6	50.8	118.4	4318	7.3%	5.6%	5.6%	0.0%
	Non-Metro	57	77.5	5.1	4.6	21.7	45.2	125.2	2002	16.4%	10.2%	5.1%	0.0%
	City	109	90.9	4.6	5.2	32.0	77.3	213.7	7025	19.4%	8.8%	4.1%	0.8%
	Non-City	141	70.5	4.3	4.6	25.9	53.8	135.9	4318	12.6%	5.4%	3.4%	0.0%
	Northeast	31	150.2	3.6	14.8	53.8	115.2	357.6	4318	27.5%	4.1%	3.5%	0.0%
	Midwest	63	113.3	6.0	4.6	21.7	58.9	162.3	2752	24.1%	19.0%	8.9%	0.0%
	South	111	57.9	3.3	5.2	26.3	52.7	115.1	7025	6.6%	1.3%	1.3%	0.9%
	West	45	47.3	4.0	4.8	25.9	60.4	197.9	2019	9.0%	2.7%	1.4%	0.0%
Baltimore Urban Garden		422	NA	NA	1.0	24.5[4]	100.0	421.0[5]	10900	NA	NA	NA	NA
Baltimore 3-City	Top 2 cm	181[6]	442.3	1.7	103.7	308.4	479.3	688.4	1793	59.7%	2.8%	0.0%	0.0%
Boston 3-City	Top 2 cm	101[6]	2430.9	1.6	744.3	1678.0	2380.0	3600.0	7070	100.0%	93.1%	65.3%	6.9%
Boston Brigham and Women		195	360.8	3.3	7.0	193.0	374.0	796.0	13237	49.2%	13.8%	6.7%	1.0%
Cincinnati 3-City	Overall	7[6]	133.1	1.9	55.8	86.4	112.9	257.3	285	0.0%	0.0%	0.0%	0.0%
	Full Grass	4[6]	138.9	2.4	49.9	73.9	144.9	294.3	397	0.0%	0.0%	0.0%	0.0%
	>1/2 Grass	6[6]	115.7	1.5	71.5	77.8	126.3	151.6	182	0.0%	0.0%	0.0%	0.0%
	<1/2 Grass	4[6]	149.6	2.3	56.8	91.6	142.1	300.1	442	25.0%	0.0%	0.0%	0.0%
	All Bare	3[6]	103.6	1.6	60.8	60.8	118.1	154.7	155	0.0%	0.0%	0.0%	0.0%
HUD Grantees Evaluation	All Grantees	314	857.5	3.8	0.0	479.0	920.5	1730.0	15535	82.8%	40.1%	21.0%	4.1%
	Alameda	58	669.8	2.5	39.5	352.5	588.5	1348.0	12848	70.7%	34.5%	6.2%	1.7%
	California	8	341.8	2.1	58.0	357.5	415.8	512.5	560	62.5%	0.0%	0.0%	0.0%
	Cleveland	99	1620.7	2.2	315.0	940.0	1545.0	2840.0	14180	99.0%	61.6%	38.4%	8.1%
	Minnesota	41	563.1	2.4	49.5	339.5	591.5	857.5	4800	70.7%	17.1%	7.3%	0.0%
	Rhode Island	40	1146.0	3.3	35.5	608.5	1227.8	2875.0	15535	87.5%	52.5%	32.5%	7.5%
	Wisconsin	38	318.3	11.8	0.0	316.0	536.8	917.0	3852	71.1%	15.8%	7.9%	0.0%

Table 3-22a. (cont.)

Study	Subset of Units or Type of Soil/cover	Yard-Wide Average Soil-Lead Concentration (µg/g)								Percentage of Homes with Yard-Wide Average Soil-Lead Concentration ≥			
		N	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
	Milwaukee	10	1530.5	2.0	766.0	829.0	1184.8	2287.5	5800	100.0%	40.0%	30.0%	10.0%
	Vermont	20	707.9	3.0	38.5	393.8	850.9	1868.8	3695	75.0%	35.0%	15.0%	0.0%
Milwaukee		92	1640.5	2.1	449.0	903.5	1605.5	2472.0	15814	100.0%	63.0%	32.6%	7.6%
Omaha	Urban Commercial	69	262.0	NA	53.0	NA	NA	NA	1615	NA	NA	NA	NA
	Urban Mixed	56	339.0	NA	20.0	NA	NA	NA	4792	NA	NA	NA	NA
	Suburban	51	81.0	NA	16.0	NA	NA	NA	341	NA	NA	NA	NA
Rochester Lead-in-Dust	Total soil	82	880.2	3.5	46.7	487.5	807.8	1736.6	55617	76.8%	32.9%	22.0%	9.8%
	Fine soil only	82	670.8	2.7	51.1	419.0	626.8	1150.5	10721	79.3%	23.2%	12.2%	3.7%

NA = Not Available

1 Mass-weighted arithmetic average soil-lead concentration as reported in the HUD National Survey; summarized without weighting by sample weights.
2 Mass-weighted arithmetic average soil-lead concentration as calculated in Chapter 3 of the 403 risk analysis; summarized by weighting each average to reflect the 1997 U.S. housing stock.
3 Mass-weighted arithmetic average soil-lead concentration as reported in the HUD National Survey; summarized by weighting with the National Survey sample weights.
4 20th percentile
5 80th percentile
6 An initial unweighted arithmetic average of soil lead levels at the specified locations was taken prior to calculation of statistics within this table. The number in this column represents the number of properties, not necessarily the number of houses.

- For the HUD National Survey data, which are associated with sampling weights from the original survey and revised sampling weights for the §403 risk analyses, all results in Table 3-22a are portrayed three times: under each of these two sets of weights as well as without regard to weights.

- For the HUD National Survey data, which are associated with sampling weights from the original survey and revised sampling weights for the §403 risk analyses, all results in Table 3-22a are portrayed three times: under each of these two sets of weights as well as without regard to weights.

- In addition to summarizing results across all housing units or samples in a study, results for selected studies are also summarized for specific subsets of housing units, soil types, or soil samples. In particular, HUD National Survey results are portrayed according to urbanicity and Census region, results from the HUD Grantees evaluation are portrayed by grantee, and the Rochester study results are portrayed for the fine soil fraction as well as for total soil.

Refer to Table 3-17 to verify the types of results being summarized in Table 3-22a (i.e., housing unit averages versus averages for single analytical samples).

Table 3-22b contains the same descriptive statistics as those portrayed in Table 3-22a, but they represent average soil-lead concentration for specific locations, such as dripline, play areas, remote areas, geographical areas, and other locations that were considered within the individual studies. As in Table 3-22a, the statistics in Table 3-22b are given over the entire study, as well as for specified sets of units that are determined by urbanicity and other factors.

Summary statistics by housing age category

As housing age category is generally regarded as an important influence on soil-lead concentrations, the above summaries are also presented according to the housing age categories considered in the HUD National Survey (pre-1940, 1940-1959, 1960-1979, post-1979). Figure 3-17 presents boxplots for pre-1980 housing data from the HUD National Survey and the Rochester Lead-in-Dust study (total soil and fine soil), non-control houses in the Baltimore R&M study, and pre-1978 data from the HUD Grantees evaluation (data combined across grantees). As all non-control units in the Baltimore R&M study were built prior to 1941, the only boxplot for this study in Figure 3-17 appears in the "pre-1940" category. Caution must be taken when interpreting results in Figure 3-17 for the Rochester study, as the actual age of certain houses may be older than what was specified in the Rochester study database (see Section 3.3.1.3 of the §403 risk analysis report).

Many of the other studies listed in Table 3-17 did not have information readily available on housing age. Thus, no corresponding figure portraying distributions according to housing age was prepared to represent these other studies.

Table 3-22b. Descriptive Statistics of Average Soil-Lead Concentrations in Specific Yard Areas and/or for Certain Subsets of the Sampled Housing Within a Study

Study	Yard Area/ Subset of Housing Units		Average Soil-Lead Concentration (µg/g)								Percentag of Homes with Soil-Lead Concentration ≥			
			N	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
HUD National Survey (unweighted) [1]	Dripline/ Entryway	All Units	263	91.3	4.3	5.2	32.4	75.2	237.3	13596	16.4%	4.9%	3.0%	1.1%
		Big City/ Metro	76	101.8	3.7	7.9	40.2	81.5	226.9	2571	17.1%	5.3%	3.9%	0.0%
		Big City/ Suburb	62	89.5	4.0	7.5	29.1	82.0	263.3	1661	19.4%	1.6%	0.0%	0.0%
		Small City/ Metro	44	146.9	6.7	5.2	31.8	127.2	695.6	13596	31.8%	11.4%	6.8%	6.8%
		Small City/ Suburb	24	83.5	3.3	10.5	36.1	71.1	148.5	1684	8.3%	4.2%	0.0%	0.0%
		Non-Metro	57	57.9	3.8	5.6	26.0	45.5	131.9	3999	3.5%	3.5%	3.5%	0.0%
		City	120	116.4	4.7	5.2	37.8	85.6	294.0	13596	22.5%	7.5%	5.0%	2.5%
		Non-City	143	74.4	3.8	5.6	29.4	67.4	178.8	3999	11.2%	2.8%	1.4%	0.0%
		Northeast	38	212.5	3.9	20.6	75.2	209.5	534.7	2571	36.8%	13.2%	7.9%	0.0%
		Midwest	66	95.2	4.8	5.8	31.7	73.1	261.6	5336	18.2%	6.1%	4.5%	1.5%
		South	113	70.1	3.8	5.2	31.8	62.5	126.0	13596	8.0%	3.5%	1.8%	1.8%
		West	46	81.8	4.3	7.5	26.6	65.2	263.3	1149	17.4%	0.0%	0.0%	0.0%
HUD National Survey (403 RA) [2]	Dripline/ Entryway	All Units	312	72.7	4.6	5.2	31.8	76.7	245.7	13596	15.5%	4.0%	2.9%	1.1%
		Big City/ Metro	96	102.4	4.4	7.9	44.2	91.9	426.0	2571	0.0%	1.7%	1.2%	0.0%
		Big City/ Suburb	73	68.0	3.9	7.5	28.9	78.7	242.0	1661	16.7%	0.5%	0.0%	0.0%
		Small City/ Metro	50	96.2	6.0	5.2	27.6	124.7	589.2	13596	22.2%	8.7%	6.5%	6.5%
		Small City/ Suburb	28	60.6	2.9	10.5	35.4	70.0	108.2	1684	4.9%	3.8%	0.0%	0.0%
		Non-Metro	65	48.7	4.9	5.6	20.3	37.6	112.9	3999	6.8%	6.8%	6.8%	0.0%
		City	146	99.7	5.0	5.2	37.3	96.1	439.2	13596	22.5%	4.6%	3.4%	2.7%
		Non-City	166	58.5	4.1	5.6	27.5	61.9	131.9	3999	10.7%	3.5%	2.6%	0.0%
		Northeast	53	251.0	3.6	20.6	85.3	373.2	1007.0	2571	48.5%	5.1%	1.8%	0.0%
		Midwest	73	94.5	6.3	5.8	28.4	61.9	283.0	5336	20.6%	10.9%	10.4%	3.5%
		South	134	51.8	3.3	5.2	29.4	59.4	123.3	13596	4.3%	1.7%	0.8%	0.8%
		West	52	40.3	3.7	7.5	24.7	56.6	216.4	1149	6.8%	0.0%	0.0%	0.0%

Table 3-22b. (cont.)

Study	Yard Area/ Subset of Housing Units		Average Soil-Lead Concentration (µg/g) N	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	Percentag of Homes with Soil-Lead Concentration 2 400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
HUD National Survey (HUD NS weights) [3]	Dripline/ Entryway	All Units	263	92.8	4.6	5.2	32.4	75.2	237.3	13596	18.5%	5.7%	4.3%	1.6%
		Big City/ Metro	76	90.2	3.7	7.9	40.2	81.5	226.9	2571	16.7%	2.3%	1.6%	0.0%
		Big City/ Suburb	62	86.1	4.1	7.5	29.1	82.0	263.3	1681	22.5%	0.7%	0.0%	0.0%
		Small City/ Metro	44	147.4	7.0	5.2	31.8	127.2	695.6	13596	33.2%	12.9%	9.7%	9.7%
		Small City/ Suburb	24	75.2	3.1	10.5	36.1	71.1	148.5	1684	7.3%	5.6%	0.0%	0.0%
		Non-Metro	57	82.0	5.1	5.6	26.0	45.5	131.9	3999	10.2%	10.2%	10.2%	0.0%
		City	120	109.9	5.0	5.2	37.8	85.6	294.0	13596	23.3%	6.6%	4.9%	3.9%
		Non-City	143	82.5	4.3	5.6	29.4	67.4	178.8	3999	15.1%	5.2%	3.9%	0.0%
		Northeast	38	195.9	3.5	20.6	75.2	209.5	534.7	2571	42.0%	6.5%	2.2%	0.0%
		Midwest	66	132.0	6.0	5.8	31.7	73.1	261.6	5336	24.4%	13.6%	13.0%	4.3%
		South	113	66.9	3.6	5.2	31.8	62.5	126.0	13596	7.2%	2.8%	1.3%	1.3%
		West	46	53.2	3.9	7.5	26.6	65.2	263.3	1149	9.6%	0.0%	0.0%	0.0%
Baltimore R&M	Dripline		28	444.5	5.1	28.9	71.5	686.9	1767.5	3539	60.7%	42.9%	10.7%	0.0%
Baltimore 3-City	Dripline	Top 2 cm	196 [10]	635.9	2.0	96.0	390.2	666.6	1035.6	4400	72.4%	18.9%	2.6%	0.0%
	Midyard	Top 2 cm	183 [10]	287.0	1.9	31.0	199.0	286.0	425.0	2500	29.0%	1.1%	0.5%	0.0%
	Remote	Top 2 cm	197 [10]	337.0	1.7	77.2	230.0	351.8	465.6	1850	35.5%	1.0%	0.0%	0.0%
CAP Study	Dripline	All Homes	117 [7]	182.1	2.7	11.0	97.8	190.4	331.8	3351	20.5%	3.4%	0.9%	0.0%
		Unabated Homes	37 [7]	91.3	2.6	11.0	55.8	112.0	137.0	1016	5.4%	0.0%	0.0%	0.0%
		Abated Homes	80 [7]	250.6	2.3	51.4	122.6	257.1	412.4	3351	27.5%	5.0%	1.3%	0.0%
	Entryway	All Homes	109 [7]	143.5	2.7	4.6	72.9	148.5	265.2	1068	17.4%	0.0%	0.0%	0.0%
		Unabated Homes	37 [7]	101.3	3.3	4.6	47.4	129.5	215.8	655	13.5%	0.0%	0.0%	0.0%
		Abated Homes	72 [7]	171.6	2.3	42.9	88.0	152.8	343.4	1068	19.4%	0.0%	0.0%	0.0%
	Remote	All Homes	120 [7]	120.4	2.1	15.0	70.3	120.0	197.6	1073	5.0%	0.0%	0.0%	0.0%
		Unabated Homes	39 [7]	85.3	2.2	15.0	53.4	87.9	131.3	1073	2.6%	0.0%	0.0%	0.0%
		Abated Homes	81 [7]	142.2	1.9	28.7	79.1	150.2	228.8	615	6.2%	0.0%	0.0%	0.0%
California	Oakland		292	897.0	NA	56.0	NA	880.0	NA	88176	NA	NA	NA	NA
	Los Angeles		327	188.0	NA	30.0	NA	190.0	NA	1973	NA	NA	NA	NA
	Sacramento		227	234.0	NA	26.0	NA	229.0	NA	2664	NA	NA	NA	NA

Table 3-22b. (cont.)

Study	Yard Area/ Subset of Housing Units		Average Soil-Lead Concentration (µg/g)								Percentag of Homes with Soil-Lead Concentration z			
			N	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
Cincinnati 3-City	Building	Overall	100 [10]	233.9	4.6	7.1	91.3	260.9	842.4	6340	35.0%	17.0%	7.0%	1.0%
		Full Grass	22 [10]	286.7	5.7	16.9	55.4	249.2	1554.0	4533	45.5%	27.3%	13.6%	0.0%
		>1/2 Grass	35 [10]	303.5	4.3	6.2	102.0	290.9	1133.6	4963	34.3%	22.9%	8.6%	0.0%
		<1/2 Grass	56 [10]	184.2	4.8	7.1	57.6	219.9	569.8	4897	33.9%	10.7%	5.4%	0.0%
		All Bare	46 [10]	410.1	3.3	35.0	214.4	296.9	1130.7	7602	43.5%	23.9%	13.0%	2.2%
	Bare Areas	Overall	74 [10]	220.9	5.5	5.4	77.1	223.9	800.2	4552	39.2%	17.6%	12.2%	0.0%
		Full Grass	3 [12]	340.2	17.1	12.9	12.9	1491.3	2047.3	2047	66.7%	66.7%	33.3%	0.0%
		>1/2 Grass	8 [10]	106.7	2.9	42.3	52.1	77.6	163.1	1128	12.5%	0.0%	0.0%	0.0%
		<1/2 Grass	8 [12]	30.1	4.4	7.8	9.7	21.0	70.1	609	12.5%	0.0%	0.0%	0.0%
		All Bare	63 [10]	272.6	4.7	5.4	111.1	256.7	861.6	4552	41.3%	17.5%	12.7%	0.0%
	Play Area	Overall	11 [10]	94.6	1.9	20.0	70.7	103.4	155.4	192	0.0%	0.0%	0.0%	0.0%
		Full Grass	5 [10]	122.1	1.6	69.3	80.2	129.1	164.3	230	0.0%	0.0%	0.0%	0.0%
		>1/2 Grass	7 [12]	86.3	1.4	55.8	68.3	82.0	123.4	124	0.0%	0.0%	0.0%	0.0%
		<1/2 Grass	7 [10]	129.8	1.8	62.0	72.6	155.4	211.4	299	0.0%	0.0%	0.0%	0.0%
		All Bare	8 [10]	60.0	2.3	18.2	32.1	75.7	98.2	192	0.0%	0.0%	0.0%	0.0%
	Other	Overall	9 [10]	107.8	4.0	5.8	61.3	137.5	248.1	743	11.1%	0.0%	0.0%	0.0%
		Full Grass	3 [10]	172.8	1.6	122.6	122.6	139.4	301.7	302	0.0%	0.0%	0.0%	0.0%
		>1/2 Grass	2 [10]	222.7	1.3	180.4	180.4	227.7	274.9	275	0.0%	0.0%	0.0%	0.0%
		<1/2 Grass	6 [10]	95.9	5.1	5.8	59.7	104.4	275.6	743	16.7%	0.0%	0.0%	0.0%
		All Bare	4 [10]	65.4	1.9	35.7	42.8	55.7	114.1	167	0.0%	0.0%	0.0%	0.0%
Cincinnati Longitudinal	Play Area and/or Entryway	All Units	80	1360.3	4.7	76.0	NA	NA	NA	54519	NA	NA	NA	NA
		20th Century Public	14	572.0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
		19th Century Rehabbed	18	804.0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
		19th Century Satisfactory	7	2540.0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
		19th Century Deteriorated	13	2670.0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Cincinnati Roadside	Pavement Edge	< 1950	60 [9]	1256.0 [9]	1254.3 [9]	NA	NA	NA	NA	NA	NA	NA	NA	NA
		> 1960		752.0 [9]	557.4 [9]	NA	NA	NA	NA	NA	NA	NA	NA	NA
Charleston	Play Area		164	NA	NA	9.0	173.0	585.0	1400.0	7890	NA	NA	NA	NA

Table 3-22b. (cont.)

Study	Yard Area/ Subset of Housing Units		Average Soil-Lead Concentration (µg/g) N	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	Percentag of Homes with Soil-Lead Concentration ≥ 400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
Corpus Christi	All Samples		485 [7]	208.0 [8]	NA	8.0	NA	NA	NA	2969	NA	NA	NA	NA
	Parks		94 [7]	55.0 [8]	NA	8.0	NA	NA	NA	318	NA	NA	NA	NA
	Schools		12 [7]	57.0 [8]	NA	11.0	NA	NA	NA	258	NA	NA	NA	NA
	All Others		379 [7]	250.0 [8]	NA	8.0	NA	NA	NA	2969	NA	NA	NA	NA
I-880 (Alameda County)	East		116 [7]	594.3 [8]	NA	22.3	NA	NA	NA	3187	NA	NA	NA	NA
	West		22 [7]	263.3 [8]	NA	89.7	NA	NA	NA	862	NA	NA	NA	NA
HUD Abatement Demonstration Study	Dripline		455 [7]	755.0 [8]	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dripline - Post-Abatement		455 [7]	867.5 [8]	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
HUD Grantees Evaluation	Dripline	All Grantees	557	1182.0	3.7	0.1	557.0	1252.0	2580.0	52700	83.7%	53.1%	32.1%	11.3%
		Alameda	97	776.3	2.7	30.0	395.0	710.0	1387.0	21131	73.2%	32.0%	13.4%	4.1%
		California	8	330.7	1.9	94.0	270.0	360.0	460.0	780	37.5%	0.0%	0.0%	0.0%
		Cleveland	99	2380.5	2.3	420.0	1350.0	2140.0	4517.0	16380	100.0%	80.8%	54.6%	20.2%
		Minnesota	44	593.2	2.7	45.0	280.0	559.5	1150.5	8120	59.1%	25.0%	9.1%	4.5%
		Rhode Island	60	1392.4	3.3	66.0	639.5	1500.0	2779.0	26159	88.3%	60.0%	36.7%	15.0%
		Wisconsin	66	563.7	7.3	0.1	400.0	859.0	1500.0	5733	77.3%	37.9%	16.7%	3.0%
		Milwaukee	12	1974.2	2.5	327.0	1166.5	1690.0	3496.0	10300	91.7%	75.0%	33.3%	16.7%
		Vermont	171	1536.5	3.3	25.0	692.0	1560.0	3380.0	52700	88.9%	60.8%	41.5%	14.0%
Maine Urban	Homes		75	1275.0	NA	50.0	NA	NA	NA	10900	50.0% [5]	37.0% [6]	NA	NA
	Parks and Playgrounds		25	205.0	NA	50.0	NA	NA	NA	700	8.0% [5]	0.0% [6]	0.0%	0.0%
Milwaukee	Perimeter		93	2343.7	2.2	587.0	1248.0	1990.0	3655.0	29335	100.0%	77.2%	50.0%	18.3%
	Play Area		92	626.3	2.3	130.0	378.0	556.0	860.0	9459	73.9%	20.7%	9.8%	3.3%
Minneapolis Clean-up	Minneapolis	Foundation	12	NA	NA	34.0	184.0	795.0	1265.0	2240	NA	NA	NA	NA
		Mid-yard	12	NA	NA	6.0	55.0	272.0	411.0	680	NA	NA	NA	NA
		Street	10	NA	NA	96.0	138.0	255.0	282.0	373	NA	NA	NA	NA
	St. Paul	Foundation	10	NA	NA	22.0	178.0	561.0	980.0	2960	NA	NA	NA	NA
		Mid-yard	10	NA	NA	44.0	70.0	108.0	284.0	414	NA	NA	NA	NA
		Street	10	NA	NA	33.0	106.0	153.0	282.0	470	NA	NA	NA	NA

Table 3-22b. (cont.)

Study	Yard Area/ Subset of Housing Units		Average Soil-Lead Concentration (µg/g)								Percentag of Homes with Soil-Lead Concentration >			
			N	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
Minnesota	Entire Study, All Samples		2454 [7]	NA	NA	NA	NA	NA	NA	NA	11.0% [5]	5.0% [6]	2.0%	NA
	St. Paul	Foundation	127 [7]	472.0	4.5	3.0	268.0	576.0	1246.0	7994	70.7% [4]	26.3%	NA	NA
		Garden	8 [7]	174.0	6.2	14.0	40.0	147.0	1177.0	2846	NA	NA	NA	NA
		Backyard	114 [7]	119.0	3.8	1.0	65.0	161.0	300.0	1366	21.0% [4]	2.5%	NA	NA
		Front yard	108 [7]	90.0	3.0	1.0	44.0	104.0	192.0	1377				
		Side yard	46 [7]	96.0	6.1	1.0	42.0	133.0	364.0	2385				
		Street side	170 [7]	113.0	2.2	6.0	64.0	127.0	204.0	575	8.3% [4]	0.0%	0.0%	0.0%
		Open	95 [7]	66.0	3.7	2.0	26.0	76.0	177.0	1466	NA	NA	NA	NA
		Play area	164 [7]	24.0	7.0	1.0	4.0	36.0	104.0	607	NA	NA	NA	NA
	Minneapolis	Foundation	199 [7]	665.0	3.5	35.0	305.0	689.0	1496.0	20136	56.60 [4]	32.6%	NA	NA
		Garden	28 [7]	253.0	3.2	34.0	109.0	264.0	445.0	3858	NA	NA	NA	NA
		Backyard	61 [7]	212.0	3.3	4.0	110.0	247.0	520.0	1210	31.4% [4]	0.8%	NA	NA
		Front yard	131 [7]	173.0	2.1	18.0	107.0	185.0	289.0	1345				
		Side yard	170 [7]	177.0	2.2	27.0	106.0	165.0	297.0	1326				
		Street side	119 [7]	186.0	2.6	3.0	108.0	223.0	338.0	1876	25.0% [4]	0.6%	NA	NA
		Open	51 [7]	39.0	3.7	1.0	24.0	34.0	73.0	878	NA	NA	NA	NA
		Play area	139 [7]	22.0	6.9	1.0	4.0	33.0	110.0	788	NA	NA	NA	NA
New Haven, Connecticut	Near (near the house)		260	712.9	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Far (near the street)		260	597.0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
New Orleans	Inner City	Foundation	201 [7]	NA	NA	8.0	249.0	840.0	2370.0	69000	NA	NA	NA	NA
		Streetside	723 [7]	NA	NA	4.0	142.0	342.0	620.0	9450	55.5% [4]	9.1%	NA	NA
		Open Area	74 [7]	NA	NA	10.0	76.0	212.0	460.0	10600	NA	NA	NA	NA
	Mid-City	Foundation	220 [7]	NA	NA	1.0	32.0	110.0	446.0	24400	NA	NA	NA	NA
		Streetside	765 [7]	NA	NA	1.0	30.0	110.0	246.0	6340	21.2% [4]	2.9%	NA	NA
		Open Area	80 [7]	NA	NA	2.0	16.0	40.0	98.0	3960	NA	NA	NA	NA
	Suburban	Foundation	332 [7]	NA	NA	2.0	18.0	50.0	154.0	5650	NA	NA	NA	NA
		Streetside	1195 [7]	NA	NA	2.0	40.0	86.0	171.0	2150	9.2% [4]	0.3%	NA	NA
		Open Area	114 [7]	NA	NA	4.0	14.0	28.0	78.0	540	NA	NA	NA	NA
Rochester Study	Dripline		185	992.6	4.2	17.8	545.8	1117.5	2380.2	110834	79.5%	47.6%	31.4%	11.4%
	Dripline (fine soil only)		185	732.0	3.7	12.3	412.0	959.0	1648.0	21049	76.2%	38.4%	18.4%	3.8%

Table 3-22b. (cont.)

Study	Yard Area/ Subset of Housing Units		Average Soil-Lead Concentration (µg/g)								Percentag of Homes with Soil-Lead Concentration [2]			
			N	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
Washington, D.C.	Front yard	Ward 1	30	NA	NA	36.4	228.0	444.2	1145.0	4905	NA	NA	NA	NA
		Ward 2	30	NA	NA	48.3	344.8	471.4	975.0	4520	NA	NA	NA	NA
		Ward 3	30	NA	NA	10.2	25.1	53.7	105.7	815	NA	NA	NA	NA
		Ward 4	30	NA	NA	32.7	95.5	198.9	294.9	4575	NA	NA	NA	NA
		Ward 5	30	NA	NA	12.0	101.3	221.9	380.4	5056	NA	NA	NA	NA
		Ward 6	30	NA	NA	13.8	125.0	260.4	427.9	1720	NA	NA	NA	NA
		Ward 7	30	NA	NA	36.2	70.3	144.4	274.9	3740	NA	NA	NA	NA

NA = Not Available

1 Mass-weighted arithmetic average soil-lead concentration as reported in the HUD National Survey; summarized without weighting by sample weights.
2 Mass-weighted arithmetic average soil-lead concentration as calculated in Chapter 3 of the 403 risk analysis; summarized by weighting each average to reflect the 1997 U.S. housing stock.
3 Mass-weighted arithmetic average soil-lead concentration as reported in the HUD National Survey; summarized by weighting with the National Survey sample weights.
4 Percent of samples that exceed 300 ppm
5 Percent of samples that exceed 500 ppm
6 Percent of samples that exceed 1000 ppm
7 Number of samples (multiple samples taken at many sites).
8 Arithmetic Mean or SD
9 60 houses total; reference used did not provide number of houses by house age.
10 An initial unweighted arithmetic average of soil lead levels at the specified locations was taken prior to calculation of statistics within this table. The number in this column represents the number of properties, not necessarily the number of houses.

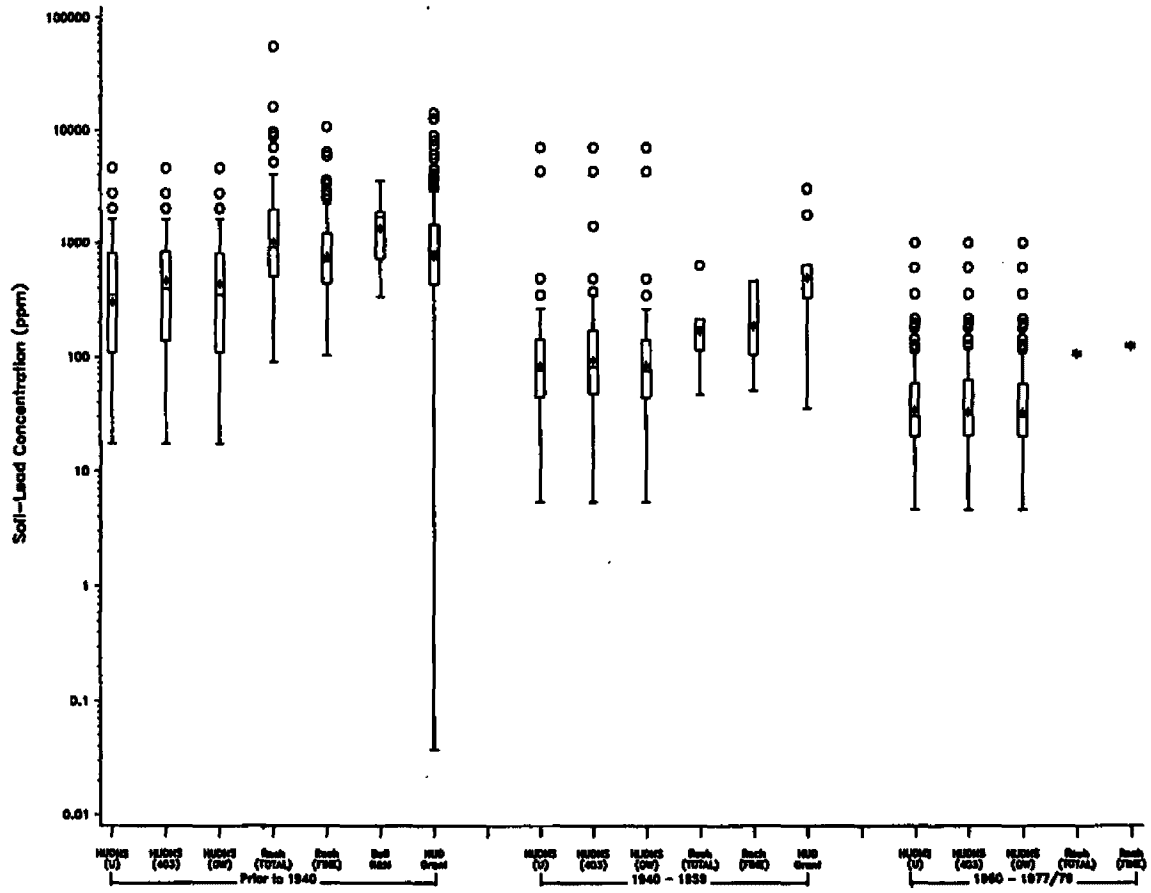

Figure 3-17. Boxplots of Household Average Soil-Lead Concentrations (μg/g) for Houses in the HUD National Survey, Baltimore R&M Study, Rochester Lead-in-Dust Study, and HUD Grantees Evaluation, by Housing Age Category (pre-1979 only)

(Note: Data for the Baltimore R&M study are dripline results. See text for definitions of labels along the horizontal axis. Caution must be taken when categorizing houses in the Rochester study by age of the house.)

The summary statistics found in Tables 3-22a and 3-22b were calculated according to housing age category for relevant studies. These summaries are found in Tables 3-23a (for yard-wide average soil-lead concentration) and 3-23b (for average soil-lead concentration for specific locations). Note that these tables also include summary statistics for housing units built after 1979 (although the Rochester study units may not have actually been built in this time period, as mentioned in Section 3.2.1.2). The post-1979 results labeled as "HUD National Survey (§403 RA)" represent surveyed homes built from 1960-1979 that contain no lead-based paint (Section 3.3.1.5 of the §403 risk analysis report).

3.2.2.3 Calculating National Exceedance Percentages for Yardwide Average Soil-Lead Concentration. The soil-lead data summaries presented above suggest that the distribution of measured soil-lead concentrations as reported for the HUD National Survey are reasonably consistent with the distributions suggested by other studies, including the interim NSLAH data. Thus, these two national surveys are expected to generate similar national distributions for yardwide average soil-lead concentration, from which the estimated percentages of housing units whose yardwide average soil-lead concentrations exceed specified thresholds ("exceedance percentages") could be calculated. These percentages give some indication of the frequency with which intervention activities might be prompted by regulations that target alleviating soil-lead exposure. Soil abatement practices are often recommended both within the literature and by the HUD "Guidelines for the Evaluation and Control of Lead-Based Paint Hazards in Housing" (USHUD, 1995b; pages 12-47 to 12-56).

The methods detailed in Section 3.2.1.3, which were used to fit lognormal distributions to household average floor dust-lead loadings based on data from the two national surveys, were also used to fit lognormal distributions to yardwide average soil-lead concentration data from these two surveys. As discussed in Section 3.2.1.3, the key objective to fitting the lognormal distribution was to use the distribution to estimate exceedance percentages for specified soil-lead concentration thresholds. Therefore, in order to ensure that the upper tail of the distribution was as accurately portrayed as possible within the fitted distribution, this method treated a certain percentage of the lowest data values as censored data when fitting the distribution. In this exercise, four thresholds were of interest for yardwide average soil-lead concentration: 400, 1200, 2000, and 5000 ppm.

Figure 3-18 contains plots of the fitted lognormal distributions (superimposed on bar charts of the observed data) and the estimated exceedance probabilities corresponding to these distributions, for residential yard-wide average soil-lead concentrations, based on the HUD National Survey data (top plot) and the interim NSLAH data (bottom plot). Recall that the sampling weights corresponding to the HUD National Survey data were revised in the §403 risk analysis to reflect the 1997 national housing stock. The same soil-lead concentration (horizontal) axis is used for both plots, so that the two plots can be directly compared. The similarity of the two distributions is noted in this plot, as the fitted distributions are nearly the same shape and cover approximately the same ranges of data. Furthermore, the estimated exceedance percentages for a given threshold differ by less than one percentage point between the two

Table 3-23a. Descriptive Statistics of Yard-Wide Average Soil-Lead Concentrations, According to Study and Housing Age Category and Within Specific Subsets of the Sampled Housing Within a Study

Study	Subset of Units or Type of Soil/Cover	Yard-Wide Average Soil-Lead Concentration (µg/g)								Percentage of Homes with Yard-Wide Average Soil-Lead Concentration ≥			
		N	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
Houses Built Prior to 1940													
HUD National Survey (unweighted)	All	58	298.3	3.8	17.4	109.4	346.5	805.3	4619	43.1%	17.2%	6.9%	0.0%
	City Only	29	375.6	4.0	17.4	110.2	533.9	1159.2	4619	55.2%	24.1%	6.9%	0.0%
	Northeast	11	453.8	1.9	136.7	289.6	443.6	627.9	1427	54.5%	9.1%	0.0%	0.0%
	Midwest	15	476.9	3.9	49.8	109.4	679.1	1497.0	2752	53.3%	40.0%	13.3%	0.0%
	South	19	166.3	4.3	17.4	47.4	125.2	613.4	4619	26.3%	5.3%	5.3%	0.0%
	West	13	286.2	4.0	25.9	112.7	393.5	711.2	2019	46.2%	15.4%	7.7%	0.0%
	Northeast - City Only	3	545.1	2.4	255.8	255.8	443.6	1427.1	1427	66.7%	33.3%	0.0%	0.0%
	Midwest - City Only	6	794.2	3.7	80.4	371.5	1427.8	1497.0	2752	66.7%	66.7%	16.7%	0.0%
	South - City Only	13	221.4	4.9	17.4	59.6	258.1	717.5	4619	38.5%	7.7%	7.7%	0.0%
	West - City Only	7	449.7	2.8	84.2	137.7	585.6	1159.2	1244	71.4%	14.3%	0.0%	0.0%
HUD National Survey (403 RA) [2]	All	77	462.7	3.1	17.4	137.7	393.5	840.7	4619	59.2%	19.6%	9.4%	0.0%
	City Only	45	509.4	3.0	17.4	258.8	613.4	840.7	4619	67.4%	20.7%	8.6%	0.0%
	Northeast	26	490.7	1.6	136.7	289.6	554.1	840.7	1427	64.7%	0.8%	0.0%	0.0%
	Midwest	19	940.7	2.7	49.8	162.3	834.7	1463.0	2752	77.3%	54.1%	25.2%	0.0%
	South	19	173.6	3.7	17.4	47.4	125.2	613.4	4619	27.7%	2.9%	2.9%	0.0%
	West	13	295.5	3.8	25.9	112.7	393.5	711.2	2019	45.5%	18.0%	9.0%	0.0%
	Northeast - City Only	16	525.4	1.6	255.8	278.9	642.1	840.7	1427	72.0%	1.5%	0.0%	0.0%
	Midwest - City Only	9	1405.1	2.1	80.4	641.9	840.7	1463.0	2752	92.4%	77.8%	34.0%	0.0%
	South - City Only	13	223.8	4.0	17.4	59.6	258.1	717.5	4619	39.8%	4.2%	4.2%	0.0%
	West - City Only	7	434.4	2.8	84.2	137.7	585.6	1159.2	1244	66.9%	16.6%	0.0%	0.0%
Rochester Lead-in-Dust	Total Soil	75	1018.6	3.3	91.7	508.6	911.9	1971.8	55617	81.3%	36.0%	24.0%	10.7%
	Fine Soil Fraction	75	749.3	2.6	102.9	438.5	686.0	1205.0	10721	82.7%	25.3%	13.3%	4.0%

Table 3-23a. (cont.)

Study	Subset of Units or Type of Soil/Cover	Yard-Wide Average Soil-Lead Concentration (µg/g)								Percentage of Homes with Yard-Wide Average Soil-Lead Concentration ≥			
		N	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
Houses Built Prior to 1940 (cont.)													
HUD National Survey (HUD NS weights) [3]	All	58	433.9	3.4	17.4	109.4	346.5	805.3	4619	56.1%	24.2%	11.6%	0.0%
	City Only	29	478.7	3.6	17.4	110.2	533.9	1159.2	4619	67.5%	29.9%	12.5%	0.0%
	Northeast	11	433.3	1.5	136.7	289.6	443.6	627.9	1427	62.5%	1.4%	0.0%	0.0%
	Midwest	15	955.4	2.8	49.8	109.4	679.1	1497.0	2752	75.1%	59.3%	27.7%	0.0%
	South	19	173.6	3.7	17.4	47.4	125.2	613.4	4619	27.7%	2.9%	2.9%	0.0%
	West	13	295.5	3.8	25.9	112.7	393.5	711.2	2019	45.5%	18.0%	9.0%	0.0%
	Northeast - City Only	3	444.9	1.4	255.8	255.8	443.6	1427.1	1427	90.8%	4.6%	0.0%	0.0%
	Midwest - City Only	6	1559.6	2.1	80.4	371.5	1427.8	1497.0	2752	91.1%	91.1%	39.8%	0.0%
	South - City Only	13	223.8	4.0	17.4	59.6	258.1	717.5	4619	39.8%	4.2%	4.2%	0.0%
	West - City Only	7	434.4	2.8	84.2	137.7	585.6	1159.2	1244	66.9%	16.6%	0.0%	0.0%
HUD Grantees Evaluation	All Grantees	181	757.2	4.2	0.0	431.0	835.0	1455.0	14180	79.0%	35.4%	18.8%	3.9%
	Alameda	39	650.9	2.7	39.5	316.0	582.0	1348.0	12648	69.2%	33.3%	7.7%	2.6%
	California	7	325.1	2.2	58.0	325.0	405.0	540.0	560	57.1%	0.0%	0.0%	0.0%
	Cleveland	64	1629.1	2.2	431.0	922.0	1430.0	2857.5	14180	100.0%	59.4%	37.5%	7.8%
	Minnesota	18	442.8	2.6	49.5	265.0	471.8	790.0	4492	61.1%	5.6%	5.6%	0.0%
	Rhode Island	11	835.1	3.6	65.0	281.5	1205.5	1496.0	5648	72.7%	54.5%	18.2%	9.1%
	Wisconsin	28	303.4	11.9	0.0	260.3	556.0	908.5	3852	67.9%	10.7%	7.1%	0.0%
	Milwaukee	6	1085.9	1.5	766.0	804.0	1005.0	1188.5	2288	100.0%	16.7%	16.7%	0.0%
	Vermont	8	395.9	3.6	38.5	178.5	503.8	1033.9	2078	50.0%	25.0%	12.5%	0.0%
Houses Built From 1940-1959													
HUD National Survey (unweighted)	All	77	83.1	2.9	5.4	44.3	75.8	141.6	7025	3.9%	2.6%	2.6%	1.3%
	City Only	37	81.0	3.2	5.4	43.5	77.3	129.6	7025	2.7%	2.7%	2.7%	2.7%
	Northeast	10	96.9	4.2	33.7	52.4	62.0	77.3	4318	10.0%	10.0%	10.0%	0.0%
	Midwest	19	85.2	2.4	9.3	52.4	90.5	145.3	346	0.0%	0.0%	0.0%	0.0%
	South	33	84.1	3.3	5.4	43.5	81.0	135.1	7025	6.1%	3.0%	3.0%	3.0%
	West	15	70.7	2.2	24.9	34.6	60.4	145.5	214	0.0%	0.0%	0.0%	0.0%
	Northeast - City Only	4	96.4	1.8	63.9	70.6	77.3	151.4	225	0.0%	0.0%	0.0%	0.0%
	Midwest - City Only	3	104.3	3.1	36.3	36.3	90.5	345.9	346	0.0%	0.0%	0.0%	0.0%
	South - City Only	20	91.5	4.1	5.4	46.2	94.5	139.5	7025	5.0%	5.0%	5.0%	5.0%
	West - City Only	10	55.0	2.1	24.9	26.0	49.7	108.5	214	0.0%	0.0%	0.0%	0.0%

Table 3-23a. (cont.)

Study	Subset of Units or Type of Soil/Cover	Yard-Wide Average Soil-Lead Concentration (µg/g)								Percentage of Homes with Yard-Wide Average Soil-Lead Concentration ≥			
		N	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
Houses Built From 1940-1959 (cont.)													
HUD National Survey (403 RA) [2]	All	87	92.6	3.2	5.4	47.6	81.4	170.7	7025	5.2%	4.3%	3.2%	1.1%
	City Only	46	101.5	3.4	5.4	49.0	103.7	218.2	7025	4.7%	4.7%	2.3%	2.3%
	Northeast	17	136.4	4.4	33.7	53.8	77.3	313.3	4318	14.3%	14.3%	9.3%	0.0%
	Midwest	21	92.6	2.4	9.3	58.9	123.3	182.0	372	0.0%	0.0%	0.0%	0.0%
	South	33	83.1	3.3	5.4	43.5	81.0	135.1	7025	6.1%	3.3%	3.3%	3.3%
	West	16	72.1	2.2	24.9	39.4	70.9	171.7	220	0.0%	0.0%	0.0%	0.0%
	Northeast - City Only	10	255.8	2.3	63.9	77.3	269.4	313.3	1412	12.6%	12.6%	0.0%	0.0%
	Midwest - City Only	5	140.6	2.4	36.3	90.5	216.7	345.9	372	0.0%	0.0%	0.0%	0.0%
	South - City Only	20	91.8	4.1	5.4	46.2	94.5	139.5	7025	5.7%	5.7%	5.7%	5.7%
	West - City Only	11	56.3	2.1	24.9	26.0	51.8	129.6	220	0.0%	0.0%	0.0%	0.0%
HUD National Survey (HUD NS weights) [3]	All	77	83.9	3.1	5.4	44.3	75.8	141.6	7025	4.4%	3.4%	3.4%	1.2%
	City Only	37	80.8	3.2	5.4	43.5	77.3	129.6	7025	2.8%	2.8%	2.8%	2.8%
	Northeast	10	102.8	4.5	33.7	52.4	62.0	77.3	4318	12.3%	12.3%	12.3%	0.0%
	Midwest	19	85.7	2.4	9.3	52.4	90.5	145.3	346	0.0%	0.0%	0.0%	0.0%
	South	33	83.1	3.3	5.4	43.5	81.0	135.1	7025	6.1%	3.3%	3.3%	3.3%
	West	15	69.9	2.2	24.9	34.6	60.4	145.5	214	0.0%	0.0%	0.0%	0.0%
	Northeast - City Only	4	138.8	1.8	63.9	70.6	77.3	151.4	225	0.0%	0.0%	0.0%	0.0%
	Midwest - City Only	3	104.3	2.5	36.3	36.3	90.5	345.9	346	0.0%	0.0%	0.0%	0.0%
	South - City Only	20	91.8	4.1	5.4	46.2	94.5	139.5	7025	5.7%	5.7%	5.7%	5.7%
	West - City Only	10	53.1	2.0	24.9	26.0	49.7	108.5	214	0.0%	0.0%	0.0%	0.0%
HUD Grantees Evaluation	All Grantees	11	492.0	3.0	35.5	328.0	479.0	640.0	3024	72.7%	18.2%	9.1%	0.0%
	Alameda	2	1059.0	2.1	632.0	632.0	1203.2	1774.5	1774	100.0%	50.0%	0.0%	0.0%
	Rhode Island	5	409.2	5.0	35.5	328.0	509.5	640.0	3024	60.0%	20.0%	20.0%	0.0%
	Wisconsin	3	404.8	1.2	316.0	316.0	450.0	466.5	466	66.7%	0.0%	0.0%	0.0%
	Vermont	1	479.0	.	479.0	479.0	479.0	479.0	479	100.0%	0.0%	0.0%	0.0%
Rochester Lead-in-Dust [4]	Total Soil	5	166.8	2.6	46.7	113.9	180.6	212.8	632	20.0%	0.0%	0.0%	0.0%
	Fine Soil Fraction	5	186.3	2.6	51.1	104.0	198.5	458.5	465	40.0%	0.0%	0.0%	0.0%

Table 3-23a. (cont.)

Study	Subset of Units or Type of Soil/Cover	Yard-Wide Average Soil-Lead Concentration (µg/g)								Percentage of Homes with Yard-Wide Average Soil-Lead Concentration ≥			
		N	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
Houses Built From 1960-1979 (1977 for HUD Grantees Evaluation)													
HUD National Survey (unweighted)	All	115	33.9	2.6	4.6	20.0	30.1	58.3	996	1.7%	0.0%	0.0%	0.0%
	City Only	43	39.2	2.7	5.2	21.1	33.3	79.3	996	2.3%	0.0%	0.0%	0.0%
	Northeast	10	61.2	2.2	14.8	41.1	62.2	115.2	196	0.0%	0.0%	0.0%	0.0%
	Midwest	29	26.5	2.4	4.6	17.1	23.4	39.2	355	0.0%	0.0%	0.0%	0.0%
	South	59	36.4	2.5	5.2	22.6	32.1	66.4	996	1.7%	0.0%	0.0%	0.0%
	West	17	28.4	3.7	4.8	14.2	23.7	39.5	604	5.9%	0.0%	0.0%	0.0%
	Northeast - City Only	3	98.7	2.2	42.5	42.5	115.2	196.2	196	0.0%	0.0%	0.0%	0.0%
	Midwest - City Only	6	19.6	1.4	13.7	13.8	20.6	21.1	33	0.0%	0.0%	0.0%	0.0%
	South - City Only	25	48.5	2.7	5.2	26.4	39.4	81.6	996	4.0%	0.0%	0.0%	0.0%
	West - City Only	9	25.4	2.6	5.4	18.8	23.7	31.8	186	0.0%	0.0%	0.0%	0.0%
HUD National Survey (403 RA) [2]	All	120	32.8	2.6	4.6	20.4	31.5	62.5	996	1.2%	0.0%	0.0%	0.0%
	City Only	46	36.2	2.4	5.2	21.3	34.8	68.5	996	0.9%	0.0%	0.0%	0.0%
	Northeast	10	60.7	2.2	14.8	41.1	62.2	115.2	196	0.0%	0.0%	0.0%	0.0%
	Midwest	29	27.1	2.3	4.6	17.1	23.4	39.2	355	0.0%	0.0%	0.0%	0.0%
	South	64	36.5	2.3	5.2	23.0	35.1	64.9	996	0.8%	0.0%	0.0%	0.0%
	West	17	23.8	3.0	4.8	14.2	23.7	39.5	604	3.7%	0.0%	0.0%	0.0%
	Northeast - City Only	3	115.0	1.9	42.5	42.5	115.2	196.2	196	0.0%	0.0%	0.0%	0.0%
	Midwest - City Only	6	20.1	1.4	13.7	13.8	20.6	21.1	33	0.0%	0.0%	0.0%	0.0%
	South - City Only	28	48.8	2.2	5.2	26.8	42.7	80.4	996	2.0%	0.0%	0.0%	0.0%
	West - City Only	9	23.5	2.1	5.4	18.8	23.7	31.8	186	0.0%	0.0%	0.0%	0.0%
HUD National Survey (HUD NS weights) [3]	All	115	32.4	2.6	4.6	20.0	30.1	58.3	996	1.2%	0.0%	0.0%	0.0%
	City Only	43	35.9	2.5	5.2	21.1	33.3	79.3	996	0.9%	0.0%	0.0%	0.0%
	Northeast	10	60.7	2.2	14.8	41.1	62.2	115.2	196	0.0%	0.0%	0.0%	0.0%
	Midwest	29	27.1	2.3	4.6	17.1	23.4	39.2	355	0.0%	0.0%	0.0%	0.0%
	South	59	35.7	2.3	5.2	22.6	32.1	66.4	996	0.8%	0.0%	0.0%	0.0%
	West	17	23.8	3.0	4.8	14.2	23.7	39.5	604	3.7%	0.0%	0.0%	0.0%
	Northeast - City Only	3	115.0	1.9	42.5	42.5	115.2	196.2	196	0.0%	0.0%	0.0%	0.0%
	Midwest - City Only	6	20.1	1.4	13.7	13.8	20.6	21.1	33	0.0%	0.0%	0.0%	0.0%
	South - City Only	25	49.3	2.3	5.2	26.4	39.4	81.6	996	2.2%	0.0%	0.0%	0.0%
	West - City Only	9	23.5	2.1	5.4	18.8	23.7	31.8	186	0.0%	0.0%	0.0%	0.0%

Table 3-23a. (cont.)

Study	Subset of Units or Type of Soil/Cover	Yard-Wide Average Soil-Lead Concentration (µg/g)								Percentage of Homes with Yard-Wide Average Soil-Lead Concentration ≥			
		N	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
Houses Built From 1960-1979 (1977 for HUD Grantees Evaluation) (cont.)													
Rochester Lead-In-Dust [4]	Total Soil	1	106.2	.	106.2	106.2	106.2	106.2	106	0.0%	0.0%	0.0%	0.0%
	Fine Soil Fraction	1	124.5	.	124.5	124.5	124.5	124.5	124	0.0%	0.0%	0.0%	0.0%
Houses Built After 1979 (1977 for HUD Grantees Evaluation)													
HUD National Survey (403 RA) [2]	All	28	22.4	2.3	5.4	13.6	21.2	45.0	97	0.0%	0.0%	0.0%	0.0%
	City Only	9	24.8	2.3	5.4	20.4	21.3	29.7	97	0.0%	0.0%	0.0%	0.0%
	Midwest	4	11.5	1.7	6.7	7.0	12.4	19.0	20	0.0%	0.0%	0.0%	0.0%
	South	18	29.7	2.1	5.6	21.0	25.0	58.3	97	0.0%	0.0%	0.0%	0.0%
	West	6	15.0	2.3	5.4	6.2	13.6	29.7	62	0.0%	0.0%	0.0%	0.0%
	Midwest - City Only	1	20.4	1.0	20.4	20.4	20.4	20.4	20	0.0%	0.0%	0.0%	0.0%
	South - City Only	5	38.5	2.0	21.0	21.3	24.5	79.3	97	0.0%	0.0%	0.0%	0.0%
	West - City Only	3	12.8	2.0	5.4	5.4	13.0	29.7	30	0.0%	0.0%	0.0%	0.0%
HUD Grantees Evaluation	Minnesota	1	405.5	.	405.5	405.5	405.5	405.5	406	100.0%	0.0%	0.0%	0.0%
Rochester Lead-In-Dust [4]	Total Soil	1	521.9	.	521.9	521.9	521.9	521.9	522	100.0%	0.0%	0.0%	0.0%
	Fine Soil Fraction	1	545.5	.	545.5	545.5	545.5	545.5	546	100.0%	0.0%	0.0%	0.0%

1 Mass-weighted arithmetic average soil-lead concentration as reported in the HUD National Survey; summarized without weighting by sample weights.
2 Mass-weighted arithmetic average soil-lead concentration as calculated in Chapter 3 of the 403 risk analysis; summarized by weighting each average to reflect the 1997 U.S. housing stock.
3 Mass-weighted arithmetic average soil-lead concentration as reported in the HUD National Survey; summarized by weighting with the National Survey sample weights.
4 Some houses in this housing age category may belong to an earlier age category, as some houses may have actually been built earlier than the year specified within the study's database.

Table 3-23b. Descriptive Statistics of Average Soil-Lead Concentrations in Specific Yard Areas and/or for Certain Subsets of the Sampled Housing Within a Study, Presented by Housing Age Category

Study	Location		Average Soil-Lead Concentration (µg/g)								Percentage of Homes with Average Soil-Lead Concentration >			
			N	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
Houses Built Prior to 1940														
HUD National Survey (unweighted)	Dripline/ Entryway	All	64	347.2	4.0	19.7	127.9	418.2	987.6	8960	51.6%	12.5%	7.8%	3.1%
		City Only	33	426.2	4.0	23.1	182.9	504.3	1106.5	8960	60.6%	15.2%	9.1%	6.1%
		Northeast	15	556.9	2.0	211.8	377.9	523.7	887.7	2334	73.3%	13.3%	6.7%	0.0%
		Midwest	17	515.9	4.0	34.7	259.8	424.8	1165.5	5336	52.9%	23.5%	17.6%	5.9%
		South	19	207.5	5.0	23.1	49.4	182.9	842.9	8960	31.6%	10.5%	5.3%	5.3%
		West	13	254.4	4.1	19.7	88.1	423.6	932.3	1149	53.8%	0.0%	0.0%	0.0%
		Northeast - City Only	6	509.3	2.4	211.8	241.9	458.5	700.8	2334	66.7%	16.7%	16.7%	0.0%
		Midwest - City Only	7	702.0	3.8	93.7	261.6	702.0	1932.8	5336	71.4%	28.6%	14.3%	14.3%
		South - City Only	13	299.4	5.7	23.1	59.0	296.3	1106.5	8960	46.2%	15.4%	7.7%	7.7%
		West - City Only	7	428.0	3.1	66.5	123.8	652.8	1038.2	1149	71.4%	0.0%	0.0%	0.0%
HUD National Survey (403 RA) [2]	Dripline/ Entryway	All	77	529.0	3.4	19.7	184.0	466.4	1126.5	8960	68.1%	15.1%	12.7%	4.5%
		City Only	45	622.7	3.3	23.1	261.6	700.8	1126.5	8960	76.7%	12.7%	9.2%	8.6%
		Northeast	26	664.4	1.6	211.8	453.0	622.4	1126.5	2334	93.6%	2.4%	0.8%	0.0%
		Midwest	19	924.7	3.6	34.7	259.8	702.0	1165.5	5336	64.7%	39.7%	37.9%	12.6%
		South	19	215.9	4.3	23.1	49.4	182.9	842.9	8960	33.8%	9.1%	2.9%	2.9%
		West	13	241.4	3.8	19.7	88.1	423.6	932.3	1149	50.2%	0.0%	0.0%	0.0%
		Northeast - City Only	16	743.6	1.6	211.8	453.0	913.7	1126.5	2334	94.1%	1.5%	1.5%	0.0%
		Midwest - City Only	9	1387.1	3.0	93.7	424.8	1114.5	1126.5	5336	92.4%	38.9%	34.0%	34.0%
		South - City Only	13	301.3	4.6	23.1	59.0	296.3	1106.5	8960	48.7%	13.1%	4.2%	4.2%
		West - City Only	7	375.0	2.9	66.5	123.8	652.8	1038.2	1149	66.9%	0.0%	0.0%	0.0%

Table 3-23b. (cont.)

Study	Location		Average Soil-Lead Concentration (µg/g)								Percentage of Homes with Average Soil-Lead Concentration ≥			
			N	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
Houses Built Prior to 1940 (cont.)														
HUD National Survey (HUD NS weights) [3]	Dripline/ Entryway	All	64	481.8	3.7	19.7	127.9	418.2	987.6	8960	62.9%	17.5%	14.7%	5.2%
		City Only	33	545.8	3.8	23.1	182.9	504.3	1106.5	8960	71.7%	16.8%	12.2%	11.4%
		Northeast	15	562.0	1.5	211.8	377.9	523.7	887.7	2334	90.4%	3.6%	1.2%	0.0%
		Midwest	17	917.8	3.6	34.7	259.8	424.8	1165.5	5336	63.3%	41.2%	39.3%	13.1%
		South	19	215.9	4.3	23.1	49.4	182.9	842.9	8960	33.8%	9.1%	2.9%	2.9%
		West	13	241.4	3.8	19.7	88.1	423.6	932.3	1149	50.2%	0.0%	0.0%	0.0%
		Northeast - City Only	6	572.4	1.6	211.8	241.9	458.5	700.8	2334	66.8%	3.3%	3.3%	0.0%
		Midwest - City Only	7	1418.8	3.1	93.7	281.6	702.0	1932.8	5336	91.6%	43.1%	37.7%	37.7%
		South - City Only	13	301.3	4.6	23.1	59.0	296.3	1106.5	8960	48.7%	13.1%	4.2%	4.2%
		West - City Only	7	375.0	2.9	66.5	123.8	652.8	1038.2	1149	66.9%	0.0%	0.0%	0.0%
Baltimore R&M	Dripline		28	444.5	5.1	28.9	71.5	686.9	1767.5	3539	60.7%	42.9%	10.7%	0.0%
California	Oakland, LA, Sacramento		377	NA	NA	NA	NA	NA	NA	NA	66.0% [4]	NA	NA	NA
	Oakland		174	NA	NA	NA	NA	NA	NA	NA	90.0% [4]	NA	NA	NA
HUD Grantees Evaluation	Dripline	All Grantees	266	1025.9	3.9	0.1	534.0	1077.5	2150.0	50600	80.8%	46.2%	29.3%	8.6%
		Alameda	57	733.3	2.8	30.0	370.0	652.0	1317.0	21131	71.9%	28.1%	14.0%	3.5%
		California	7	321.9	1.9	94.0	250.0	350.0	520.0	780	28.6%	0.0%	0.0%	0.0%
		Cleveland	64	2491.7	2.3	540.0	1370.0	2150.0	4638.0	16380	100.0%	79.7%	59.4%	21.9%
		Minnesota	21	455.7	2.8	45.0	275.0	400.0	770.0	8120	52.4%	9.5%	4.8%	4.8%
		Rhode Island	13	1251.5	3.0	112.0	744.0	1511.0	2401.0	10209	84.6%	61.5%	30.8%	7.7%
		Wisconsin	44	504.9	7.6	0.1	353.5	796.5	1205.0	5733	75.0%	29.5%	9.1%	4.5%
		Milwaukee	6	1224.7	2.2	327.0	1070.0	1166.5	1910.0	3727	83.3%	50.0%	16.7%	0.0%
		Vermont	54	1355.8	3.2	28.0	645.0	1425.0	3180.0	50600	88.9%	55.6%	40.7%	5.6%
New Haven, Connecticut	Near (near the house)		112	1252.5	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Far (near the street)		112	816.5	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Rochester Lead-In-Dust	Dripline		158	1329.8	3.5	29.1	668.8	1322.0	2755.0	110834	88.0%	55.1%	36.7%	13.3%
	Dripline (fine soil only)		158	937.8	3.2	12.3	640.0	1076.5	1816.0	21049	85.4%	44.3%	21.5%	4.4%

Table 3-23b. (cont.)

Study	Location		Average Soil-Lead Concentration (µg/g)								Percentage of Homes with Average Soil-Lead Concentration ≥			
			N	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
Houses Built From 1940-1959														
HUD National Survey (unweighted)	Dripline/ Entryway	All	82	107.7	3.2	8.0	55.8	89.6	179.4	13596	8.5%	4.9%	3.7%	1.2%
		City Only	42	116.8	4.1	8.0	55.8	87.7	187.5	13596	14.3%	7.1%	7.1%	2.4%
		Northeast	13	170.4	4.6	35.9	73.5	83.7	246.0	2571	23.1%	23.1%	15.4%	0.0%
		Midwest	20	121.8	2.6	11.6	61.9	116.7	244.9	689	10.0%	0.0%	0.0%	0.0%
		South	33	97.9	3.4	8.0	45.5	89.6	145.3	13596	6.1%	3.0%	3.0%	3.0%
		West	16	77.2	2.5	9.5	43.1	71.0	178.7	284	0.0%	0.0%	0.0%	0.0%
		Northeast - City Only	7	256.8	5.1	73.5	83.7	88.0	2570.5	2571	28.6%	28.6%	28.6%	0.0%
		Midwest - City Only	4	218.5	3.3	51.3	91.3	311.1	590.0	689	50.0%	0.0%	0.0%	0.0%
		South - City Only	20	114.0	4.4	8.0	53.9	94.2	175.3	13596	10.0%	5.0%	5.0%	5.0%
		West - City Only	11	58.9	2.5	9.5	31.8	57.4	177.9	188	0.0%	0.0%	0.0%	0.0%
HUD National Survey (403 RA) [2]	Dripline/ Entryway	All	87	108.6	3.1	8.0	55.8	90.0	218.9	13596	6.5%	4.3%	2.2%	1.1%
		City Only	46	119.3	3.8	8.0	57.4	94.2	246.0	13596	9.6%	4.7%	4.7%	2.3%
		Northeast	17	152.7	3.6	35.9	73.5	88.0	373.2	2571	14.3%	14.3%	5.0%	0.0%
		Midwest	21	125.3	2.5	11.6	61.9	131.4	249.0	689	7.0%	0.0%	0.0%	0.0%
		South	33	96.8	3.3	8.0	45.5	89.6	145.3	13596	4.9%	3.3%	3.3%	3.3%
		West	16	75.7	2.5	9.5	43.1	71.0	178.7	284	0.0%	0.0%	0.0%	0.0%
		Northeast - City Only	10	294.4	2.8	73.5	83.7	309.6	373.2	2571	12.6%	12.6%	12.6%	0.0%
		Midwest - City Only	5	224.0	2.7	51.3	131.4	373.2	490.7	689	33.0%	0.0%	0.0%	0.0%
		South - City Only	20	114.0	4.4	8.0	53.9	94.2	175.3	13596	8.6%	5.7%	5.7%	5.7%
		West - City Only	11	55.2	2.4	9.5	31.8	57.4	177.9	188	0.0%	0.0%	0.0%	0.0%
HUD National Survey (HUD NS weights) [3]	Dripline/ Entryway	All	82	103.0	3.1	8.0	55.8	89.6	179.4	13596	6.9%	4.5%	2.3%	1.1%
		City Only	42	105.7	3.9	8.0	55.8	87.7	187.5	13596	10.6%	5.2%	5.2%	2.6%
		Northeast	13	134.9	3.8	35.9	73.5	83.7	246.0	2571	17.2%	17.2%	6.0%	0.0%
		Midwest	20	118.7	2.5	11.6	61.9	116.7	244.9	689	7.4%	0.0%	0.0%	0.0%
		South	33	96.8	3.3	8.0	45.5	89.6	145.3	13596	4.9%	3.3%	3.3%	3.3%
		West	16	75.7	2.5	9.5	43.1	71.0	178.7	284	0.0%	0.0%	0.0%	0.0%
		Northeast - City Only	7	255.2	3.5	73.5	83.7	88.0	2570.5	2571	20.2%	20.2%	20.2%	0.0%
		Midwest - City Only	4	193.4	2.9	51.3	91.3	311.1	590.0	689	42.5%	0.0%	0.0%	0.0%
		South - City Only	20	114.0	4.4	8.0	53.9	94.2	175.3	13596	8.6%	5.7%	5.7%	5.7%
		West - City Only	11	55.2	2.4	9.5	31.8	57.4	177.9	188	0.0%	0.0%	0.0%	0.0%

Table 3-23b. (cont.)

Study	Location		N	Average Soil-Lead Concentration (µg/g) Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	Percentage of Homes with Average Soil-Lead Concentration > 400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
Houses Built From 1940-1959 (cont.)														
California	Oakland, LA, Sacramento		163	NA	NA	NA	NA	NA	NA	NA	19.6% [4]	NA	NA	NA
	Oakland		17	NA	NA	NA	NA	NA	NA	NA	70.6% [4]	NA	NA	NA
HUD Grantees Evaluation	Dripline	All Grantees	17	478.0	3.2	66.0	174.0	530.0	925.0	5389	64.7%	23.5%	11.8%	5.9%
		Alameda	4	484.3	2.2	174.0	273.0	645.5	922.0	925	50.0%	0.0%	0.0%	0.0%
		Rhode Island	6	509.1	4.8	66.0	140.0	536.0	1217.0	5389	66.7%	33.3%	16.7%	16.7%
		Wisconsin	5	516.5	2.7	139.0	400.0	516.0	593.0	2160	80.0%	20.0%	20.0%	0.0%
		Vermont	2	317.5	6.6	84.0	84.0	642.0	1200.0	1200	50.0%	50.0%	0.0%	0.0%
New Haven, Connecticut	Near (near the house)		115	534.9	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Far (near the street)		115	500.2	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Rochester Lead-in-Dust [5]	Dripline		13	282.6	3.5	27.5	170.5	259.0	843.0	1790	38.5%	7.7%	0.0%	0.0%
	Dripline (fine soil only)		13	276.5	3.4	29.7	146.0	272.0	851.0	1788	30.8%	7.7%	0.0%	0.0%
Houses Built From 1960-1979 (1977 for HUD Grantees and New Haven)														
HUD National Survey (unweighted)	Dripline/ Entryway	All	117	39.1	2.8	5.2	21.3	33.6	70.0	1713	2.6%	0.9%	0.0%	0.0%
		City Only	45	44.8	2.9	5.2	21.7	37.3	79.3	1713	2.2%	2.2%	0.0%	0.0%
		Northeast	10	66.8	2.1	20.6	35.3	73.1	118.6	207	0.0%	0.0%	0.0%	0.0%
		Midwest	29	29.8	2.5	5.8	19.4	28.4	39.9	685	3.4%	0.0%	0.0%	0.0%
		South	61	41.7	2.8	5.2	27.0	35.9	72.3	1713	1.6%	1.6%	0.0%	0.0%
		West	17	36.2	4.0	7.5	15.9	26.6	36.5	910	5.9%	0.0%	0.0%	0.0%
		Northeast - City Only	3	101.1	2.5	35.3	35.3	141.2	207.2	207	0.0%	0.0%	0.0%	0.0%
		Midwest - City Only	6	21.3	1.7	10.1	15.8	20.4	35.5	40	0.0%	0.0%	0.0%	0.0%
		South - City Only	27	56.1	2.8	5.2	28.2	49.5	91.7	1713	3.7%	3.7%	0.0%	0.0%
		West - City Only	9	28.7	2.9	7.9	20.3	26.6	34.3	337	0.0%	0.0%	0.0%	0.0%

Table 3-23b. (cont.)

Study	Location		N	Average Soil-Lead Concentration (µg/g) Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	Percentage of Homes with Average Soil-Lead Concentration ≥ 400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
Houses Built From 1960-1979 (1977 for HUD Grantees and New Haven) (cont.)														
HUD National Survey (403 RA) [2]	Dripline/ Entryway	All	120	38.0	2.7	5.2	21.4	34.2	75.8	1713	2.0%	0.3%	0.0%	0.0%
		City Only	46	40.9	2.7	5.2	21.7	37.8	83.9	1713	0.9%	0.9%	0.0%	0.0%
		Northeast	10	66.7	2.1	20.6	35.3	73.1	118.6	207	0.0%	0.0%	0.0%	0.0%
		Midwest	29	30.6	2.4	5.8	19.4	28.4	39.9	685	3.7%	0.0%	0.0%	0.0%
		South	64	41.9	2.6	5.2	27.2	36.8	83.4	1713	0.8%	0.8%	0.0%	0.0%
		West	17	29.6	3.3	7.5	15.9	26.6	36.5	910	3.7%	0.0%	0.0%	0.0%
		Northeast - City Only	3	118.6	2.1	35.3	35.3	141.2	207.2	207	0.0%	0.0%	0.0%	0.0%
		Midwest - City Only	6	22.1	1.6	10.1	15.8	20.4	35.5	40	0.0%	0.0%	0.0%	0.0%
		South - City Only	28	57.3	2.5	5.2	30.3	50.8	87.9	1713	2.0%	2.0%	0.0%	0.0%
		West - City Only	9	26.0	2.4	7.9	20.3	26.6	34.3	337	0.0%	0.0%	0.0%	0.0%
HUD National Survey (HUD NS weights) [3]	Dripline/ Entryway	All	117	37.5	2.7	5.2	21.3	33.6	70.0	1713	2.0%	0.3%	0.0%	0.0%
		City Only	45	40.6	2.7	5.2	21.7	37.3	79.3	1713	0.9%	0.9%	0.0%	0.0%
		Northeast	10	66.7	2.1	20.6	35.3	73.1	118.6	207	0.0%	0.0%	0.0%	0.0%
		Midwest	29	30.6	2.4	5.8	19.4	28.4	39.9	685	3.7%	0.0%	0.0%	0.0%
		South	61	40.8	2.6	5.2	27.0	35.9	72.3	1713	0.8%	0.8%	0.0%	0.0%
		West	17	29.6	3.3	7.5	15.9	26.6	36.5	910	3.7%	0.0%	0.0%	0.0%
		Northeast - City Only	3	118.6	2.1	35.3	35.3	141.2	207.2	207	0.0%	0.0%	0.0%	0.0%
		Midwest - City Only	6	22.1	1.6	10.1	15.8	20.4	35.5	40	0.0%	0.0%	0.0%	0.0%
		South - City Only	27	56.8	2.5	5.2	28.2	49.5	91.7	1713	2.0%	2.0%	0.0%	0.0%
		West - City Only	9	26.0	2.4	7.9	20.3	26.6	34.3	337	0.0%	0.0%	0.0%	0.0%
California	Oakland, LA, Sacramento		93	NA	NA	NA	NA	NA	NA	NA	16.1% [4]	NA	NA	NA
	Oakland		12	NA	NA	NA	NA	NA	NA	NA	58.3% [4]	NA	NA	NA
New Haven, Connecticut	Near (near the house)		33	286.5	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Far (near the street)		33	382.2	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Rochester Lead-in-Dust [5]	Dripline		4	66.3	2.1	27.5	37.9	69.6	125.6	160	0.0%	0.0%	0.0%	0.0%
	Dripline (fine soil only)		4	66.3	1.8	29.0	49.5	78.0	98.5	111	0.0%	0.0%	0.0%	0.0%

Table 3-23b. (cont.)

Study	Location		Average Soil-Lead Concentration (µg/g)								Percentage of Homes with Average Soil-Lead Concentration ≥			
			N	Geometric Mean	Geometric Std. Dev.	Minimum	25th Percentile	Median	75th Percentile	Maximum	400 µg/g	1200 µg/g	2000 µg/g	5000 µg/g
Houses Built After 1979 (1977 for HUD Grantees and New Haven)														
HUD National Survey (403 RA) [2]	Dripline/ Entryway	All	28	27.4	2.5	5.6	11.9	28.3	52.3	144	0.0%	0.0%	0.0%	0.0%
		City Only	9	32.4	2.5	7.9	21.3	31.5	37.3	144	0.0%	0.0%	0.0%	0.0%
		Midwest	4	15.4	1.8	7.5	9.0	15.4	27.9	35	0.0%	0.0%	0.0%	0.0%
		South	18	34.5	2.5	5.6	19.9	32.8	70.0	144	0.0%	0.0%	0.0%	0.0%
		West	6	20.2	2.4	7.9	9.5	17.9	31.5	105	0.0%	0.0%	0.0%	0.0%
		Midwest - City Only	1	35.5	1.0	35.5	35.5	35.5	35.5	35	0.0%	0.0%	0.0%	0.0%
		South - City Only	5	52.7	2.2	21.3	27.6	37.3	128.3	144	0.0%	0.0%	0.0%	0.0%
		West - City Only	3	14.0	1.8	7.9	7.9	11.0	31.5	31	0.0%	0.0%	0.0%	0.0%
HUD Grantees Evaluation	Dripline	Minnesota	1	330.0	.	330.0	330.0	330.0	330.0	330	0.0%	0.0%	0.0%	0.0%
Rochester Lead-in-Dust [5]	Dripline		10	147.6	3.7	17.8	66.0	125.8	705.0	874	30.0%	0.0%	0.0%	0.0%
	Dripline (fine soil only)		10	135.3	3.1	26.0	72.0	125.5	169.0	876	20.0%	0.0%	0.0%	0.0%

NA = Not Available

1 *Mass-weighted arithmetic average soil-lead concentration as reported in the HUD National Survey; summarized without weighting by sample weights.*
2 Mass-weighted arithmetic average soil-lead concentration as calculated in Chapter 3 of the 403 risk analysis; summarized by weighting each average to reflect the 1997 U.S. housing stock.
3 Mass-weighted arithmetic average soil-lead concentration as reported in the HUD National Survey; summarized by weighting with the National Survey sample weights.
4 Percent of samples that exceed 500 µg/g.
5 Some houses in this housing age category may belong to an earlier age category, as some houses may have actually been built earlier than the year specified within the study's database.

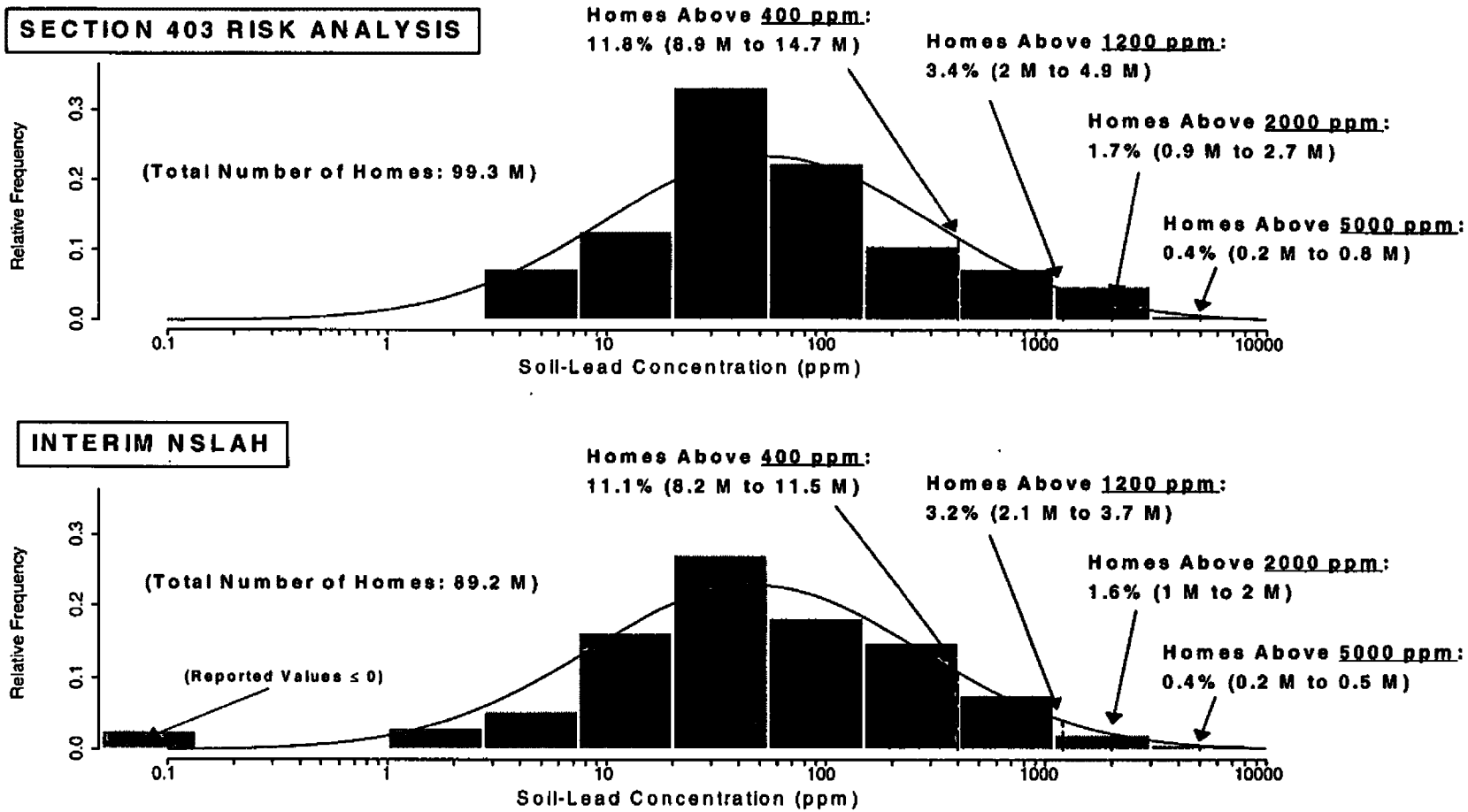

Figure 3-18. Estimated Distribution of Yardwide Average Soil-Lead Concentration in the Nation's Housing Stock, and Corresponding Estimates of the Percentage of Homes Exceeding Specified Thresholds (with 95% Confidence Intervals on the Corresponding Number of Homes, in Millions), Based on Data from the HUD National Survey (top plot) and the Interim NSLAH (bottom plot)

Note: The estimated exceedance percentages are calculated based on the fitted distribution (solid curve).

surveys. Each estimated exceedance percentage is accompanied by an approximate 95% confidence interval on the number of homes in the U.S. housing stock that exceeds the threshold (given in millions).

In Figure 3-18, the distribution based on the HUD National Survey data used in the §403 risk analysis was determined by censoring data values below 24.5 ppm (i.e., the bottom 30 percent of the data, taking into account the sample weights). The distribution based on the interim NSLAH data was determined by censoring data values below 2.01 ppm, which corresponds to the bottom 5% of the observed weighted distribution, including negative values.

Among these four thresholds, the estimated percentage of residences that exceed the threshold vary widely. For a threshold of 2000 ppm, the estimated percentage is 1.6% to 1.7% for the two surveys, while the percentage increases to from 11.1% to 11.8% for the two surveys when the threshold is lowered to 400 ppm.

For both surveys, the estimated exceedance percentages specified within Figure 3-18 for yardwide average soil-lead concentration, based on the fitted lognormal distribution, are also included within Table 3-24 (columns 2 and 4) for the same four thresholds. Also included in Table 3-24 (columns 3 and 5) are estimated exceedance percentages that were determined solely by the proportion of total sampling weights in the survey that corresponded to surveyed units whose household average floor dust-lead loadings exceeded the given threshold (i.e., information from the bar charts within Figure 3-18). The two types of estimates are very similar for the interim NSLAH data except at the highest threshold, while for the HUD National Survey data, differences between the estimates increase as the threshold decreases. It should be noted that the lognormal-based estimates for the exceedance percentages (which were also portrayed in Figure 3-18) should be used when making inferences on the nation's housing stock.

Table 3-24. Estimated Percentages of 1997 U.S. Housing Exceeding Specified Thresholds of Yardwide Average Soil-Lead Concentration

Soil-Lead Conc. Threshold (ppm)	§403 Risk Analysis – Based on Data from the HUD National Survey (n=284)		Data from the Interim NSLAH (n=706)	
	Based on the Fitted Lognormal Distribution (i.e., the curve in Figure 3-18)	Based on the Weighted Observed Data (i.e., the bar chart in Figure 3-18)	Based on the Fitted Lognormal Distribution (i.e., the curve in Figure 3-18)	Based on the Weighted Observed Data (i.e., the bar chart in Figure 3-18)
400	11.8%	13.2%	11.1%	11.2%
1200	3.4%	4.7%	3.2%	2.9%
2000	1.7%	2.5%	1.6%	1.7%
5000	0.4%	0.2%	0.4%	0.1%

Note: Data are imputed for those surveyed units with missing data prior to calculating the above statistics (34 observations in the HUD National Survey had either dripline or remote soil-lead concentration imputed prior to calculating a yardwide average; 42 observations in the interim NSLAH had an imputed yardwide average). The estimates based on the weighted observed data are simple weighted percentiles that do not originate from a fitted distribution.

It was also desired to calculate exceedance percentages for only urban residences within the U.S. housing stock, as urban soil has the potential for being more likely to be contaminated by lead than non-urban soil (in the absence of a particular lead source). Thus, the procedure used to fit the distributions in Figure 3-18 was also applied to the HUD National Survey data for only the 146 surveyed homes labeled as being located in urban areas. (The interim NSLAH data were not included in this exercise because homes were not characterized by urbanicity.)

Figure 3-19 plots the distribution and documents the exceedance percentages for urban residential soil-lead concentrations as estimated using the HUD National Survey data. Based on the fitted lognormal distribution, this figure indicates that approximately 2.8 percent of the roughly 40 million homes in urban areas are estimated to exceed a yardwide average soil-lead concentration of 2000 ppm[11]. This corresponds to approximately 1.1 million homes. However, because the sampling weights in the HUD National Survey (and revised in the §403 risk analysis) were not necessarily determined to ensure that the weights assigned to the homes in urban areas would be representative of the entire urban housing stock, caution must be taken in making inferences on the national urban housing stock based on these estimates.

3.2.2.4 Interpreting the Observed Differences with Other Studies. Contrasting the measured soil-lead concentrations from one study to another is complicated by differences in study designs, sampling locations, and sampling and laboratory protocols and practices used by these studies. As areal patterns in the lead concentration of residential soil have long been recognized, different locations within the same yard can have widely different soil-lead concentrations. For example, levels along the foundation of the residence are typically highest, reflecting the presence of deteriorated lead-based paint formerly on the residence or deposited leaded gasoline emissions washed off the roof. Also, distinct sampling protocols may impact the amount of lead measured in a collected sample. The Rochester and Milwaukee studies, for example, partitioned a collected soil sample into fine- and coarse-sieved fractions. Finally, various laboratory practices and procedures can leach more or less lead from the digested soil sample. Some studies seek to mimic "bioavailable" lead by using an acidic digestion meant to mimic human stomach acids.

Unfortunately, insufficient data were available from the various studies in Table 3-17 to consider fully any distinctions in soil-lead concentration that would be prompted exclusively by a study's collection and measurement practices. Undoubtedly, soil collection and measurement practices partially explain the observed differences across the studies, but their effects cannot be quantified at this stage. The data summaries in Section 3.2.2.2 attempted to express soil-lead concentrations in the Rochester study as reflecting the total sample (as is done in many studies) rather than only the fine-sieved portion of the sample by adjusting the data based on relationships observed in the Milwaukee study among fine-, coarse- and total-sieved soil fraction data.

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[11] The sum of the sampling weights (adjusted in the §403 risk analysis to represent the 1997 housing stock) for the 146 urban homes in the HUD National Survey is roughly 40 million. The fitted lognormal distribution in Figure 3-19 treats the bottom 20 percent of the HUD National Survey (based on the sample weights) as censored data at 21.3 μg/g.

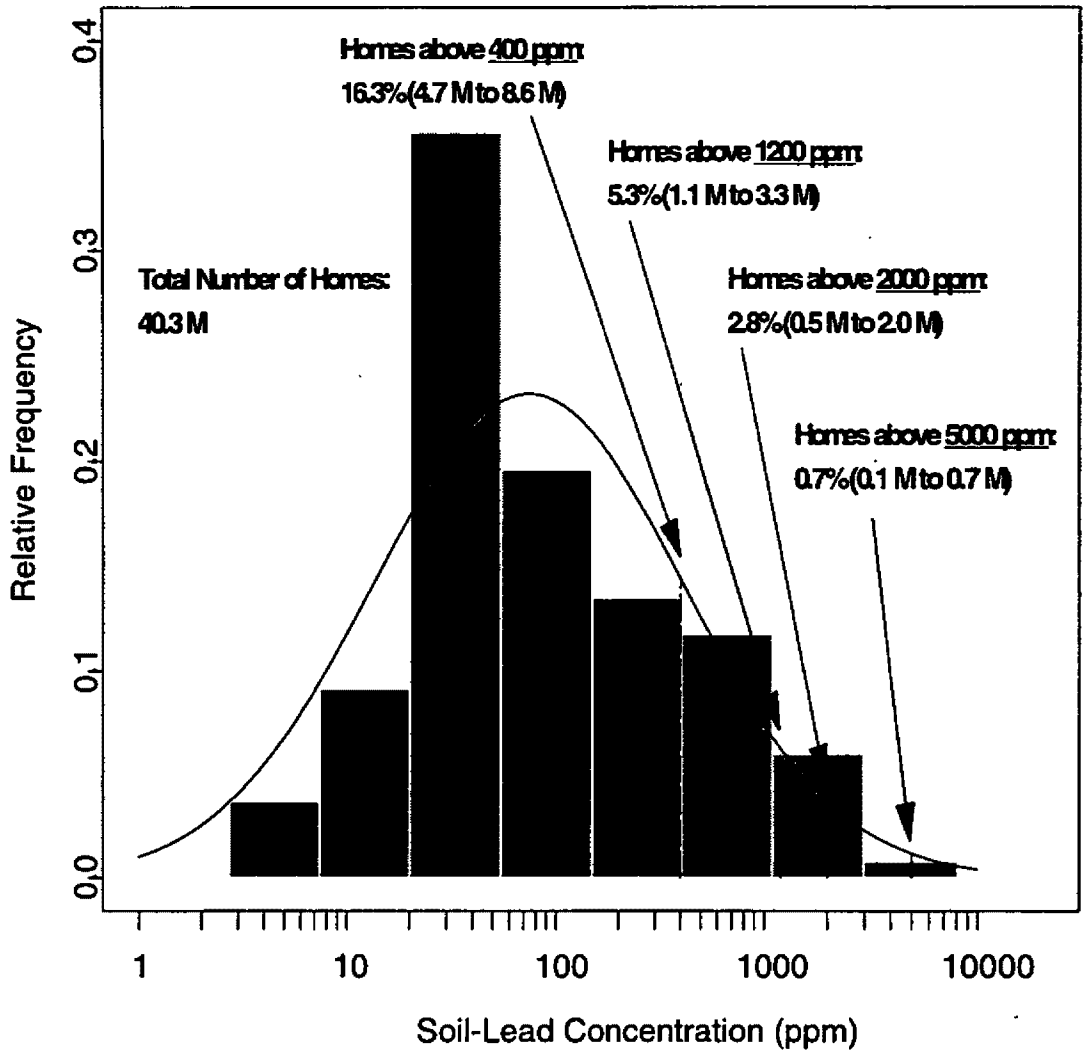

Figure 3-19. Estimated Distribution of Yardwide Average Soil-Lead Concentration Among Urban Housing in the HUD National Survey, and Corresponding Estimates of the Percentage of Urban Homes That Exceed Specified Thresholds (with 95% Confidence Intervals on the Corresponding Number of Urban Homes in the Nation, in Millions)

Note: Because the HUD National Survey was not necessarily conducted in a manner such that the sample weights for urban housing are representative of urban housing in the entire country, caution should be made when attempting to use this information to infer about urban housing for the entire nation.

It is possible to discuss study-specific caveats about how the housing selection procedure and sample collection and analysis procedures differ between the studies and, therefore, can contribute to the differences observed in the plots and tables in Section 3.2.2.2. For the Baltimore R&M, the Rochester, HUD Grantee, and HUD National Survey studies, this information was summarized in Tables 3-3a through 3-3f of the §403 risk analysis report. For the interim NSLAH, this information was summarized in Section 3.1 of this report. Some of the study differences mentioned in Section 3.2.1.4 as possibly contributing to differences in dust-lead loading data would also be contributors to differences in the reported soil-lead concentration data. Other differences among the studies in Table 3-17 include the following:

- The neighborhoods surveyed within the Baltimore R&M study, 3-City study, Cincinnati Longitudinal study, California Lead study, and HUD Grantees evaluation had a high prevalence of homes with lead-based paint hazards, along with a history of children with elevated blood-lead concentrations and/or considered at high-risk for lead poisoning.

- For the HUD Grantees evaluation, 28% of the homes were single-family buildings, 32% were single-family detached, and 12% were single-family attached (rowhouses). All homes in the R&M intervention group within the Baltimore R&M study were urban rowhouses (single-family attached). Eighty percent of the homes in the HUD National Survey were single-family dwellings. In the 3-Cities study, 100% of the Boston homes were single-family detached residences, most of the Baltimore homes were single-family attached dwellings, and the majority of Cincinnati homes were multi-story, multi-family structures.

- The dates of environmental sampling were 11/89-3/90 for the HUD National Survey, 12/93-1/99 for the HUD Grantees evaluation, 8/93-11/93 for the Rochester study, 3/93-11/94 for the Baltimore R&M study, 2/89-2/90 for the Baltimore 3-City study, 7/89-12/89 for the Boston 3-City study, and 1/89-8/89 for the Cincinnati 3-City study. Therefore, the HUD National Survey performed sampling roughly three years before the three major studies in this report, but near in time to others (such as the 3-Cities study).

- The New Orleans, Baltimore Garden, Minneapolis Clean-Up and Minnesota studies have sometimes been identified as using distinct laboratory practices, producing higher soil-lead concentrations than might be otherwise measured. The published literature regarding these studies, however, cites nothing unusual.

Because the HUD Grantees evaluation emphasizes local control of the individual programs, each grantee is responsible for designing and implementing lead-hazard reduction approaches applicable to its specific needs and objectives. These responsibilities include the recruitment methods, enrollment criteria, and intervention strategies. However, to enable comparison of results from the various approaches, grantees participating in the evaluation follow the same sampling protocols and use standard data collection forms developed

specifically for this evaluation. Table 3-4 of the §403 risk analysis report documented the differences between grantees in their enrollment/recruitment criteria. As a result, the summaries in Section 3.2.2.2 were also presented by grantee.

3.2.2.5 Conclusions of the Soil-Lead Data Comparisons. The following can be concluded from review of the boxplots and tables within Section 3.2.2 of this report, especially in regard to how the reported soil-lead concentration data for various studies compare with data from the HUD National Survey (as portrayed in the §403 risk analysis):

- Geometric mean (yard-wide) average soil-lead concentration was quite lower for the HUD National Survey relative to the yardwide estimates for most of the other studies cited in Table 3-17. However, the interim NSLAH (Section 3.2.2.1), as well as such studies as the Cincinnati 3-City and Baltimore Garden studies, did report geometric mean soil-lead concentrations that were comparable to that for the HUD National Survey. Otherwise, the distributions of soil-lead concentrations were rather consistent across the studies and available grantees.

- Among the housing age categories, the greatest difference in observed soil-lead concentration between the HUD National Survey (as portrayed in the §403 risk analysis) and the interim NSLAH was for housing built prior to 1940, where nearly a 50% decline in the estimated median was seen from the HUD National Survey to the interim NSLAH. The two sets of results were comparable among the other housing age categories.

- The low geometric mean soil-lead concentration in the HUD National Survey compared to other studies within Table 3-17 was most dramatic for homes built from 1940 to 1959. For homes built prior to 1940, the geometric mean reported in the §403 risk analysis (463 µg/g) was within 150 µg/g of that for three grantees within the HUD Grantees evaluation: California, Minnesota, and Vermont. However, for homes built from 1940 to 1959, the geometric mean soil-lead concentration across all units in the HUD Grantees evaluation (492 µg/g) was over four times higher than that reported in the §403 risk analysis (92.6 µg/g). Insufficient numbers of housing units built after 1959 in the other studies prevent reliable comparisons of soil-lead concentrations with these studies.

- Overall, the importance of housing age is evident in the summaries within the four housing age categories. Older housing is more likely to contain higher average soil-lead concentrations compared to newer housing. However, within an age category, the summaries were reasonably consistent across studies.

- As expected, dripline/entryway soil-lead concentrations consistently exceeded yard-wide average levels for all studies with sampling plans permitting such comparisons. That soil-lead concentrations exhibit an areal pattern is well-known and documented throughout the scientific literature, and suggests caution when

comparing the HUD National Survey yard-wide average results to those of other studies.

In general, the soil-lead concentrations observed in the HUD National Survey seem lower than many studies, but not necessarily beyond reason. Several of these other studies were conducted in urban neighborhoods already recognized to either have elevated environmental-lead levels or high incidence rates of elevated blood-lead concentrations among resident children. As such, higher soil-lead concentrations among these residences may be entirely consistent. Furthermore, soil-lead levels in the HUD National Survey were found to be comparable with those reported in the interim NSLAH, which reflects the entire nation's housing stock, and in other studies such as the Cincinnati 3-City and the Baltimore Garden study. Even studies conducted within the same urban area can differ considerably in the reported soil-lead concentrations; for example, the Baltimore 3-City study had levels about five times higher than the Baltimore Garden study.

3.3 EVALUATION OF SOIL PICA IN CHILDREN

This section investigates what has been published in the literature concerning the potential effects that pica for soil may have on children's exposure to lead, over and above the exposure associated with pica for paint that was considered when estimating risks in the §403 risk analysis. While the analysis did not consider the independent impact of soil pica over and above paint pica, it considered the impact of soil pica as part of the relation between soil-lead concentration and blood-lead concentration. While this section does not change the approach taken in the original §403 risk analysis, it documents information obtained on the component of soil-lead exposure that may be attributable to soil pica.

This section summarizes information on pica behavior for soil and paint for the three studies constituting the Urban Soil Lead Abatement Demonstration Project (USLADP) (USEPA, 1996a), the Rochester Lead-in-Dust study (USHUD, 1995a; Lanphear et al., 1996a), and the Baltimore Repair and Maintenance (R&M) study (USEPA, 1996c). The percentage of children who ingest soil, the frequency of soil ingestion episodes, and the amount of soil ingested by children with pica are estimated.

3.3.1 What Is Soil Pica?

Definitions. The literature provides varying definitions of pica. Pica is generally accepted to be the consumption of non-food items and there are at least nine different types of pica, including soil pica (Lacey, 1990). Some authors also consider mouthing of non-food items a pica behavior. Usually, pica is seen as normal behavior in young children, but abnormal in older children and adults. Exceptions occur, however, for some individuals, such as children and pregnant women in certain ethnic groups, the socially disadvantaged, groups of low income and socioeconomic status, developmentally delayed individuals, and the mentally retarded.

The American Psychiatric Association (DSM-III-R) has clinically defined pica as the ingestion of non-nutritive or inedible substances and requires repeated ingestion of a non-nutritive substance for at least one month before pica is considered a diagnosis. However, in practical research, authors tend to use less rigorous definitions of pica. For example, Shellshear et al., (1975) defined pica simply as an unusual appetite for non-food items.

Some authors consider pica a common occurrence in young children while others view pica behavior as abnormal. Sedman (1989) included in his definition of pica the ingestion of foreign substances by children that occurs during the course of normal development. This is consistent with Karam et al., (1990) who stated that pica includes the ingestion of some non-food items and that pica is a relatively common occurrence in small children. Barltrop et al., (1974) defined soil pica as the habitual insertion of soiled fingers or toys into a child's mouth, in addition to the direct consumption of soil. In contrast, Lyngbye et al., (1990) loosely defined pica as a mouthing habit more pronounced than in other children at the same age. Calabrese et al., (1991) defined soil pica as the ingestion of soil in amounts far exceeding those observed in the average child.

Pica for soil is considered by most authors to be the purposeful ingestion of soil. This definition is used throughout this report. Estimates of intentional soil ingestion, such as would occur in an actual "pica" episode, range from 500 to 13,000 mg soil/day, according to the studies cited in Table 3-25. To put this in perspective, quantitative estimates of inadvertent soil ingestion by normal children range from 9 to 246 mg/day (see Table 3-25), which are consistent with the estimates used in the §403 Risk Analysis.

Methods Used to Measure Soil Ingestion. Average daily soil ingestion can be quantified using a mass-balance approach, in which concentrations of tracer elements in fecal matter are measured and used to estimate the amount of soil ingested. The tracer elements typically used in soil ingestion studies include barium (Ba), manganese (Mn), silicon (Si), aluminum (Al), titanium (Ti), vanadium (V), yttrium (Y), and zirconium (Zr). However, in an adult validation study investigating the recovery of different tracer elements, Calabrese et al., (1989) concluded that the most reliable elements for this type of study are Al, Si, and especially Y. In addition, the authors indicated that when using these tracers, 500 mg/day could reliably be detected, and 100 mg/day could also be reliably detected but with a higher degree of variability. These levels are greater than most estimates of average daily soil ingestion in children. Tracer elements are generally selected due to their high concentration in soil relative to food products, and their low level of absorption in the gastrointestinal tract. Thus, the quantities of these tracer elements present in the feces, corrected for "background" or intake levels, can be attributed to the ingestion of soil (assuming there is no other non-food ingestion occurring, e.g., paint). Using the concentration of a tracer element in the bulk soil, the total quantity of soil ingested can be calculated. Concentrations of the tracer elements in the bulk soil are determined from soil samples around the child's home and play area. Samples are typically taken from the upper layers of soil (as this is where children are assumed to play), and finer size fractions may be separated out (as this size fraction is preferentially ingested) (Calabrese et al., 1989; Sheppard,

Table 3-25. Results of Literature Review on Children's Exposure to Lead Through Soil Pica

Author(s) / Publication Date	Methods	Pica Prevalence (% exhibiting soil pica)	Soil Intake Amount (soil ingestion rate in mg/day)	Pica Frequency (# days on which soil was ingested)
Mass-Balance / Chemical Tracer Studies				
Calabrese et al., (1989)	64 children, ages 1-4 years; pica measured by fecal analysis using chemical tracers and mass-balance methodology; 8-day study [a]	1.6 % (1 child in 64)	• non-pica: 9-40 mg/day • pica: 5,000-8,000 mg/day	Information Not Given
Calabrese et al., (1991)	Follow-up study of pica child in Calabrese et al., (1989) [a]	Not Applicable	• 5,000-7,000 mg/day over 2 weeks • 10,000-13,000 mg/day during week 2	Episodic • pica occurred only during week 2 of observation
Calabrese et al., (1993)	Follow-up study of pica child in Calabrese et al., (1989) [a]	Not Applicable	• lead consumed in soil: 0.96 μg/day - 11.6 μg/day (where soil lead = 22 ppm)	pica occurred on 2 days out of 8
Stanek and Calabrese, (1995)	Re-analysis of Calabrese et al., (1989) [a]	1.6 % (1 child in 64)	• 12 mg/day or less for 50% of the children (median) • 138 mg/day or less for 95% of the children (median) • 10% of subjects ate 1,200 mg/day (n = 64, mean value)	• 33% of children are expected to ingest > 10 g soil on 1-2 days/year • 16% of children are expected to ingest >1 g soil on 35-40 days/year
Calabrese and Stanek (1993)	Critique of Wong, M.S. (1988) "The Role of Environmental and Host Behavioral Factors in Determining Exposure to Infection With *Ascaris Lumbricoides* And *Trichuris Trichluta*." [Ph.D. thesis], University of the West Indies, Mona, Jamaica. 28 children, ages 1.8-14 years, and 24 children, ages 0.3-7.5 years;[b] ingestion measured by fecal analysis using chemical tracers and correction for "background"; 4 month study (1 day/month) [c]	• older group: 3.6% (1 in 28 children; child with pica was mentally retarded) • younger group: 20.8% (5 of 24 children)	• older group: 58 mg/day based on the mean (mentally retarded child excluded) • younger group: mean 470 ± 370 mg/day • highly variable for pica episodes: 1,000 - 10,300 mg/day (8 episodes over 5 children, mentally retarded child excluded)	High degree of daily variation • 3 of 6 pica children exhibited behavior on only 1 of 4 days, others did more often

Table 3-25. (cont.)

Author(s) / Publication Date	Methods	Pica Prevalence (% exhibiting soil pica)	Soil Intake Amount (soil ingestion rate mg/day)	Pica Frequency (# days on which soil was ingested)
Calabrese et al., (1997)	12 children, ages 1-3 years; children preselected as exhibiting soil ingestion (Stanek et al., 1998); ingestion measured by fecal analysis using chemical tracers and mass-balance methodology; 7-day study [4]	• 8.3% (1 child in 12) clearly exhibited pica / high soil ingestion	• non-pica: < 10 mg/day (5 children); 10-20 mg/day (4 children) • pica: 500-3,050 mg/day (median 1,320 mg/day)	• pica child ingested between 0.5-3.0 g/day on 4 of 7 days
Davis et al., (1990)	104 children, aged 2-7 years; ingestion measured by fecal analysis using chemical tracers and mass-balance methodology; study over 7 consecutive days.	0% pica reported	• median soil ingestion estimates ranged from 25.3-81.3 mg/day (n= 101), depending on the tracer • mean soil ingestion estimates ranged from 38.9 ± 12.2 to 245.5 ± 119.7 mg/day (n = 101), depending on the tracer	Information Not Given
Studies Based on Interviews and Questionnaires				
Stanek et al., 1998	553 children assessed at well-child pediatric visits; presence of pica behavior assessed via parent interview[5]	Reported by age; pica prevalence of 38% at age 1 year declines to 21% at age 2 years and <10 % for ages 3-6 years; overall estimate of 18% for children aged 1 to 6 years	Information Not Given	• 38% at least monthly, 24% at least weekly, 11% daily at age 1 year • 21% at least monthly, 7% weekly, 0% daily at age 2 years
Abadin et al., (1997)	Discussion of ATSDR method to estimate blood-lead levels in children, where soil ingestion is one predictor	Not Applicable	• use assumed soil ingestion rate of 200 mg/day	Not Applicable
Bates et al., (1995)	143 children, aged 12-23 months; soil ingestion assessed via questionnaire [6]	• 62% (89 of 143) ate soil • 38% (54 of 143) never ate soil	Information Not Given	Information Not Given
Sedman and Mahmood, (1994)	Estimated average daily and lifetime soil ingestion in young children using results of two previous chemical tracer studies (Davis et al., 1990 and Calabrese et al, 1989); age adjusted for a 2 year-old child	Not Applicable	• mean estimate (2 year-old): 195 mg/day (std. err. - 53 mg/day) • estimated average daily soil ingestion over a lifetime: 70 mg/day (accounting for changes in soil ingestion with age)	Information Not Given
Greene et al., (1992)	270 socioeconomically disadvantaged children; pica assessed via caretaker interview at ages 2 years, 3 years, and 4 years 10 months [6]	• 19% at 2 years of age • 13% at 3 years of age • 6% at 4 years 10 months of age	Information Not Given	Information Not Given

Table 3-25. (cont.)

Author(s)/ Publication Date	Methods	Pica Prevalence (% exhibiting soil pica)	Soil Intake Amount (soil ingestion rate mg/day)	Pica Frequency (# days on which soil was ingested)
Annest and Mahaffey (1984)	2,195 children, ages 1-5 years; presence of pica behavior assessed by household interview [e]	• 6 month-3 year age group: 11.0% • 4-5 year age group: 3.2% • annual family income < $10K: 11.9% • annual family income ≥ $10K: 6.0%	Information Not Given	Information Not Given
Shellshear et al., (1975)	170 children, ages 1-5 years; pica assessed via parent interview [f]	10.6% (18 children of 170)	Information Not Given	Information Not Given
Barltrop et al., (1974)	119 children in two towns, ages 2-3 years; 48 children in high soil lead area; presence of pica behavior assessed via parent interview [e]	• 51 of 119 (43%) conformed to pica definition; 11 of 119 (9%) known to swallow soil • 33% (16 of 48) of children had pica for soil (in high soil lead area)	Information Not Given	Information Not Given
Cohen et al., (1973)	230 rural and 272 urban children, mean age of 4 years; history pica assessed via parent questionnaire[g]	• 50% of the children had a history of pica at some time for dirt, cigarettes, or other non-food items • 10% reported ingestion of paint or plaster	Information Not Given	Information Not Given

[a] *soil pica defined as ingestion of soil in quantities far exceeding those observed in the average child*
[b] subject population consisted of Jamaican children of normal intelligence in long-term institutional settings, with the exception of one child (i.e., the child exhibiting extreme pica in the older group) who was mentally retarded
[c] pica defined as ingestion of > 1 g soil/day
[d] high soil ingestion defined as >0.5 g soil/day on a regular or nearly daily basis
[e] soil pica not quantitatively defined in this study; pica only indicates the occurrence of ingestion
[f] soil pica defined as "an unusual appetite" for soil
[g] definition of pica for soil included children who habitually put fingers, etc., in mouth while playing in their gardens, as well as children who actually put soil directly into their mouths

1998). Most quantitative estimates of the amount of soil ingested that were reviewed in this report were obtained using the mass-balance/tracer element approach.

Incidence rates (i.e., prevalence) of pica for soil in young children may be estimated from parental questionnaires. This approach can yield biased results, however, as it relies on the observation and accurate reporting of pica by the adult. In addition, the response depends on the wording of the question. Various surveys have asked whether the child eats dirt (Annest and Mahaffey, 1984; Bates et al., 1995; USEPA, 1996a - Boston and Baltimore portions), whether the child puts dirt or sand in mouth while playing outside (USEPA, 1996a - Cincinnati portion; USEPA, 1996c), or whether the child puts fingers or toys in mouth while playing outside (Barltrop et al., 1974). Clearly, these questions would elicit different responses from the same caregiver. In addition, response choices may be simply yes or no, may specify a timeframe (e.g., in the past month), or may be open-ended. These choices, too, would result in differing responses. Thus, care must be taken in comparing soil pica prevalence rates originating from parental questionnaire data.

While mass-balance studies provide soil ingestion rates to support prevalence data, these studies are also subject to error and have disadvantages. For example, Calabrese et al., (1989) acknowledge that analyzing chemical tracers without the use of a mass-balance approach (i.e., not correcting for intake) can result in soil ingestion estimates that are increased by factors of 2 to 6. In addition, the particular tracer used, the duration of the study, and the frequency of sampling may also influence reported results (Calabrese et al., 1989; Calabrese et al., 1997). For example, the short duration of most mass-balance studies makes it difficult to determine a "normal" rate of soil ingestion for a child. Approaches using chemical tracers also have disadvantages in that they are more expensive and generally have small sample sizes.

3.3.2 How Does the §403 Risk Analysis Account for Soil Pica?

Within the exposure assessment (Chapter 3) portion of the §403 risk analysis report (USEPA, 1998a), soil was considered an indirect source of lead exposure, although summary information on soil pica frequency from two lead exposure studies was presented in Table 3-3b of the §403 risk analysis report.

An indicator of soil pica was considered as a candidate predictor variable in the development of the empirical model for the §403 risk analysis. The soil pica variable was based on the parental questionnaire administered in the Rochester Lead-in-Dust study. This variable measured the child's tendency to put dirt or sand in the mouth using a scale of 0 (never) to 4 (always). The soil pica variable was borderline significant in single media models, which assessed the relationship between blood-lead concentration and each predictor variable under consideration. These single media models were the first step in developing the empirical model. Variable selection for the multimedia exposure model was based on several properties, including the strength of the relationship with blood-lead concentration as estimated using the bivariate statistical models, predictive power of each variable when included into a model with competing sources of lead exposure, and interpretability of parameter estimates. The soil pica variable was

dropped during this phase of the empirical model development. Additional information on the development of the empirical model can be found in Appendix G of the §403 risk analysis report.

Age-dependant soil and dust ingestion rates for the Integrated Exposure, Uptake, and Biokinetic (IEUBK) model were taken from the IEUBK guidance manual (USEPA, 1994) and represent central tendencies within the range of values seen in different studies. Combined soil and dust ingestion amounts ranged from 85 to 135 mg/day, as shown in Table 4-1 of the §403 risk analysis report, of which 45 percent was assumed to be from soil. Thus, soil ingestion was assumed to be between 38 and 61 mg/day for children aged 0 to 7 years, with the maximal ingestion estimated for children aged 1 to 3 years. These ingestion rates are consistent with estimates of inadvertent soil ingestion presented in this report, but are not representative of pica episodes. While IEUBK model predicted blood-lead levels were adjusted in the §403 risk analysis to allow consideration of paint pica in homes with damaged lead-based paint, as described in Section 4.1 and Appendix D1 of the §403 risk analysis report, no such adjustment was made for the effect of soil pica.

It should be noted that while neither model used in the §403 risk analysis had explicitly accounted for soil pica as a separate factor independent of paint pica, the impact of soil pica was included in the analysis as part of the relation between soil-lead concentration and blood-lead concentration which the analysis characterized.

3.3.3 Prevalence of Soil Pica Behavior

Estimates reported in the scientific literature of the percentage of children who ingest soil are summarized in this section. From the literature, it was not possible to estimate the percentage of children who exhibit pica for soil but not paint. The §403 risk analysis did account for the effect of paint pica on blood-lead concentration estimates. For children who ingest both paint chips and soil, it is reasonable to assume that the effect of soil pica is insignificant compared to that of paint pica. Thus, in estimating the percentage of children who ingest soil, it is important to exclude those who also ingest paint chips. It was possible to estimate the percentage of children who exhibit soil pica, but not paint pica, using information from parental questionnaires administered in the USLADP study (USEPA, 1996a), Baltimore R&M study (USEPA, 1996c), and Rochester Lead-in-Dust study (USHUD, 1995a; Lanphear et al., 1996a). This information is also summarized in this section.

3.3.3.1 Literature Review. Most sources in the literature reported prevalence rates for general pica behavior (mouthing or eating non-food items) or for soil pica (eating dirt). One source (Stanek et al., 1998) reported pica rates for a variety of specific non-food items (soil, paint chips, paper, toys, etc), but did not cross-tabulate [e.g., 18 percent of children ages 1 - 6 years, as assessed by parent interview, were reported to ingest/mouth dirt at least monthly and 3 percent to ingest/mouth paint chips, but information was not provided on how many children eat both soil and paint chips (Stanek et al., 1998)]. An overview of selected studies estimating pica prevalence is shown in Table 3-25 above. It is important to note that in the cited studies, various definitions of "pica" were used in reporting the prevalence of pica behavior. For example,

Greene et al., (1992) defined soil pica only as the occurrence of soil ingestion and reported the percentage of children who ingest soil based on caretaker interview. In comparison, Calabrese et al., (1997) defined soil pica as consumption exceeding 0.5 grams per day and reported the prevalence of pica behavior as assessed quantitatively by mass-balance methods. It should be noted that only primary research findings are reported in Table 3-25, with the exception of Calabrese and Stanek (1993). Several review articles were also obtained. These were excluded from Table 3-25, as insufficient details of the source studies were provided. General findings of the review articles are cited in the text.

Table 3-25 shows that the estimated percentage of children ingesting soil ranged from 1.6 to 62 percent and varied with definition/criteria for soil pica used, age group of children, and socioeconomic status. In general, 12 of 16 observations in the table report a prevalence of soil ingestion in children of 13 percent or lower (where, at a minimum, limiting criteria are defined as ingesting soil at least once). "Normal" mouthing behavior, however, is typically exhibited more commonly, particularly in the younger age groups. For example, Barltrop et al., (1974) reported that 43 percent of children exhibited pica defined to include mouthing behavior, but that 9 percent were estimated to swallow soil. Stanek et al., (1998) assessed non-food ingestion and mouthing behaviors in 533 children, ages 1 to 6, by parental interview. Results of the survey indicated that 38 percent of 1 year old children and 21 percent of 2-year old children ingest/mouth soil at least monthly. In contrast, at ages 3 to 6 years, less than 10 percent of children were observed to ingest/mouth soil at least monthly. At age 1 year, 11 percent of children were observed to ingest/mouth soil daily compared to one percent or less among children aged 2 to 6 years.

Of the studies that used mass-balance methodology, prevalence of soil pica ranged from 1.6 to 20.8 percent. For studies that employed parent or caretaker interview methodology, soil pica prevalence ranged from 3.2 to 19 percent, although one study reported a rate of 62 percent, which appears to be more consistent with studies that monitored general mouthing behavior. For both methodologies, the prevalence of soil ingestion tended to be higher in the younger age groups and for children in families with lower socioeconomic status. Although, as mentioned in Section 3.3.1 above, techniques utilizing parental interview are generally considered less reliable than quantitative methodologies, the issue of consistently defining "pica" when reporting study results is an issue not only in studies using questionnaire methodology, but also in the mass-balance/chemical tracer studies. Differences in values reported, both between and within the various assessment techniques, may largely be due to differences in how pica behavior is being defined in the study. Thus, many studies estimating soil ingestion prevalence may not consistently monitor the actual overall risk associated with soil pica behavior in children.

3.3.3.2 Prevalence of Soil Pica Separate from Paint Pica. Although the literature review provided several estimates of the prevalence of soil pica behavior, none of the cited sources provided information about concurrent paint pica behavior. For the purpose of this report, the prevalence of soil pica behavior in the absence of paint pica is of interest, as the §403 risk analysis did account for paint pica behavior. For children who ingest both paint chips and soil, it is reasonable to assume that the effect of soil pica is insignificant compared to that of

paint pica. Unless there is industrial contamination, or the home is in an area with heavy traffic, where residual leaded gasoline emissions are present, lead in residential soil is usually derived primarily from lead-based paint. Thus, soil pica can be considered an indirect pathway of exposure to leaded paint, whereas paint pica is a direct exposure pathway.

Information on pica behavior for paint, soil, and other objects was collected in the three USLADP studies (USEPA, 1996a), the Rochester Lead-in-Dust study (USHUD, 1995a; Lanphear et al., 1996a), and the Baltimore R&M study (USEPA, 1996c), through parental reporting of observed behaviors. Therefore, it is possible to use these data to estimate the prevalence of soil pica separately from paint pica. This information is summarized in Table 3-26. As can be seen in this table, rates of soil pica only range from 9.1 to 40.9 percent, while rates of both soil and paint pica range from 1.4 to 7.4 percent.

Some of the disparity in the rates reported in Table 3-26 can be explained by the survey questions and other factors associated with the study. For example, in the Rochester Lead-in-Dust, Baltimore R&M, and Cincinnati USLADP studies, parents were asked how frequently the child put dirt or sand in his or her mouth. In contrast, parents in the Boston and Baltimore portions of the USLADP were asked how frequently the child ate dirt or sand. The paint pica questions were more consistent across studies, querying how frequently the child put paint chips in his or her mouth. In the Cincinnati USLADP study, the time-period of observation for both soil and paint was limited to the previous month, whereas the other studies used open-ended time periods. Response rates in Rochester were consistent with literature estimates of soil pica that included mouthing behavior, while the Baltimore R&M and Cincinnati USLADP studies provided substantially lower estimates. Because most homes in the Baltimore R&M study had small or no yards, the low estimates of soil mouthing behavior are not unexpected. The lower response in Cincinnati is probably due to the limited period of observation.

Since inadvertent soil ingestion due to mouthing behavior was included in the IEUBK model analysis for the §403 risk analysis, the prevalence of soil ingestion, rather than mouthing behavior, is of interest in the context of this report. Thus, the Boston and Baltimore portions of the USLADP study provide the best estimates of soil pica behavior in the absence of paint pica. These estimates are 14.4 and 16.3 percent in Boston and Baltimore, respectively. These estimates are greater than those derived from the mass-balance studies, but consistent with other studies that rely on parental reporting methods. The prevalence of pica for both paint and soil was low in Boston (1.4 %), but somewhat higher in Baltimore (6.0 %). Adding these rates to the reported rates for soil pica alone does not substantially increase the estimates, however, which remain in the range of other studies that rely on parental reporting.

Table 3-26. Estimated Rates of Paint and Soil Pica Behavior Reported in the USLADP Studies, the Rochester Lead-in-Dust Study, and the Baltimore R&M Study

Study	Type of Pica Behavior	Study Children Exhibiting Such Pica Behavior		
		Percent (#) of Study Children[1]	Average Age (months)	Geometric Mean, Blood-Lead Conc. (µg/dL)
Boston USLADP (146 children)	Soil only	14.4% (21)	NA	12.5
	Paint only	9.6% (14)	NA	12.7
	Soil and Paint	1.4% (2)	NA	13.4
	neither	74.7% (109)	NA	11.7
Baltimore USLADP (400 children)	Soil only	16.3% (65)	35.7	12.0
	Paint only	10.5% (42)	33.1	11.7
	Soil and Paint	6.0% (24)	26.5	15.2
	neither	67.3% (269)	41.7	10.3
Cincinnati USLADP (220 children)	Soil only	23.2% (51)	35.9	12.8
	Paint only	2.7% (6)	19.7	15.3
	Soil and Paint	2.3% (5)	28.0	14.7
	neither	71.8% (158)	28.1	8.9
Baltimore R&M Pre-intervention (165 children)	Soil only	9.1% (15)	27.5	10.5
	Paint only	7.3% (12)	26.7	15.3
	Soil and Paint	7.3% (12)	24.2	20.7
	neither	76.4% (126)	31.1	9.4
Rochester Lead-in-Dust (203 children)	Soil only	40.9% (83)	21.0	6.1
	Paint only	2.5% (5)	23.0	11.5
	Soil and Paint	7.4% (15)	20.0	8.5
	neither	49.3% (100)	21.4	6.2

[1] A response of "Unknown" was treated as missing and was not included in the calculation of these percentages.
NA = Not applicable

3.3.4 Estimating the Frequency of Ingestion and Amount of Soil Ingested by Children Who Exhibit Soil Pica

As discussed in Section 3.3.3.1 above, studies reporting soil ingestion prevalence have the potential to misrepresent the extent of soil pica behavior in children due to differences in methodology and criteria for defining pica. Therefore, estimates of ingestion quantity and frequency may also be employed to assess the severity of soil pica behavior.

Mass-balance studies provide data on the frequency of ingestion and amount of soil ingested by children who exhibit soil pica. These studies estimate the typical amounts of soil inadvertently ingested by normal children as ranging from 9 to 246 mg/day. The estimated

quantities ingested in actual "pica" episodes are between 500 and 13,000 mg/day (Table 3-25). The literature generally reports pica behavior to be episodic in nature, varying both amongst different children and within individual children. In addition, the occurrence (including both frequency and quantity) of soil ingestion was observed to be influenced by the age of the child (Stanek et al., 1998; Annest and Mahaffey, 1984), as well as by variety of factors that may alter the child's access to soil, including seasonal variation and/or climate/vegetation differences (Simon, 1998; Calabrese and Stanek, 1993), socioeconomic status (Annest and Mahaffey, 1984; Bhatia, 1988), and parental supervision (Calabrese and Stanek, 1993; Bhatia, 1988). However, Davis et al., (1990) found that although there was considerable variability in soil ingestion estimates among children, there was no consistent demographic or behavioral factor that was predictive of soil ingestion.

Calabrese et al., (1989) estimated median soil ingestion rates, including those involved in non-pica behavior, between 9 and 40 mg/day (n = 64). Calabrese et al., (1997) also observed median soil ingestion rates under 40 mg/day in 12 children (selected from the population described in Stanek et al., 1998) identified by their parents as likely to ingest soil at a high rate. These levels of soil ingestion typically would not be considered pica behavior. Each of these studies, however, did report observations of a child exhibiting extreme soil pica behavior, with one child ingesting from 5 to 8 grams of soil per day (Calabrese et al., 1989) and another child ingesting between 0.5 and 3.0 grams of soil per day on 4 of 7 days (Calabrese et al., 1997). Calabrese et al., (1991) found that the soil pica behavior for the former child occurred only on two days during the two weeks of observation with an ingestion rate ranging from 10-13 grams of soil per day, suggesting that the issue of variability in soil pica behavior may be very important, meriting further research. Implications of these patterns were demonstrated by Calabrese et al., (1993), who observed that on the two days when the child displayed soil pica behavior, she also displayed striking increases in fecal lead excretory values. In contrast, the pica child reported in Calabrese et al., (1997) consistently ingested large quantities of soil (0.5-3 g soil/day on 4 of 7 days).

Calabrese and Stanek (1993) presented results of a 4-month mass-balance/chemical tracer study performed by M.S. Wong of 52 Jamaican children of generally normal intelligence in an institutional setting. The children were partitioned into a younger (0.3-7.5 years) group and an older (1.8-14 years) group. One of the children in the older group exhibited mental retardation. This was the only child in the older group (of 28 children) that exhibited soil pica exceeding one gram of soil per day. This child had an average ingestion rate of 41 g soil/day over 4 months (observations on 1 day per month). In the younger group, 10.5 percent of total observations (n = 84) included soil pica, and five of the 24 children exhibited soil pica on at least one occasion. The Wong study showed that soil pica occurred more frequently in younger children, and there was a fairly high degree of daily variation in soil ingestion among the children exhibiting soil pica. For example, 3 of 6 children displayed pica on only 1 of 4 days. Furthermore, even for the children who consumed soil more consistently with regards to frequency, the rates were still variable (e.g., 1.0-10.3 g/day). Calabrese and Stanek (1993) suggest that although this study confirms that soil pica, strictly defined as ingestion greater than 1.0 g/day, is likely to be rare in

older children, the Wong study is important in that it challenges the idea that pica is a rare event in younger children.

Using daily soil ingestion data from their 1989 study, Stanek and Calabrese (1995) developed annual soil ingestion distribution estimates as follows. First, the mean and variance of daily soil ingestion were estimated for each of the 64 children in the 1989 study, based on 4 to 8 daily estimates for each child. Then 365 daily soil ingestion amounts for each child were calculated as percentiles of a log-normal distribution with the estimated mean and variance, in increments of 1/365. Based on these distributions, Stanek and Calabrese conclude that 33 percent of children are expected to ingest more than 10 grams of soil on 1-2 days per year and that 16 percent of children are expected to ingest more than 1 gram of soil on 35-40 days per year. These ingestion levels are consistent with amounts estimated for soil pica episodes. The median and 95th percentile for average daily soil ingestion resulting from this method were 75 mg/day and 1,751 mg/day, respectively. While the median estimate is similar to previous estimates, the estimated 95th percentile is substantially greater than most other estimates.

Assumptions and limitations of this approach include:

1. The assumption of a log-normal distribution for daily soil ingestion. Insufficient data were available to determine whether this assumption is reasonable.

2. The estimation of the mean and variance for each child based on very small sample sizes. The annual estimates were strongly affected by the tails of the distribution, which are imprecise due to large variability in the estimates of the mean and variance.

3. The extrapolation of daily soil ingestion estimates from a 2-week period in autumn to the remainder of the year, without regard to possible seasonal effects. In addition, the children studied were a nonrandom sample residing in or near an academic community in western Massachusetts. Thus, the soil ingestion behavior of these children may not be representative of those living in other climates, geographic regions, or in inner-city or rural areas.

4. The presence of trace elements in fecal matter was assumed to be entirely due to soil consumption, after correcting for food consumption, with no contribution from indoor dust.

Many of these assumptions and limitations serve to introduce positive bias to the daily soil ingestion estimate, while the effect of others is unclear. Nonetheless, this analysis is at present, the only available source of both frequency of soil pica episodes and amount ingested during soil pica episodes.

3.3.5 Conclusions on Soil Pica

The following conclusions can be made from the findings presented in this section:

- The prevalence of soil pica, exclusive of paint pica, is most likely between 10 and 20 percent in young children. For the purpose of this report, the Boston and Baltimore portions of the USLADP provide the best estimates of soil pica behavior in the absence of paint pica (14.4 and 16.3 percent, respectively).

- Soil pica behavior is episodic in nature. The frequency of soil pica episodes depends on many factors, including climate, access to bare soil, socioeconomic standing, age of child, and parental supervision. In one study of 12 children identified by their parents to be predisposed to pica for soil, only one child displayed soil pica during the two week observation period (Calabrese et al., 1997). Only one study estimated annual rates for pica episodes (Stanek and Calabrese, 1995). This study suggested that 33 percent of children would ingest more than 10 grams of soil on 1-2 days per year, and that 16 percent of children are expected to ingest more than 1 gram of soil on 35-40 days per year.

- Estimates of the amount of soil ingested during pica episodes vary widely among the mass balance studies, from 500 to 13,000 mg/day. The average daily ingestion over a year, however, may be much lower. Assuming the frequencies estimated by Stanek and Calabrese (1995), children who ingest 15 grams of soil on 1-2 days per year and 50 mg/day on remaining days would have an average daily soil intake of 132 mg/day over the course of a year. Children who ingest 1.5 grams of soil on 40 days per year and 50 mg/day on remaining days would have an average daily soil intake of 209 mg/day. A question, however, is whether the amount of lead in soil ingested on the small number of days where pica episodes occurred would be sufficient to elevate the blood-lead concentration to unsafe levels.

3.4 CHARACTERIZING THE POPULATION OF CHILDREN IN THE NATION'S HOUSING STOCK

For the §403 risk analysis, it was necessary to estimate numbers of children of specific age groups who reside within the 1997 national housing stock in order to characterize the extent to which various environmental-lead levels provide exposures to children and to characterize the benefits associated with performing interventions under §403 rules. These estimates were based on numbers of housing units determined by sampling weights within the HUD National Survey (conducted in 1989-1990), revised to represent the 1997 national occupied housing stock and on average numbers of children per housing unit determined from the 1993 American Housing Survey (AHS). The estimates used in the §403 risk analysis were presented in Section 3.3.2 and Appendix C of the §403 risk analysis report. This section provides alternative estimates using

more recent data (i.e., interim data from the NSLAH and data from the 1997 American Housing Survey).

The method to calculating the alternative estimates involved determining the numbers of children in a given age group for each of the 706 housing units surveyed within the NSLAH whose interim data were made available to this effort. Methods used to obtain these estimated numbers of children were similar to those presented in Section 1.2 of Appendix C1 of the §403 risk analysis report. For a given age group of children, the estimated number of children associated with a given NSLAH-surveyed unit was determined by the following formula:

$$\#\text{children} = (1997\ \text{weight}) * (\text{Average}\ \#\ \text{residents per unit}) * (\#\ \text{children per person}) \quad (1)$$

The "1997 weight" factor in equation (1) was the interim sampling weight from the NSLAH for the unit. The factor "average # residents per unit" in equation (1) was calculated for the housing group based on information obtained from the 1997 AHS. The 1997 AHS database provided information on up to 18 residents within each housing unit in the AHS. Once units surveyed in the 1997 AHS were placed within the four year-built categories (pre-1940, 1940-1959, 1960-1979, post-1979), the average number of people residing in a unit (regardless of their ages) was calculated for each group. This average ranged from 2.5 to 2.7 across the four year-built categories. Therefore, a common average of 2.6 residents per unit was used for the entire national housing stock. The third factor in equation (1), "# children per person," represented the average number of resident children (of the given age group) in a housing unit. This factor was determined by dividing the total number of residents in the housing stock of a given age group by the total number of residents regardless of age, where both totals were calculated from data in the 1997 AHS. The method for calculating this third factor differed from the approach used in the §403 risk analysis, where forecasted birth rate and population estimates from the Bureau of the Census were used.

Table 3-27 contains estimates of average number of children per unit in the 1997 national housing stock, according to age group. These number are the product of the final two factors in equation (1). Therefore, these number are multiplied by the sampling weights for each housing unit in the interim NSLAH to obtain a revised number of children per housing unit. For children aged 12-35 months, the estimated average of 0.073 children per unit is about 9% lower than the estimate of 0.080 used in the §403 risk analysis.

Table 3-27. Alternative Estimates of the Average Number of Children Per Unit in the 1997 National Housing Stock, by Age of Child

Age Group	Estimated Average Number of Children Per Unit
12-35 months	2.6*0.0281 = 0.073
12-71 months	2.6*0.0732 = 0.190

By summing the estimates across surveyed units in the interim NSLAH, the updated number of children aged 12-35 months and 12-71 months residing within the 1997 national housing stock is obtained by year-built category and for the nation. Table 3-28 provides these alternative estimates on the number of children residing in the 1997 housing stock according to age of housing unit and age of child. The overall estimate of approximately 6.51 million children aged 1-2 years is approximately 18% lower than the estimate of 7.96 million made in the §403 risk analysis. The lower estimates are due to the lower per-unit estimate from Table 3-27 and on the lower sample weight total in the interim NSLAH data compared to the HUD National Survey (Table 3-2). They are also likely to be underestimates of the numbers of children of the given age category, based upon population projections previously published by the U.S. Bureau of the Census (e.g., Day, 1993).

Table 3-28. Alternative Estimates of the Average Number of Children in the 1997 National Housing Stock, by Age of Child and Year-Built Category, Based on Data Obtained Since the §403 Risk Analysis

Years in Which Housing Units Were Built	Age of Child Within These Housing Units	
	1-2 Years	1-5 Years
Prior to 1940	1,053,000	2,743,000
1940-1959	1,234,000	3,214,000
1960-1977	1,877,000	4,889,000
After 1977	1,759,000	4,582,000
Unknown[1]	591,000	1,540,000
All Housing[2]	6,513,000	16,967,000

[1] There are 66 units in the interim NSLAH which have missing age of house data.
[2] Values in this row may differ from sum of previous rows due to rounding.

3.5 SUMMARIES OF DUST-LEAD LEVELS ON SURFACES OTHER THAN UNCARPETED FLOORS AND WINDOW SILLS

The exposure assessment in Chapter 3 of the §403 risk analysis report concluded that even at low to moderate lead levels, lead-contaminated dust can affect children's blood-lead concentration. The assessment focused on dust-lead found on floor and window sill surfaces, for which §403 regulatory standards were proposed. However, dust found on other surfaces, such as exterior dust and dust in air ducts, carpeted floors, window troughs (also known as window wells), and upholstery, may also potentially present a lead exposure hazard. Many issues concerning potential exposure to dust-lead on these other surfaces were raised throughout the §403 Dialogue Process as well as in comments received on preliminary drafts of the §403 risk analysis and on the proposed rule. For example, there was extensive discussion during the Dialogue Process concerning whether standards were necessary for window troughs (i.e., window wells) as long as there are standards for window sills and the window troughs are

thoroughly cleaned. Concern was also expressed about sampling on carpeted and upholstered surfaces.

The purpose of this section is to supplement information in the original exposure assessment by assessing the potential exposure to dust-lead found on surfaces other than floors and window sills. In particular this assessment seeks to answer the following questions:

1. What information is available to assess residential lead exposure resulting from dust on surfaces other than floors and window sills?

2. What does this information say about the distribution of environmental lead levels for dust on these other types of surfaces?

3. Is there evidence of a relationship between these exposures and children's blood-lead concentrations?

4. Is the information sufficient to set a regulatory standard and is a standard necessary?

The exposure assessment is based on a review of the literature to identify studies with potentially useful data for assessing lead hazards due to dust-lead on these other surfaces. It should be noted that this section deals only with several specific surfaces other than uncarpeted floors and window sills. These surfaces include exterior dust, air ducts, window troughs, and upholstery. Hazards associated with lead-contaminated dust from carpeted floors are addressed in Section 6.5 and Appendix I of this report.

The literature review for this assessment drew upon previous literature reviews conducted for the §403 risk analysis and reviews conducted for other EPA published reports (e.g., USEPA, 1997b). Most of the studies that were found that addressed the various surfaces are included here, regardless of whether there is any information specifically relating dust-lead levels and blood-lead levels. For those studies where blood samples were collected from resident children, those results are also presented. Table 3-29 provides a summary of the studies that were examined. The table indicates the surfaces from which dust samples were collected and in the case of exterior dust samples, where those samples were collected.

Table 3-29 indicates that the review of the literature found fourteen studies that have examined exterior dust as a source of lead exposure and seven studies that similarly assessed window troughs. Only four studies were located with significant information on dust-lead levels in air ducts, and four with similar information on upholstery.

3.5.1 Distribution of Dust-Lead on Surfaces Other than Floors and Window Sills

Tables 3-30 through 3-33 present summary information from the studies related to exterior dust samples, air duct samples, window trough samples, and upholstery samples,

Table 3-29. Studies for Which Dust Samples Have Been Collected from Exterior Areas, Air Ducts, Window Troughs, and Upholstery for Lead Analysis

✓ = medium was sampled in the given study

Study	Exterior Dust (Sampling Location)	Air Ducts	Window Troughs	Upholstery	Blood
Lead-Based Paint Abatement and Repair & Maintenance (R&M) Study (USEPA, 1996c, 1997c)	✓ (Entryway)	✓	✓	✓	✓
University of Rochester Lead-In-Dust Study (USHUD, 1995a)	✓ (Play area, Porch, Entryway?)		✓		✓
Urban Soil Lead Abatement Demonstration Project (USEPA, 1996a)	✓ (Entryway, Mat)				✓
The National Survey of Lead-Based Paint in Housing (USEPA, 1995)			✓		
The HUD Lead-Based Paint Abatement Demonstration (USHUD, 1991)			✓		✓
The Comprehensive Abatement Performance (CAP) Study (USEPA, 1996b)	✓ (Entryway)	✓	✓		
Birmingham Urban Lead Uptake Study (Davies et al., 1990)	✓ (Playground, Doormat, Pavement, Roadside)				✓
Mexico City Study (Romieu et al., 1995)	✓ (Street)				✓
Butte-Silver Bow Environmental Health Lead Study (Butte-Silver Bow Dept. of Health et al., 1991)	✓ (Entrance)				✓
Midvale Community Lead Study (Bornschein et al., 1990)	✓ (Entrance)				✓
Philadelphia Neighborhood Lead Study (USDHHS, 1991)	✓ (Street)				✓
The Arnhem, Netherlands Lead Study (Brunekreef et al., 1981)	✓ (Street)				✓
Belgium Lead Smelter Study (Roels et al., 1980)	✓ (School playground)				✓
Mount Pleasant Household Lead Study (Francek et al., 1994)	✓ (Entrance?)				
Baltimore Experimental Paint Abatement Study (Farfel et al., 1991)			✓		
Traditional vs. Modified Practices of Lead Abatement in Baltimore (Farfel et al., 1990)			✓		✓
Wales Environmental Lead Study (Gallacher et al., 1984)	✓ (Pavement)				✓
New Orleans Day Care Center Lead Study (viverette et al., 1996)	✓ (Play area)				
Omaha Study of Childhood Lead (Angle et al., 1995)		✓			✓
Renovation & Remodeling Study (USEPA, 1997a)		✓			
HVFS Pilot Study (Roberts et al., 1996)				✓	
Throop, PA, Superfund Cleanup (Steuteville, 1990)				✓	

Table 3-30. Summary of Data from Studies Where Exterior Dust Samples Were Collected for Lead Analysis

Study	Environmental-Lead Measurements					Blood-Lead Measurements		
	Group of Homes/ Sampling Location	Sampling Method	N	Concentration Statistics (units)	Loading Statistics (units)	Group of Homes/Children	N	Statistics (units)
Lead-Based Paint Abatement and Repair & Maintenance (R&M) Study (1993-present)				GM [95% CI] (µg/g)	GM [95% CI] (µg/ft²)			GM [95% CI] (µg/dL)
	Entryway:	BRM						
	R&M I, Init. Camp.		25	2219 [1218 - 4043]	242 [109 - 539]	R&M I, Init. Camp.	33	9.9 [7.9 - 12.3]
	R&M II, Init. Camp.		23	4265 [2588 - 7029]	187 [102 - 340]	R&M II, Init. Camp.	32	13.8 [11.2 - 16.9]
	R&M III, Init. Camp.		26	6936 [3549 - 13555]	342 [184 - 637]	R&M III, Init. Camp.	33	14.2 [11.3 - 16.1]
	Pre. Abt., Init. Camp.		15	2073 [1232 - 3488]	227 [76 - 676]	Pre. Abt., Init. Camp.	23	12.8 [10.2 - 16.1]
	Mod. Urb., Init. Camp.		15	137 [75 - 250]	335 [188 - 597]	Mod. Urb., Init. Camp.	19	4.8 [3.8 - 6.1]
University of Rochester Lead-In-Dust Study (1993)				GM [± 2 SD] (µg/g)	GM [± 2 SD] (µg/ft²)			GM [SD] (µg/dL)
	Porch	BRM		1132 [42 - 30150]	548 [7 - 43370]	All Children	205	7.7 [5.1]
		DVM		557 [52 - 6017]	17 [1 - 446]	Levels < 10 µg/dL	157	5.5 [2.2]
		Wipe			57 [4 - 871]	Levels ≥ 10 µg/dL	48	15.1 [5.0]
	Entryway	BRM		468 [19 - 11243]	88 [0 - 15881]			
		DVM		329 [18 - 5967]	3 [0 - 124]			
		Wipe			18 [2 - 215]			
	External	BRM	145		335 [7 - 17271]			
	Combined	DVM	150		18 [1 - 576]			
The Comprehensive Abatement Performance (CAP) Study (1992)				GM [Range] (µg/g)	GM [Range] (µg/ft²)			
	Entryway	Cyclone vacuum	97	237 [9-16,355]	384 [4.0 - 14021]			
Birmingham Urban Lead Uptake Study (1984-1985)				GM [5th, 95th %] AM [Range] (µg/g)				GM [5th, 95th %] (µg/g)
	Doormat	Vacuum	42	615 [120 - 4300]				
				1436 [79 - 15000]				
	Pavement	Vacuum	97	360 [127 - 1340]				11.7 [6.24]
				506 [62 - 5100]				
	Roadside	Vacuum	97	527 [195 - 1170]				
				805 [80 - 2100]				

Table 3-30. (cont.)

Study	Environmental-Lead Measurements					Blood-Lead Measurements		
	Group of Homes/ Sampling Location	Sampling Method	N	Concentration Statistics (units)	Loading Statistics (units)	Group of Homes/Children	N	Statistics (units)
Urban Soil Lead Abatement Demonstration Project (1988-1992)				Med. (µg/g)				GM (µg/dL)
	Cincinnati Mat:	Personal Air						
	Soil, Dust Abt. Rd. 1	Monitoring		109		Soil, Dust Abt. Rd. 1		8.8
	Soil, Dust Abt. Rd. 2	Vacuum		738		Soil, Dust Abt. Rd. 2		-
	Soil, Dust Abt. Rd. 3	Pump		549		Soil, Dust Abt. Rd. 3		6.9
	Soil, Dust Abt. Rd. 4			767		Soil, Dust Abt. Rd. 4		8.8
	Soil, Dust Abt. Rd. 5			659		Soil, Dust Abt. Rd. 5		-
	Dust, (Soil) Abt. Rd. 1			132		Soil, Dust Abt. Rd. 6		8.2
	Dust, (Soil) Abt. Rd. 2			939		Soil, Dust Abt. Rd. 7		8.7
	Dust, (Soil) Abt. Rd. 3			702		Dust, (Soil) Abt. Rd. 1		10.8
	Dust, (Soil) Abt. Rd. 4			722		Dust, (Soil) Abt. Rd. 2		-
	Dust, (Soil) Abt. Rd. 5			889		Dust, (Soil) Abt. Rd. 3		9.3
	No Treatment Rd. 1			100		Dust, (Soil) Abt. Rd. 4		8.6
	No Treatment Rd. 2			373		Dust, (Soil) Abt. Rd. 5		-
	No Treatment Rd. 3			349		Dust, (Soil) Abt. Rd. 6		7.6
	No Treatment Rd. 4			405		Dust, (Soil) Abt. Rd. 7		8.9
	No Treatment Rd. 5			332		No Treatment Rd. 1		8.3
	Cincinnati Entryway:	Personal Air				No Treatment Rd. 2		-
	Soil, Dust Abt. Rd. 1	Monitoring		334		No Treatment Rd. 3		5.7
	Soil, Dust Abt. Rd. 2	Vacuum		606		No Treatment Rd. 4		6.8
	Soil, Dust Abt. Rd. 3	Pump		433		No Treatment Rd. 5		-
	Soil, Dust Abt. Rd. 4			491		No Treatment Rd. 6		7.2
	Soil, Dust Abt. Rd. 5			211		No Treatment Rd. 7		7.8
	Soil, Dust Abt. Rd. 6			382				
	Soil, Dust Abt. Rd. 7			488				
	Dust, (Soil) Abt. Rd. 1			425				
	Dust, (Soil) Abt. Rd. 2			492				
	Dust, (Soil) Abt. Rd. 3			468				
	Dust, (Soil) Abt. Rd. 4			632				
	Dust, (Soil) Abt. Rd. 5			102				
	Dust, (Soil) Abt. Rd. 6			598				
	Dust, (Soil) Abt. Rd. 7			615				
	No Treatment Rd. 1			290				
	No Treatment Rd. 2			367				
	No Treatment Rd. 3			317				
	No Treatment Rd. 4			286				
	No Treatment Rd. 5			84				
	No Treatment Rd. 6			317				
	No Treatment Rd. 7			284				

Table 3-30. (cont.)

Study	Environmental-Lead Measurements					Blood-Lead Measurements		
	Group of Homes/ Sampling Location	Sampling Method	N	Concentration Statistics (units)	Loading Statistics (units)	Group of Homes/Children	N	Statistics (units)
Mexico City Study (1992-1994)				AM [IQR] (µg/g)				AM [SD] (µg/dL)
	Street	Broom	200	206 [89.5 - 270]		<18 months old	52	7.38 [4.81]
						18-35 months old	55	10.13 [5.92]
						35-49 months old	44	11.07 [5.83]
						50 months old	49	11.40 [5.76]
						Total	200	9.91 [5.78]
Butte-Silver Bow Environmental Health Lead Study (1990)				GM [GSD] (µg/g)				GM [GSD] (µg/dL)
	Entrance:	DVM						
	All Locations		210	541 [2.96]		All Locations	183	3.69 [1.84]
	Location A		141	921 [2.59]		Location A	15	2.27 [1.67]
	Location B		10	302 [1.72]		Location B	12	4.59 [1.89]
	Location C		7	439 [3.56]		Location C	11	4.56 [1.79]
	Location D		9	218 [2.43]		Location D	27	2.72 [1.50]
	Location E		21	188 [2.26]		Location E	17	3.02 [1.52]
	Location F		11	273 [2.56]		Location F	17	3.02 [1.52]
	Location G		11	924 [1.45]		Location G	13	3.81 [1.67]
Midvale Community Lead Study (1989)				GM [(Range] (µg/g)				GM [Range] (µg/dL)
	Entryway	Vacuum	112	466 [[79 - 2984]				5.2 [0.5 - 14.5]
Philadelphia Neighborhood Lead Study (1989)				AM (µg/g)				AM [SD] (µg/dL)
	Street:	Spatula						
	1 Block from Facility			1087		L. Pt. Richm. 0-5 yrs.	122	9.7 (4.8)
	2 Blocks from Facility			1078		Comparison 0-5 yrs.	96	9.5 (3.5)
	3 Blocks from Facility			907		U. Pt. Richm. 0-5 yrs.	55	9.1 (3.7)
	4 Blocks from Facility			882		Manayunk 0-5 yrs.	41	10.0 (3.3)
						L. Pt. Richm. 6-15 yrs.	41	7.8 (3.3)
						Comparison 6-15 yrs.	41	7.4 (2.9)
						U. Pt. Richm. 6-15 yrs.	29	6.7 (2.9)
						Manayunk 6-15 yrs.	12	9.0 (2.4)
						L. Pt. Richm. ≥16 yrs.	197	6.4 (3.3)
						Comparison ≥16 yrs.	239	7.3 (4.3)
						U. Pt. Richm. ≥16 yrs.	142	6.9 (5.0)
						Manayunk ≥16 yrs.	97	7.7 (3.0)
						L. Pt. Richm. Total	360	7.7 (4.1)
						Comparison Total	376	7.8 (4.1)
						U. Pt. Richm. Total	226	7.4 (4.6)
						Manayunk Total	150	8.4 (3.2)

Table 3-30. (cont.)

Study	Environmental-Lead Measurements					Blood-Lead Measurements		
	Group of Homes/ Sampling Location	Sampling Method	N	Concentration Statistics (units)	Loading Statistics (units)	Group of Homes/Children	N	Statistics (units)
The Arnhem Netherlands Study (1978)				GM [Range] (mg/kg) AM				GM (µg/dL)
	Street	Vacuum		690 [77 - 2667] 859				16.1
Mount Pleasant Household Study (1991)				GM [SD] AM [Range] (µg/g) Med.				
	Entrance?	Wipe	42	12 [105] 53 [0 - 594] 7				
Wales Environmental Lead Study (unknown)	Pavement			AM [95% CI] (µmol)				AM [SD] (µg/dL)
	Area 1 Area 2	Unknown	42 30	2 [0.8 - 3.5] 1 [0.4 - 4.2]		Area1, Children Area1, Mother Area2, Children Area2, Mother	42 42 30 30	16.56 [8.49] 8.90 [2.48] 14.70 [3.73] 8.90 [2.90]
New Orleans Day Care Center Lead Study (unknown)					Med. [Range] (µg/ft²)			
	Play area Private inner city Private outer city Public inner city Public outer city	Unknown	 5 4 5 5		 412 [44 - 690] 3 [2.2 - 8.0] 11 [8 - 33] 11 [9.5 - 18.4]			

N = Sample size
GM = Geometric mean
AM = Arithmetic mean
Med. = Median
SD = Standard deviation
GSD = Geometric standard deviation
IQR = Interquartile range (75th percentile - 25th percentile)
BRM = Baltimore R&M vacuum method
DVM = Dust vacuum method
Rd = Sampling round

Table 3-31. Summary of Data from Studies Where Air Duct Dust Samples Were Collected for Lead Analysis

Study	Environmental-Lead Measurements					Blood-Lead Measurements			
	Group of Homes/ Sampling Location	Sampling Method	N	Concentration Statistics (units)	Loading Statistics (units)	Group of Homes/Children	N	Statistics (units)	
Lead-Based Paint Abatement and Repair & Maintenance (R&M) Study (1993-present)				GM [95% CI] (µg/g)	GM [95% CI] (µg/ft²)			GM	[SD] (µg/dL)
	R&M I, Init. Camp.	BRM	1	- -	- -	R&M I, Init. Camp.	33	9.9	[7.9 - 12.3]
	R&M II, Init. Camp.		12	1445 [617-3,388]	51405 [33,671-78,480]	R&M II, Init. Camp.	32	13.8	[11.2 - 16.9]
	R&M III, Init. Camp.		15	1491 [945-2,354]	30046 [18,399-49,066]	R&M III, Init. Camp.	33	14.2	[11.3 - 16.1]
	Pre. Abt., Init. Camp.		1	- -	- -	Pre. Abt., Init. Camp.	23	12.8	[10.2 - 16.1]
	Mod. Urb., Init. Camp.		0	- -	- -	Mod. Urb., Init. Camp.	19	4.8	[3.8 - 6.1]
The Comprehensive Abatement Performance (CAP) Study (1992)				GM [Range] (µg/g)	GM [Range] (µg/ft²)				
	Denver Pilot Study	Blue Nozzle	10	749 [363-1,699]	308 [27-3,910]				
	Denver Full Study	CAPS Cyclone	109	427 [59-5,640]	120 [2 - 40,900]				
R&R Study (1993-1995)					GM [Range] (µg/ft²)				
	Baltimore & Denver	Wipe	21		2,900 [205-30,900]				
Omaha Study of Childhood Lead (1995)					AM (µg/g)				
	Omaha City	Unknown	21		383				

N = Sample size
GM = Geometric mean
AM = Arithmetic mean
Med. = Median
SD = Standard deviation
BRM = Baltimore R&M vacuum method
DVM = Dust vacuum method
Rd = Sampling round

Table 3-32. Summary of Data from Studies Where Window Trough Dust Samples Were Collected for Lead Analysis

Study	Environmental-Lead Measurements					Blood-Lead Measurements			
	Group of Homes/ Sampling Location	Sampling Method	N	Concentration Statistics (units)	Loading Statistics (units)	Group of Homes/Children	N	Statistics (units)	
Lead-Based Paint Abatement and Repair & Maintenance (R&M) Study (1993-present)				GM [95% CI] (µg/g)	GM [95% CI] (µg/ft²)			GM	[SD] (µg/dL)
	R&M I, Init. Camp.	BRM	43	22144 [15,091-32,495]	7051 [4,896-10,156]	R&M I, Init. Camp.	33	9.9	[7.9 - 12.3]
	R&M II, Init. Camp.		45	20462 [15,106-27,717]	9900 [7,245-13,529]	R&M II, Init. Camp.	32	13.8	[11.2 - 16.9]
	R&M III, Init. Camp.		54	21600 [12,751-36,590]	13916 [10,104-19,167]	R&M III, Init. Camp.	33	14.2	[11.3 - 16.1]
	Pre. Abt., Init. Camp.		31	2251 [1,247-4,062]	802 [501-1,284]	Pre. Abt., Init. Camp.	23	12.8	[10.2 - 16.1]
	Mod. Urb., Init. Camp.		30	338 [239-479]	1021 [515-2,024]	Mod. Urb., Init. Camp.	19	4.8	[3.8 - 6.1]
University of Rochester Lead-In-Dust Study (1993)				GM [± 2 SD] (µg/g)	GM [± 2 SD] (µg/ft²)			AM	[SD] (µg/dL)
	Rochester, NY	BRM		6114 [65 - 579,533]	11874 [26 - 5,365,819]	All Children	205	7.7	[5.1]
		DVM		1709 [17 - 171,081]	370 [3 - 45,177]	Levels < 10 µg/dL	157	5.5	[2.2]
		Wipe			2759 [29 - 264,752]	Levels ≥ 10 µg/dL	48	15.1	[5.0]
The National Survey of Lead-Based Paint in Housing (1989-1990)[1]				GM [Range] (µg/g)	GM [Range] (µg/ft²)				
	Homes Built Prior to 1940	Blue Nozzle	77	8389 [189.8 - 74980]	929 [1.6 - 23798]				
	Homes Built 1940-1959		87	1972 [5.2 - 41429]	140 [0.3 - 5312]				
	Homes Build 1960-1979		120	1016 [19.2 - 17725]	110 [0.04 - 3244]				
	All Surveyed Homes		284	1965 [5.2 - 74980]	177 [0.04 - 23798]				
	Homes Built Prior to 1940	Wipe-Equivalents	77		2017 [8.7 - 33391]				
	Homes Built 1940-1959		87		389 [1.9 - 9446]				
	Homes Build 1960-1979		120		296 [0.4 - 6169]				
	All Surveyed Homes		284		460 [0.4 - 33391]				
The HUD Lead-Based Paint Abatement Demonstration (1990-1993)					> 800 µg/ft² [#] [%]				
	Albany	Wet Wipe	98		[2] [2.0%]				
	Cambridge		119		[5] [4.2%]				
	Omaha		161		[0] [0%]				
The Comprehensive Abatement Performance (CAP) Study (1992)				GM [Range] (µg/g)	GM [Range] (µg/ft²)				
		Cyclone Vacuum	98	1439 [72.9 - 45229]	2515.6 [19.1 - 244581]				

Table 3-32. (cont.)

Study	Environmental-Lead Measurements					Blood-Lead Measurements		
	Group of Homes/ Sampling Location	Sampling Method	N	Concentration Statistics (units)	Loading Statistics (units)	Group of Homes/Children	N	Statistics (units)
Baltimore Experimental Paint Abatement Study (1986-1987)					GM (µg/ft²)			
	New Wndw., Pre-Abt.	Wipe	11		15663			
	New Wndw., Pst.-Abt.		13		2238			
	New Wndw., Pst.-Trt.		13		1212			
	New Wndw., Pst. C.-u.		14		466			
	New Wndw., 1m. Pst.		10		186			
	New Wndw., 3m. Pst.		13		466			
	New Wndw., 6-9m. Pst.		14		466			
	On-sit. C. S., Pre-Abt.		17		44566			
	On-sit. C. S., Pst.-Abt.		16		29462			
	On-sit. C. S., Pst.-Trt.		12		5687			
	On-sit. C. S., Pst. C.-u.		17		1585			
	On-sit. C. S., 1m. Pst.		15		5314			
	On-sit. C. S., 3m. Pst.		15		7179			
	On-sit. C. S., 6-9m. Pst.		10		2984			
Traditional vs. Modified Practices of Lead Abatement in Baltimore (1984-1985)					GM (µg/ft²)			
	Traditional	Wet Wipe			15570			
	Modified				18367			

[1] Area-weighted household averages are summarized in this table for this study, using sampling weights modified in the §403 risk analysis to represent the 1997 housing stock. Wipe-equivalents represent converting the Blue-Nozzle dust-lead loadings to wipe-equivalent loadings as documented in Chapter 4 of the §403 risk analysis report. Data were imputed for surveyed homes without data (see Chapter 3 of the §403 risk analysis report).

N = Sample size
GM = Geometric mean
AM = Arithmetic mean
Med. = Median
SD = Standard deviation
BRM = Baltimore R&M vacuum method
DVM = Dust vacuum method
Rd = Sampling round

Table 3-33. Summary of Data from Studies Where Upholstery Dust Samples Were Collected for Lead Analysis

Study	Environmental-Lead Measurements					Blood-Lead Measurements		
	Group of Homes/ Sampling Location	Sampling Method	N	Concentration Statistics (units)	Loading Statistics (units)	Group of Homes/Children	N	Statistics (units)
Lead-Based Paint Abatement and Repair & Maintenance (R&M) Study (1998-present)				GM [95% CI] (µg/g)	GM [95% CI] (µg/ft²)			GM [95% CI] (µg/dL)
	R&M I, Init. Camp.		23	699 [493-992]	95 [49-186]	R&M I, Init. Camp.	33	9.9 [7.9 - 12.3]
	R&M II, Init. Camp.		7	700 [180-2,722]	92 [39-218]	R&M II, Init. Camp.	32	13.8 [11.2 - 16.9]
	R&M III, Init. Camp.		0	- -	- -	R&M III, Init. Camp.	33	14.2 [11.3 - 16.1]
	Pre. Abt., Init. Camp.		14	503 [353-718]	101 [55-186]	Pre. Abt., Init. Camp.	23	12.8 [10.2 - 16.1]
	Mod. Urb., Init. Camp.		16	142 [101-200]	61 [35-138]	Mod. Urb., Init. Camp.	19	4.8 [3.8 - 6.1]
Mexico City Study (1992-1994)					AM [SD] (µg/ft²)			AM [SD] (µg/dL)
					70 0.11 [0.19]	<18 months old	52	7.38 [4.81]
						18-35 months old	55	10.13 [5.92]
						35-49 months old	44	11.07 [5.83]
						50 months old	49	11.40 [5.76]
						Total	200	9.91 [5.78]
HVFS Pilot Study (1993)				AM [Range] µg/g	AM [Range] µg/ft²			
	Seattle	HVFS	5	229 [130-380]	27.8 [2.7-94.9]			
Throop, PA Superfund Cleanup (1989-1990)					AM [Range] µg/ft²			
	Throop, PA Pre-Clean	Vacuum (15.2/ min air pump)	5		28.5 [19.9-34.3]			
	Throop, PA Post-Clean	Vacuum (15.2/ min air pump)	5		23.1 [13.7-35.5]			

N = Sample size
GM = Geometric mean
AM = Arithmetic mean
Med. = Median
SD = Standard deviation
BRM = Baltimore R&M vacuum method
DVM = Dust vacuum method
Rd = Sampling round

respectively. Included in the tables are sampling location, dust collection methods used, number of dust samples taken, and distribution statistics available from the literature on dust-lead concentrations and loadings. Also presented in the tables are simple summaries of blood-lead concentrations for those studies which also sampled blood.

Exterior Dust

Table 3-30 summarizes the data from the fourteen studies in which exterior dust samples were collected and analyzed for lead content. In addition, there are also results for several studies in which dust samples were collected inside the home in the entryway. These can occasionally be good representations of exterior dust samples. The summary consists of descriptive statistics for the dust-lead measures and also for blood-lead measures, when available. Summary statistics are presented separately for different study groups and different sampling periods, where appropriate. As indicated in Table 3-29, exterior dust samples were not collected at a consistent location across all studies. Sampled locations included the house entrance (including doormats and porches), street, and the child's play area. Likewise, samples were collected by a variety of methods including surface scraping, vacuum sampling, and wipe sampling. The sampling location and method have a significant impact on the lead loading and concentration estimates.

Overall, even with the variability in sampling location and method, Table 3-30 indicates the potential for significant amounts of lead in exterior dust, with concentrations often exceeding 400 $\mu g/g$ and loadings often exceeding 100 $\mu g/ft^2$.

Air Ducts

As indicated in Table 3-31, only four studies were identified that contained information on lead levels in dust within air ducts in residential housing. While only limited information was encountered, a consensus across studies was that air ducts can contain high amounts of dust and lead. This was due partially to the general lack of cleaning of air ducts over time and the ability of lead particles to enter air ducts from outside of the unit via ventilation filters.

In units with a potential for containing lead hazards, dust-lead loadings in air ducts typically exceeded 100 $\mu g/ft^2$, with individual samples often exceeding 1,000 $\mu g/ft^2$. Lead levels can vary considerably among dust samples within the same unit and in different units. Older ductwork and HVAC systems, as well as vacant units in which no cleaning is performed and HVAC systems may not be used, tend to have high dust loadings, and therefore, higher dust-lead loadings when a lead source is present. Several methods were used across studies to collect dust in air ducts. As air ducts often have metal surfaces, issues concerning static electricity must be considered when sampling dust from air ducts.

The EPA Comprehensive Abatement Performance (CAP) study involving occupied homes assumed to be free of lead-based paint for at least two years, provided the greatest amount of information on lead in dust within air ducts; levels were relatively low in this study compared

to the others. Nevertheless, in a typical housing unit in the CAP study, average dust-lead loadings from air ducts exceeded all other sampled surfaces except for window troughs and entryways. In general, air duct dust-lead levels in the Baltimore R&M study and the Renovation and Remodeling study were considerably higher than in the CAP study, as these studies included older, vacant units in need of repair and maintenance.

The relative sparsity of published information indicates that many open questions exist on the nature of lead contamination of dust within residential air ducts and whether the lead in this dust is available for exposure to residents (especially children). Nevertheless, evidence exists that air ducts can contain some of the highest levels of lead in dust within a housing unit.

Window Troughs

Table 3-32 presents summary information on seven studies that sampled dust-lead levels in window troughs (also known as window wells). The HUD Grantees evaluation is a significant data source on window trough dust-lead levels in high risk housing that is not included in Table 3-32, as these data are still being collected and reported.

In general, partially because of the published data summarized in Table 3-32, and partially because a standard for window troughs has been historically used in risk assessments and to determine clearance (following EPA's Interim Guidance for §403 standards and the HUD Guidelines), window troughs are widely recognized as a major reservoir of dust-lead in residences. As shown in Table 3-32, levels often exceed 800 $\mu g/ft^2$ and it is not uncommon to see levels above 10,000 $\mu g/ft^2$ in high risk housing. However, unlike the other surfaces discussed in this report, national estimates of the distribution of dust-lead in window troughs are available from the HUD National Survey. The estimated national geometric mean dust-lead loading in window troughs from the HUD National Survey (as modified in the §403 risk analysis to reflect the 1997 housing stock and wipe techniques) was 460 $\mu g/ft^2$ (Table 3-32), with 30% of homes estimated to have average window trough dust-lead loadings at or above 800 $\mu g/ft^2$.

Upholstery

Table 3-33 summarizes the data from the four studies which collected dust-lead samples from upholstery. In general, dust-lead loadings for these surfaces averaged below 100 $\mu g/ft^2$. As the sample sizes in all of these studies were small, and sampling techniques, sampling locations, and study goals varied considerably from study to study, more information would be necessary to fully characterize potential lead hazards associated with upholstery.

3.5.2 Evidence of a Relationship Between Children's Blood-Lead Concentrations and Dust-Lead on Surfaces Other Than Floors and Window Sills

The information available to assess children's exposure to dust-lead on surfaces covered in this section is discussed in detail for each surface type below. It should be noted, however, that in general it is difficult to establish the causal link between these surfaces and children's blood-lead concentrations. This is true for many reasons. Often other important sources of lead exposure are not well characterized in the studies that provide data on these special surfaces. Correlations are often estimated based on small sample sizes and without adjusting for other exposure variables such as lead in floor-dust and soil. Moreover, there is often correlation between lead levels on these surfaces and lead levels on floors, window sills, and in soil. For all of these reasons, it must be noted that even significant correlation coefficients should not be interpreted as the degree to which dust-lead on these surfaces causes a change in blood-lead concentration. In almost all cases, in order to characterize the pathway of lead from these surfaces to children's blood, additional data collection or analyses are needed.

Exterior Dust

Table 3-30 (in the previous subsection) contains a summary of the dust-lead data and blood-lead data separately for studies which collected exterior dust. However, it contains no results providing information about the relationship between exterior dust-lead levels and blood-lead levels. The reports describing these analyses were examined to assess the relationship between exterior dust-lead levels and blood-lead levels. In the Repair and Maintenance Study (USEPA, 1996c, 1997c), exterior dust samples were collected at five separate times. The (Pearson) correlation coefficients between blood-lead concentrations and entryway dust-lead concentrations ranged from 0.23 to 0.49, and the correlation coefficients between blood-lead concentrations and entryway dust-lead loadings ranged from 0.10 to 0.46. In most cases, those correlation coefficients were statistically significant at the α=0.01 level. In the Rochester Study (USHUD, 1995a), the University of Cincinnati Dust Vacuum Method (DVM) and Baltimore Repair and Maintenance vacuum method (BRM) were used to collect external dust samples. The correlation coefficients for the BRM and DVM techniques were 0.21 and 0.27 for the correlation between blood-lead concentrations and exterior dust-lead concentrations and 0.34 and 0.18 for the correlation between blood-lead concentrations and exterior dust-lead loadings, respectively. The correlations were all statistically significant at the α=0.05 level, with the correlation for the DVM measured loading significant at the α=0.01 level. On the other hand, the Mexico City Study (Romieu et. al., 1995), Arnhem Study (Brunekreef et. al., 1981) and the Midvale Study (Bornschein et. al., 1990) reported the correlations between external dust measurements and blood-lead levels to be statistically insignificant.

Multivariate regression and structural equation modeling was used in some of the studies to examine how multiple sources of environmental lead exposure and other factors affect blood-lead levels. Regression analyses were carried out in most of the studies where both external dust-lead and blood-lead measurements were collected, but the external dust-lead measurements were

not included as an explanatory variable in any of the reported regression models. Reasons for excluding external dust-lead were not clearly stated. Speculatively, such reasons may include a lack of interest in the relationship, poor data quality in the external dust-lead measurements, colinearity of external with internal dust-lead measurements and omission of the variable through step-wise regression. Structural equation modeling was carried out in the (Three Cities) Urban Soil Lead Abatement Demonstration Project (USEPA, 1996a), Butte-Silver Bow Study (Butte-Silver Bow DoH et. al., 1991) and Midvale Study (Bornschein et. al., 1990). In the Three Cities Study, exterior dust was lead considered as a component of the lead exposure pathway in the general structural equation model, but the component was excluded in the actual implementation of the model. In the Butte-Silver Bow Study, external dust-lead was also excluded from the structural equation model, but external dust-lead was observed to be correlated (Pearson correlation r=0.64) with soil lead, which was included as a component of the lead exposure pathway in the model. The Midvale Study was the only one to include external dust-lead in the actual implementation of the structural equation model, but the dust-lead to blood-lead relationship was reported as being statistically insignificant.

In summary, there is much difficulty in distinguishing between direct and indirect exposure in cases where external dust-lead levels is closely related to levels in other sources of environmental lead. Correlation and univariate regressions with external dust-lead and blood-lead fail to account for the possibility that external dust-lead by itself may only play a small part in aggregate lead exposure when other sources of lead and exposure pathways are considered. Multivariate regressions using lead measurements from multiple sources do not solve this problem due to problems with colinearity. The preferred approach would be to use structural equation models, which allow multiple source and exposure pathways to be modeled in a reasonable way, but this approach requires more effort in terms of implementation and interpretation of the model, and is not well-reported in the literature. Therefore, quantitative estimates of the effect of external dust-lead on children's blood-lead concentrations have not been well established in the literature.

Air Ducts

Most of the encountered articles provided only preliminary information on lead exposures associated with air ducts. It is unclear to what extent dust-lead in air ducts is accessible to children. Children would not typically be expected to encounter the dust lodged in air ducts directly. One case study found that dust-lead levels in living areas outside of contaminated air ducts can be orders of magnitude lower than what is found in the air ducts. However, if dust in air ducts is disturbed, it is more likely to be introduced to the air and to nearby surfaces with which children can come into direct contact. In particular, HVAC ductwork removal can yield extensive contamination of surfaces in the general area of the ductwork.

Only one study (the Baltimore R&M Study) estimated (in a quantitative manner) the association between blood-lead concentrations in children and dust-lead levels found in air ducts. This relationship was expressed as a simple correlation coefficient. Unlike correlations between blood-lead concentrations and dust-lead levels on other surfaces, the correlation coefficient

involving dust-lead levels from air ducts was not significant at the 0.05 level. However, this analysis was based on a small sample size and did not adjust for the effects of other exposure variables such as lead in floor-dust and soil. Moreover, as evidence of a significant correlation was observed between air duct dust-lead levels and lead levels on other surfaces, such as floors, even significant correlation coefficients should not be interpreted as the degree to which air duct dust causes a change in blood-lead concentration. In order to characterize the pathway of lead from air ducts to children's blood, additional data collection and analyses are needed.

Window Troughs

Of the seven studies listed in Table 3-32 above that collected information on dust-lead in window troughs (also known as window wells), three also collected blood-lead data from resident children. Correlation coefficients between blood-lead levels and window-trough dust-lead concentrations in the R&M study ranged from 0.20 to 0.39, and correlation coefficients between blood-lead levels and window-trough dust-lead loadings ranged from 0.06 to 0.44. The correlations between dust-lead concentrations and blood-lead concentrations were statistically significant in 4 of the 5 sampling campaigns, and the correlation between dust-lead loading and blood-lead concentration was statistically significant only in the pre-maintenance sampling. In the Rochester Study, correlation coefficients between blood-lead concentrations and dust-lead loadings were 0.35 for the BRM samples, 0.31 for the DVM samples, and 0.29 for wipe samples, while correlation coefficients between blood-lead concentrations and dust-lead concentrations were 0.23 for both BRM and DVM samples.

Previous analyses (Battelle, 1996a; Battelle, 1996b) have examined whether the predictive ability of a model improves when adding window trough lead levels to a model which already accounts for dust-lead on floors and window sills. Results of these analyses on the Rochester Lead-in-Dust Study and Baltimore R&M study data indicated that the estimated effect of window trough dust-lead on blood-lead was either not statistically significant or only marginally significant after adjusting for the effects of floor lead, sill lead, and temporal variation. A pathways analysis (USEPA, 1998c) using structural equations modeling concluded that window troughs were a significant pathway for lead exposure, both as a direct pathway of lead to children's blood-lead concentration (seen when Rochester Lead-in-Dust study data were analyzed) and as an indirect pathway through window sills and floors to blood-lead concentrations (seen when both the Rochester and Baltimore R&M study data were analyzed).

In summary, the association between blood-lead concentrations and window trough dust-lead has been well established in the literature. The more difficult question of the degree to which window troughs contribute directly or indirectly to children's lead exposure is not well established.

Upholstery

Table 3-33 in the previous subsection included results for two studies which measured both children's blood-lead concentrations and dust-lead on upholstery. In the Baltimore R&M study, correlation coefficients were calculated between blood-lead concentrations in children and both the loading and concentration of lead in upholstery dust. These correlation coefficients ranged from 0.19 to 0.61 for dust-lead concentrations and from 0.06 to 0.47 for dust-lead loading. These correlations were statistically significant in the pre-intervention sampling. As with most of the other surfaces discussed in this section, upholstery dust-lead levels were not included in any analyses to determine which lead sources were most significantly related to blood-lead levels. In the Mexico City Study, the correlation between upholstery dust-lead levels was not statistically significant, resulting in the absence of upholstery dust-lead levels from models linking blood-lead levels and environmental lead levels.

The results of the two studies assessing the importance of upholstery dust as a source for lead exposure in children differ. In one case, the relationship between blood-lead and upholstery-dust-lead is significant, while in the other it is not. Moreover, as upholstery dust-lead is often correlated with other lead exposure variables, such as floor dust-lead and soil-lead, as cautioned earlier, the positive correlation coefficient should not be interpreted as the degree to which upholstery dust causes a change in blood-lead concentration. In order to characterize the pathway of lead from upholstery to children's blood (and perhaps hands), additional data collection and analyses are needed.

3.5.3 Implications of the Available Information For Regulatory Standards

Two primary questions related to the need and feasibility of regulatory standards for dust-lead on surfaces other than floors and window sills are:

1. Is there sufficient information available on which to base a standard?

2. Is the standard necessary to either identify a lead hazard at a residence or to characterize the risk to determine appropriate corrective actions?

The answers to these questions are discussed for each surface type below.

Exterior Dust

In general, there is a fair amount of data on exterior dust, including studies where exterior dust has been measured along with other lead exposure variables and blood-lead concentrations. The amount of data implies that analyses could be conducted to provide a quantitative basis for an exterior dust standard. However, implementation and interpretation of such analyses for exterior dust will face many difficulties. For example, in many of the studies it is difficult to distinguish between exterior dust and soil samples because of aggregation of the samples or of the measurements. Some external sampling for lead was carried out using surface scrapings

which measures lead levels from a mix of both soil and dust-lead and some analyses averaged the external soil and dust measurements and recorded the value as a single external lead measurement. (Hence only studies for which a clear distinction between external soil and dust-lead levels is possible were included in this summary.) It is also difficult to determine what locations for exterior dust should be included. Should the focus be on enclosed spaces or also include unenclosed areas such as sidewalks, stoops, and unenclosed porches? One primary reason to focus only on enclosed areas is because exposures to unenclosed areas are not under the direct control of property owner. Exposure and cleaning scenarios for enclosed versus unenclosed areas are likely to be very different as well. In conclusion, decisions on the specific focus of a standard for exterior dust would impact the feasibility of establishing a good quantitative basis on which to set the standard.

The question of whether a standard for exterior dust is necessary is also a difficult one, for which the literature does not have a clear answer. While it is reasonable to assume that measurements of lead in interior dust and exterior soil might capture a lead hazard if one exists, there is not a strong body of information on which to base this conclusion. A separate standard may not be necessary if risk assessors are aware of the potential hazard from exterior dust, and include testing or corrective actions in cases where it is suspected to be an important pathway of exposure (for example, in the case where a child spends a considerable amount of time on a paved surface, such as a driveway or patio).

Air Ducts

There is insufficient data upon which to develop a hazard standard for lead in air duct dust, or upon which to draw conclusions about the necessity of a standard to either identify a hazard or determine corrective actions.

Window Troughs

The fact that regulatory standards have been proposed for dust-lead on floors and window sills based on data sets (most notably the Rochester Lead-In-Dust Study and the HUD National Survey) that also include window troughs implies that sufficient data exists on which to base a standard for window troughs.

However, while there is sufficient information on which to base a standard, analyses conducted to assess the necessity of a window trough standard given the existence of a floor and window sill standard suggest that a window trough standard may not be necessary to identify a residence with a lead-based paint hazard. These analyses include the sensitivity/specificity analyses included in a companion §403 report as well as the analyses that examine the effect of adding window troughs to a statistical model that already includes floors and window sills (Battelle, 1996a; Battelle, 1996b). Given the correlation between window trough and window sill lead levels, it is likely that if more sampling is to be done beyond a minimal risk assessment, more benefit will be obtained from sampling more windows at the sill rather than sampling fewer windows but at both the sill and trough. Moreover, cleaning of window troughs is

recommended for all homes that require a dust intervention, and clearance standards have been proposed to guide assessment of the effectiveness of the cleaning. For these reasons, it does not appear that an additional standard for window troughs is necessary either to identify a home with a hazard or to guide corrective actions.

Upholstery

There is insufficient data upon which to develop a hazard standard for lead in upholstery dust, or upon which to draw conclusions about the necessity of a standard to either identify a hazard or determine corrective actions.

3.6 DISTRIBUTION OF CHILDHOOD BLOOD-LEAD

This section updates the information presented in Section 3.4 of the §403 risk analysis report on the distribution of childhood blood-lead concentration in the United States, with a focus on the 1-2 year (12-35 month) age range as the population of interest. In addition to a national characterization based on data from Phase 2 of the Third National Health and Nutrition Examination Survey (NHANES III), Section 3.4 of the §403 risk analysis report summarized data from other studies (e.g., the Baltimore R&M study, the Rochester Lead-in-Dust study, and the HUD Grantees evaluation) to provide supporting information on the prevalence of elevated blood-lead concentrations in children living in urban locations and in older housing or housing likely to contain lead-based paint. Blood-lead data from these other studies were also considered because the NHANES III did not collect environmental-lead data, despite having the most nationally representative data on blood-lead levels.

Section 3.6.1 below is an update of Section 3.4.4 of the §403 risk analysis report. It contains revised data summaries of pre-intervention blood-lead concentrations in children monitored within the HUD Grantees evaluation and revised regression model fits to predict blood-lead concentration as a function of dust-lead loading for each individual grantee, as well as for the Rochester Lead-in-Dust study (i.e., the study that provided the data used to developed the empirical model developed within the §403 risk analysis. These revisions were possible as additional pre-intervention data from the HUD Grantees evaluation (through 1/99) have been made available to the risk analysis since the report was released.

Section 3.6.2 provides information from the Cincinnati Prospective Lead study (Clark et al., 1985) and summarized by the Centers for Disease Control and Prevention (CDC) on the relationship between children's blood-lead concentration and housing age/condition, and how this relationship may change with the age of the child (CDC, 1991). CDC used this information in their recommendations for blood-lead screenings of young children.

3.6.1 Evaluation of the HUD Lead-Based Paint Hazard Control Grant Program ("HUD Grantees")

Blood-lead concentrations of children residing in households participating in the evaluation phase of the HUD Grantees evaluation (Section 3.2.2.3 of the §403 risk analysis report) were measured, along with environmental-lead levels in various media. The population of children targeted for participation in the program differed among the fourteen grantee recipients, due to the different enrollment criteria among the grantees (see Table 3-4 of the §403 risk analysis report). These criteria included targeting high-risk neighborhoods, enrolling only homes with a lead-poisoned child, and considering unsolicited applications. Pre-intervention data collected through January 1999 are presented in this section; these data provide some of the most recent information on the relationship between children's blood-lead concentration and environmental-lead levels.

Across all grantees, pre-intervention blood-lead concentration data through 1/99 were available for 526 children aged 1-2 years and for 764 children aged 3-5 years. For these children, Table 3-34 summarizes measured blood-lead concentration for each combination of blood collection type (venipuncture, fingerstick) and age of child (1-2 years, 3-5 years, and 1-5 years). Table 3-34 also summarizes measured blood-lead concentration for children aged 1-2 years for each combination of blood collection type and grantee. Note that fingerstick methods were predominant for Wisconsin, Milwaukee, and Vermont, while Rhode Island used both methods for similar numbers of children. The remaining nine grantees (excluding New Jersey) used the venipuncture either exclusively or predominantly.

According to Table 3-34, the geometric mean blood-lead concentration via the venipuncture collection method was 9.3 µg/dL for children aged 1-2 years and 8.0 µg/dL for children aged 3-5 years. In contrast, the geometric means based on data from Phase 2 of NHANES III were 3.1 µg/dL for children aged 1-2 years and 2.5 µg/dL for children aged 3-5 years (Table 3-36 of the §403 risk analysis report). The larger values in the HUD Grantees evaluation reflect the HUD Grantees program's procedure of selecting high-risk children for monitoring. The differing enrollment criteria across grantees also contributed to considerable differences in the geometric mean blood-lead concentration among the grantees.

Under venipuncture, the geometric means of children aged 1-2 years for individual grantees reporting more than three blood-lead results ranged from 4.2 µg/dL (California, which only targeted older units) to 15.9 µg/dL (Cleveland, which targeted units with lead-poisoned children).

The geometric mean blood-lead concentration via the fingerstick collection method was 9.4 µg/dL for children aged 1-2 years and 8.9 µg/dL for children aged 3-5 years. When data were available for more than one child under fingerstick collection methods, the geometric means for children aged 1-2 years ranged from 5.9 µg/dL (Wisconsin) to 13.5 µg/dL (Milwaukee).

Table 3-34. Summary of Children's Pre-Intervention Blood-Lead Concentration in the HUD Grantees Evaluation According to Blood Collection Method, Child Age Category, and Grantee (ages 1-2 years only)

	Number of Children	Blood-Lead Concentration (μg/dL)							
		Arithmetic Mean	Geometric Mean	Geometric Standard Deviation	Minimum	25th Percentile	Median	75th Percentile	Maximum
Age Category	**Blood Collection Method = Venipuncture**								
1-2 Years	361	12.5	9.3	2.3	0.7	5.4	10.0	17.0	53.0
3-5 Years	536	10.6	8.0	2.2	0.0	4.5	8.6	15.0	48.0
1-5 Years	897	11.4	8.5	2.2	0.0	5.0	9.0	16.0	53.0
Grantee	**Blood Collection Method = Venipuncture (Children Aged 1-2 Years only)**								
Alameda County	27	6.5	4.7	2.2	1.4	3.0	4.7	6.6	24.8
Baltimore	25	9.3	7.7	1.9	2.0	6.0	7.0	10.0	26.0
Boston	20	12.7	10.4	2.0	3.0	6.0	14.5	19.0	27.0
California	21	5.3	4.2	2.0	1.4	3.2	3.8	6.0	16.9
Cleveland	64	19.3	15.9	1.9	4.0	11.5	17.0	28.0	53.0
Massachusetts	43	11.2	9.1	1.9	3.0	6.0	9.0	16.0	40.0
Minnesota	75	14.5	10.7	2.4	0.7	6.0	11.0	22.0	43.0
New Jersey	1	3.0	3.0	--	3.0	3.0	3.0	3.0	3.0
Rhode Island	14	10.0	8.1	2.0	2.0	6.0	8.5	14.0	21.0
Wisconsin	9	10.2	8.7	1.8	4.0	6.0	8.0	12.0	24.0
Milwaukee	3	26.0	25.1	1.4	18.0	18.0	25.0	35.0	35.0
Chicago	28	13.9	11.7	2.0	1.0	9.5	12.0	19.0	35.0
New York City	23	5.2	4.7	1.6	2.0	4.0	5.0	7.0	12.0
Vermont	8	13.5	12.4	1.6	6.0	8.5	14.5	17.0	22.0
Age Range	**Blood Collection Method = Fingerstick**								
1-2 Years	164	11.7	9.4	1.9	2.0	6.0	9.0	15.0	48.0
3-5 Years	232	11.5	8.9	2.0	2.0	5.0	9.0	15.0	62.0
1-5 Years	396	11.6	9.1	2.0	2.0	5.0	9.0	15.0	62.0
Grantee	**Blood Collection Method = Fingerstick (Children Aged 1-2 Years only)**								
Cleveland	1	13.0	13.0	--	13.0	13.0	13.0	13.0	13.0
Massachusetts	4	7.8	7.3	1.5	4.0	6.0	8.5	9.5	10.0
Minnesota	1	33.0	33.0	--	33.0	33.0	33.0	33.0	33.0
Rhode Island	9	8.8	8.2	1.5	5.0	7.0	7.0	11.0	15.0
Wisconsin	43	6.2	5.8	1.4	3.5	4.0	6.0	8.0	14.0
Milwaukee	82	16.0	13.2	1.9	2.0	9.0	14.5	20.0	48.0
Vermont	24	7.9	7.0	1.6	3.5	5.0	6.5	11.0	16.0

Note: All pre-intervention blood-lead concentration data available and collected through 1/99 are included in the above summaries.

The percentages of children with elevated blood-lead concentrations (i.e., concentrations at or above 10, 15, 20 or 25 μg/dL) at pre-intervention are summarized in Table 3-35. According to this table, 51 percent of children aged 1-2 years sampled via venipuncture methods had blood-lead concentrations at or above 10 μg/dL, compared to the estimates of 5.88% for Phase 2 of NHANES III, 53.8% for the Baltimore R&M study (pre-intervention), and 23.4% for the Rochester Lead-In-Dust study (Tables 3-37, 3-41, and 3-42, respectively, of the §403 risk analysis report). For individual grantees having more than three children with a measured blood-lead concentration, the percentage of children aged 1-2 years with blood-lead concentrations (venipuncture) at or above 10 μg/dL varied from 4% (New York City, which targeted housing and neighborhoods rather than lead-poisoned children) to 80% (Cleveland). The range of percentages under the fingerstick method were similar to that under the venipuncture method, but less data were available to estimate them.

Figures 3-20 and 3-21 illustrate the nature of the linear relationship observed in the HUD Grantees evaluation between a child's (log-transformed) blood-lead concentration and the household's (log-transformed) area-weighted arithmetic average wipe dust-lead loading for floors and window sills, respectively. The figures portray fitted linear regression models for each grantee, as well as for the Rochester Lead-In-Dust study and, in Figure 3-21, the Baltimore R&M study (for comparison purposes). The regression model used only the log-transformed average dust-lead loading as a predictor variable; the impact of other potentially important predictor variables on blood-lead concentration was not considered in the model fittings. The regression lines span the ranges of the observed area-weighted average dust-lead loadings, except data for five HUD Grantees households (three from Cleveland and one each from Baltimore and Rhode Island) were omitted from Figure 3-21 as their average window sill dust-lead loadings were extremely low (less than 0.05 $\mu g/ft^2$) compared to the other households and were considered too influential to the model fittings.

When fitting the regression models in Figures 3-20 and 3-21 to the HUD Grantees data, it was desired to have each household having blood-lead and dust-lead data be represented by only a single data point. This was possible only if blood-lead data were considered for a single child in that household. In situations where data for multiple children were available for a single household, only data for the youngest child older than 12 months of age were considered. This approach resulted in a single blood-lead result for each household with blood-lead data. In addition, only data for children meeting the following criteria were included in the regression modeling:

- Children who lived in the sampled housing unit for at least three months and before dust and soil samples were collected;
- Children whose blood samples were taken within four months of dust and soil sample collection;
- Children not having medical treatment for lead poisoning.

Table 3-35. Percentage of Children with Elevated Blood-Lead Concentration (at Pre-Intervention) in the HUD Grantees Evaluation According to Blood Collection Method, Child Age Category, and Grantee (ages 1-2 years only)

	Number of Children	Percentage of Children with Elevated Blood-Lead Concentration (%)			
		≥ 10 µg/dL	≥ 15 µg/dL	≥ 20 µg/dL	≥ 25 µg/dL
Age Range	**Blood Collection Method = Venipuncture**				
1-2 Years	361	51	35	18	12
3-5 Years	536	43	27	12	7
1-5 Years	897	46	30	14	9
Grantee	**Blood Collection Method = Venipuncture (Children Aged 1-2 Years only)**				
Alameda County	27	22	11	4	0
Baltimore	25	36	20	8	4
Boston	20	55	50	15	5
California	21	14	5	0	0
Cleveland	64	80	59	38	30
Massachusetts	43	47	30	9	7
Minnesota	75	61	44	31	21
New Jersey	1	0	0	0	0
Rhode Island	14	36	21	7	0
Wisconsin	9	44	22	11	0
Milwaukee	3	100	100	67	67
Chicago	28	75	39	14	7
New York City	23	4	0	0	0
Vermont	8	63	50	13	0
Age Range	**Blood Collection Method = Fingerstick**				
1-2 Years	164	46	28	13	9
3-5 Years	232	44	26	15	8
1-5 Years	396	45	27	14	9
Grantee	**Blood Collection Method = Fingerstick (Children Aged 1-2 Years only)**				
Cleveland	1	100	0	0	0
Massachusetts	4	25	0	0	0
Minnesota	1	100	100	100	100
Rhode Island	9	33	11	0	0
Wisconsin	43	9	0	0	0
Milwaukee	82	70	50	26	17
Vermont	24	29	13	0	0

Note: All pre-intervention blood-lead concentration data available and collected through 1/99 are included in the above summaries.

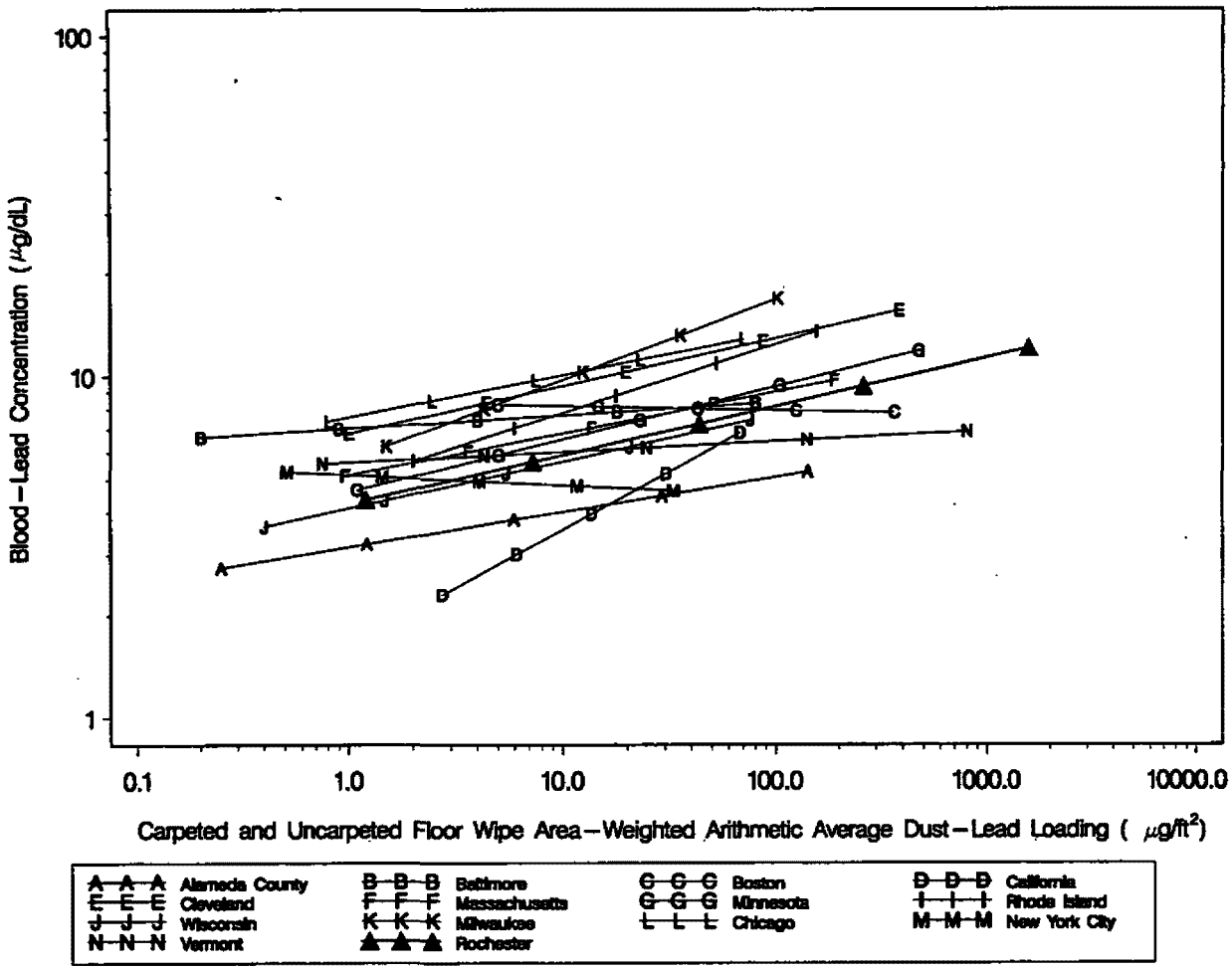

Figure 3-20. Fitted Regression Models Predicting Children's Blood-Lead Concentration as a Function of Area-Weighted Arithmetic Average Floor Dust-Lead Loading (Wipe Collection Method), for the Various Grantees in the HUD Grantees Evaluation and for the Rochester Lead-In-Dust Study

(Note: Venipuncture blood-lead data were exclusively used in each fitting except for Wisconsin, Milwaukee, and Vermont, where fingerprick blood-lead data were exclusively used.)

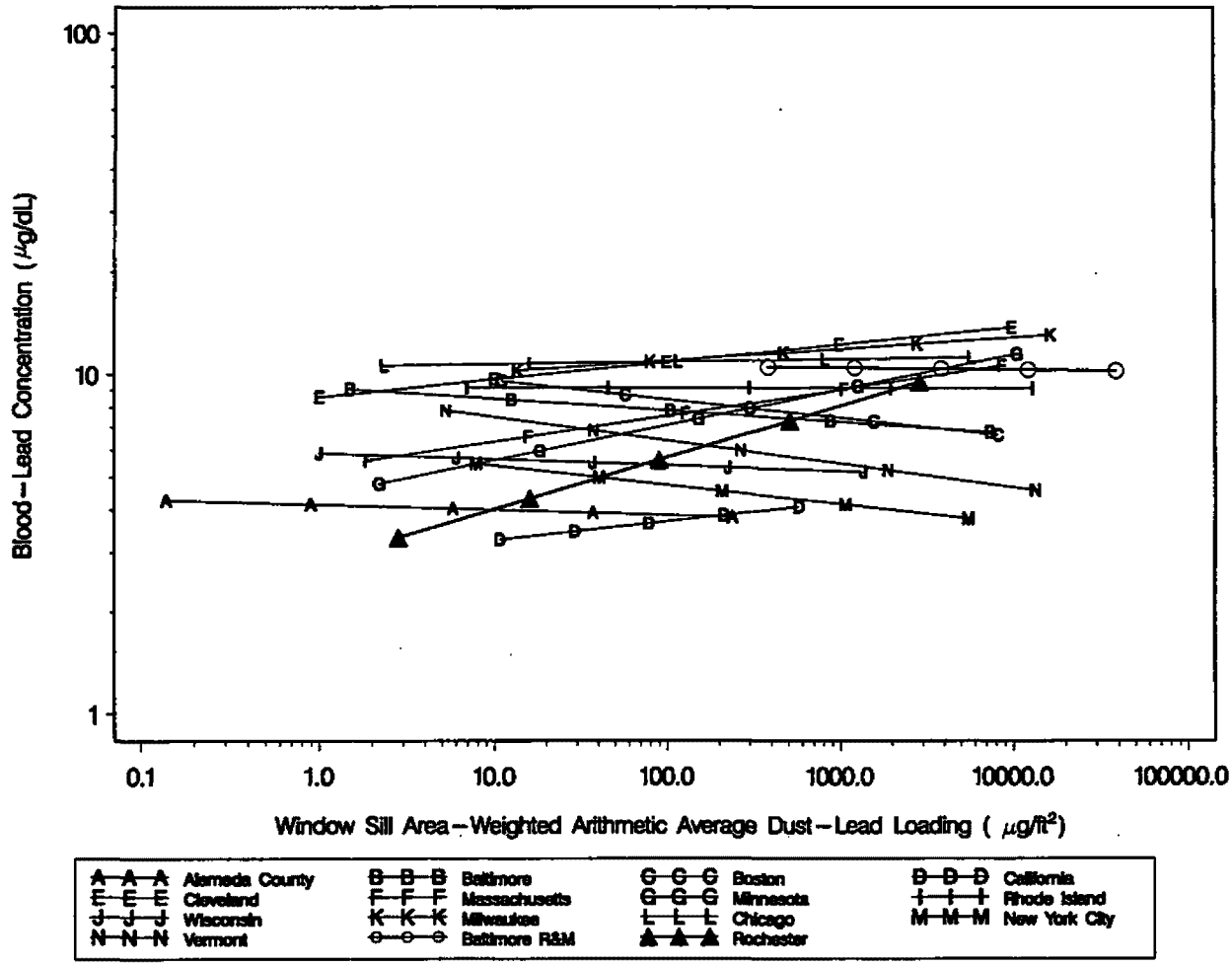

Figure 3-21. Fitted Regression Models Predicting Children's Blood-Lead Concentration as a Function of Area-Weighted Arithmetic Average Window Sill Dust-Lead Loading (Wipe Collection Method), for the Various Grantees in the HUD Grantees Evaluation and for the Rochester Lead-In-Dust Study

(Note: Venipuncture blood-lead data were exclusively used in each fitting except for Wisconsin, Milwaukee, and Vermont, where fingerprick blood-lead data were exclusively used.)

The regression models in Figures 3-20 and 3-21 were fitted to blood-lead concentration data only under the venipuncture method for all but the three grantees (Wisconsin, Milwaukee and Vermont) for which fingerstick sample results were predominant. For these three grantees, only fingerstick blood-lead concentration data were used in the regressions.

Note that the slopes of the fitted regression lines in Figures 3-20 and 3-21 are generally similar in sign and magnitude (given expected ranges of variability) across the grantees and the two other studies. This suggests that the relationships between blood-lead concentration and household average dust-lead loading were relatively consistent across grantees. In particular,

these relationships were similar to that observed for data from the Rochester study (i.e., the data used to develop the empirical model presented in Chapter 4 of the §403 risk analysis). This conclusion is important in that the data from the HUD Grantees evaluation reflect a much larger geographical area than the Rochester study and represent several types of exposure conditions.

3.6.2 Evidence of the Impact of Housing Age/Condition on Blood-Lead Concentration

The role that housing age plays in the increased likelihood of a resident child having an elevated blood-lead concentration has been well-documented and is accepted by many experts in residential lead exposure. Older housing is more likely to contain lead-based paint in a deteriorated condition, which contributes to lead in other environmental media within the residence, especially those media that is most likely to come into direct contact with children. In particular, the importance that the level of deterioration plays in the accessibility of lead-based paint hazards implies that housing condition is an additional key factor in predicting blood-lead concentration.

Table 3-39 of the §403 risk analysis report summarized data from Phase 2 of NHANES III to illustrate how geometric mean blood-lead concentration and the percentage of elevated blood-lead concentrations (i.e., percentage exceeding a given threshold) for children are related to housing age category. For example, the percentage of children aged 1-5 years with blood-lead concentration of at least 10 µg/dL increases from 1.6% for children living in post-1973 housing to 8.6% for children living in pre-1946 housing, with a corresponding geometric mean increase from 2.0 to 3.8 µg/dL. The Centers for Disease Control and Prevention (CDC) cited these same results in their 1997 document, *Screening Young Children for Lead Poisoning*, to support their conclusion that older housing (i.e., housing built prior to 1950) contained the greatest risk for lead-based paint hazards.

Figure 6-1 of the CDC's 1991 document, *Preventing Lead Poisoning in Young Children – A Statement by the Centers for Disease Control*, presents results from the Cincinnati Prospective Lead Study (Clark et al., 1985) to illustrate how the combination of housing age and condition is related to children's blood-lead concentration and how this relationship changes with the age of the child. This figure is duplicated in Figure 3-22. This figure shows that children's blood-lead concentration tends to peak at 18-24 months, with the most rapid increase occurring between 6-12 months. The highest blood-lead levels are associated with housing built prior to World War II, as well as older housing (predominantly 19th century) that once contained considerable lead-based paint but which later underwent rehabilitation. Within these groups of housing, children living in units in a deteriorated or dilapidated condition had consistently higher geometric mean blood-lead concentrations through their first three years, with this geometric mean exceeding 20 µg/dL from about 12 to 24 months of age. CDC used the information presented in Figure 3-22 to prepare a recommended screening schedule for testing children's blood-lead levels.

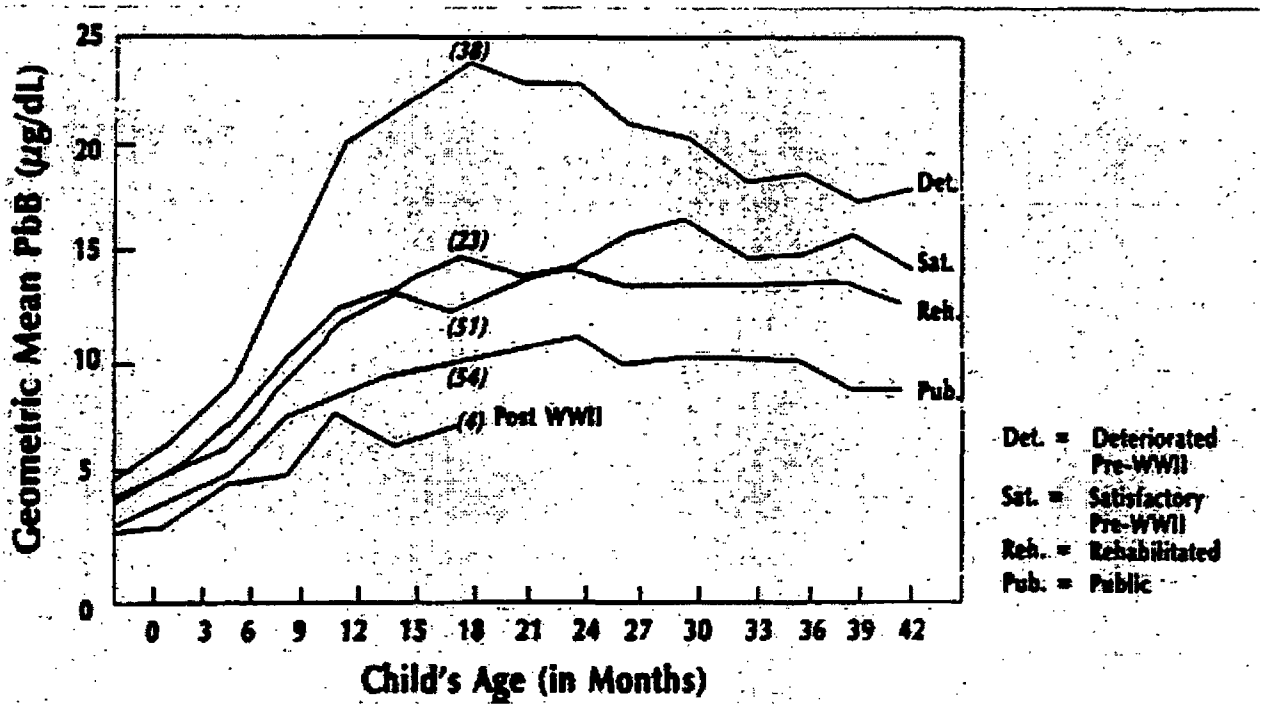

Figure 3-22. Geometric Mean Blood-Lead Concentration Versus Child Age, As Reported Within the Cincinnati Prospective Lead Study and Presented According to Housing Age and Condition

(Note: Duplicated from Figure 6-1 of CDC, 1991. Blood-lead concentrations for the same cohort of children were measured over time. Numbers in parentheses indicate numbers of children with blood-lead information at 18 months of age.)

4.0 DOSE-RESPONSE ASSESSMENT

The objective of dose-response assessment was to characterize the relationship between environmental-lead exposure and the resulting adverse health effects in young children. The foundation of this characterization was the relationship between environmental-lead levels and blood-lead concentration. EPA's Integrated Exposure, Uptake, and Biokinetic (IEUBK) model and an empirical model developed for this risk assessment (Sections 4.1 and 4.2 of the §403 risk analysis report) were employed to make this characterization.

Section 4.1 of this chapter documents an additional tool, obtained since the §403 risk analysis report was published, for predicting blood-lead concentration as a function of environmental-lead levels. This tool is a regression model developed from epidemiological data collected from 12 studies and was suggested for use in the §403 risk analysis by some commenters on the §403 proposed rule. As the U.S. Department of Housing and Urban Development (HUD) sponsored the development of this model, it is referred to in this report as the "HUD Model." The goal of this model was to "estimate the contribution of lead-contaminated house dust and soil to children's blood-lead levels" (Lanphear et al., 1998). This goal is consistent with the objectives of the §403 risk assessment, and so the model merits consideration for this analysis. Section 4.1 includes documentation on the HUD model, key steps that were taken in its development, and issues that are necessary to consider when interpreting the results of the model fits.

Section 4.2 of this chapter contains a revision of the Rochester Multimedia model, introduced in Section 4.2.3 of the §403 risk analysis report, to allow the model to predict results that are more comparable to the results of the performance characteristics analysis presented in the preamble to the §403 proposed rule. The Rochester Multimedia model predicted a geometric mean blood-lead concentration as a function of average dust-lead loadings in floors and window sills, dripline soil-lead concentration, and a variable which indicates the presence of deteriorated lead-based paint and a child with paint pica tendencies. In contrast, the performance characteristics analysis in the preamble estimated risks associated with dust-lead loadings on uncarpeted floors, dust-lead loadings on window sills, yardwide average soil-lead concentration, and the percentage of painted components with deteriorated lead-based paint. Because the definitions of the data inputs were not always consistent between these two statistical approaches, their findings were not comparable. Thus, the revised model presented in Section 4.2 uses the same types of data inputs as those used for the performance characteristics analysis. Section 4.2 also documents multimedia models that omit one or more of the dust, soil, and paint input variables to obtain predicted blood-lead concentration in instances where data for one or more of these media were not available.

Section 4.3 provides additional information regarding key assumptions made in the risk characterization process: the "scaling" algorithm used to determine a post-intervention blood-lead concentration distribution that is comparable to the baseline distribution, and the issue of

adjusting for measurement error when deriving the empirical model used in the §403 risk analysis.

4.1 HUD MODEL

The HUD model (Lanphear et al., 1998) was developed by a team of researchers and sponsored by HUD's Office of Lead Hazard Control. This modeling effort used data from 12 epidemiologic studies (hence its frequent reference as a "pooled analysis" model) to make statistical inferences on the contribution of lead-contaminated house dust and residential soil to children's blood-lead concentration.

The HUD model predicts a geometric mean blood-lead concentration for children aged 6-36 months (i.e., the age range of data considered from the 12 studies) as a function of exposure to specific lead levels in dust, soil, paint, and water, and as a function of other important demographic variables. Therefore, the model is used to estimate individual risks, or the risks associated with a class of children determined by specified environmental-lead levels to which they are exposed. EPA addressed minimizing individual risks when establishing "levels of concern" for lead in dust and soil within the §403 proposed rule. However, EPA was obliged to consider population-based risks within a cost-benefit analysis to establish lead hazard standards within the proposed rule.

4.1.1 Form of the HUD Model

The HUD model takes the following form:

$$
\begin{aligned}
(1)\quad Ln(PbB) = {} & 1.496 + 0.183 \cdot Ln(DustLead) + 0.01398 \cdot Ln(WaterLead) + 0.02116 \cdot Ln(ExtLead) + \\
& 0.005787 \cdot Ln(ExtLead) \cdot ExtType + 0.4802 \cdot Ln(ExtLead) \cdot ExtLoc - 0.1336 \cdot ExtType + 0.5858 \cdot ExtLoc \\
& - 0.02199 \cdot Ln(MaxXRF) + 0.03811 \cdot Ln(MaxXRF) \cdot PaintCond - 0.0808 \cdot PaintCond + 0.02126 \cdot Age - \\
& 0.001399 \cdot Age2 + 0.00007854 \cdot Age3 - 0.3932 \cdot Boston - 0.01167 \cdot Butte + 0.2027 \cdot Bcreek + \\
& 0.2392 \cdot Cpgm + 0.5383 \cdot Csoil + 0.05717 \cdot Leadville + 0.1761 \cdot Magna - 0.04209 \cdot RochLong + \\
& 0.07257 \cdot RochLID - 0.3712 \cdot Sandy + 0.1777 \cdot Midvale + 0.123 \cdot Race + 0.3175 \cdot SES1 + 0.2138 \cdot SES2 \\
& + 0.1799 \cdot SES3 + 0.1691 \cdot SES4 - 0.03233 \cdot MouthOften - 0.2454 \cdot MouthRare - 0.1397 \cdot MouthSome \\
& + 0.002649 \cdot Ln(DustLead) \cdot Age - 0.0003381 \cdot Ln(DustLead) \cdot Age2 - 0.00001281 \cdot Ln(DustLead) \cdot Age3 \\
& + 0.2212 \cdot Ln(ExtLead) \cdot MouthOften + 0.07892 \cdot Ln(ExtLead) \cdot MouthRare + \\
& 0.1663 \cdot Ln(ExtLead) \cdot MouthSome + 0.5305 \cdot Ln(WaterLead) \cdot SES1 - 0.0136 \cdot Ln(WaterLead) \cdot SES2 + \\
& 0.1033 \cdot Ln(WaterLead) \cdot SES3 - 0.09098 \cdot Ln(WaterLead) \cdot SES4 + 0.01192 \cdot Age \cdot Race - \\
& 0.01023 \cdot Age \cdot SES1 + 0.003849 \cdot Age \cdot SES2 + 0.00008468 \cdot Age \cdot SES3 - 0.01679 \cdot Age \cdot SES4 + error
\end{aligned}
$$

where

Ln(PbB) = log-transformed blood-lead concentration (µg/dL)

Ln(DustLead) = log-transformed interior (wipe) floor dust-lead loading (µg/ft^2), minus the mean of the log-transformed data used to develop the model (2.605 µg/ft^2)

Ln(WaterLead) = log-transformed water-lead concentration (ppb), minus the mean of the log-transformed data used to develop the model (0.785 ppb)

Ln(ExtLead) = log-transformed exterior-lead concentration (ppm), minus the mean of the log-transformed data used to develop the model (6.232 ppm), where the exterior

sample is either soil collected at the perimeter of the foundation, soil from the child's play area, or exterior dust

ExtType = indicator of the type of exterior sample (1 = dust, 0 = soil)

ExtLoc = indicator of whether exterior sample is represented by soil at the perimeter of the house's foundation (1 = exterior sample is not from perimeter soil, 0 = exterior sample is from perimeter soil)

Ln(MaxXRF) = log-transformed maximum lead-paint measurement on interior surfaces (mg/cm^2, as measured by XRF), minus the mean of the log-transformed data used to develop the model (0.921 mg/cm^2)

PaintCond = indicator of paint condition (1 = damaged, 0 = undamaged)

Age = age of child (months) minus the mean age of children whose data were used to develop the model (16.3 months)

Age2 = Age^2 - (85.5 + 4.82·Age) (quadratic orthogonal polynomial)

Age3 = Age^3 - (-490.71 + 10.32·Age2 + 122.3·Age) (cubic orthogonal polynomial)

Boston, Butte, Bcreek, Cpgm, Csoil, Leadville, Magna, RochLong, RochLID, Sandy, and *Midvale* are indicators that the data come from the particular study being represented (1 = data comes from the particular study, 0 = otherwise)

Race = Race indicator (0 = white, 1 = other)

SES1 = indicator of whether the pseudo-Hollingshead measure of socioeconomic status is equal to 1 (1 = yes, 0 = no)

SES2 = indicator of whether the pseudo-Hollingshead measure of socioeconomic status is equal to 2 (1 = yes, 0 = no)

SES3 = indicator of whether the pseudo-Hollingshead measure of socioeconomic status is equal to 3 (1 = yes, 0 = no)

SES4 = indicator of whether the pseudo-Hollingshead measure of socioeconomic status is equal to 4 (1 = yes, 0 = no)

MouthOften = indicator of whether mouthing behavior occurs often in the child (1 = often, 0 = otherwise)

MouthRare = indicator of whether mouthing behavior occurs rarely in the child (1 = rarely, 0 = otherwise)

MouthSome = indicator of whether mouthing behavior occurs sometimes in the child (1 = sometimes, 0 = otherwise)

error = random error between the observed log-transformed blood-lead concentration and what is predicted by the model.

4.1.2 Development of the HUD Model

This section presents several issues on how the HUD model was developed that have a direct impact on the predicted blood-lead concentration and how this prediction should be interpreted. These issues include how studies were selected, how study effects were represented in the model, and how data were handled or adjusted prior to or during the model development exercise.

Study Selection and Potential Selection Bias

The HUD model was developed from environmental-lead, blood-lead, and demographic data from 12 studies performed over a 15-year time frame (1982-1997). These studies investigated the relationship between environmental-lead levels and children's blood-lead levels in various locations and subpopulations. Five of the studies (representing 62% of the data used to fit the model) were conducted in urban environments:

- Boston Longitudinal Study (Rabinowitz et al., 1985)
- Cincinnati Longitudinal Study (Bornschein et al., 1985b)
- Cincinnati Soil Study (Clark et al., 1991)
- Rochester Longitudinal Study (Lanphear et al., unpublished)
- Rochester Lead-in-Dust Study (Lanphear et al., 1996a,b)

The remaining seven studies (representing 38% of the data used to fit the model) were conducted in milling, mining, or smelter environments:

- Bingham Creek, Utah (1993)
- Butte, Montana (1990)
- Leadville, Colorado (1991)
- Magna, Utah (1994)
- Midvale, Utah (1989)
- Palmerton, Pennsylvania (1994)
- Sandy, Utah (1994)

According to Lanphear et al. (1998), these 12 studies were selected based on the following criteria:

- The studies had well-defined sampling protocols for blood and environmental media (particularly dust, soil, and paint).
- The studies took measures of dust-lead levels, soil-lead levels, paint-lead content (via XRF), and paint condition.
- The original data were available and could be reanalyzed.
- Dust samples were collected via wipe techniques or by the Dust Vacuum Method (DVM)
- Dust samples were taken within three months of collecting blood samples from the resident child(ren) (to address seasonal variation in blood-lead concentration).
- Children were not selected on the basis of having a high blood-lead concentration.

In addition, only cross-sectional data were considered (i.e., data were not considered that could reflect changes in environmental-lead levels over time).

As a result of the inclusion criteria, data for at least seven other studies considered by HUD were excluded from the model development process. These studies and the primary reasons for their exclusion (when specified within Lanphear et al., 1998) were

- UK Study (Davies et al., 1990) – dust collection method not wipe or DVM
- Boston and Baltimore segments of the EPA Urban Soil Lead Abatement Demonstration Study (USEPA, 1996a; Weitzman et al., 1993) – dust collection method not wipe or DVM
- Australian National Survey (1996) – lack of XRF paint-lead levels
- Baltimore R&M Study (USEPA, 1996c; USEPA, 1997c) – method for selecting children did not meet the criteria, and data were not available to the analysis
- Telluride, CO (1987) – reason for exclusion not given
- Trail, BC (1992) – reason for exclusion not given.

Lanphear et al. (1998) indicates that the 12 studies were not chosen to represent the entire nation or even communities like those in which the studies were conducted. In fact, these studies were conducted in communities with a recognized environmental-lead hazard, and any abatement efforts within each study targeted those hazards. Furthermore, the study effects included in the model were treated as fixed effects (i.e., they are the only studies of interest in the model-building process) rather than random effects (i.e., they are assumed to be a random sample of a larger population of studies). Thus, if the model is used to estimate risks to a broader population of children than simply those within the 12 studies, additional information is needed to determine the extent to which the pooled data used to develop the model are representative of the U.S. housing stock.

Fixed vs. Random Study Effects and Interaction with the Study Effects

As stated in the previous paragraph, the study effect in the HUD model is a series of fixed effects. If the study effect was assumed to be random instead of fixed (i.e., the studies can be considered a random selection of all such residential-lead exposure studies), then study-to-study variation would become a contributor to total variation in the prediction. Based on work with previous models that incorporate a random study effect, the study-to-study component is typically a major portion of total variability in the prediction. Thus, the variability associated with predictions by the HUD model is likely underestimated.

Additional underestimation in variability may result from the absence of interaction terms in the model between study effects and other environmental exposure factors. This can underestimate variability associated with inferences involving the environmental exposure factors, including the principal inferences which involve dust-lead and exterior-lead levels.

Adjusting for Measurement Error in Environmental-Lead Predictor Variables

The HUD model parameter estimates were determined from Simulation Extrapolation (SIMEX) methods, which attempted to quantify the theoretical relationship between blood-lead

and "error-free" measures of environmental-lead. As a result, the parameter estimates were adjusted to reflect measurement error present among the environmental-lead variables. However, the goal is to predict children's blood-lead concentrations as a function of wipe dust-lead loadings and soil-lead concentrations as they would be measured in a risk assessment, not their true, "error-free" (but unobservable) values. Carroll et al. (1995) states that for prediction problems, adjusting for the effects of measurement error in predictor variables is rarely necessary. Adjusting for measurement error in these predictor variables tends to increase the values of the slope parameters associated with these variables, which in turn can inflate predicted blood-lead concentrations. Thus any predictions from the HUD model fits should be properly labeled that values of the predictor variables are assumed to be "error-free" rather than measures of environmental-lead levels taken from activities such as a risk assessment (as the predictor variables in the models used in the §403 risk analysis were assumed to represent).

Making Survey Variable Definitions Consistent Across Studies

Because different survey designs in different studies can result in different definitions for a common survey measure (e.g., SES, mouthing behavior, paint condition), which in turn can introduce considerable complication when interpreting model predictions, certain survey measures were redefined to make them more consistent across studies. Each redefinition transforms the original data values to values generated from a domain that is consistent across the studies. However, such a transformation does not remove all study-to-study differences in these values. In particular, it does not consider factors that impact how the specific study measurements were obtained and which differ from study to study, such as the use of different survey instruments and different approaches to administering the instruments.

Converting DVM Dust-Lead Loadings to Wipe-Equivalents

While it was desired to have floor dust-lead loading assuming wipe dust collection as a predictor variable in the HUD model, some of the 12 studies used DVM methods to collect dust samples. Rather than exclude data from these studies from consideration in the model development effort, a procedure was derived to convert DVM dust-lead loadings reported in these studies to wipe-equivalent loadings. This procedure used data from the Butte study to develop the following conversion equations:

- $Log_{10}(Wipe) = 0.7727 + 0.9821 \cdot Log_{10}(DVM)$ *for carpeted floors,*
- $Log_{10}(Wipe) = 0.1762 + 0.4839 \cdot Log_{10}(DVM)$ *for hard floors.*

where "Wipe" and "DVM" indicate wipe dust-lead loadings and DVM dust-lead loadings, respectively (Westat, 1998). Note that these same two equations were used to convert DVM dust-lead loadings in each study, regardless of whether the relationship between DVM and wipe dust-lead loadings differed among the studies.

The method for deriving these conversion equations included a procedure to adjust for measurement error in the DVM dust-lead loading measurements. However, the purpose of the

conversion was to predict a measured wipe dust-lead loading based on a measured DVM dust-lead loading, not the (unobservable) "true" DVM dust-lead loading. Thus, some bias may have been introduced in this conversion process.

Interpreting the "Exterior-Lead" Predictor

Certain households whose data were used in the HUD model development effort did not have soil-lead data for various reasons (e.g., no bare soil). In these instances, exterior dust-lead concentration was generally measured instead. As a result, among the predictor variables in the HUD model was an indicator variable that identified whether or not an exterior-lead measurement was from dust or soil. This indicator allowed both the model intercept and the slope factor associated with exterior-lead concentration to change according to its value. However, no other consideration was made for differences in sampling and analysis methods between soil and exterior dust and the impact such differences can have on the reported lead levels. In addition, no indication was given that differences in bioavailability between soil-lead and exterior dust-lead were considered in developing the model.

When a household had soil-lead data available, data from foundation perimeter (i.e., dripline) soil were used when available; otherwise, play-area soil-lead data were used. Like the soil vs. exterior-dust issue in the previous paragraph, the HUD model includes an indicator variable that identified whether or not soil-lead levels represent dripline soil. This indicator allowed both the model intercept and the slope factor associated with exterior-lead concentration to change according to its value. However, certain study-to-study differences in collecting soil samples or obtaining a soil-lead measurement were not considered in model development, such as the depth of soil sampling, soil surface type (e.g., covered vs. bare), chemical methods for the digestion and analysis of soil samples, and soil compositing.

Handling Missing Water-Lead Measurements

While the HUD model included water-lead concentration as a predictor variable, it was necessary to impute values for this measurement during model development when data for a given household were not available. Water-lead data were unavailable for all households in two studies and up to 12% of study households in the other ten studies. In such instances, imputed measurements were randomly generated from a lognormal distribution with geometric mean equal to that observed from data for other study households (if data for other households were available) or to the community-wide average (if data for other households were not available).

Handling Data Reported Below a Detection Limit

When data values reflected measurements at or below some detection limit, the HUD model development effort replaced them with probability-based values between zero and the detection limit, where probabilities were determined from a lognormal distribution associated with data above the detection limit. According to Table 2.15 of Westat (1998), the incidence of not-detected results was high with XRF paint-lead level, and to a lesser extent, with water-lead

concentration. In half of the studies, the percentage of not-detected paint-lead results ranged from 20% to 86% (with the detection limit being reported at either 0.1 or 0.7 mg/cm^2). The percentage of not-detected water-lead concentrations (i.e., results somewhere below 5 ppb) exceeded 80% in two studies. In contrast, none of the lead levels in blood, dust, or soil samples were reported below a detection limit in 10 of the 12 studies, and lead levels in no more than 9% of these samples were reported below a detection limit in the other two studies.

Selecting from Multiple Observations Within a Household

When blood-lead concentration data were available for multiple children within a household (as occurred in nine of the studies), only data for one child selected at random from the household were considered in the HUD model development. If blood-lead data existed at multiple time points for the same child (such as at 6, 18, and 24 months of age in the Boston Longitudinal study), those time points whose data met the initial inclusion criteria were identified (e.g., lead interventions did not occur between the time points), and data for the time point having dust-lead measurements taken more closely in time were selected for the model development. Similarly, when environmental-lead measurements were repeatedly taken over time for a given household, data for the time point closest to a blood-lead measurement were used.

Handling Seasonality Effects

The effect of seasonality on blood-lead concentrations was given some consideration in the modeling effort (e.g., blood and dust samples must have been collected within three months of each other for their data to be included). However, there is no effect of seasonality included in the final model. It is unclear whether seasonality was determined not to be a significant effect among the pooled data, or whether a seasonality term was intentionally left out of the model.

4.1.3 Interpreting Results of Fitting the HUD Model

The previous section discussed issues concerning the pooled study data and development of the HUD model that should be understood when using the model to estimate risks, as is done in Section 5.1.1 and Appendix F. This section addresses the interpretation of results from fitting the HUD model and, in particular, caveats associated with certain interpretations.

Individual risks vs. population-based risks

As mentioned earlier, the HUD model estimates individual risks associated with lead exposure to children aged 6-36 months. While the §403 risk analysis included individual risks analyses, which EPA used in efforts to establish levels of concern for lead in environmental media, EPA was required to employ cost-benefit analysis to select §403 hazard standards. The cost-benefit analyses used population-based risks (i.e., risks posed by childhood lead exposure to the nation as a whole) to estimate the benefit and cost associated with performing interventions and other activities in response to §403 rules.

Individual risks and population-based risks are generally not comparable. This must be understood when attempting to compare the individual risks estimated by fitting the HUD model at specified environmental-lead levels with the population-based risk estimates found in the §403 risk analysis.

Interpreting Model Parameter Estimates

The prediction parameters in the HUD model are not independent. For example, it is known that soil-lead and dust-lead measures are correlated. Therefore, it is not appropriate to interpret the parameter estimates in the HUD model (or in the models developed for the §403 risk analysis) in isolation. Using the parameter estimates to characterize a cause-and-effect relationship that is attributable to a single parameter alone, such as measuring the extent of an increase in blood-lead concentration associated with a given increase in dust-lead loading, is very problematic.

One example of how correlation among the predictor variables can influence the model parameter estimates is seen with maximum XRF paint-lead measurement. One would expect a positive correlation between maximum XRF paint-lead measurement and blood-lead concentration, and as a result, a positive slope parameter. However, the estimated slope parameter is negative (-0.022), although not significantly different from zero. The negative estimate is likely due to confounding between paint-lead measurements and other predictor variables. The likelihood of confounding increases with the number of parameters in the model.

Problems with interpreting model parameter estimates in isolation emphasizes the need to consider total exposure (i.e., prediction based on considering the joint effect of all model parameters) rather than exposure associated with a single environmental medium. In the §403 situation, protectiveness needs to be judged by recognizing that hazard standards exist for dust, soil, and paint, and that resulting actions from these multiple standards will determine the level of protection, not just the actions associated with a single standard. For example, the level of protection associated with a dust-lead loading standard of 5 $\mu g/ft^2$, without consideration of other standards, may equal that associated with a joint set of standards that involve a higher dust-lead loading standard.

Interpreting Results at Low Environmental-Lead Exposures

The HUD model and the models developed for the EPA risk analysis are "log-log" models. That is, they predict log-transformed blood-lead concentration as a linear function of log-transformed environmental-lead levels. As the log transformation "stretches out" the lower portion of the scale and contracts the upper portion of the scale, very low environmental-lead levels and blood-lead concentrations have undue influence on inferences made from the models. For example, the effect of increasing dust-lead loading from 1 to 10 $\mu g/ft^2$ is equal to the effect of increasing dust-lead loading from 10 to 100 $\mu g/ft^2$. Therefore, inferences at such low levels can be overestimated and misleading. Thus, any inferences at very low dust-lead loadings, such as 1 or 5 $\mu g/ft^2$, should be made with caution.

4.1.4 Conclusions

The following conclusions can be made on the HUD model and the comparison of risk estimates originating from this model versus those originating from models used in the §403 risk analysis:

- As the HUD model parameters associated with environmental-lead measurements in specific media have been adjusted for measurement error, the input parameters to this model are assumed "true" lead levels in these media. This can provide biased results when the model is used to predict blood-lead concentration associated with lead levels measured in current risk assessments. The Rochester multimedia model and the empirical model did not have such an adjustment incorporated.

- While the HUD model contains study effects, they are considered fixed effects and therefore allow the model to make predictions for only the group of children represented by the 12 studies. Furthermore, the study effects impact only the intercept of the model, and any study-to-study differences that may be present in other model terms (such as in environmental-lead measurements) are not represented.

- The HUD model handles "exterior-lead measurements" (e.g., soil) differently than the §403 models; the impact of such difference has not been determined.

4.2 ALTERNATIVE MULTIMEDIA MODELS FOR PREDICTING A GEOMETRIC MEAN BLOOD-LEAD CONCENTRATION BASED ON ENVIRONMENTAL-LEAD LEVELS

As discussed in the introduction to this chapter, the Rochester multimedia model, presented in Section 4.2.3 of the §403 risk analysis report, was developed using data from the Rochester Lead-in-Dust study to explain children's blood-lead concentration as a function of dust-lead loadings from floors (carpeted and uncarpeted) and window sills, dripline soil-lead concentration, and an indicator variable on the presence of deteriorated lead-based paint and a child with paint pica tendencies. This model was used in the risk characterization (Section 5.3 of the §403 risk analysis report) to determine the probability that a child exposed to specific levels of lead in paint, dust, and soil would have a blood-lead concentration at or above 10 µg/dL. EPA used these estimates of individual risk, as well as the findings of performance characteristics analyses detailed in Section 6.1 of this report, in proposing levels of concern for lead in dust (page 30318 in the §403 proposed rule).

The §403 proposed rule considered uncarpeted floors and a yard-wide average soil-lead concentration when proposing dust and soil standards and levels of concern. The performance characteristics analysis cited in the proposed rule considered these types of dust-lead and soil-

lead measures. However, in the Rochester multimedia model, the floor dust-lead loading measure did not limit the type of floor surface to uncarpeted floors, the soil-lead measure represented only dripline soil, and the paint/pica indicator variable was different from the paint measure used in the performance characteristics analysis (the percentage of tested components in the home with deteriorated lead-based paint). For these reasons, it was difficult to compare estimates of individual risks based on this model to results obtained from the performance characteristics analyses. Thus, it was desired to fit an alternative multimedia model (cited as "Model A" in this section) that replaced the floor dust-lead and soil-lead predictor variables used in the Rochester multimedia model with uncarpeted floor dust-lead loading and yard-wide average soil-lead concentration, respectively, and replaced the paint/pica indicator variable with a measure of the percentage of tested components containing lead-based paint.

While a household risk assessment for lead-based paint hazards is expected, at a minimum, to characterize lead levels in floor dust and to identify the extent of deteriorated lead-based paint, it is possible that some risk assessments may not measure lead levels in soil or window sill dust. Therefore, to investigate how individual risks would be characterized in these types of risk assessments, two alternative multimedia models were fitted that were reduced versions of Model A. One model excluded soil-lead concentration as a predictor variable ("Model B"), and the other model excluded both soil-lead concentration and window sill dust-lead loading as predictor variables ("Model C").

The three alternative multimedia models were fitted using data from the Rochester Lead-in-Dust study, using the same approach used to fit the Rochester multimedia model in the §403 risk analysis. The models were log-linear in nature, where the dust-lead and soil-lead measures were log-transformed, and the models predicted a log-transformed blood-lead concentration. For example, Model A took the following form:

$$\log(PbB) = \beta_0 + \beta_1 * \log(PbF) + \beta_2 * \log(PbW) + \beta_3 * \log(PbS) + \beta_4 * PbP$$

where PbB represents blood-lead concentration (μg/dL), PbF represents household average dust-lead loading for uncarpeted floors (μg/ft^2), PbW represents household average dust-lead loading for window sills (μg/ft^2), PbS represents yard-wide average soil-lead concentration (μg/g), and PbP represents the larger (between the interior and exterior of the housing unit) of the percentages of tested components containing deteriorated lead-based paint. As with the Rochester multimedia model, ordinary least squares regression methods were used to fit the models to the Rochester data.

Table 4-1 presents the estimates of the model parameters for each of the three alternative multimedia models. Note that the model fits were based on different numbers of housing units, as eliminating certain predictor variables from the above model resulted in more housing units that had all necessary data available for fitting the model. An investigation of model diagnostics showed that the extent of collinearity among the predictor variables in these models was low. Generally, the slope estimates associated with the paint variable were very low, and except for

Table 4-1. Parameter Estimates and Associated Standard Errors for the Three Alternative Multimedia Models Fitted to Rochester Study Data to Predict Log-Transformed Blood-Lead Concentration[1]

Parameter	Predictor Variable	Parameter Estimate (Standard Error)		
		Model A	Model B	Model C
β_0	Intercept	0.331 (0.263)	0.899 (0.183)	1.337 (0.122)
β_1	log (PbF): Area-Weighted Arithmetic Mean (Wipe) Dust-Lead Loading from Uncarpeted Floors	0.114 (0.049)	0.130 (0.048)	0.140 (0.043)
β_2	log (PbW): Area-Weighted Arithmetic Mean (Wipe) Dust-Lead Loading from Window Sills	0.082 (0.037)	0.101 (0.035)	--
β_3	log (PbS): Yardwide Average Soil-Lead Concentration[2] (fine soil fraction)	0.115 (0.040)	--	--
β_4	PbP: The larger of the following two percentages: % of interior tested surfaces that contain deteriorated LBP, and % of exterior tested surfaces that contain deteriorated LBP[3]	0.001 (0.002)	0.002 (0.002)	0.004 (0.002)
R^2	Coefficient of Determination	20.03%	15.62%	11.38%
σ_{Error}	Error	0.561	0.572	0.580
n	# data points included in model fitting	177	188	196

"--" indicates that the variable is not included in the model as a predictor. The models are log-linear in nature.

[1] One housing unit (cid=01689) had an uncarpeted floor dust-lead loading measurement of 18130.0 $\mu g/ft^2$ (only one uncarpeted floor wipe sample was collected in this unit). This data value was omitted when fitting the above models as it was highly influential and led to a noticeable reduction in the estimate of β_1.

[2] Yardwide soil-lead concentration at a given housing unit was calculated as the unweighted arithmetic average of dripline and play area soil-lead concentrations. If one or the other is missing (but both are not missing), yardwide concentration was set equal to the non-missing value. If both are missing, yardwide concentration is missing.

[3] If one or the other of these two percentages is missing (but both are not missing), the value of this variable is set equal to the non-missing value. If both are missing, the value is missing.

Model C, were not significantly different from zero at the 0.05 level. See footnotes to this table for additional details on the model fits.

Other alternative multimedia models were considered when initially developing the Rochester multimedia model. These models, and information used to evaluate these models in selecting the final version used in the §403 risk analysis, were presented in Appendix G of the §403 risk analysis report.

4.3 SUPPLEMENTAL INFORMATION ON MODEL-BASED APPROACHES IN THE §403 RISK ANALYSIS

Some comments on the §403 proposed rule addressed issues concerning approaches taken in the §403 risk analysis in which statistical models were used to predict a post-intervention distribution of blood-lead concentration, and therefore, a means of assessing how health risks associated with lead-based paint hazards would change as a result of implementing the §403 rule. The types of models used in this analysis and the approach to characterize post-intervention health risks were presented in Chapters 4 and 6 of the §403 risk analysis report. In addition, technical details on these models and approaches were provided in appendices to the report. However, certain comments on the §403 proposed rule indicated that this information may not have been provided in sufficient detail or would have benefitted from additional clarity. In particular, two issues in question involved how the §403 risk analysis estimated a post-intervention blood-lead concentration distribution that was comparable to the baseline distribution that was characterized by data from Phase 2 of NHANES III, and how the empirical model (Section 4.2 of the §403 risk analysis report) account for measurement error issues associated with the predictor variables. The following subsections provide additional information on these two issues, specifically geared toward addressing the specific areas raised by selected public comments.

4.3.1 The "Scaling" Algorithm Used to Determine a Post-Intervention Blood-Lead Concentration Distribution

In the §403 risk analysis, EPA used data from Phase 2 of NHANES III (collected from 1991-1994) as the basis for the baseline ("pre-§403 rule") characterization of children's blood-lead concentration in the U.S. housing stock. As discussed in Section 3.5 of the §403 risk analysis report, EPA took this approach because these data were considered the best available data (as well as the most recent data) on blood-lead measures, the data consisted of actual blood-lead measurements from a nationally-representative survey, and it was preferred (and considered more defensible) to use such data to characterize the baseline distribution rather than data generated from statistical prediction models. However, because the "post-§403 rule" time period has not yet occurred, and it was desired to compare the blood-lead distribution in this time period to the baseline blood-lead distribution, it was necessary to use statistical prediction models to generate how the baseline distribution would change following interventions performed as a result of the §403 rule.

When estimating the blood-lead concentration and health effect endpoints used in the §403 risk analysis, it was assumed that the national distribution of blood-lead concentration was lognormally distributed. Initial investigations into the weighted NHANES III blood-lead concentration data used in the §403 risk analysis (Figure 5-3 of the §403 risk analysis report) suggested that this was a satisfactory assumption. The lognormal distribution is characterized by the geometric mean and geometric standard deviation (GSD) of the data (or, equivalently, the exponentiated mean and exponentiated standard deviation of the log-transformed data). Once the geometric mean and GSD were calculated from the weighted NHANES III data, the additional assumption of lognormality was used to obtain baseline estimates of the health effect and blood-lead concentration endpoints considered in the §403 risk analysis (e.g., probability that a child's blood-lead level was at or above 10 μg/dL). These estimates were presented in Table 5-1 of the §403 risk analysis report.

To estimate how the blood-lead distribution changed between the "pre-§403 rule" and "post-§403 rule" environments (based on a given set of candidate §403 standards and on assumed changes in environmental-lead levels resulting from implementing the §403 rule), model-based estimates of the blood-lead distribution were made for both environments. For reasons explained in the §403 risk analysis report, EPA chose to characterize both the pre-§403 and post-§403 blood-lead distributions twice, with the first characterization using the IEUBK model and the second characterization using the empirical model. Each characterization involved fitting the given model to environmental-lead data separately for each home in the HUD National Survey and weighting each prediction appropriately to represent a given proportion of the nation's children. Therefore, although the HUD National Survey homes do not represent a random sample of homes in the national housing stock, and therefore, the set of predicted blood-lead concentrations do not themselves represent a random sample of blood-lead levels in the nation's children, the fact that each prediction is weighted appropriately to represent a given proportion of the nation's children allows the total set of predictions to be a good estimate of the national blood-lead distribution, in either a pre-§403 or post-§403 environment.

Appendix E2 of the §403 risk analysis report discusses how model-predicted blood-lead concentrations generated for the HUD National Survey homes are used to estimate the geometric mean and GSD associated with the national blood-lead distribution. Recall that for a given HUD National Survey home, the model-predicted blood-lead level represents a geometric mean of children whose exposure is characterized by the environmental-lead levels in that home. Therefore, the estimated GSD of the national blood-lead distribution is characterized not only by the variability among the predicted blood-lead levels, but also by the assumed variability in blood-lead levels among individual children exposed to the same environmental-lead levels.

Under a given model (i.e., either the IEUBK or empirical model), the "scaling" algorithm (Appendix F1 of the risk analysis report) involves calculating the proportional change in the geometric mean and GSD of the model-predicted blood-lead distribution from the "pre-§403 rule" to the "post-§403 rule" environment. Then, the same proportional change in both statistics was applied to the geometric mean and GSD of the baseline distribution determined from the NHANES III data:

$$GM_{post\text{-}403} = GM_{baseline} * (GM_{model\text{-}based\ post\text{-}403} / GM_{model\text{-}based\ pre\text{-}403})$$
$$GSD_{post\text{-}403} = GSD_{baseline} * (GSD_{model\text{-}based\ post\text{-}403} / GSD_{model\text{-}based\ pre\text{-}403})$$

The resulting geometric mean and GSD, along with an assumption that lognormality still holds, characterized a blood-lead concentration that represented the post-§403 environment and that was considered comparable to the baseline distribution.

This type of algorithm was necessary due to the difficulties associated with comparing a model-based post-§403 blood-lead distribution directly with a baseline distribution that was characterized from observed (NHANES III) data. While the empirical model was calibrated so that its estimate for the baseline national geometric mean blood-lead concentration for children aged 1-2 years (as obtained within the approach taken in the §403 risk analysis) equaled the NHANES III Phase 2 estimate (although the predicted GSD was not similarly calibrated), such a calibration was not possible for the IEUBK model. As a result, using the HUD National Survey data as input, these models could not both predict the same national estimate of the geometric mean blood-lead concentration as Phase 2 of NHANES III. In addition, the empirical model could not be developed based on data from a national survey that measured both blood-lead and environmental-lead levels, which would have facilitated direct comparisons of the predicted blood-lead distribution between pre-§403 and post-§403 environments. Therefore, the "post-§403 rule" blood-lead distributions predicted by the two models had some inconsistency with the baseline distribution estimated from the NHANES III data, making direct comparisons problematic. For example, if a model underestimates the geometric mean, the benefits associated with the §403 rule could be overestimated, while if a model overestimates the geometric mean, this could result in estimates of negative benefits. Therefore, the "scaling" algorithm used in the risk analysis used the models to predict the change in the geometric mean and GSD that occurs from a pre- to post-§403 rule environment, then applied this same change to the baseline distribution.

Note that the scaling algorithm does not require that the two model-based blood-lead distributions (pre-§403 and post-§403) be independent of each other. In fact, the two distributions are dependent, because the post-§403 environmental-lead levels, used to predict post-§403 blood-lead levels, are dependent on the pre-§403 levels. Only the geometric mean and GSD of these two distributions are necessary to characterize, and they are used simply to estimate the proportional change in the geometric mean and GSD of the national blood-lead distribution between pre-§403 and post-§403 conditions.

While the geometric mean and GSD are scaled separately, one change is not necessarily independent of the other. For example, if the pre-§403 geometric mean and GSD both have high values, they are both likely to be reduced at a greater rate than at lower values. The approach was kept as simple as possible while retaining scientific defensibility, in order that it be easily applied during risk characterization and in the economic analysis.

In the peer review of the §403 risk analysis report, EPA specifically asked the peer reviewers to comment on whether the scaling procedure was scientifically defensible in general,

and in particular, whether it was relevant in the situation where the environmental-lead data (from the HUD National Survey) and the blood-lead data (from NHANES III) were collected at different periods in time. None of the peer reviewers specifically criticized the scaling algorithm. Furthermore, the Science Advisory Board reviewing the §403 risk analysis stated that, in general, the approach was scientifically defensible as presented, and specifically, the multi-step approach was warranted due to the need to use various datasets of differing sources and representing different time periods to make the characterization.

See Section 6.4.4 below for an alternative approach to applying this scaling algorithm where the probability of a child's blood-lead concentration exceeding 10 µg/dL is scaled rather than the GSD.

4.3.2 Adjusting the Empirical Model Parameter Estimates to Reflect Measurement Error

The approach to the measurement error adjustment, discussed in Section 4.2.4 of the §403 risk analysis report, attempted to correct for the fact that while the empirical model was developed using data from the Rochester Lead-in-Dust study, it was used to predict a (pre-403) geometric mean blood-lead concentration assuming that the data input to the model originated from the HUD National Survey. Because the Rochester study and the HUD National Survey used different sampling schemes involving different collection devices and instruments, as well as different analytical methods, and because the ranges of observed environmental-lead levels differed between the two studies, it was necessary to adjust the model parameter estimates to reflect these differences prior to allowing the model to accept HUD National Survey environmental-lead data as input to the prediction.

Note that the measurement error adjustment made to the empirical model was not to address the more-standard "errors in variables" issue (Carroll et al., 1995) which attempts to take into account that a value input to the model represents a measurement subject to error, rather than a "true" value. As discussed in Section 4.2.4 and Section G4.2 (Appendix G) of the §403 risk analysis report, the empirical model was not intended to be used in the risk analysis as a dose-response model, which would have required the predictor variables to reflect actual exposures. Instead, the model assumed that its input environmental-lead information reflected measurements that would have been made as a result of a risk assessment within a home. Therefore, adjusting the model for the fact that its inputs reflect measured rather than actual lead levels was considered inappropriate for this analysis. This decision in the type of application that is represented by the §403 risk analysis has been concurred upon in the published literature (e.g., Carroll and Galindo, 1999).

When using the empirical model to predict a post-403 geometric mean blood-lead concentration, some of the HUD National Survey dust-lead and soil-lead data (i.e., those data for homes that exceed the candidate 403 standards) were modified to reflect the impact of performing interventions in response to the 403 rule on these measured data values (see Table 6-2 of the §403 risk analysis report), then the model is fitted to the modified data. These

modified data are still considered to be measured lead levels, rather than actual (or "true") lead levels. However, the modified data values must first be transformed to represent measurements that would have made under the methods used in the HUD National Survey. For example, the assumed post-intervention floor wipe dust-lead loading of 40 $\mu g/ft^2$ must be converted to a Blue Nozzle vacuum-equivalent loading prior to using it as input to the empirical model.

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5.0 RISK CHARACTERIZATION

Chapter 5 of the §403 risk analysis report documented the final portion of the risk assessment phase of the §403 risk analysis, in which the methods introduced in the earlier chapters of the §403 risk analysis report were applied to characterize risks associated with current (i.e., baseline) lead exposures for children aged 1-2 years. The baseline distribution of blood-lead concentration in this population was characterized using data from Phase 2 of the Third National Health and Nutritional Examination Survey (NHANES III), conducted from 1991 to 1994. Alternative pre-§403 risk estimates were also calculated as a function of environmental-lead levels by using data from the HUD National Survey as input to the IEUBK and empirical models. Both individual risk estimates (i.e., risks associated with specific environmental-lead levels) and population-based risk estimates (i.e., average risks over the entire nation) were presented. As mentioned in Section 2.0, the specific blood-lead concentration and health effect endpoints used to measure the risks of lead exposure to children aged 1-2 years were

- Incidence of blood-lead concentration greater than or equal to 10 μg/dL
- Incidence of blood-lead concentration greater than or equal to 20 μg/dL
- Incidence of IQ score less than 70 in the population of U.S. children, which results from lead exposure
- Incidence of IQ score decrement (in the population of U.S. children) greater than or equal to 1 resulting from lead exposure
- Incidence of IQ score decrement (in the population of U.S. children) greater than or equal to 2 resulting from lead exposure
- Incidence of IQ score decrement (in the population of U.S. children) greater than or equal to 3 resulting from lead exposure
- Average IQ decrement within the population of U.S. children that results from lead exposure.

The risk characterization included a sensitivity and uncertainty analysis where possible alternatives to various approaches taken and assumptions made in the risk characterization were identified and incorporated into the analysis, and the resulting impact on the risk estimates was evaluated. This analysis resulted in a measure of the uncertainty associated with the risk estimates due to methodological assumptions, thereby producing a range of estimates within which the true risk may reasonably be expected to fall. Section 5.1 of this chapter contains the following additional sensitivity and uncertainty analyses that were performed and documented since the §403 risk analysis report was published:

- Calculate individual risks associated with specified lead levels in floor-dust and soil, as predicted by the HUD model introduced in Section 4.1 and the alternative multimedia models presented in Section 4.2.

- Calculate estimates assuming a 50% decline in the estimated geometric mean blood-lead concentration of children aged 1-2 years from the estimate generated

from Phase 2 of NHANES III (in addition to the estimates associated with 10%, 20%, and 30% declines that were presented in Section 5.4.3 of the §403 risk analysis report).

- Calculate model-based estimates of the pre-§403 blood-lead distribution under revised environmental-lead levels (from the HUD National Survey) input to the models, with the revisions representing the potential change in these levels that may have occurred since the survey was performed.

- Calculate baseline estimates of the IQ-related health effect endpoints assuming that specified non-zero thresholds exist in the relationship between blood-lead concentration and IQ.

5.1 RISK CHARACTERIZATION SENSITIVITY AND UNCERTAINTY ANALYSIS

The following subsections present the results of additional sensitivity and uncertainty analyses performed to gauge the level of uncertainty in baseline risk estimates associated with methodological assumptions. These results should be considered with those presented in the sensitivity and uncertainty analyses in Section 5.4 of the §403 risk analysis report to characterize overall uncertainty associated with the methods and assumptions taken in the risk assessment.

5.1.1 Estimates of Individual Risks from Applying the HUD Model

In Section 5.3 of the §403 risk analysis report, the concept of *individual risks* was introduced, and estimates of individual risks associated with lead exposure in children were generated by fitting the IEUBK and Rochester multimedia models to specified environmental-lead levels. Briefly, within the context of the §403 risk analysis, individual risks refer to the risks associated with a young child's exposure to specified levels of environmental-lead. Once environmental-lead levels were specified for each medium, the model-predicted blood-lead concentration at these levels, along with the assumption that blood-lead concentrations have a lognormal distribution with a specified variability, were used to estimate the percentage of children exposed to the specified set of environmental-lead levels that would have elevated blood-lead concentrations (i.e., at or above 10 μg/dL). Then, those sets of environmental-lead levels associated with estimated elevated blood-lead percentages of 1%, 5%, and 10% were identified and presented in Tables 5-5 through 5-7 and Figures 5-7 and 5-8 of Section 5.3 of the §403 risk analysis report. The IEUBK model was used to identify soil-lead concentrations associated with these elevated blood-lead percentages (at specified dust-lead loadings), while the Rochester multimedia model was used to identify (wipe) dust-lead loadings associated with these elevated blood-lead percentages (at specified soil-lead concentrations). These results contributed to the information which EPA used in proposing dust and soil levels of concern in the §403 proposed rule. See Section 5.3 of the §403 risk analysis report for additional details.

As discussed in Section 4.1 of this report, the HUD model was published shortly after the §403 risk analysis report was finalized, and some commenters to the §403 proposed rule

suggested that it be considered within the §403 risk analysis. The HUD model can be used in the same manner as the §403 risk analysis models to predict individual risks associated with exposure to a specified set of environmental-lead levels. Therefore, this section presents estimates of individual risks based on fitting the HUD model to specified environmental-lead levels and compares these risk estimates with those based on the IEUBK model (for soil) or the Rochester multimedia model (for dust). Supporting summaries and discussion for comparing HUD model results with those of the multimedia models developed for the §403 risk analysis are presented in Appendix F.

Soil-Lead Concentrations

When fitting the HUD model to evaluate individual risks associated with yard-wide average soil-lead concentration, the model's soil-lead parameters were set to the following values:

- ExtType = 0 (indicating that soil was sampled rather than exterior dust)
- ExtLoc = 0.5 (indicating that the total soil sampled was a rough average of drip-line and non-drip-line soil)

Figure 5-1 plots estimates of the percentage of children's blood-lead concentrations at or above 10 µg/dL as a function of soil-lead concentration, as predicted by the IEUBK model and the HUD model. The left-most panel in Figure 5-1 corresponds to the results of IEUBK model fits at specified dust-lead concentrations (100, 200, and 500 µg/g) and is identical to Figure 5-7 in the §403 risk analysis report. The middle and right-most panels of Figure 5-1 correspond to fits of the HUD model at specified dust-lead loadings (5, 10, 20, 25, 40, 50, 100 and 200 $\mu g/ft^2$) and differ according to the assumed geometric standard deviation (GSD) associated with the blood-lead concentration distribution (GSD=1.6 for the middle panel; GSD=1.72 for the right-most panel). A GSD of 1.72 was estimated within the HUD model publication (Lanphear et al., 1998).

The IEUBK and HUD model fits portrayed in Figure 5-1 are not directly comparable as the IEUBK model controls for dust-lead *concentration* while the HUD model controls for dust-lead *loading*. However, the plots do suggest that the predicted patterns of change in blood-lead concentration with soil-lead concentration differ considerably for the two models.

Table 5-1a, identical to Table 5-5 of the §403 risk analysis report, presents the soil-lead concentrations for which the IEUBK model-predicted percentages of children having blood-lead concentrations of at least 10 µg/dL equal 1%, 5%, and 10% assuming dust-lead concentrations of 100, 200, and 500 µg/g. Table 5-1b presents the soil-lead concentrations for which the HUD model-predicted percentages of children having blood-lead concentrations of at least 10 µg/dL equal 1%, 5%, and 10% assuming the dust-lead loadings considered in the HUD model panels of Figure 5-1. Table 5-1b (HUD model) indicates that, for all dust-lead loadings considered, the soil-lead concentrations estimated to maintain risks at 1% and 5% are less than 400 µg/g; even a soil-lead concentration of less than 6 µg/g would not achieve these levels of protection if children

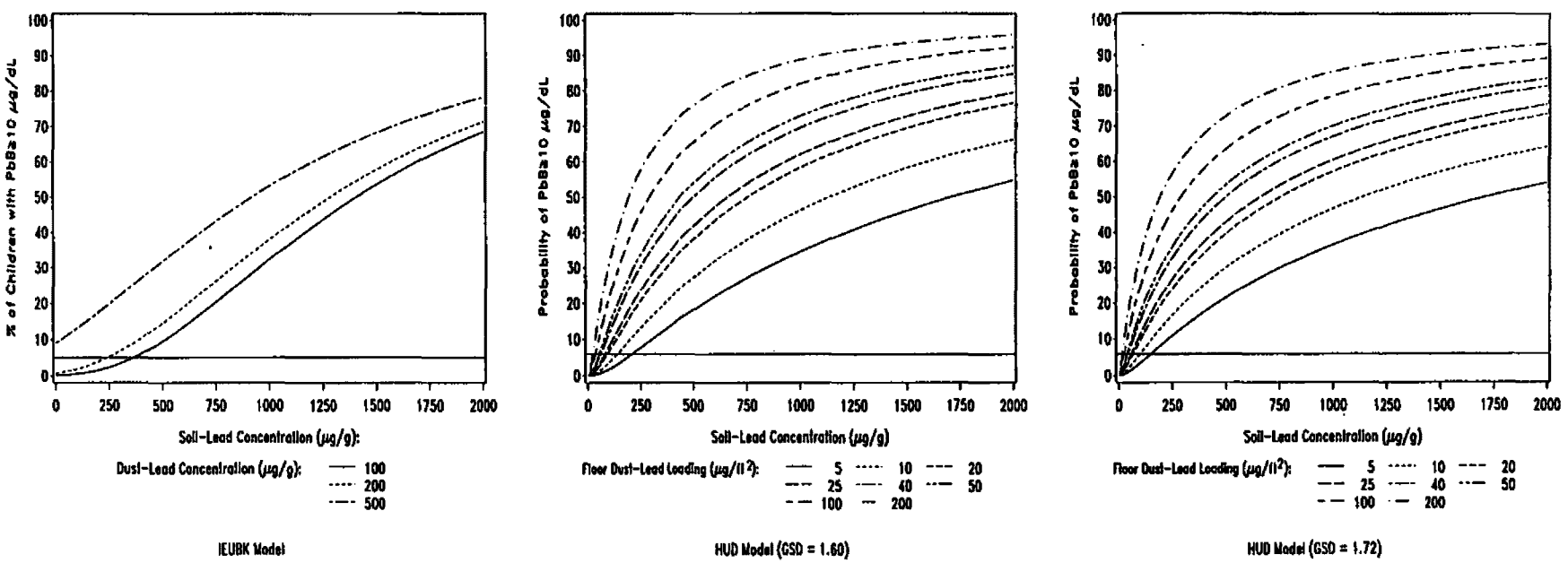

Figure 5-1. Percentage of Children's Blood-Lead Concentrations, as Predicted by the IEUBK and HUD Models, That Will Exceed or Equal 10 µg/dL as a Function of Yard-Wide Average Soil-Lead Concentration and at Fixed Levels of Dust-Lead Concentrations or Loadings

Table 5-1a. Yard-Wide Average Soil-Lead Concentrations at Which the Percentage of Children Aged 1-2 Years With Blood-Lead Concentration At or Above 10 μg/dL is Estimated by the IEUBK Model at 1, 5, or 10%, for Three Assumed Dust-Lead Concentrations (Table 5-5 in §403 risk analysis report).

Floor Dust-Lead Concentration (μg/g)	Soil-Lead Concentration (μg/g)		
	1%	5%	10%
100	155	365	515
200	35	245	395
500	Not achievable	Not achievable	25

Table 5-1b. Yard-Wide Average Soil-Lead Concentrations at Which the Percentage of Children Aged 1-2 Years With Blood-Lead Concentration At or Above 10 μg/dL is Estimated by the HUD Model at 1, 5, or 10%, for Eight Assumed Dust-Lead Loadings and Two Assumed Geometric Standard Deviations

Floor Dust-Lead Loading (μg/ft²)	Soil-Lead Concentration (μg/g)		
	1%	5%	10%
GSD = 1.60			
5	74.4	186.2	303.7
10	49.4	123.7	201.8
20	32.9	82.3	134.2
25	28.8	72.1	117.6
40	21.8	54.7	89.2
50	19.1	47.9	78.2
100	12.7	31.9	52.0
200	8.5	21.2	34.5
GSD = 1.72			
5	45.9	132.4	232.8
10	30.5	88.0	154.8
20	20.3	58.5	102.9
25	17.8	51.3	90.2
40	13.5	38.9	68.4
50	11.8	34.1	60.0
100	7.9	22.7	39.9
200	5.2	15.1	26.5

were exposed to dust-lead loadings of 40 $\mu g/ft^2$ or more. Based on Table 5-1b, if dust-lead loadings are equal to 5 $\mu g/ft^2$, a soil-lead concentration of approximately 300 $\mu g/g$ (100 $\mu g/g$) maintains the percentage of blood-lead concentrations greater than or equal to 10 $\mu g/dL$ at 5% for a GSD of 1.60 (1.72).

Floor Dust-Lead Loadings

Figures 5-2 and 5-3 plot the estimated percentages of children having blood-lead concentrations at or above 10 $\mu g/dL$ as a function of floor dust-lead loadings as predicted by the HUD model and the Rochester multimedia model assuming GSDs of 1.60 and 1.72, respectively, on the blood-lead distribution. Soil-lead concentrations are assumed to be fixed at 100, 400, 1200, 2000 and 5000 $\mu g/g$ and, for the Rochester multimedia model, window sill dust-lead loadings are assumed to be fixed at 200 and 500 $\mu g/ft^2$. In each figure, the left-most panel contains estimates based on fitting the HUD model. The Rochester multimedia model panels for GSD equal to 1.60 and soil-lead concentrations of 100 and 400 $\mu g/g$ were presented in Figure 5-8 in the §403 risk analysis report.

Tables 5-2 and 5-3 present the floor dust-lead loadings that are predicted by the HUD model and Rochester multimedia model, respectively, to maintain the percentage of children having blood-lead concentrations above or equal to 10 $\mu g/dL$ at 1%, 5%, and 10% for specified levels of soil-lead concentration, window sill dust-lead loading and GSD. Approximate 95% upper confidence bounds, which account for the variability of parameter estimates from the Rochester multimedia model, are also provided in Table 5-3. See Appendix C2, Section 5.0 of the §403 risk analysis report for the methodology used to compute these confidence bounds. The rows of Table 5-3 corresponding to a GSD of 1.60 and soil-lead concentrations of 100 and 400 $\mu g/g$ are identical to Table 5-6 in the §403 risk analysis report (after correcting an error in the computation of the confidence bounds).

Some key findings noted when comparing the individual risk estimates presented in Appendix F between the HUD model and Rochester multimedia model include the following:

- At very low floor dust-lead loadings (i.e., 1-5 $\mu g/ft^2$), the HUD model and the Rochester multimedia model yield similar predictions for the geometric mean blood-lead concentration, which also results in similar predictions for the health-effect endpoints that are calculated directly from this geometric mean (e.g., percentage of children with blood-lead concentration at or above a specified threshold; average IQ decrement resulting from lead exposure). However, due to the forms of these models and concerns involving the accuracy of very low dust-lead measurements, any conclusions made at such low dust-lead loadings must be made with caution.

- The predicted geometric mean blood-lead concentration under the HUD model ranges from 20% to nearly 60% higher than the prediction under the Rochester multimedia model as floor dust-lead loadings increase from 15 to 100 $\mu g/ft^2$ and

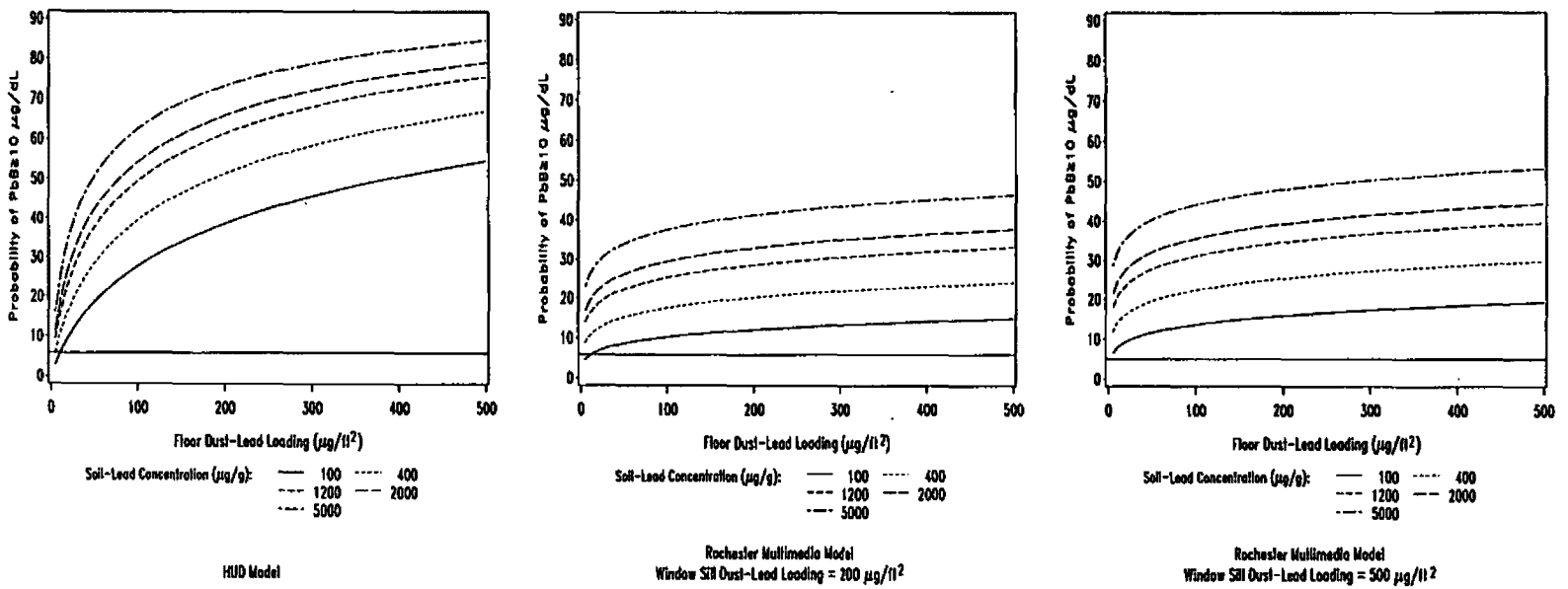

Figure 5-2. Percentage of Children's Blood-Lead Concentrations, As Predicted By the HUD Model and the Rochester Multimedia Model, That Will Exceed or Equal 10 µg/dL as a Function of Floor Dust-Lead Loading for Five Soil-Lead Concentrations and Two Window Sill Dust-Lead Loadings (Geometric Standard Deviation=1.60)

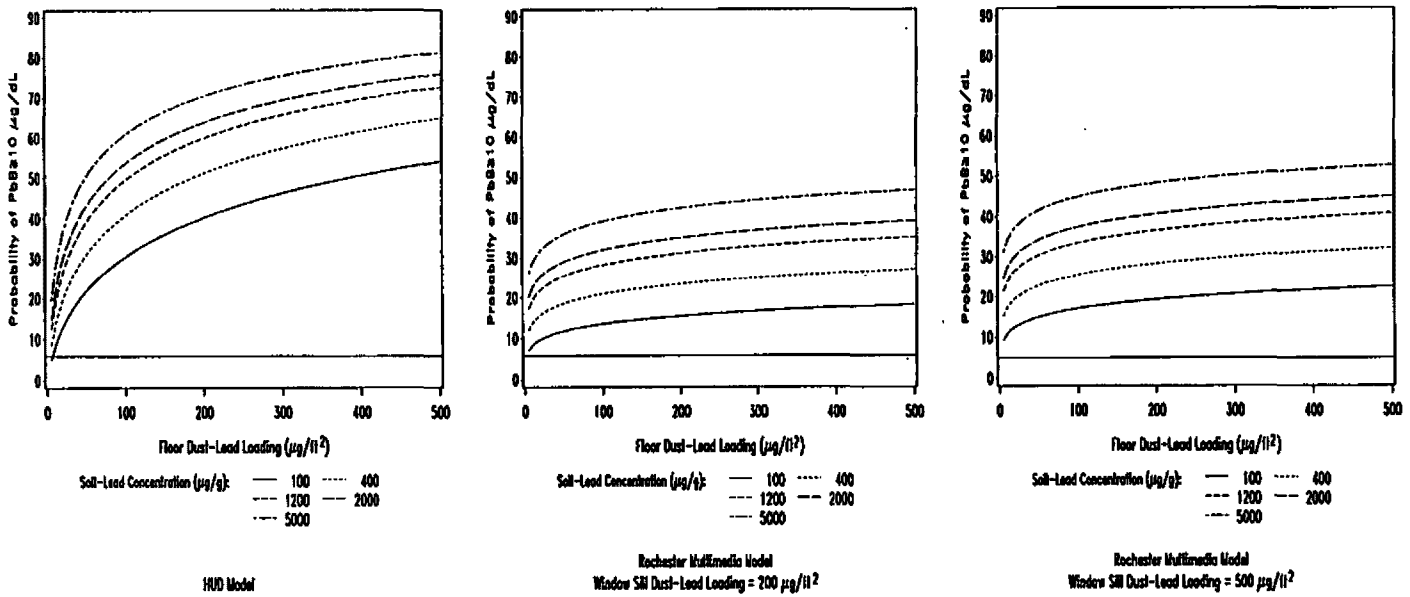

Figure 5-3. Percentage of Children's Blood-Lead Concentrations, As Predicted By the HUD Model and the Rochester Multimedia Model, That Will Exceed or Equal 10 µg/dL as a Function of Floor Dust-Lead Loading for Five Soil-Lead Concentrations and Two Window Sill Dust-Lead Loadings (Geometric Standard Deviation=1.72)

Table 5-2. Floor Dust-Lead Loadings at Which the Percentage of Children Aged 1-2 Years With Blood-Lead Concentration At or Above 10 μg/dL is Estimated by the HUD Model at 1, 5, or 10%, for Five Assumed Soil-Lead Concentrations and Two Assumed Geometric Standard Deviations

Soil-Lead Concentration (μg/g)	Floor Dust-Lead Loading (μg/ft²)		
	1%	5%	10%
GSD = 1.60			
100	1.88	8.93	20.49
400	0.90	4.29	9.83
1200	0.50	2.40	5.49
2000	0.39	1.83	4.19
5000	0.24	1.12	2.58
GSD = 1.72			
100	0.83	5.01	13.05
400	0.40	2.40	6.26
1200	0.22	1.34	3.50
2000	0.17	1.02	2.67
5000	0.10	0.63	1.64

Table 5-3. Floor Dust-Lead Loadings at Which the Percentage of Children Aged 1-2 Years With Blood-Lead Concentration At or Above 10 μg/dL is Estimated by the Rochester Multimedia Model at 1, 5, or 10%, for Five Assumed Soil-Lead Concentrations, Two Assumed Window Sill Dust-Lead Loadings, and Two Assumed Geometric Standard Deviations (expanded version of Table 5-6 in §403 risk analysis report).

Soil-Lead Concentration (μg/g)	Window Sill Dust-Lead Loading (μg/ft²)	Floor Dust-Lead Loading (μg/ft²)					
		1%		5%		10%	
		Estimate	95% Upper Confidence Bound [1]	Estimate	95% Upper Confidence Bound [1]	Estimate	95% Upper Confidence Bound [1]
GSD = 1.60							
100	200	0.05	0.37	6.70	22.00	89.08	327.73
	500	0.02	0.14	2.00	8.93	26.62	92.01
400	200	0.00	0.04	0.61	2.81	8.13	20.32
	500	0.00	0.02	0.18	1.12	2.43	9.24
1200	200	0.00	0.01	0.09	0.59	1.22	4.86
	500	0.00	0.00	0.03	0.22	0.36	1.99
2000	200	0.00	0.00	0.04	0.28	0.50	2.47
	500	0.00	0.00	0.01	0.10	0.15	0.96
5000	200	0.00	0.00	0.01	0.07	0.10	0.69
	500	0.00	0.00	0.00	0.02	0.03	0.25
GSD = 1.72							
100	200	0.00	0.04	1.11	4.90	21.87	67.96
	500	0.00	0.01	0.33	1.95	6.54	24.54
400	200	0.00	0.00	0.10	0.64	2.00	7.04
	500	0.00	0.00	0.03	0.24	0.60	3.00
1200	200	0.00	0.00	0.02	0.12	0.30	1.59
	500	0.00	0.00	0.00	0.04	0.09	0.61
2000	200	0.00	0.00	0.01	0.06	0.12	0.77
	500	0.00	0.00	0.00	0.02	0.04	0.29
5000	200	0.00	0.00	0.00	0.01	0.03	0.20
	500	0.00	0.00	0.00	0.00	0.01	0.07

[1] The 95% upper confidence bounds here differ from those in Table 5-6 of the §403 risk analysis report. The values here are corrected for a mistake in the original computations.

as soil-lead concentrations decrease from 2000 ppm to 10 ppm (assuming, for the Rochester multimedia model, that window sill dust-lead loadings are at their estimated national median level; Tables F-1 and F-2 of Appendix F). Note that for a fixed value of the geometric standard deviation (GSD) for the blood-lead distribution, the average IQ decrement in the population that is associated with lead exposure is a multiple of the geometric mean (as calculated in the §403 risk analysis). Therefore, similar differences in predictions between the two models would occur for average IQ decrement.

- If the geometric standard deviation (GSD) associated with the blood-lead distribution is fixed, then as floor dust-lead loadings increase beyond 10 $\mu g/ft^2$, the predicted percentage of children with blood-lead levels at or above 10 µg/dL increases at a much faster rate under the HUD model (at a constant soil-lead level). For example, if window sill dust-lead loading is at its estimated national median and soil-lead concentration is below 2000 ppm, the predicted percentage under the HUD model is at a minimum twice as large as the prediction under the Rochester multimedia model . This difference in predictions gets even greater as the assumed soil-lead concentration gets lower. For example, at a GSD of 1.6, a floor dust-lead loading of 100 $\mu g/ft^2$, and a soil-lead concentration of 10 ppm, the prediction is over 7 times higher for the HUD model compared to the Rochester multimedia model (13.1% versus 1.76%; Tables F-3 and F-4 of Appendix F).

5.1.2 Estimates of Individual Risks from Applying the Alternative Rochester Multimedia Model

The approach to estimating individual risks that was discussed and applied (using the IEUBK model and Rochester multimedia model) in Section 5.3 of the §403 risk analysis report and was applied using the HUD model in Section 5.1.1 above was also applied using the alternative Rochester multimedia model ("Model A") that was documented in Section 4.2 of this report. Recall that this model uses 1) average dust-lead loadings on uncarpeted floors, 2) average dust-lead loadings on window sills, 3) yardwide average dust-lead concentration, and 4) the percentage of painted components containing deteriorated lead-based paint as predictor variables. The blood-lead concentration predicted by this model is an estimate of the geometric mean of the distribution of blood-lead concentration, which is assumed to be lognormal with geometric standard deviation 1.6. The resulting distribution is then used to estimate the percentage of children with blood-lead concentration at or above 10 µg/dL. This subsection documents the results of estimating individual risks based on the alternative Rochester multimedia model.

In this analysis, it is assumed that the given residential environment for which individual risks will be estimated has no deteriorated lead-based paint (as was done in Section 5.3 of the §403 risk analysis report). Then, levels of two of the remaining three predictor variables are fixed, and the level of the third variable is determined so that either 1%, 5%, or 10% of children

exposed to the combined lead levels given by the three predictor variables would be predicted to have a blood-lead concentration at or above 10 µg/dL.

Table 5-4a contains the estimated floor dust-lead loading at which the predicted percentage of children with blood-lead concentration at or above 10 µg/dL is either 1%, 5%, or 10%, given that window sill dust-lead loading is at either 200 or 500 µg/ft^2, and soil-lead concentration is at either 100 or 400 µg/g. Table 5-4b contains the estimated window sill dust-lead loading at which the predicted percentage of children with blood-lead concentration at or above 10 µg/dL is either 1%, 5%, or 10%, given that floor dust-lead loading is at either 25 or 100 µg/ft^2, and soil-lead concentration is at either 100 or 400 µg/g. These two tables correspond to Tables 5-6 and 5-7, respectively, in Section 5.3 of the §403 risk analysis report, where the estimates were based on the original Rochester multimedia model. Finally, Table 5-4c contains the estimated yard-wide average soil-lead concentration at which the predicted percentage of children with blood-lead concentration at or above 10 µg/dL is either 1%, 5%, or 10%, given that floor dust-lead loading is at either 25 or 100 µg/ft^2, and window sill dust-lead loading is at either 200 or 500 µg/ft^2. (A corresponding table for the soil-lead concentration does not exist in Section 5.3 of the §403 risk analysis report as the soil-lead concentration predictor variable in the original Rochester multimedia model assumed only dripline soil rather than a yard-wide average, and the IEUBK model did not accept dust-lead loadings as input.)

Effect on risk analysis: If the target percentage of children with elevated blood-lead concentration is 5%, the estimates in Table 5-4a (fourth column) are only slightly higher than the corresponding estimates in Table 5-6 of the §403 risk analysis report, suggesting that at the fixed levels of soil-lead concentration and window sill dust-lead loading, the corresponding floor dust-lead loading is nearly the same for the two methods determined by the two forms of the multimedia model. However, at a soil-lead concentration of 100 µg/g, the estimated floor dust-lead loadings are considerably smaller (and below 50 µg/ft^2) than in Table 5-6 of the §403 risk analysis report under 10% risk and considerably larger (but still below 1 µg/ft^2) under a 1% risk.

For window sills (Table 5-4b), estimated dust-lead loadings are reduced under the alternative Rochester multimedia model compared to the estimates in Table 5-7 of the §403 risk analysis report at the 5% and 10% risk levels. At the 5% risk level, the estimated window sill dust-lead loadings were at 40 µg/ft^2 or lower at the specified soil-lead and uncarpeted floor dust-lead levels (Table 5-4b, fourth column).

Yardwide average soil-lead concentration needed to be below 150 µg/g at each of the specified levels of floor and window sill dust-lead loadings to achieve risks of 10% or lower, and at 32 µg/g or lower to achieve risks of 5% or lower (Table 5-4c). The estimated soil-lead concentration increases as the two specified dust-lead loadings decrease, illustrating how the soil-lead standard could become less stringent as the dust-lead loading standards became more stringent.

Table 5-4a. Uncarpeted Floor Dust-Lead Loadings at Which the Percentage of Children Aged 1-2 Years With a Blood-Lead Concentration At or Above 10 μg/dL is Estimated by the Alternative Rochester Multimedia Model (A) at 1%, 5%, or 10%, Under Fixed Levels of Yardwide Average Soil-Lead Concentration and Window Sill Dust-Lead Loading

Yardwide Average Soil-Lead Conc. (μg/g)	Window Sill Dust-Lead Loading ($\mu g/ft^2$)	Uncarpeted Floor Dust-Lead Loading ($\mu g/ft^2$) at Which the Estimated % of Children With Blood-Lead Concentration At or Above 10 μg/dL is ...		
		1%	5%	10%
100	200	0.48	7.9	35
	500	0.25	4.1	18
400	200	0.12	1.9	8.7
	500	0.06	1.0	4.5

Note: The percentages of 1%, 5%, and 10% were determined assuming that the blood-lead distribution is lognormal with geometric mean as predicted by the alternative Rochester multimedia model (A) and a geometric standard deviation of 1.6.

Table 5-4b. Window Sill Dust-Lead Loadings at Which the Percentage of Children Aged 1-2 Years With a Blood-Lead Concentration At or Above 10 μg/dL is Estimated by the Alternative Rochester Multimedia Model (A) at 1%, 5%, or 10%, Under Fixed Levels of Yardwide Average Soil-Lead Concentration and Uncarpeted Floor Dust-Lead Loading

Yardwide Average Soil-Lead Conc. (μg/g)	Uncarpeted Floor Dust-Lead Loading ($\mu g/ft^2$)	Window Sill Dust-Lead Loading ($\mu g/ft^2$) at Which the Estimated % of Children With Blood-Lead Concentration At or Above 10 μg/dL is ...		
		1%	5%	10%
100	25	0.78	40	320
	100	0.11	5.7	46
400	25	0.11	5.6	45
	100	0.02	0.80	6.5

Note: The percentages of 1%, 5%, and 10% were determined assuming that the blood-lead distribution is lognormal with geometric mean as predicted by the alternative Rochester multimedia model (A) and a geometric standard deviation of 1.6.

Table 5-4c. Yardwide Average Soil-Lead Concentration at Which the Percentage of Children Aged 1-2 Years With a Blood-Lead Concentration At or Above 10 μg/dL is Estimated by the Alternative Rochester Multimedia Model (A) at 1%, 5%, or 10%, Under Fixed Levels of Dust-Lead Loadings for Uncarpeted Floors and Window Sills

Uncarpeted Floor Dust-Lead Loading ($\mu g/ft^2$)	Window Sill Dust-Lead Loading ($\mu g/ft^2$)	Yardwide Average Soil-Lead Concentration ($\mu g/g$) at Which the Estimated % of Children With Blood-Lead Concentration At or Above 10 μg/dL is ...		
		1%	5%	10%
25	200	2.0	32	140
	500	1.0	17	73
100	200	0.50	8.0	35
	500	0.26	4.2	18

Note: The percentages of 1%, 5%, and 10% were determined assuming that the blood-lead distribution is lognormal with geometric mean as predicted by the alternative Rochester multimedia model (A) and a geometric standard deviation of 1.6.

5.1.3 Considering Potential Declines in Blood-Lead Concentration from NHANES III Phase 2 Measures

The results of this subsection are an extension of the analysis in Section 5.4.3 of the §403 risk analysis report. In that subsection, the geometric mean blood-lead concentration of 3.14 μg/dL for children aged 1-2 years, estimated from data collected in Phase 2 of NHANES III (1991-1994), was assumed to be either 10%, 20%, or 30% lower, and the resulting impact on the baseline risk estimates was investigated. This analysis was performed due to the likelihood of continued decline in blood-lead concentrations in the U.S. population that has occurred in recent years. It was desired to augment this analysis by considering an additional assumption on the percentage decline since Phase 2 of NHANES III: 50%.

Table 5-5 presents the baseline estimates of the blood-lead concentration and health effect endpoints for children aged 1-2 years, where each blood-lead concentration measurement in Phase 2 of NHANES III was reduced by the same amount: 10%, 20%, 30%, or 50%. Thus, the analysis assumed a constant percentage decline for the entire blood-lead concentration distribution as characterized by Phase 2 of NHANES III. This table is an extension of the results presented in Table 5-11 of the §403 risk analysis report and includes the baseline estimates reported in the risk analysis for comparison purposes (i.e., where no reduction is assumed).

Note that within NHANES III, the estimated geometric mean blood-lead concentration for children aged 1-2 years declined from 4.05 μg/dL in Phase 1 to 3.14 μg/dL in Phase 2, representing a 22.5% decline. This is within the range of declines being considered in the

Table 5-5. Sensitivity Analysis for the Estimated Baseline Number and Percentage of Children Aged 1-2 Years Having Specific Health Effect and Blood-Lead Concentration Endpoints, Assuming Various Percentage Declines in Blood-Lead Concentration Since Phase 2 of NHANES III

Health Effect and Blood-Lead Concentration Endpoints	Numbers (%) of Children Aged 1-2 Years				
	Risk Analysis Estimate (Table 5-1 of the §403 risk analysis report)	Percentage Decline in Blood-Lead Concentration Since NHANES III Phase 2			
		10%	20%	30%	50%
PbB ≥ 20 μg/dL	46,800 (0.588%)	30,900 (0.388%)	18,900 (0.238%)	10,600 (0.133%)	2,130 (0.0268%)
PbB ≥ 10 μg/dL	458,000 (5.75%)	340,000 (4.27%)	239,000 (3.00%)	156,000 (1.96%)	46,800 (0.588%)
IQ score less than 70	9,130 (0.115%)	8,610 (0.108%)	8,160 (0.102%)	7,760 (0.098%)	7,140 (0.0897%)
IQ score decrement ≥ 1	3,060,000 (38.5%)	2,640,000 (33.2%)	2,190,000 (27.6%)	1,740,000 (21.8%)	863,000 (10.8%)
IQ score decrement ≥ 2	863,000 (10.8%)	669,000 (8.40%)	493,000 (6.19%)	340,000 (4.27%)	117,000 (1.47%)
IQ score decrement ≥ 3	294,000 (3.70%)	213,000 (2.68%)	146,000 (1.83%)	91,900 (1.15%)	25,200 (0.317%)
Average IQ score decrement	1.06	0.951	0.845	0.740	0.528
Geometric Mean (μg/dL)	3.14	2.82	2.51	2.20	1.57

sensitivity analysis within Table 5-5. However, due to the NHANES III survey design and how this survey was performed, caution must be taken when interpreting observed differences in results between the two phases of this survey.

Effect on risk analysis: According to Table 5-5, if it were assumed that a 50% across-the-board decline in blood-lead concentration (resulting in a national geometric mean blood-lead concentration of 1.57 μg/dL), this would reduce the estimated number of children whose blood-lead concentration was at or above 20 μg/dL from 46,800 to 2,130, a decline of 95%, while the estimated number at or above 10 μg/dL would be reduced by nearly 90%, to 46,800 children. The 50% decline resulted in percentage declines of 72%, 86%, and 91% for numbers of children with IQ score decrements of 1, 2, or 3, respectively, as a result of lead exposure. The estimated average IQ decrement in the population due to lead exposure is cut in half under this assumption (from 1.06 to 0.53 points), matching the assumed 50% decline in blood-lead concentration because the IQ/blood-lead concentration relationship is assumed to be linear across the entire range of blood-lead concentration. The effects of the lower assumed percentage declines (10%-30%) were discussed in Section 5.4.3 of the §403 risk analysis report.

The results in Table 5-5 are based on the assumption that the blood-lead concentrations for each child in the population have been reduced by the same percentage since Phase 2 of NHANES III. In reality, different subgroups have achieved different rates of change over this time. However, considering different percentage declines for different subgroups would be very difficult, and the resulting estimates of the health effect and blood-lead concentration endpoints would likely differ only slightly from that observed in Table 5-5.

5.1.4 Considering How Baseline Environmental-Lead Levels May Have Changed Since the HUD National Survey

Although interim data from the NSLAH (Section 3.1) have recently been made available to this risk analysis and have been summarized throughout this report, the fact that the public could not have reviewed these summaries during the public comment period limits the extent to which these data could be considered in the rulemaking. Therefore, for purposes of the rulemaking, data from the HUD National Survey continue to be the only nationally-representative data source on baseline environmental-lead levels in the nation's housing stock. Nevertheless, it was desired to estimate how changes in these environmental-lead levels that have occurred since the HUD National Survey was conducted would affect the baseline (i.e., pre-§403) risk characterization. Therefore, a sensitivity analysis was performed where the HUD National Survey data was adjusted to reflect possible change in the distribution over time. The adjusted data would yield a surrogate distribution of baseline environmental-lead levels. Several alternative adjustments would be considered, and risk estimates based on each set of adjusted data would be calculated.

To help in determining appropriate adjustments to the HUD National Survey data, the summaries presented in Section 3.2 of this document compared the distribution of dust-lead and soil-lead data reported in the HUD National Survey with distributions from other studies performed more recently, but typically in specific locations that may not necessarily be nationally-representative. These summaries showed that the distributions were quite consistent across studies, suggesting that the distributions based from the HUD National Survey data, even after converting from Blue Nozzle dust-lead loadings to wipe-equivalent loadings, are likely adequate for characterizing environmental-lead levels even up to ten years after the survey. In fact, the HUD National Survey data distributions were often centered at lower lead levels than in the other studies. (A primary exception was household average dust-lead loadings, where data from the interim NSLAH were considerably lower than in other studies, including the HUD National Survey. It is currently uncertain of the degree to which the observed distribution from the interim NSLAH reflects actual declines in dust-lead levels since the HUD National Survey.) Furthermore, the nature of the distribution appears to be affected by housing age, with higher lead levels associated with older housing.

For this sensitivity analysis, the following five alternatives for adjusting the HUD National Survey data were made, in an effort to reflect more current environmental-lead levels in households:

- Average dust-lead loading and dust-lead concentration reduced by 20%, (yard-wide) average soil-lead concentration reduced by 20%

- Average dust-lead loading/concentration reduced by 50%, average soil-lead concentration reduced by 50%

- Average dust-lead loading/concentration reduced by 50%, average soil-lead concentration reduced by 0%

- Average dust-lead loading/concentration reduced by 0%, average soil-lead concentration reduced by 50%

- Average dust-lead loading/concentration increased by 25%, average soil-lead concentration increased by 25%.

The dust-lead loading assumptions are assumed to be for both floors and window sills and are made to the reported Blue Nozzle loadings (i.e., those estimates used as input to the empirical model). The same changes are assumed for Blue Nozzle floor dust-lead concentration, which is used as input to the IEUBK model.

Each of the above five sets of alternatives implies that the same percentage change would be applied to data from each housing unit in the HUD National Survey. Thus, the resulting national distribution of baseline environmental-lead levels would be a simple shift in the current distribution used in the §403 risk analysis, with no change in the variability associated with the distribution. Insufficient data exist to determine how a distribution's variability may have changed, so it is assumed to remain unchanged. Within each set, different percentage changes are considered for dust-lead loading and soil-lead concentration to allow for added flexibility in how lead levels may have changed in different media. Four of the five sets represent declines over time, which are expected due to the increased prevalence of homes with no lead-based paint in the housing stock and the reduced likelihood of residual contamination associated with leaded gasoline emissions. Nevertheless, one set representing an increase is considered, due to the potential for the new survey to include housing with generally higher levels than those houses included in the original survey and the continued potential for deteriorated lead-based paint and other lead sources to contaminate dust and soil.

Note that in all five sets of adjustments, the assumed within-house geometric standard deviation (GSD) remains equal to 1.6. Alternative values of this GSD assumption were considered in the sensitivity analysis presented in Section 5.4.6 of the §403 risk analysis report.

Table 5-6 presents the pre-§403 model-based estimates for the health effect and blood-lead concentration endpoints, under each of the above five sets of data adjustments, as calculated based on blood-lead distribution generated from IEUBK and empirical model fits. For comparison purposes, the table includes the estimates assuming no adjustments (i.e., as the data were used in the §403 risk analysis) as reported in Table 5-2 of the §403 risk analysis report.

Table 5-6. Sensitivity Analysis on How Changes in Household Average Baseline Dust-Lead Loadings/Concentrations and Soil-Lead Concentration Impact Pre-§403 Estimates of Health Effect and Blood-Lead Concentration Endpoints for Children Aged 1-2 Years

Assumed Percentage Change in Average Dust-Lead Loadings and Concentrations (Both Floor and Window Sill) and in Yard-wide Average Soil-Lead Concentration						
Dust:	No change	20% decrease	50% decrease	50% decrease	No change	25% increase
Soil:	No change	20% decrease	50% decrease	No change	50% decrease	25% increase
Predicted Health Effect And Blood-Lead Concentration Endpoints (Based on Empirical Model)						
PbB ≥20 (%)	0.0278	0.0212	0.0117	0.0187	0.0176	0.0364
PbB ≥10 (%)	1.54	1.28	0.849	1.17	1.13	1.85
IQ < 70 (%)	0.0997	0.0983	0.0957	0.0977	0.0974	0.101
IQ decrement ≥1 (%)	34.5	31.8	26.5	30.6	30.1	37.2
IQ decrement ≥2 (%)	4.53	3.87	2.74	3.61	3.49	5.27
IQ decrement ≥3 (%)	0.718	0.584	0.373	0.532	0.509	0.877
Avg. IQ decrement	0.932	0.896	0.825	0.880	0.873	0.969
Predicted Health Effect And Blood-Lead Concentration Endpoints (Based on IEUBK Model)						
PbB ≥20 (%)	2.24	1.39	0.427	0.957	1.44	3.06
PbB ≥10 (%)	12.4	9.33	4.60	7.28	9.70	15.3
IQ < 70 (%)	0.146	0.131	0.110	0.121	0.132	0.160
IQ decrement ≥1 (%)	50.4	45.1	34.6	40.3	46.4	55.4
IQ decrement ≥2 (%)	19.9	15.8	8.97	12.8	16.4	23.8
IQ decrement ≥3 (%)	8.95	6.46	2.90	4.92	6.72	11.3
Avg. IQ decrement	1.40	1.24	0.978	1.12	1.26	1.56

Effect on risk analysis: The greatest total decline in baseline environmental-lead levels being considered is the set containing 50% declines in both dust- and soil-lead levels (i.e., the fourth column of Table 5-6). Under the empirical model, Table 5-6 indicates that the most sensitive endpoints to the 50% decline in both dust-lead and soil-lead are the incidence of IQ decrement of at least 3 and the incidence of blood-lead concentration of at least 10 μg/dL, where declines of 48% and 45%, respectively, were observed in these estimates relative to no decline in environmental-lead levels. The empirical model-based estimates appear to be more sensitive to changes in soil-lead concentration than in dust-lead concentration, as lower estimates were observed when soil-lead concentrations declined by 50% (and no change was made to dust-lead loadings) than when dust-lead loadings declined by 50% (and no change was made to soil-lead concentrations). This is explained by the empirical model's larger slope estimate for soil-lead

concentration than for dust-lead loading in either floors or window sills (Table 4-3 of the §403 risk analysis report).

When the IEUBK model is used to estimate the distribution of blood-lead concentration, corresponding declines of 68% and 63% were observed for incidence of IQ score decrement at or above 3 and for incidence of blood-lead concentration at or above 10 µg/dL, respectively, when 50% declines are assumed for both dust-lead and soil-lead concentration (Table 5-6). However, in this same scenario, the greatest decline (81%) among the endpoints is observed with the incidence of blood-lead concentration at or above 20 µg/dL. This is a considerable decline compared to the 24% decline for this endpoint observed under the empirical model. Contrary to the type of finding observed under the empirical model, the IEUBK model-based estimates appear to be more sensitive to changes in dust-lead concentration than in soil-lead concentration, as lower estimates were observed when dust-lead concentrations declined by 50% (and no change was made to soil-lead concentrations) than when soil-lead concentrations declined by 50% (and no change was made to dust-lead concentrations).

Under both models, the last column in Table 5-6 shows that only modest increases in the risk estimates were observed under the one adjustment assumption involving increases in environmental-lead levels (i.e., 25% increases in both dust-lead and soil-lead levels).

5.1.5 Impact on the Estimated Incidence of IQ Point Decrement Assuming Certain Thresholds on the IQ/Blood-Lead Relationship

As discussed in Chapter 4 of the §403 risk analysis report, 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, with no evidence of a threshold in this relationship (i.e., non-zero blood-lead concentration below which the predicted IQ point loss is zero). These results were used in the §403 risk analysis to characterize the IQ/blood-lead relationship. Section 2.3 of this report provides additional justification for making these assumptions.

As discussed in Section 2.3, some researchers have suggested that a non-zero threshold exists in the IQ/blood-lead relationship. While no consensus on a single threshold has been adopted among those making this conclusion, and such conclusions are occasionally made by visual inspection of data rather than on statistical criteria, this sensitivity analysis considers the impact of assuming non-zero blood-lead concentration thresholds on the baseline and model-based pre-§403 risk estimates. A non-zero threshold will result in reduced estimates for health effects measured by IQ decrement, as children with blood-lead concentrations below the threshold will have an estimated IQ decrement of zero due to lead exposure.

Estimates of the IQ decrement parameters under the following thresholds are presented in this subsection: 1, 2, 3, 5, 8, and 10 µg/dL. In addition, the estimates under an assumed "threshold" of 0 µg/dL (i.e., those measured in the §403 risk analysis and meaning that any blood-lead level, regardless of how small, would have an adverse effect on a child's IQ score) are

presented for comparison purposes. The candidate threshold of 8 µg/dL has been suggested by Rabinowitz et al. (1992), as discussed in Section 2.3. The candidate threshold of 10 µg/dL was selected as it represents the action level reported by the Centers for Disease Control and Prevention (Section 2.5.1 of the §403 risk analysis report). It also is representative of the higher-level thresholds reported by some early studies; thresholds any higher than 10 µg/dL would result in extremely low (and likely very underestimated) risk estimates and have been discounted by more recent studies. Levels of 1, 2, 3, and 5 µg/dL represents possible candidates of a very low, but positive, threshold. Such thresholds would not be detectable by many studies in the literature as they tend to fall below the range of observed data or the detection limit of the blood-lead measurement procedure.

The assumption of a positive threshold requires a minor modification to the method used to predict IQ score decrement based on blood-lead concentration. An average IQ point loss of 0.257 continues to be predicted for every 1.0 µg/dL increase in blood-lead concentration, but only above the assumed blood-lead concentration threshold value. Thus, if T represents the threshold, then the predicted IQ score decrement at a blood-lead concentration of C would equal 0.257*(C-T) if C is greater than T, or zero if C is less than or equal to T. While the methodology used to obtain risk estimates remains the same as that documented in Appendices E1 and E2 of the §403 risk analysis report, slight differences were required for calculating the average and standard deviation of IQ decrement, as this measure was no longer assumed to be lognormally distributed. See Appendix B for how these statistics are calculated assuming a non-zero threshold.

Table 5-7 presents the estimated percentages of children with IQ score decrements greater than or equal to 1, 2, or 3, and the average and standard deviation IQ point decrement under assumptions of an IQ score decline of 0.257 points for every 1.0 µg/dL increase in blood-lead concentration above the specified threshold. These estimates are presented assuming the baseline blood-lead distribution (top section of the table), the pre-§403 distribution as generated by IEUBK model fits (middle section of the table), and the pre-§403 distribution as generated by the empirical model fits (bottom section of the table) for children aged 1-2 years.

Effect on risk analysis: The magnitude of the assumed blood-lead concentration threshold has a considerable impact on the percentage of children affected by decrements in IQ score. As seen in Table 5-7, while the §403 risk analysis estimated an average IQ decrement of 1.06 points occurs due to lead exposure across the population of children aged 1-2 years, this average declines by approximately 44% under a assumed threshold of 2 µg/dL (0.588 points) and by 90% under a threshold of 8 µg/dL (0.103 points). An estimated 38.5% of children aged 1-2 years were expected to experience an IQ score decrement of at least 1 if a threshold was not assumed. This percentage is decreased by approximately 50% under a threshold of 2 µg/dL (19.6%) and by 90% under a threshold of 8 µg/dL (3.5%). The percentage decline is decreased in magnitude as the lower limit of IQ score decrement increases to 3, but it remains at least a 39% decline for a threshold of 2 µg/dL and 83% for a threshold of 8 µg/dL.

Table 5-7. Sensitivity Analysis on the Assumed Blood-Lead Concentration Threshold on IQ Decrement and Its Impact on the Pre-§403 Estimates of IQ Decrement Endpoints for Children Aged 1-2 Years

Assumed Threshold (μg/dL)	% of Children Aged 1-2 Years with a Specified IQ Decrement Due to Lead Exposure[1]			Average IQ Decrement (# points)[2]	Standard Deviation of IQ Decrement[2]
	IQ Decrement ≥ 1	IQ Decrement ≥ 2	IQ Decrement ≥ 3		
Baseline Estimates (Section 5.1.1 of §403 risk analysis report)					
0	38.5	10.8	3.70	1.06	0.895
1	27.3	8.08	2.88	0.804	0.891
2	19.6	6.10	2.26	0.588	0.860
3	14.2	4.66	1.80	0.428	0.802
5	7.83	2.80	1.16	0.233	0.666
8	3.50	1.40	0.627	0.103	0.494
10	2.15	0.915	0.429	0.0638	0.408
Pre-§403 Estimates Based on IEUBK Model-Generated PbB Distribution (Section 5.1.2 of §403 risk analysis report)					
0	50.4	19.9	8.95	1.40	1.35
1	39.3	16.0	7.42	1.15	1.35
2	30.8	13.0	6.19	0.921	1.33
3	24.4	10.6	5.20	0.738	1.28
5	15.7	7.27	3.73	0.483	1.15
8	8.58	4.31	2.35	0.273	0.964
10	5.96	3.13	1.76	0.194	0.854
Pre-§403 Estimates Based on Empirical Model-Generated PbB Distribution (Section 5.1.2 of §403 risk analysis report)					
0	34.5	4.53	0.718	0.932	0.538
1	20.4	2.76	0.464	0.675	0.537
2	12.0	1.71	0.330	0.442	0.514
3	7.14	1.07	0.202	0.271	0.453
5	2.61	0.443	0.0926	0.0972	0.309
8	0.652	0.130	0.0312	0.0224	0.162
10	0.278	0.0613	0.0158	0.00912	0.108

[1] A 0.257 IQ decrement is assumed for each 1.0 μg/dL increase in PbB above the assumed threshold (see Section 4.4.1 of the §403 risk analysis report). Thus, the following hold:

- P[IQ ≥ 1] = P[PbB ≥ (threshold + 3.9 μg/dL]
- P[IQ ≥ 2] = P[PbB ≥ (threshold + 7.8 μg/dL)]
- P[IQ ≥ 3] = P[PbB ≥ (threshold + 11.7 μg/dL)]

[2] Average and standard deviation of IQ decrement are calculated assuming no IQ decrement occurs below the assumed threshold, and a 0.257 IQ decrement is assumed for each 1.0 μg/dL increase in PbB above the threshold.

Similar patterns of decline were seen for the pre-§403 estimates generated under the IEUBK and empirical model-based blood-lead distributions, with the empirical model predicting greater reductions for the larger thresholds. These model-based estimates were used in the procedure to characterize changes from baseline that occur in a post-§403 environment, which is addressed in Chapter 6.

6.0 ANALYSIS OF EXAMPLE OPTIONS FOR THE §403 STANDARDS

Chapter 6 of the §403 risk analysis presented the methodology used to characterize reductions to childhood health effect and blood-lead concentration endpoints expected to result after interventions are conducted in response to the proposed §403 rule and applied this methodology to a broad range of example options for standards. Assumptions were made on post-intervention environmental-lead levels, which were applied to those HUD National Survey housing units where a particular intervention was triggered as a result of having environmental-lead levels that exceeded an example standard. Then, the IEUBK and empirical models were used to generate the post-§403 blood-lead concentration distribution given post-§403 environmental-lead levels. These results, combined with similar model-based estimates in the pre-§403 environment presented in Chapter 5, were used to obtain a final post-§403 blood-lead distribution which was comparable to the baseline distribution generated by data from Phase 2 of NHANES III. This procedure was detailed in Chapter 6 and Appendix F1 of the §403 risk analysis report. This was the distribution upon which the health effects and blood-lead concentration endpoints were estimated in the post-§403 environment.

The risk management procedure in Chapter 6 of the §403 risk analysis report considered example standards for the following risk assessment measures:

- Average floor dust-lead loading
- Average window sill dust-lead loading
- Average soil-lead concentration
- Amount of deteriorated lead-based paint requiring paint maintenance
- Amount of deteriorated lead-based paint requiring paint abatement

Note that the lead-based paint standards considered in the risk management procedure differed somewhat from the standards proposed in the §403 rule (see Chapter 1 of this report), as the rule considered only a single tier rather than a two-tiered standard.

Section 6.1 presents additional detail and results on the performance characteristics analyses, a non-modeling data analysis procedure used by EPA to help establish levels of concern within the §403 rule. Performance characteristics analyses cited in the §403 proposed rule are detailed, and additional performance characteristics analyses performed after the proposed rule to address public comments and to finalize the rule are presented.

Section 6.2 investigates the incidence of children with elevated blood-lead concentrations in homes where no candidate standard is met or exceeded (i.e., children who would be "missed" by a specified set of candidate standards).

Since the §403 risk analysis report was published, public comment resulted in an additional investigation into the assumptions made in the risk management on average dust-lead loading following an intervention involving dust cleaning (40 $\mu g/ft^2$ on floors, 100 $\mu g/ft^2$ on

window sills). The results of this investigation are presented in Section 6.3. Based on this investigation, the impact of alternative assumptions on post-intervention dust-lead loadings on characterizing the reduction in risk as a result of implementing §403 rules was evaluated through a sensitivity analysis presented in Section 6.4. Also included in Section 6.4 are sensitivity analyses applied to baseline (pre-§403) data within Section 5.1 of this report to evaluate the impact of potential changes to the HUD National Survey data and assumptions on non-zero thresholds for the IQ/blood-lead relationship, where the analyses are implemented on data representing the post-§403 environment.

6.1 PERFORMANCE CHARACTERISTICS ANALYSES

The procedures defined and discussed in the §403 risk analysis report used statistical modeling techniques to characterize risks of lead exposure to children in the nation's housing stock and how these risks may be reduced as a result of interventions performed to reduce lead-based paint hazards in the housing stock under the §403 rule. While using the findings of this risk analysis to evaluate options for the standards specified in the §403 rule, EPA also wished to base its evaluation partially on a non-modeling approach using data from field studies that measured lead levels in both children's blood and in the same environmental media targeted by the §403 rule. In particular, given the data reported in these studies, EPA was interested in observing how often a specified set of candidate standards would "trigger" interventions in housing units within these studies and the extent to which these units contained a child with an elevated blood-lead concentration (≥ 10 µg/dL). Such an investigation provided useful information on the performance of a specified set of candidate standards without some of the complexities associated with making conclusions from statistical modeling analyses.

EPA employed *performance characteristics analysis*, sometimes referred to as *sensitivity/specificity analysis*, as a non-modeling approach to evaluating candidate §403 standards. The underlying statistical principle of this approach involves conditional probabilities and has been documented in references such as Fleiss (1981, Section 1.2). This chapter presents the findings of performance characteristics analyses applied to data from the Rochester Lead-in-Dust study. Applying data from this study was highly appropriate under the objective to evaluate candidate lead standards in the §403 rulemaking. The form of the study data used in this analysis is discussed in detail within Section 6.1.1. The methods used to perform this performance characteristics analysis are presented in Section 6.1.2. Section 6.1.3 presents the results of performance characteristics analysis presented in the preamble and which were used in the §403 rulemaking. Finally, Section 6.1.4 presents additional performance characteristics analyses performed after the §403 proposed rule was published, where these analyses considered other sets of standards (including the standards specified in the §403 proposed rule) and other means of handling data on amount of deteriorated paint within a household.

6.1.1 Data Used in The Performance Characteristics Analysis

The performance characteristics analysis was applied to data from the recently-conducted Rochester Lead-in-Dust study. A summary of objectives and design information for this study is

found in Section 3.2.2.2 of the §403 risk analysis report. The Rochester study data were selected for this analysis for the following reasons:

- The study reported information for all media for which §403 standards were proposed (e.g., dust-lead on floors and window sills, soil-lead, condition of lead-based paint).

- The study measured blood-lead concentration in 205 children aged 12-31 months who resided in the selected homes.

- The dust sampling methods used in this study included the wipe technique, from which dust-lead loadings were measured.

- For some homes, soil was sampled from multiple locations (i.e., dripline and play areas), allowing for yardwide average soil-lead concentration to be estimated.

- While homes and children were targeted for selection in this study, the selection process was more random and more representative of a general population than is the case with other lead exposure studies.

The primary concern with using data from the Rochester study in this analysis is the degree to which the study may be considered representative of the nation as a whole. The study selected a targeted sample which was limited to a single geographic area. The sample consisted of children who had moderate exposure to lead in their home environment and did not necessarily include children with very high or very low exposure to lead. In particular,

- 22.9% of the children in this study (47 children total) had blood-lead concentrations at or above 10 µg/dL, compared to the national estimate of 5.9% for children aged 1-2 years according to Phase 2 of the Third National Health and Nutrition Examination Survey (NHANES III) (CDC, 1997).

- The geometric mean blood-lead concentration for the study children was 6.38 µg/dL with a geometric standard deviation (GSD) of 1.85. This compares with a geometric mean of 3.1 µg/dL and GSD of 2.09 estimated for U.S. children aged 1-2 years according to Phase 2 of NHANES III (CDC, 1997).

- At least 84% of the housing units included in this study were built prior to 1940, compared to the estimated 20% of the entire U.S. housing stock made within the §403 risk analysis (Table 3-5 of USEPA, 1998). There is a well-documented relationship between age of housing and presence of lead-based paint hazards.

- While geometric mean floor dust-lead loadings were comparable between the Rochester study and the HUD National Survey (after converting the data to wipe-equivalent loadings), whose results are considered nationally-representative,

geometric mean estimates of window sill dust-lead loading and soil-lead concentration were higher for the Rochester study relative to HUD National Survey estimates (Section 3.2).

Despite these limitations, the Rochester study is considered one of the best resources of data for characterizing the relationship between children's blood-lead concentration and residential environmental-lead levels, and therefore, for evaluating national standards for lead in the nation's housing stock.

While data were available for 205 units in the Rochester study, somewhat fewer of these units had values for all required data endpoints for this analysis. In particular, 177 units had data reported on the amount of deteriorated lead-based paint, plus lead measurements for floor dust (wipe), window sill dust (wipe), and soil (dripline and/or play area). Of these units, 77 had soil-lead data for both dripline and play areas, thereby allowing an average concentration across these two areas to be calculated.

For the analysis presented in the §403 proposed rule, the following five data endpoints were calculated for each Rochester study housing unit:

- Area-weighted average uncarpeted floor wipe dust-lead loading (i.e., the measured loading for each sample was weighted by the area of the sample when averaged)
- Area-weighted average window sill wipe dust-lead loading
- Average of dripline and play area soil-lead concentrations
- The percentage of interior painted components tested in the study that contained lead-based paint (measurements at or above 1.0 mg/cm^2) and some level of deterioration (paint condition listed as fair or poor)
- The percentage of exterior painted components tested in the study that contained lead-based paint and some level of deterioration.

Note that these endpoints are comparable to the standards included in the §403 proposed rule, with the exception of the latter two paint-lead measurements. While the proposed §403 standard for the paint component is expressed as a square footage of deteriorated lead-based paint for components with large surface areas (2 ft^2 for interior surfaces, 10 ft^2 for exterior surfaces) or as the percentage of total painted surface area that is deteriorated for components with small surface areas (10%), no indication on the amount of deteriorated lead-based paint on a given component (either in square feet or as a percentage of the total surface area) was recorded in the Rochester study. Instead, each paint-lead measurement was associated with an indicator of the paint's condition (good, fair, poor). Therefore, for this analysis, the amount of deteriorated lead-based paint in a housing unit was taken to be the percentage of tested components in the housing unit that contained lead-based paint along with some level of deterioration (i.e., condition of paint

either fair or poor). This result was assumed to be a good estimate of the total amount of lead-based paint in the unit that was deteriorated.

6.1.2 Analysis Approach

The performance characteristics analysis classified each housing unit in the Rochester study according to two different criteria:

1. Whether or not the unit exceeded any of the candidate standards for the various media being controlled.

2. Whether or not the unit contained a child with elevated blood-lead concentration ($\geq$ 10 µg/dL).

The first criterion represented whether a housing unit was "triggered" for any intervention by exceeding at least one candidate standard, while the second represented whether the unit contained a child requiring attention as a result of having an elevated blood-lead concentration. The first criterion was determined by noting whether the value for at least one of the five endpoints mentioned at the end of the previous section exceeded the standard associated with the type of measurement represented by that endpoint.

For a given set of candidate standards, the set of housing units in the Rochester study was identified that had data for all of the above five endpoints. These units were classified according to whether or not they achieved the above two criteria. These results are summarized in the manner illustrated within the 2x2 frequency table in Table 6-1. From this information, the four performance characteristics defined in Table 6-1 were then calculated: sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV). These characteristics provide the necessary information for evaluating the sets of standards on their ability to target the proper set of units for intervention.

In this analysis, a "false positive" corresponds to triggering a housing unit for intervention when it does not contain a child with an elevated blood-lead concentration, while a "false negative" corresponds to not triggering a housing unit containing a child with an elevated blood-lead concentration. Note that the proportion of false positives is equal to one minus the specificity, while the proportion of false negatives is equal to one minus the sensitivity.

While information from all four performance characteristics are important for evaluating the performance of a given set of standards, typically one or two characteristics are given more weight than the others in the performance evaluation process. For example, in the preamble, EPA evaluated candidate standards for dust-lead loading on uncarpeted floors and window sills according to whether the performance characteristics analysis yielded a value of NPV from 95 to 99 percent under the given set of standards. This implied that no more than 5% of children living in housing units with environmental-lead levels below the standards would have elevated blood-lead concentrations (i.e., at or above 10 µg/dL). More recent Agency inquiries have focused on

Table 6-1. Definitions of Performance Characteristics Used to Evaluate How Various Combinations of Environmental-Lead Standards Classify Housing Units in the Rochester Lead-in-Dust Study

		Any of the Standards Exceeded?	
		No	Yes
Blood-Lead Concentration At or Above 10 μg/dL?	Yes	a	b
	No	c	d

In the above table, the letter 'b' represents the number of children which have a blood-lead concentration at or above 10 μg/dL who live in a residence with environmental-lead levels that exceed at least one of the specified standards. Letters 'a', 'c', and 'd' represent similar counts. The total number of housing units equals a+b+c+d. From these counts, the following performance characteristics are calculated:

Performance Characteristic	Definition	Calculation
Sensitivity (or True Positive Rate, or 1 - False Negative Rate)	Probability of a housing unit exceeding at least one standard given that there is a resident child with an elevated blood concentration (≥10 μg/dL)	b/(a+b)
Specificity (or True Negative Rate, or 1 - False Positive Rate)	Probability of a housing unit not exceeding at least one standard given that a resident child has a low blood-lead concentration (< 10 μg/dL).	c/(c+d)
Positive Predictive Value (PPV)	Probability of a resident child having an elevated blood-lead concentration (≥10 μg/dL) given that the housing unit exceeds at least one standard.	b/(b+d)
Negative Predictive Value (NPV)	Probability of a resident child having a low blood-lead concentration (< 10 μg/dL) given that the housing unit does not exceed at least one standard.	c/(a+c)

the ability of candidate standards to "trigger" housing units containing elevated blood-lead children, which corresponds to maximizing the sensitivity.

Figure 6-1 provides an example (based on hypothetical data) of an ideal situation for selecting a single standard (e.g., dripline soil-lead concentration). In this example, a dripline soil-lead concentration standard of 400 μg/g would result in all four performance characteristics achieving their maximum value of 1 (or 100%). Thus, all homes triggered for intervention (i.e., exceeding the standard) would contain a child with an elevated blood-lead concentration, and all homes containing a child with an elevated blood-lead concentration would be triggered for intervention. This situation is very unlikely to occur typically. Therefore, in a less than ideal situation (i.e., with typical data), one may wish to maximize each characteristic or some subset of the most important characteristic(s). If all four characteristics are equally important, one approach is to maximize the unweighted sum of the four characteristics. In the ideal situation represented by Figure 6-1, this sum would equal 4 (or 400%). With actual data, however, this sum will be less than 4. Figure 6-2 illustrates a situation (again, based on hypothetical data) where both the NPV and sensitivity equal 100%, but the PPV and specificity are less than 100%. This situation would be acceptable if only the NPV and sensitivity needed to be maximized.

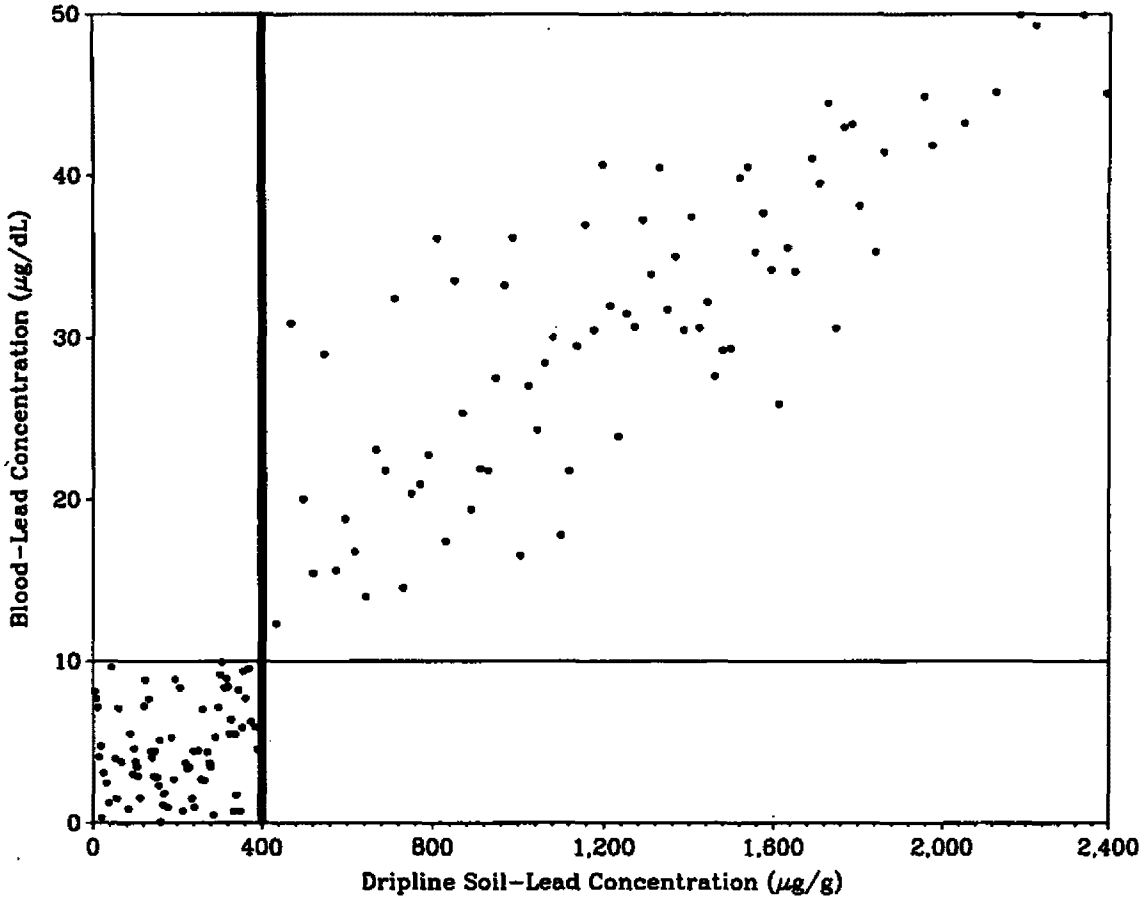

Figure 6-1. Example of an Ideal Situation for Establishing Potential Dripline Soil-Lead Standards

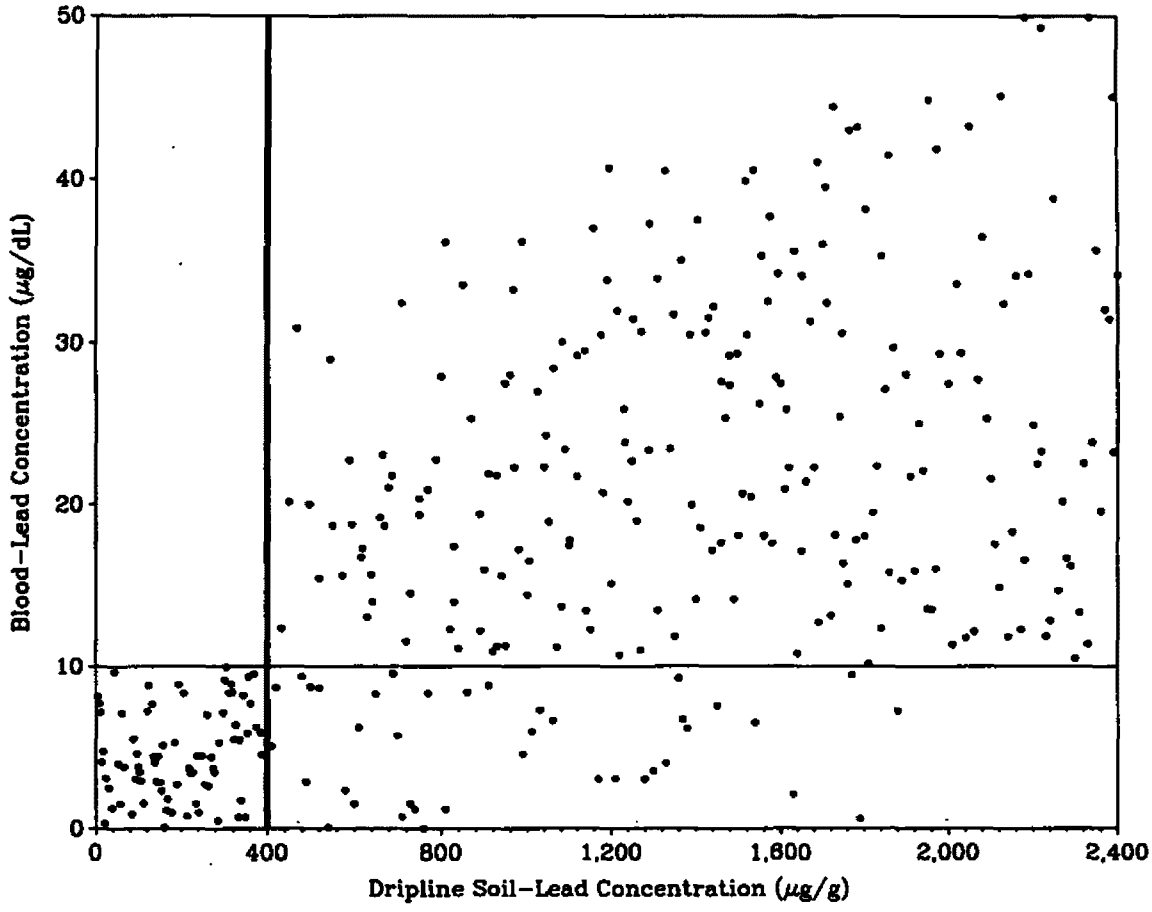

Figure 6-2. Example of a Situation Where the Negative Predictive Value and Sensitivity Equal 100%, but the Positive Predictive Value and Specificity are Less than 100%

The performance characteristics analysis was repeated for different sets of standards. For each analysis, the information within Table 6-1 was calculated, and those sets of standards that maximized the desired performance criteria were identified.

The different analyses presented in the subsequent sections of this chapter were performed on different subsets of housing units in the Rochester study. The results are purely descriptive in that they represent combinations of candidate standards that meet the specified performance criteria when considering the housing units in the Rochester study and are not based on any underlying probability model. Different results are possible if this analysis were to be applied to data from different studies. In addition, only point estimates of the performance characteristics are presented. The uncertainty in these estimates is primarily dependent on sample size, and to a lesser degree on measurement error.

6.1.3 Results Cited in the §403 Proposed Rule

The analysis presented in this section were cited in section B.1.d of Part IV of the preamble. This section of the preamble contained a brief presentation of the information presented in Section 6.1.2 above, then cited findings of analyses documented in a memorandum dated 9/3/97 from Battelle (Ronald Menton and Warren Strauss) to EPA (Todd Holderman). EPA requested that Battelle perform this analysis in an action item of a meeting between Battelle and EPA on August 27, 1997. A copy of the cited memorandum is found in Appendix G.

The analyses presented in Appendix G were performed on data for the 77 housing units in the Rochester study that had all necessary data for the analysis, including soil-lead concentrations for both dripline and play areas. As the §403 proposed rule was to contain a yardwide average soil-lead standard, it was desired to consider only those housing units that had soil-lead data for both locations. The considerable reduction in the number of Rochester study housing units whose data were considered in this analysis (from 205 to 77 units) was due primarily to the fact that play-area soil-lead concentration was measured for less than half of the study units.

The combinations of candidate standards considered in this analysis were those requested by EPA at the time, when EPA was actively considering candidate standards in the rulemaking. These combinations included all 8x4x9x3=864 combinations of the following:

- uncarpeted floor dust-lead loading: 50, 75, 100, 125, 150, 175, 200, 400 $\mu g/ft^2$
- window sill dust-lead loading: 100, 300, 500, 800 $\mu g/ft^2$
- average soil-lead concentration: 200, 300, 400, 500, 600, 700, 900, 1000, 1500 $\mu g/g$
- maximum of percent of interior/exterior painted surfaces with deteriorated lead-based paint: 5, 10, 20%

Note that the type of endpoint that represented the paint-lead measurement in this analysis (i.e., the last bullet) differed from the type of paint-lead standard that EPA ultimately proposed in the §403 proposed rule.

The purpose of this analysis was to identify those sets of candidate standards (from the 864 combinations above) which, when applying the performance characteristics analysis under those sets of standards, resulted in values of negative predictive value (as defined in Table 6-1 above) that met one of the following three criteria:

- $NPV \geq 99\%$
- $95\% \leq NPV < 99\%$
- $90\% \leq NPV < 95\%$.

The findings of this analysis are documented in Tables 1 and 2 of Appendix G.

Twenty-one of the 77 housing units whose data were included in the analysis did not exceed any of the candidate standards in at least one of the 864 combinations of candidate standards. These housing units, with the values of the endpoints used to compare to the candidate standards and children's blood-lead concentration, are listed in Table 6-2. This means that the denominator of NPV (i.e., the number of housing units that do not exceed at least one of the candidate standards being considered) never exceeded 21 across the 864 combinations. For some combinations, the denominator was as small as 2. Furthermore, all but two of the 21 units in Table 6-2 contained children with blood-lead concentrations below 10 µg/dL. As a result, the value of NPV was no lower than 84.6% across all 864 combinations of candidate standards. At least one of the above three criteria for NPV was met for 808 (93.5%) of the combinations. Of these 808 combinations, NPV equaled 100% for 690 of the combinations, equaled 95% for seven combinations, and was at least 90% but below 95% for the remaining 111 combinations.

All of the remaining 56 housing units in the analysis that are not represented in Table 6-2 exceeded either the soil-lead standard or one of the two paint standards (i.e., interior and/or exterior) in each of the 864 combinations of candidate standards. That is, each of these houses had at least one of the following:

- average soil-lead concentration of at least 1500 µg/g
- at least 20% of painted surfaces with deteriorated lead-based paint in the interior and/or exterior.

Therefore, the 56 housing units not represented in Table 6-2 were triggered in each of the 864 combinations of candidate standards, without regard to the floor or window sill standards.

The results presented in this section led to the following conclusions stated in Part IV of the preamble:

> *"For uncarpeted floors, dust-lead loadings ranged from 50 µg/ft² to 400 µg/ft² depending on the dust-lead loading on interior window sills and the soil-lead concentration. For interior window sills, dust-lead loadings ranged from 100 µg/ft² to 800 µg/ft² depending on the dust-lead loading on uncarpeted floors and the soil-lead concentration. These ranges are significantly higher than the ranges yielded by the multimedia approach."*
>
> *"Soil-lead concentrations ranged from 200 ppm to 1,500 ppm depending on dust-lead loadings on uncarpeted floors and interior window sills and the exceedance probability."*

The ranges cited in the preamble were precisely the lower and upper ranges of the candidate standards considered in this analysis. These findings reflect the very high values of the NPV across the combinations of standards considered in this analysis.

Table 6-2. Set of 21 Housing Units in the Rochester Study in Which No Standard Was Exceeded in at Least One of the 864 Combinations of Candidate Standards

Housing ID	Statistics Compared to the Candidate Standards[1]					Blood-Lead Conc. (μg/dL)
	Floor Dust-Lead Loading (μg/ft²)	Window Sill Dust-Lead Loading (μg/ft²)	Average Soil-Lead Conc. (μg/g)	% of Interior Components with Deteriorated LBP	% of Exterior Components with Deteriorated LBP	
00034	63.60	349.9	438.5	17	0	7.1
00132	17.30	90.6	268.0	0	0	6.0
00302	2.55	70.7	124.5	0	0	4.8
00637	59.00	74.9	950.0	0	0	13.3
00874	12.90	293.7	102.9	0	0	2.1
00974	14.90	45.6	51.1	0	0	8.9
01047	20.83	372.3	574.3	18	0	3.9
01062	12.40	87.1	447.5	0	0	7.4
01195	12.25	32.2	830.5	0	0	6.9
01228	3.37	16.2	419.0	0	0	4.6
01930	19.35	118.8	773.4	0	0	4.6
01971	5.10	41.9	506.0	11	0	6.1
01991	15.50	398.9	104.0	0	0	7.5
02290	2.65	74.1	465.0	11	0	4.9
02411	10.48	178.5	828.5	10	0	9.0
02837	4.29	2.8	458.5	0	0	8.9
03174	18.60	235.6	625.5	0	0	5.8
03360	12.43	702.0	912.0	0	0	11.3
03527	6.08	148.8	539.5	14	0	4.5
05343	10.30	75.7	552.0	13	0	5.6
05498	19.15	66.0	1150.5	0	0	5.8

[1] See Section 6.1.1 for the definitions of these statistics.

6.1.4 Results of Analysis on Specified Sets of Standards

The analyses presented in Section 6.1.3 were performed prior to release of the §403 proposed rule and contributed to the information presented in the preamble. Since the proposed rule was released, EPA has requested additional performance characteristics analyses be performed on various combinations of candidate standards, to address various issues raised within the public comments to the proposed rule and in support of preparing the final §403 rule. This section presents the results of these additional performance characteristics analyses. Additional performance characteristics analysis results are presented in Appendix J.

As discussed in Section 6.1.3, one of the limitations of the analyses presented in the preamble was the relatively small number of housing units (77) in the Rochester study whose data were used in the analyses. This small number was primarily due to the lack of available soil-lead concentrations from play areas and the desire to have soil-lead data for both dripline and play areas in order to calculate a yardwide average. Thus, the additional analyses presented in this section re-defined how the soil-lead measure was calculated (with different approaches taken to this re-definition), thereby increasing the number of units whose data could be included in the analysis.

6.1.4.1 Analyses Performed on 41 Combinations of Candidate Standards, in Three Iterations. The candidate standards that were considered in this analysis were the following:

- uncarpeted floor dust-lead loading: 5, 10, 20, 25, 40, 50, 100, 200 $\mu g/ft^2$
- window sill dust-lead loading: 250 $\mu g/ft^2$
- yardwide average soil-lead concentration: 400, 1200, 2000, 5000 $\mu g/g$
- amount of deteriorated lead-based paint: 2% of interior painted surfaces or 10% of exterior painted surfaces.

Thus, different candidate standards for floor dust-lead loading and soil-lead concentration were considered, while only a single candidate standard was considered for window sills (i.e., that specified in the §403 proposed rule) and deteriorated lead-based paint. This analysis considered a total of 41 combinations of candidate standards, corresponding to the 8x4=32 combinations of the above candidates, as well as the additional 9 combinations:

- only the paint standards (1 additional combination)
- only the paint and soil-lead concentration standards (4 additional combinations)
- only the paint, soil-lead concentration, and window sill dust-lead loading standards (4 additional combinations).

For each combination, the four performance characteristics were calculated and presented, as well as the number of housing units that exceed at least one of the specified standards.

Note that the above candidate paint standard (percentage of paint that is deteriorated lead-based paint) is not expressed in the manner that the proposed paint standard in the §403 proposed

rule was expressed (amount of deteriorated lead-based paint, in square feet). As discussed in Section 6.1.1 above, the Rochester study measured only lead content in paint plus an indicator of paint condition, and therefore, did not measure the surface area containing deteriorated lead-based paint. For the Rochester study data, the above paint standard triggered all units with deteriorated lead-based paint present, as the lowest observed non-zero percentage of deteriorated lead-based paint was 8% for interior surfaces and 14% for exterior surfaces.

Three iterations of this analysis was performed, with each iteration involving data for a different number of housing units:

Iteration #1: Instead of requiring soil-lead concentrations be reported for both dripline and play areas, as was done within the analysis cited in Section 6.1.3 above, average soil-lead concentration was set equal to the reported concentration at one of these areas if no concentration is reported for the other area. This approach permitted data for 177 housing units to be used in the analysis.

Iteration #2: After taking the approach in iteration #1, any units that did not have soil-lead concentration reported due to having no bare soil available from which to sample were assigned a soil-lead concentration of 0 ppm. This approach was taken as Title IV of TSCA restricts the §403 soil-lead hazard standard to bare soil and further assuming that any covered soil at these units would not pose a soil-lead hazard. This approach permitted data for 184 housing units to be used in the analysis.

Iteration #3: After taking the approach in iterations #1 and #2, the 21 remaining units having missing data for at least one endpoint had an imputed value assigned to the endpoint(s) equal to the average value across units within the same year-built category (pre-1940, 1940-1959, 1960-1979, post-1979) and having the same indicator of whether or not lead-based paint is present in the unit. This method followed the same approach taken in the §403 risk analysis (Section 3.3.1.1 of the §403 risk analysis report) to impute data for housing units in the HUD National Survey. This approach permitted data for 205 housing units to be used in the analysis.

The results of each iteration are now presented.

Iteration #1: Data for 177 Housing Units.

Table 6-3 presents the results of the performance characteristics analyses performed on data for 177 housing units (#1 above) under the 41 combinations of standards listed above. Note from this table that the fixed paint standards (which were equivalent to finding any deteriorated lead-based paint in the unit) triggered an intervention for nearly three-fourths of the 177 units. These paint standards considered jointly with a soil-lead concentration standard of 400 $\mu g/g$ resulted in 100% sensitivity and negative predictive value regardless of the dust standards. Sensitivity and negative predictive values of 100% were also met at a soil-lead concentration standard of 1200 $\mu g/g$ if the floor dust-lead loading standard was at 10 $\mu g/ft^2$ and the window sill

Table 6-3. Results of Performance Characteristics Analysis Performed on Data for 177 Units in the Rochester Lead-in-Dust Study for Specified Sets of Standards[1]

LBP = lead-based paint; EBL = elevated blood-lead level (≥ 10 μg/dL)

Set of Standards					# (%) of the 177 Housing Units That Are At or Above At Least One Standard	SENSITIVITY # (%) of the 43 Housing Units with EBL Children That Are At or Above At Least One Standard	SPECIFICITY # (%) of the 134 Housing Units with No EBL Children That Are At or Above No Standards	POSITIVE PREDICTIVE VALUE # (%) of Housing Units That Are At or Above At Least One Standard That Have EBL Children[2]	NEGATIVE PREDICTIVE VALUE # (%) of Housing Units That Are At or Above No Standards That Do Not Have EBL Children[3]	Sum of Four Performance Character-istics (%)
% of Interior Paint that is Damaged LBP	% of Exterior Paint that is Damaged LBP	Soil-Lead Conc. (ppm)	Window Sill Dust-Lead Loading (μg/ft²)	Floor Dust-Lead Loading (μg/ft²)						
2	10	--	--	--	132 (74.6%)	36 (83.7%)	38 (28.4%)	36/132 (27.3%)	38/45 (84.4%)	223.8
2	10	400	--	--	154 (87.0%)	43 (100%)	23 (17.2%)	43/154 (27.9%)	23/23 (100%)	245.1
2	10	400	250	--	156 (88.1%)	43 (100%)	21 (15.7%)	43/156 (27.6%)	21/21 (100%)	243.2
2	10	400	250	200	156 (88.1%)	43 (100%)	21 (15.7%)	43/156 (27.6%)	21/21 (100%)	243.2
2	10	400	250	100	156 (88.1%)	43 (100%)	21 (15.7%)	43/156 (27.6%)	21/21 (100%)	243.2
2	10	400	250	50	156 (88.1%)	43 (100%)	21 (15.7%)	43/156 (27.6%)	21/21 (100%)	243.2
2	10	400	250	40	156 (88.1%)	43 (100%)	21 (15.7%)	43/156 (27.6%)	21/21 (100%)	243.2
2	10	400	250	25	156 (88.1%)	43 (100%)	21 (15.7%)	43/156 (27.6%)	21/21 (100%)	243.2
2	10	400	250	20	159 (89.8%)	43 (100%)	18 (13.4%)	43/159 (27.0%)	18/18 (100%)	240.5
2	10	400	250	10	168 (94.9%)	43 (100%)	9 (6.7%)	43/168 (25.6%)	9/9 (100%)	233.3
2	10	400	250	5	173 (97.7%)	43 (100%)	4 (3.0%)	43/173 (24.9%)	4/4 (100%)	227.8
2	10	1200	--	--	137 (77.4%)	39 (90.7%)	36 (26.9%)	39/137 (28.5%)	36/40 (90.0%)	236.0
2	10	1200	250	--	141 (79.7%)	40 (93.0%)	33 (24.6%)	40/141 (28.4%)	33/36 (91.7%)	237.7
2	10	1200	250	200	141 (79.7%)	40 (93.0%)	33 (24.6%)	40/141 (28.4%)	33/36 (91.7%)	237.7
2	10	1200	250	100	141 (79.7%)	40 (93.0%)	33 (24.6%)	40/141 (28.4%)	33/36 (91.7%)	237.7

Table 6-3. (cont.)

Set of Standards					# (%) of the 177 Housing Units That Are At or Above At Least One Standard	SENSITIVITY # (%) of the 43 Housing Units with EBL Children That Are At or Above At Least One Standard	SPECIFICITY # (%) of the 134 Housing Units with No EBL Children That Are At or Above No Standards	POSITIVE PREDICTIVE VALUE # (%) of Housing Units That Are At or Above At Least One Standard That Have EBL Children[2]	NEGATIVE PREDICTIVE VALUE # (%) of Housing Units That Are At or Above No Standards That Do Not Have EBL Children[3]	Sum of Four Performance Characteristics (%)
% of Interior Paint that is Damaged LBP	% of Exterior Paint that is Damaged LBP	Soil-Lead Conc. (ppm)	Window Sill Dust-Lead Loading ($\mu g/ft^2$)	Floor Dust-Lead Loading ($\mu g/ft^2$)						
2	10	1200	250	50	142 (80.2%)	41 (95.3%)	33 (24.6%)	41/142 (28.9%)	33/35 (94.3%)	243.1
2	10	1200	250	40	143 (80.8%)	41 (95.3%)	32 (23.9%)	41/143 (28.7%)	32/34 (94.1%)	242.0
2	10	1200	250	25	143 (80.8%)	41 (95.3%)	32 (23.9%)	41/143 (28.7%)	32/34 (94.1%)	242.0
2	10	1200	250	20	147 (83.1%)	42 (97.7%)	29 (21.6%)	42/147 (28.6%)	29/30 (96.7%)	244.6
2	10	1200	250	10	165 (93.2%)	43 (100%)	12 (9.0%)	43/165 (26.1%)	12/12 (100%)	235.0
2	10	1200	250	5	171 (96.6%)	43 (100%)	6 (4.5%)	43/171 (25.1%)	6/6 (100%)	229.6
2	10	2000	--	--	135 (76.3%)	38 (88.4%)	37 (27.6%)	38/135 (28.1%)	37/42 (88.1%)	232.2
2	10	2000	250	--	140 (79.1%)	39 (90.7%)	33 (24.6%)	39/140 (27.9%)	33/37 (89.2%)	232.4
2	10	2000	250	200	140 (79.1%)	39 (90.7%)	33 (24.6%)	39/140 (27.9%)	33/37 (89.2%)	232.4
2	10	2000	250	100	140 (79.1%)	39 (90.7%)	33 (24.6%)	39/140 (27.9%)	33/37 (89.2%)	232.4
2	**10**	**2000**	**250**	**50**	**141 (79.7%)**	**40 (93.0%)**	**33 (24.6%)**	**40/141 (28.4%)**	**33/36 (91.7%)**	**237.7**
2	10	2000	250	40	142 (80.2%)	40 (93.0%)	32 (23.9%)	40/142 (28.2%)	32/35 (91.4%)	236.5
2	10	2000	250	25	142 (80.2%)	40 (93.0%)	32 (23.9%)	40/142 (28.2%)	32/35 (91.4%)	236.5
2	10	2000	250	20	147 (83.1%)	42 (97.7%)	29 (21.6%)	42/147 (28.6%)	29/30 (96.7%)	244.6
2	10	2000	250	10	165 (93.2%)	43 (100%)	12 (9.0%)	43/165 (26.1%)	12/12 (100%)	235.0
2	10	2000	250	5	171 (96.6%)	43 (100%)	6 (4.5%)	43/171 (25.1%)	6/6 (100%)	229.6

Table 6-3. (cont.)

Set of Standards					# (%) of the 177 Housing Units That Are At or Above At Least One Standard	SENSITIVITY # (%) of the 43 Housing Units with EBL Children That Are At or Above At Least One Standard	SPECIFICITY # (%) of the 134 Housing Units with No EBL Children That Are At or Above No Standards	POSITIVE PREDICTIVE VALUE # (%) of Housing Units That Are At or Above At Least One Standard That Have EBL Children[2]	NEGATIVE PREDICTIVE VALUE # (%) of Housing Units That Are At or Above No Standards That Do Not Have EBL Children[3]	Sum of Four Performance Character-istics (%)
% of Interior Paint that is Damaged LBP	% of Exterior Paint that is Damaged LBP	Soil-Lead Conc. (ppm)	Window Sill Dust-Lead Loading ($\mu g/ft^2$)	Floor Dust-Lead Loading ($\mu g/ft^2$)						
2	10	5000	--	--	133 (75.1%)	37 (86.0%)	38 (28.4%)	37/133 (27.8%)	38/44 (86.4%)	228.6
2	10	5000	250	--	138 (78.0%)	38 (88.4%)	34 (25.4%)	38/138 (27.5%)	34/39 (87.2%)	228.5
2	10	5000	250	200	138 (78.0%)	38 (88.4%)	34 (25.4%)	38/138 (27.5%)	34/39 (87.2%)	228.5
2	10	5000	250	100	138 (78.0%)	38 (88.4%)	34 (25.4%)	38/138 (27.5%)	34/39 (87.2%)	228.5
2	10	5000	250	50	139 (78.5%)	39 (90.7%)	34 (25.4%)	39/139 (28.1%)	34/38 (89.5%)	233.6
2	10	5000	250	40	140 (79.1%)	39 (90.7%)	33 (24.6%)	39/140 (27.9%)	33/37 (89.2%)	232.4
2	10	5000	250	25	140 (79.1%)	39 (90.7%)	33 (24.6%)	39/140 (27.9%)	33/37 (89.2%)	232.4
2	10	5000	250	20	145 (81.9%)	41 (95.3%)	30 (22.4%)	41/145 (28.3%)	30/32 (93.8%)	239.8
2	10	5000	250	10	164 (92.7%)	43 (100%)	13 (9.7%)	43/164 (26.2%)	13/13 (100%)	235.9
2	10	5000	250	5	171 (96.6%)	43 (100%)	6 (4.5%)	43/171 (25.1%)	6/6 (100%)	229.6

[1] Calculations are based on data from 177 of 205 units in the Rochester Lead-in-Dust study that had available data for average (wipe) floor dust-lead loading, average (wipe) window sill dust-lead loading, average soil-lead concentration (across dripline and play areas, with only one of the two areas represented if no data existed for the other area), percentage of interior lead-based paint that is deteriorated, and percentage of exterior lead-based paint that is deteriorated. Of these 177 units, 43 have children with elevated blood-lead concentrations ($\geq$ 10 $\mu g/dL$).

[2] Cell entries are as follows: (number of homes at or above at least one standard that have EBL children)/(total number of homes at or above at least one standard), followed by the corresponding percentage (in parentheses).

[3] Cell entries are as follows: (number of homes not at or above at least one standard that do not have EBL children)/(total number of homes not at or above any standard), followed by the corresponding percentage (in parentheses).

dust-lead loading was at 250 $\mu g/ft^2$, although the specificity declined considerably at these standards. When the floor dust-lead loading standard was raised to 20 $\mu g/ft^2$ in this situation, both the sensitivity and negative predictive value remained above 95%. However, at soil-lead standards of 1200 $\mu g/g$ or higher, the 95% criterion for both sensitivity and negative predictive value were no longer achieved once the floor dust-lead loading standard exceeded 20 $\mu g/ft^2$.

Among the 32 combinations of standards included in Table 6-3, the sum of the four performance characteristics (i.e., the last column of the table) was maximized at 244.6% at a floor dust-lead loading standard of 20 $\mu g/ft^2$ and a soil-lead standard of either 1200 or 2000 $\mu g/g$. (The paint and window sill standards were fixed in each combination.)

The proposed §403 standards, assuming the different approach taken in this analysis to interpreting the paint standards, resulted in a 93% sensitivity (40 of 43 units containing an elevated blood-lead child are triggered) and nearly a 92% negative predictive value (see shaded/bold row within Table 6-3). The sum of the four performance characteristics was 237.7%. Nearly 80% of the 177 units exceeded at least one of the proposed §403 standards.

Iteration #2: Data for 184 Housing Units.

Table 6-4 presents the same types of results as in Table 6-3, but it reflects analyses that included data for seven additional housing units where soil-lead concentration was assumed to be 0 $\mu g/g$ due to having no bare soil present for sampling (i.e., a total of 184 housing units). Only one of these seven additional units contained a child with an elevated blood-lead concentration.

Slight reductions in the values of the performance characteristics were seen from Table 6-3 to Table 6-4 with the addition of these seven units. The one additional unit containing a child with elevated blood-lead concentration did not exceed any of the paint, soil, or window sill standards in the table and exceeded only floor dust-lead loading standards below 50 $\mu g/ft^2$. However, as in Table 2-3, sensitivity and negative predictive values of 100% (and the considerable declines in specificity) continued to occur at a soil-lead concentration standard of 1200 $\mu g/g$ if the floor dust-lead loading standard was at 10 $\mu g/ft^2$ and the window sill dust-lead loading was at 250 $\mu g/ft^2$.

Despite the general declines in the values of the four performance characteristics from Table 6-3, the largest observed value of the sum of these characteristics among the 32 combinations of standards (245.0) was slightly larger than in Table 6-3. This value was observed for the same two combinations of standards for which the maximum occurred in Table 6-3: a floor dust-lead loading standard of 20 $\mu g/ft^2$ and a soil-lead standard of either 1200 or 2000 $\mu g/g$.

The proposed §403 standards, assuming the different approach taken in this analysis to interpreting the paint standards, resulted in nearly a 91% sensitivity and nearly a 90% negative predictive value, which were slight declines from Table 6-3 (see shaded/bold row within Table 6-4). The sum of the four performance characteristics was 233.2%.

Table 6-4. Results of Performance Characteristics Analysis Performed on Data for 184 Units in the Rochester Lead-in-Dust Study for Specified Sets of Standards[1]

LBP = lead-based paint; EBL = elevated blood-lead level (≥ 10 μg/dL)

Set of Standards						SENSITIVITY	SPECIFICITY	POSITIVE PREDICTIVE VALUE	NEGATIVE PREDICTIVE VALUE	
% of Interior Paint that is Damaged LBP	% of Exterior Paint that is Damaged LBP	Soil-Lead Conc. (ppm)	Window Sill Dust-Lead Loading (μg/ft²)	Floor Dust-Lead Loading (μg/ft²)	# (%) of the 184 Housing Units That That Are At or Above At Least One Standard	# (%) of the 44 Housing Units with EBL Children That That Are At or Above At Least One Standard	# (%) of the 140 Housing Units with No EBL Children That Are At or Above No Standards	# (%) of Housing Units That Are At or Above At Least One Standard That Have EBL Children[2]	# (%) of Housing Units That Are At or Above No Standards That Do Not Have EBL Children[3]	Sum of Four Performance Character-istics (%)
2	10	--	--	--	136 (73.9%)	36 (81.8%)	40 (28.6%)	36/136 (26.5%)	40/48 (83.3%)	220.2
2	10	400	--	--	158 (85.9%)	43 (97.7%)	25 (17.9%)	43/158 (27.2%)	25/26 (96.2%)	239.0
2	10	400	250	--	160 (87.0%)	43 (97.7%)	23 (16.4%)	43/160 (26.9%)	23/24 (95.8%)	236.9
2	10	400	250	200	160 (87.0%)	43 (97.7%)	23 (16.4%)	43/160 (26.9%)	23/24 (95.8%)	236.9
2	10	400	250	100	160 (87.0%)	43 (97.7%)	23 (16.4%)	43/160 (26.9%)	23/24 (95.8%)	236.9
2	10	400	250	50	160 (87.0%)	43 (97.7%)	23 (16.4%)	43/160 (26.9%)	23/24 (95.8%)	236.9
2	10	400	250	40	161 (87.5%)	44 (100%)	23 (16.4%)	44/161 (27.3%)	23/23 (100%)	243.8
2	10	400	250	25	161 (87.5%)	44 (100%)	23 (16.4%)	44/161 (27.3%)	23/23 (100%)	243.8
2	10	400	250	20	164 (89.1%)	44 (100%)	20 (14.3%)	44/164 (26.8%)	20/20 (100%)	241.1
2	10	400	250	10	173 (94.0%)	44 (100%)	11 (7.9%)	44/173 (25.4%)	11/11 (100%)	233.3
2	10	400	250	5	179 (97.3%)	44 (100%)	5 (3.6%)	44/179 (24.6%)	5/5 (100%)	228.2
2	10	1200	--	--	141 (76.6%)	39 (88.6%)	38 (27.1%)	39/141 (27.7%)	38/43 (88.4%)	231.8
2	10	1200	250	--	145 (78.8%)	40 (90.9%)	35 (25.0%)	40/145 (27.6%)	35/39 (89.7%)	233.2
2	10	1200	250	200	145 (78.8%)	40 (90.9%)	35 (25.0%)	40/145 (27.6%)	35/39 (89.7%)	233.2
2	10	1200	250	100	145 (78.8%)	40 (90.9%)	35 (25.0%)	40/145 (27.6%)	35/39 (89.7%)	233.2

Table 6-4. (cont.)

Set of Standards					# (%) of the 184 Housing Units That That Are At or Above At Least One Standard	SENSITIVITY # (%) of the 44 Housing Units with EBL Children That That Are At or Above At Least One Standard	SPECIFICITY # (%) of the 140 Housing Units with No EBL Children That Are At or Above No Standards	POSITIVE PREDICTIVE VALUE # (%) of Housing Units That Are At or Above At Least One Standard That Have EBL Children[2]	NEGATIVE PREDICTIVE VALUE # (%) of Housing Units That Are At or Above No Standards That Do Not Have EBL Children[3]	Sum of Four Performance Character-istics (%)
% of Interior Paint that is Damaged LBP	% of Exterior Paint that is Damaged LBP	Soil-Lead Conc. (ppm)	Window Sill Dust-Lead Loading ($\mu g/ft^2$)	Floor Dust-Lead Loading ($\mu g/ft^2$)						
2	10	1200	250	50	146 (79.3%)	41 (93.2%)	35 (25.0%)	41/146 (28.1%)	35/38 (92.1%)	238.4
2	10	1200	250	40	148 (80.4%)	42 (95.5%)	34 (24.3%)	42/148 (28.4%)	34/36 (94.4%)	242.6
2	10	1200	250	25	148 (80.4%)	42 (95.5%)	34 (24.3%)	42/148 (28.4%)	34/36 (94.4%)	242.6
2	10	1200	250	20	152 (82.6%)	43 (97.7%)	31 (22.1%)	43/152 (28.3%)	31/32 (96.9%)	245.0
2	10	1200	250	10	170 (92.4%)	44 (100%)	14 (10.0%)	44/170 (25.9%)	14/14 (100%)	235.9
2	10	1200	250	5	177 (96.2%)	44 (100%)	7 (5.0%)	44/177 (24.9%)	7/7 (100%)	229.9
2	10	2000	--	--	139 (75.5%)	38 (86.4%)	39 (27.9%)	38/139 (27.3%)	39/45 (86.7%)	228.2
2	10	2000	250	--	144 (78.3%)	39 (88.6%)	35 (25.0%)	39/144 (27.1%)	35/40 (87.5%)	228.2
2	10	2000	250	200	144 (78.3%)	39 (88.6%)	35 (25.0%)	39/144 (27.1%)	35/40 (87.5%)	228.2
2	10	2000	250	100	144 (78.3%)	39 (88.6%)	35 (25.0%)	39/144 (27.1%)	35/40 (87.5%)	228.2
2	10	2000	250	50	145 (78.8%)	40 (90.9%)	35 (25.0%)	40/145 (27.6%)	35/39 (89.7%)	233.2
2	10	2000	250	40	147 (79.9%)	41 (93.2%)	34 (24.3%)	41/147 (27.9%)	34/37 (91.9%)	237.3
2	10	2000	250	25	147 (79.9%)	41 (93.2%)	34 (24.3%)	41/147 (27.9%)	34/37 (91.9%)	237.3
2	10	2000	250	20	152 (82.6%)	43 (97.7%)	31 (22.1%)	43/152 (28.3%)	31/32 (96.9%)	245.0
2	10	2000	250	10	170 (92.4%)	44 (100%)	14 (10.0%)	44/170 (25.9%)	14/14 (100%)	235.9
2	10	2000	250	5	177 (96.2%)	44 (100%)	7 (5.0%)	44/177 (24.9%)	7/7 (100%)	229.9

Table 6-4. (cont.)

Set of Standards						SENSITIVITY	SPECIFICITY	POSITIVE PREDICTIVE VALUE	NEGATIVE PREDICTIVE VALUE	
% of Interior Paint that is Damaged LBP	% of Exterior Paint that is Damaged LBP	Soil-Lead Conc. (ppm)	Window Sill Dust-Lead Loading ($\mu g/ft^2$)	Floor Dust-Lead Loading ($\mu g/ft^2$)	# (%) of the 184 Housing Units That That Are At or Above At Least One Standard	# (%) of the 44 Housing Units with EBL Children That That Are At or Above At Least One Standard	# (%) of the 140 Housing Units with No EBL Children That Are At or Above No Standards	# (%) of Housing Units That Are At or Above At Least One Standard That Have EBL Children[2]	# (%) of Housing Units That Are At or Above No Standards That Do Not Have EBL Children[3]	Sum of Four Performance Characteristics (%)
2	10	5000	--	--	137 (74.5%)	37 (84.1%)	40 (28.6%)	37/137 (27.0%)	40/47 (85.1%)	224.8
2	10	5000	250	--	142 (77.2%)	38 (86.4%)	36 (25.7%)	38/142 (26.8%)	36/42 (85.7%)	224.6
2	10	5000	250	200	142 (77.2%)	38 (86.4%)	36 (25.7%)	38/142 (26.8%)	36/42 (85.7%)	224.6
2	10	5000	250	100	142 (77.2%)	38 (86.4%)	36 (25.7%)	38/142 (26.8%)	36/42 (85.7%)	224.6
2	10	5000	250	50	143 (77.7%)	39 (88.6%)	36 (25.7%)	39/143 (27.3%)	36/41 (87.8%)	229.4
2	10	5000	250	40	145 (78.8%)	40 (90.9%)	35 (25.0%)	40/145 (27.6%)	35/39 (89.7%)	233.2
2	10	5000	250	25	145 (78.8%)	40 (90.9%)	35 (25.0%)	40/145 (27.6%)	35/39 (89.7%)	233.2
2	10	5000	250	20	150 (81.5%)	42 (95.5%)	32 (22.9%)	42/150 (28.0%)	32/34 (94.1%)	240.4
2	10	5000	250	10	169 (91.8%)	44 (100%)	15 (10.7%)	44/169 (26.0%)	15/15 (100%)	236.7
2	10	5000	250	5	177 (96.2%)	44 (100%)	7 (5.0%)	44/177 (24.9%)	7/7 (100%)	229.9

[1] Calculations are based on data from 184 of 205 units in the Rochester Lead-in-Dust study that had available data for average (wipe) floor dust-lead loading, average (wipe) window sill dust-lead loading, average soil-lead concentration (across dripline and play areas, with only one of the two areas represented if no data existed for the other area), percentage of interior lead-based paint that is deteriorated, and percentage of exterior lead-based paint that is deteriorated. Homes having no reported soil-lead concentration but with no bare soil reported are assumed to have a soil-lead concentration of 0 ppm for these calculations. Of these 184 units, 44 have children with elevated blood-lead concentrations (≥ 10 $\mu g/dL$).

[2] Cell entries are as follows: (number of homes at or above at least one standard that have EBL children)/(total number of homes at or above at least one standard), followed by the corresponding percentage (in parentheses).

[3] Cell entries are as follows: (number of homes not at or above at least one standard that do not have EBL children)/(total number of homes not at or above any standard), followed by the corresponding percentage (in parentheses).

Iteration #3: Data for 205 Housing Units.

The third set of performance characteristics analyses was performed on data for all 205 housing units in the Rochester study. The previous analyses involved data for fewer housing units as some units did not have recorded data for the key endpoints used in the analyses to compare to the various candidate standards. Therefore, this analysis replaced incidences of missing data with data values that were imputed from information available from other study units. It was assumed that these imputed values were accurate estimates of what would have been reported for these units. This estimate for a housing unit could vary considerably from what would have been reported, however, based on actual conditions and behaviors in the household.

As all 205 housing units had reported values for child's blood-lead concentration and for the percentage of tested interior components containing deteriorated lead-based paint, no imputation was necessary for these two endpoints. The other four endpoints had at least one housing unit with missing data. For each of these four endpoints, Table 6-5 contains the number of housing units with missing data according to year-built category and whether or not the unit contains lead-based paint, along with the imputed data value assigned to these units, which equaled the average value across all units in that same category that had non-missing data. The imputed data values depended on the year-built category and lead-based paint indicator as these two variables are typically important predictors of these values. This same approach was used in the §403 risk analysis to impute environmental-lead data values for HUD National Survey units having missing data (see Section 3.3.1.1 of USEPA, 1998).

The data imputation process documented in Table 6-5 resulted in assigning imputed data to 21 units: 19 built prior to 1940, one built from 1940-1959, and one built after 1979. A total of eight average uncarpeted floor dust-lead loadings, nine average window sill dust-lead loadings, six average soil-lead concentrations, and one percentage of deteriorated lead-based paint on exterior surfaces were imputed.

Table 6-6 presents estimates of the four performance characteristics for the 41 combinations of standards, using reported and imputed data for 205 housing units in the Rochester study. These estimates are very similar to those in Table 6-4 that were calculated from data for 184 housing units. The same conclusions can be drawn from these results as were made from the results in Tables 6-3 and 6-4. This implies that at the given combinations of candidate standards considered in these analyses, the methods used in this section to estimate performance characteristics were relatively robust across the different sets of data used in the analyses (i.e., 177, 184, or 205 units).

As sensitivity and negative predictive value are the two performance characteristics of most interest to Agency reviewers, the results for these two characteristics from Tables 6-3, 6-4, and 6-6 are summarized in Table 6-7. This summary emphasizes the relative stability of the estimates across the different approaches used to make the calculations.

Table 6-5. Numbers of Housing Units with Missing Data for Four Endpoints and the Imputed Data Values Assigned to These Units in This Analysis

Year-Built Category	Lead-Based Paint Present?	Area-Weighted Average Uncarpeted Floor Dust-Lead Loading		Area-Weighted Average Window Sill Dust-Lead Loading		Average Soil-Lead Concentration		% of Exterior Components Containing Deteriorated Lead-Based Paint	
		# Units with Missing Data	Imputed Value ($\mu g/ft^2$)[1]	# Units with Missing Data	Imputed Value ($\mu g/ft^2$)[1]	# Units with Missing Data	Imputed Value ($\mu g/g$)[1]	# Units with Missing Data	Imputed Value (%)[1]
Pre-1940	Yes	6	160.2 (157)	5	633.2 (158)	5	1258 (158)	1	25.2% (162)
	No	1	13.3 (8)	3	95.2 (6)	1	631.7 (8)	0	--
1940-1959	Yes	0	--	1	569.0 (12)	0	--	0	--
	No	0	--	0	--	0	--	0	--
1960-1979	--	0	--	0	--	0	--	0	--
Post-1979	Yes	1	91.3 (3)	0	--	0	--	0	--
	No	0	--	0	--	0	--	0	--

[1] Number in parentheses equals the number of values (i.e., housing units) entering into calculation of the imputed value, which is the average of these values.

Table 6-6. Results of Performance Characteristics Analysis Performed on Data for 205 Units in the Rochester Lead-in-Dust Study for Specified Sets of Standards[1]

LBP = lead-based paint; EBL = elevated blood-lead level (≥ 10 μg/dL)

Set of Standards						SENSITIVITY	SPECIFICITY	POSITIVE PREDICTIVE VALUE	NEGATIVE PREDICTIVE VALUE	
% of Interior Paint that is Damaged LBP	% of Exterior Paint that is Damaged LBP	Soil-Lead Conc. (ppm)	Window Sill Dust-Lead Loading (μg/ft²)	Floor Dust-Lead Loading (μg/ft²)	# (%) of the 205 Housing Units That Are At or Above At Least One Standard	# (%) of the 48 Housing Units with EBL Children That Are At or Above At Least One Standard	# (%) of the 157 Housing Units with No EBL Children That Are At or Above No Standards	# (%) of Housing Units That Are At or Above At Least One Standard That Have EBL Children[2]	# (%) of Housing Units That Are At or Above No Standards That Do Not Have EBL Children[3]	Sum of Four Performance Characteristics (%)
2	10	--	--	--	151 (73.7%)	39 (81.3%)	45 (28.7%)	39/151 (25.8%)	45/54 (83.3%)	219.1
2	10	400	--	--	177 (86.3%)	47 (97.9%)	27 (17.2%)	47/177 (26.6%)	27/28 (96.4%)	238.1
2	10	400	250	--	179 (87.3%)	47 (97.9%)	25 (15.9%)	47/179 (26.3%)	25/26 (96.2%)	236.3
2	10	400	250	200	179 (87.3%)	47 (97.9%)	25 (15.9%)	47/179 (26.3%)	25/26 (96.2%)	236.3
2	10	400	250	100	179 (87.3%)	47 (97.9%)	25 (15.9%)	47/179 (26.3%)	25/26 (96.2%)	236.3
2	10	400	250	50	180 (87.8%)	47 (97.9%)	24 (15.3%)	47/180 (26.1%)	24/25 (96.0%)	235.3
2	10	400	250	40	181 (88.3%)	48 (100%)	24 (15.3%)	48/181 (26.5%)	24/24 (100%)	241.8
2	10	400	250	25	181 (88.3%)	48 (100%)	24 (15.3%)	48/181 (26.5%)	24/24 (100%)	241.8
2	10	400	250	20	184 (89.8%)	48 (100%)	21 (13.4%)	48/184 (26.1%)	21/21 (100%)	239.5
2	10	400	250	10	193 (94.1%)	48 (100%)	12 (7.6%)	48/193 (24.9%)	12/12 (100%)	232.5
2	10	400	250	5	199 (97.1%)	48 (100%)	6 (3.8%)	48/199 (24.1%)	6/6 (100%)	227.9
2	10	1200	--	--	159 (77.6%)	43 (89.6%)	41 (26.1%)	43/159 (27.0%)	41/46 (89.1%)	231.9
2	10	1200	250	--	163 (79.5%)	44 (91.7%)	38 (24.2%)	44/163 (27.0%)	38/42 (90.5%)	233.3
2	10	1200	250	200	163 (79.5%)	44 (91.7%)	38 (24.2%)	44/163 (27.0%)	38/42 (90.5%)	233.3
2	10	1200	250	100	163 (79.5%)	44 (91.7%)	38 (24.2%)	44/163 (27.0%)	38/42 (90.5%)	233.3

Table 6-6. (cont.)

Set of Standards					# (%) of the 205 Housing Units That Are At or Above At Least One Standard	SENSITIVITY	SPECIFICITY	POSITIVE PREDICTIVE VALUE	NEGATIVE PREDICTIVE VALUE	Sum of Four Performance Character-istics (%)
% of Interior Paint that is Damaged LBP	% of Exterior Paint that is Damaged LBP	Soil-Lead Conc. (ppm)	Window Sill Dust-Lead Loading ($\mu g/ft^2$)	Floor Dust-Lead Loading ($\mu g/ft^2$)		# (%) of the 48 Housing Units with EBL Children That Are At or Above At Least One Standard	# (%) of the 157 Housing Units with No EBL Children That Are At or Above No Standards	# (%) of Housing Units That Are At or Above At Least One Standard That Have EBL Children[2]	# (%) of Housing Units That Are At or Above No Standards That Do Not Have EBL Children[3]	
2	10	1200	250	50	165 (80.5%)	45 (93.8%)	37 (23.6%)	45/165 (27.3%)	37/40 (92.5%)	237.1
2	10	1200	250	40	167 (81.5%)	46 (95.8%)	36 (22.9%)	46/167 (27.5%)	36/38 (94.7%)	241.0
2	10	1200	250	25	167 (81.5%)	46 (95.8%)	36 (22.9%)	46/167 (27.5%)	36/38 (94.7%)	241.0
2	10	1200	250	20	171 (83.4%)	47 (97.9%)	33 (21.0%)	47/171 (27.5%)	33/34 (97.1%)	243.5
2	10	1200	250	10	190 (92.7%)	48 (100%)	15 (9.6%)	48/190 (25.3%)	15/15 (100%)	234.8
2	10	1200	250	5	197 (96.1%)	48 (100%)	8 (5.1%)	48/197 (24.4%)	8/8 (100%)	229.5
2	10	2000	--	--	155 (75.6%)	42 (87.5%)	44 (28.0%)	42/155 (27.1%)	44/50 (88.0%)	230.6
2	10	2000	250	--	162 (79.0%)	43 (89.6%)	38 (24.2%)	43/162 (26.5%)	38/43 (88.4%)	228.7
2	10	2000	250	200	162 (79.0%)	43 (89.6%)	38 (24.2%)	43/162 (26.5%)	38/43 (88.4%)	228.7
2	10	2000	250	100	162 (79.0%)	43 (89.6%)	38 (24.2%)	43/162 (26.5%)	38/43 (88.4%)	228.7
2	10	2000	250	50	164 (80.0%)	44 (91.7%)	37 (23.6%)	44/164 (26.8%)	37/41 (90.2%)	232.3
2	10	2000	250	40	166 (81.0%)	45 (93.8%)	36 (22.9%)	45/166 (27.1%)	36/39 (92.3%)	236.1
2	10	2000	250	25	166 (81.0%)	45 (93.8%)	36 (22.9%)	45/166 (27.1%)	36/39 (92.3%)	236.1
2	10	2000	250	20	171 (83.4%)	47 (97.9%)	33 (21.0%)	47/171 (27.5%)	33/34 (97.1%)	243.5
2	10	2000	250	10	190 (92.7%)	48 (100%)	15 (9.6%)	48/190 (25.3%)	15/15 (100%)	234.8
2	10	2000	250	5	197 (96.1%)	48 (100%)	8 (5.1%)	48/197 (24.4%)	8/8 (100%)	229.5

Table 6-6. (cont.)

Set of Standards					# (%) of the 205 Housing Units That Are At or Above At Least One Standard	SENSITIVITY # (%) of the 48 Housing Units with EBL Children That Are At or Above At Least One Standard	SPECIFICITY # (%) of the 157 Housing Units with No EBL Children That Are At or Above No Standards	POSITIVE PREDICTIVE VALUE # (%) of Housing Units That Are At or Above At Least One Standard That Have EBL Children[2]	NEGATIVE PREDICTIVE VALUE # (%) of Housing Units That Are At or Above No Standards That Do Not Have EBL Children[3]	Sum of Four Performance Characteristics (%)
% of Interior Paint that is Damaged LBP	% of Exterior Paint that is Damaged LBP	Soil-Lead Conc. (ppm)	Window Sill Dust-Lead Loading ($\mu g/ft^2$)	Floor Dust-Lead Loading ($\mu g/ft^2$)						
2	10	5000	--	--	152 (74.1%)	40 (83.3%)	45 (28.7%)	40/152 (26.3%)	45/53 (84.9%)	223.2
2	10	5000	250	--	159 (77.6%)	41 (85.4%)	39 (24.8%)	41/159 (25.8%)	39/46 (84.8%)	220.8
2	10	5000	250	200	159 (77.6%)	41 (85.4%)	39 (24.8%)	41/159 (25.8%)	39/46 (84.8%)	220.8
2	10	5000	250	100	159 (77.6%)	41 (85.4%)	39 (24.8%)	41/159 (25.8%)	39/46 (84.8%)	220.8
2	10	5000	250	50	161 (78.5%)	42 (87.5%)	38 (24.2%)	42/161 (26.1%)	38/44 (86.4%)	224.2
2	10	5000	250	40	163 (79.5%)	43 (89.6%)	37 (23.6%)	43/163 (26.4%)	37/42 (88.1%)	227.6
2	10	5000	250	25	163 (79.5%)	43 (89.6%)	37 (23.6%)	43/163 (26.4%)	37/42 (88.1%)	227.6
2	10	5000	250	20	168 (82.0%)	45 (93.8%)	34 (21.7%)	45/168 (26.8%)	34/37 (91.9%)	234.1
2	10	5000	250	10	189 (92.2%)	48 (100%)	16 (10.2%)	48/189 (25.4%)	16/16 (100%)	235.6
2	10	5000	250	5	197 (96.1%)	48 (100%)	8 (5.1%)	48/197 (24.4%)	8/8 (100%)	229.5

[1] *This analysis used the same data values used in Table 6-4, except missing values for the given endpoints were replaced by imputed numbers given in Table 6-5.* Homes having no reported soil-lead concentration but with no bare soil reported are assumed to have a soil-lead concentration of 0 ppm for these calculations. Of these 205 units, 48 have children with elevated blood-lead concentrations (≥ 10 $\mu g/dL$).

[2] Cell entries are as follows: (number of homes at or above at least one standard that have EBL children)/(total number of homes at or above at least one standard), followed by the corresponding percentage (in parentheses).

[3] Cell entries are as follows: (number of homes not at or above at least one standard that do not have EBL children)/(total number of homes not at or above any standard), followed by the corresponding percentage (in parentheses).

Table 6-7. Estimates of Sensitivity and Negative Predictive Value Presented in Tables 6-3, 6-4, and 6-6

Set of Standards					SENSITIVITY (% of Housing Units with EBL Children That Are At or Above At Least One Standard)			NEGATIVE PREDICTIVE VALUE (% of Housing Units At or Above No Standards That Do Not Have EBL Children)		
% of Interior Paint that is Damaged LBP	% of Exterior Paint that is Damaged LBP	Soil-Lead Conc. (ppm)	Window Sill Dust-Lead Loading ($\mu g/ft^2$)	Floor Dust-Lead Loading ($\mu g/ft^2$)	Data for 177 units (Table 6-3)	Data for 184 units (Table 6-4)	Data for 205 units (Table 6-6)	Data for 177 units (Table 6-3)	Data for 184 units (Table 6-4)	Data for 205 units (Table 6-6)
2	10	--	--	--	83.7%	81.8%	81.3%	84.4%	83.3%	83.3%
2	10	400	--	--	100%	97.7%	97.9%	100%	96.2%	96.4%
2	10	400	250	--	100%	97.7%	97.9%	100%	95.8%	96.2%
2	10	400	250	200	100%	97.7%	97.9%	100%	95.8%	96.2%
2	10	400	250	100	100%	97.7%	97.9%	100%	95.8%	96.2%
2	10	400	250	50	100%	97.7%	97.9%	100%	95.8%	96.0%
2	10	400	250	40	100%	100%	100%	100%	100%	100%
2	10	400	250	25	100%	100%	100%	100%	100%	100%
2	10	400	250	20	100%	100%	100%	100%	100%	100%
2	10	400	250	10	100%	100%	100%	100%	100%	100%
2	10	400	250	5	100%	100%	100%	100%	100%	100%
2	10	1200	--	--	90.7%	88.6%	89.6%	90.0%	88.4%	89.1%
2	10	1200	250	--	93.0%	90.9%	91.7%	91.7%	89.7%	90.5%
2	10	1200	250	200	93.0%	90.9%	91.7%	91.7%	89.7%	90.5%
2	10	1200	250	100	93.0%	90.9%	91.7%	91.7%	89.7%	90.5%
2	10	1200	250	50	95.3%	93.2%	93.8%	94.3%	92.1%	92.5%
2	10	1200	250	40	95.3%	95.5%	95.8%	94.1%	94.4%	94.7%
2	10	1200	250	25	95.3%	95.5%	95.8%	94.1%	94.4%	94.7%
2	10	1200	250	20	97.7%	97.7%	97.9%	96.7%	96.9%	97.1%
2	10	1200	250	10	100%	100%	100%	100%	100%	100%
2	10	1200	250	5	100%	100%	100%	100%	100%	100%
2	10	2000	--	--	88.4%	86.4%	87.5%	88.1%	86.7%	88.0%
2	10	2000	250	--	90.7%	88.6%	89.6%	89.2%	87.5%	88.4%
2	10	2000	250	200	90.7%	88.6%	89.6%	89.2%	87.5%	88.4%
2	10	2000	250	100	90.7%	88.6%	89.6%	89.2%	87.5%	88.4%
2	**10**	**2000**	**250**	**50**	**93.0%**	**90.9%**	**91.7%**	**91.7%**	**89.7%**	**90.2%**
2	10	2000	250	40	93.0%	93.2%	93.8%	91.4%	91.9%	92.3%

Table 6-7. (cont.)

Set of Standards					SENSITIVITY (% of Housing Units with EBL Children That Are At or Above At Least One Standard)			NEGATIVE PREDICTIVE VALUE (% of Housing Units At or Above No Standards That Do Not Have EBL Children)		
% of Interior Paint that is Damaged LBP	% of Exterior Paint that is Damaged LBP	Soil-Lead Conc. (ppm)	Window Sill Dust-Lead Loading ($\mu g/ft^2$)	Floor Dust-Lead Loading ($\mu g/ft^2$)	Data for 177 units (Table 6-3)	Data for 184 units (Table 6-4)	Data for 205 units (Table 6-6)	Data for 177 units (Table 6-3)	Data for 184 units (Table 6-4)	Data for 205 units (Table 6-6)
2	10	2000	250	25	93.0%	93.2%	93.8%	91.4%	91.9%	92.3%
2	10	2000	250	20	97.7%	97.7%	97.9%	96.7%	96.9%	97.1%
2	10	2000	250	10	100%	100%	100%	100%	100%	100%
2	10	2000	250	5	100%	100%	100%	100%	100%	100%
2	10	5000	–	–	86.0%	84.1%	83.3%	86.4%	85.1%	84.9%
2	10	5000	250	–	88.4%	86.4%	85.4%	87.2%	85.7%	84.8%
2	10	5000	250	200	88.4%	86.4%	85.4%	87.2%	85.7%	84.8%
2	10	5000	250	100	88.4%	86.4%	85.4%	87.2%	85.7%	84.8%
2	10	5000	250	50	90.7%	88.6%	87.5%	89.5%	87.8%	86.4%
2	10	5000	250	40	90.7%	90.9%	89.6%	89.2%	89.7%	88.1%
2	10	5000	250	25	90.7%	90.9%	89.6%	89.2%	89.7%	88.1%
2	10	5000	250	20	95.3%	95.5%	93.8%	93.8%	94.1%	91.9%
2	10	5000	250	10	100%	100%	100%	100%	100%	100%
2	10	5000	250	5	100%	100%	100%	100%	100%	100%

6.1.4.2 Considering only Soil and Dust Standards. The analysis in the previous subsection emphasized the difficulty in evaluating candidate paint standards using the Rochester data, not only due to the fact that the Rochester study did not measure total area corresponding to deteriorated lead-based paint, but also that most of the housing units with deteriorated lead-based paint exceeded the candidate standards that were considered in that analysis. In the analysis presented in this subsection, a paint standard was not considered. Instead, the performance characteristics analysis considered only candidate standards for soil-lead, floor dust-lead, and window sill dust-lead, and then investigated the percentage of painted surfaces that contained deteriorated lead-based paint for those houses that did not exceed any of these three candidate standards, in an effort to characterize the extent to which these houses would possibly exceed a paint standard. The candidate standards for dust and soil in this analysis were the same as in the previous subsection:

- uncarpeted floor dust-lead loading: 5, 10, 20, 25, 40, 50, 100, 200 $\mu g/ft^2$

- window sill dust-lead loading: 250 $\mu g/ft^2$
- yardwide average soil-lead concentration: 400, 1200, 2000, 5000 $\mu g/g$

The following 57 combinations of candidate standards were considered in this analysis:

- 8x1x4=32 combinations of the candidate floor-dust, sill-dust, and soil standards
- 4x1=4 combinations of only the candidate soil and sill-dust standards
- 1x8=8 combinations of only the candidate floor-dust and sill-dust standards
- 4 candidate soil standards without the others
- 1 sill-lead standard without the others
- 8 candidate floor-lead standards without the others.

The analysis was applied to data for housing units in the Rochester study having data that could be compared to each of the standards included in the given combinaton. Average soil-lead concentration for housing units equaled the average of the dripline and play area soil-lead measures. Units having either dripline soil-lead data or play area soil-lead data, but not both, had an average soil-lead concentration equal to the reported concentration at the area represented by the available data. An average soil-lead concentration of 0 ppm was assigned to housing units having no soil-lead data and no bare soil from which to sample.

Table 6-8 contains the results of the performance characteristics analysis, with each row of the table corresponding to one of the 57 combinations of candidate standards being considered. The following are examples of how to interpret the findings within Table 6-8:

- Consider combinations of all three standards where the candidate soil-lead standard is 400 ppm and window sill-dust standard is 250 $\mu g/ft^2$. At an uncarpeted floor-dust standard of 50 $\mu g/ft^2$, only one of the 44 homes containing children with elevated blood-lead concentration did not exceed any of these three standards and did not contain any deteriorated lead-based paint. (Two other homes with an elevated blood-lead child also do not exceed these dust or soil standards, but they do contain some deteriorated lead-based paint.) Therefore, under these standards, this particular unit would not be triggered for intervention, regardless of the paint standard, despite the unit containing a child with an elevated blood-lead concentration. However, if the uncarpeted floor-dust standard was lowered to 40 $\mu g/ft^2$, the house would exceed this lower floor standard.

- Consider the combination involving only a floor dust-lead standard of 20 $\mu g/ft^2$ and a window sill dust-lead standard of 250 $\mu g/ft^2$. A total of 106 of the 188 homes met or exceeded at least one of these two standards, including 36 of the 45 homes with elevated blood-lead children. Of the 82 homes that did not meet or exceed either dust standard, 9 contained an elevated blood-lead child, of which 2 had no deteriorated lead-based paint in either the interior or exterior. This means that if only dust and paint standards were considered, these two homes would not be triggered for any intervention, despite containing elevated blood-lead children.

Table 6-8. Results of Performance Characteristics Analysis Performed on Data for Housing Units in the Rochester Lead-in-Dust Study, for Specified Sets of Candidate Standards for Lead in Dust and Soil Only

LBP = lead-based paint (≥ 1.0 mg/cm^2); EBL = elevated blood-lead level (≥ 10 μg/dL)

"Deteriorated lead-based paint" on a tested surface implies >5% of the lead-based paint is peeling, cracking, worn, chalking, flaking, blistering, or otherwise separating from the substrate.

Set of Candidate Standards for Lead In ...[1]			# Units At or Above At Least One Standard /Total # Units[2]	Performance Characteristics				Sum of the 4 Performance Characteristics (%)	# Units with EBL Children That Are At or Above No Standard and Have No Deteriorated LBP	# Units with EBL Children That Are At or Above No Standard, Where the % of Tested Interior Paint Surfaces Having Deteriorated LBP equals[7] ...				# Units with EBL Children That Are At or Above No Standard, Where the % of Tested Exterior Paint Surfaces Having Deteriorated LBP equals[7] ...			
Soil (ppm)	Window Sill Dust (μg/ft^2)	Floor Dust (μg/ft^2)		Sensitivity # (%) of Units with EBL Children That Are At or Above At Least One Standard[3]	Specificity # (%) of Units with No EBL Children That Are At or Above No Standard[4]	PPV # (%) of Units At or Above At Least One Standard That Have EBL Children[5]	NPV # (%) of Units At or Above No Standard That Do Not Have EBL Children[6]			0%	10-30%	31-50%	>50%	0%	20-50%	51-75%	>75%
400	--	--	142/198	40/47 (85.1%)	49/151 (32.5%)	40/142 (28.2%)	49/56 (87.5%)	233.2	1	1	3	1	2	3	2	0	2
1200	--	--	55/198	22/47 (46.8%)	118/151 (78.1%)	22/55 (40.0%)	118/143 (82.5%)	247.5	5	8	9	4	4	10	8	4	3
2000	--	--	26/198	10/47 (21.3%)	135/151 (89.4%)	10/26 (38.5%)	135/172 (78.5%)	227.6	6	10	13	8	6	14	11	7	5
5000	--	--	6/198	3/47 (6.4%)	148/151 (98.0%)	3/6 (50.0%)	148/192 (77.1%)	231.5	8	12	15	10	7	19	13	7	5
--	250	--	73/195	25/45 (55.6%)	102/150 (68.0%)	25/73 (34.2%)	102/122 (83.6%)	241.4	7	10	4	4	2	12	3	2	3
--	--	200	5/196	3/47 (6.4%)	147/149 (98.7%)	3/5 (60.0%)	147/191 (77.0%)	242.0	8	12	16	9	7	20	12	7	5
--	--	100	9/196	5/47 (10.6%)	145/149 (97.3%)	5/9 (55.6%)	145/187 (77.5%)	241.0	8	12	16	9	5	20	11	7	4
--	--	50	19/196	9/47 (19.1%)	139/149 (93.3%)	9/19 (47.4%)	139/177 (78.5%)	238.3	7	11	15	7	5	19	10	5	4

Table 6-8. (cont.)

Set of Candidate Standards for Lead In ...[1]			# Units At or Above At Least One Standard /Total # Units[2]	Performance Characteristics				Sum of the 4 Performance Characteristics (%)	# Units with EBL Children That Are At or Above No Standard and Have No Deteriorated LBP	# Units with EBL Children That Are At or Above No Standard, Where the % of Tested Interior Paint Surfaces Having Deteriorated LBP equals[7] ...				# Units with EBL Children That Are At or Above No Standard, Where the % of Tested Exterior Paint Surfaces Having Deteriorated LBP equals[7] ...			
				Sensitivity # (%) of Units with EBL Children That Are At or Above At Least One Standard[3]	Specificity # (%) of Units with No EBL Children That Are At or Above No Standard[4]	PPV # (%) of Units At or Above At Least One Standard That Have EBL Children[5]	NPV # (%) of Units At or Above No Standard That Do Not Have EBL Children[6]										
Soil (ppm)	Window Sill Dust ($\mu g/ft^2$)	Floor Dust ($\mu g/ft^2$)								0%	10-30%	31-50%	>50%	0%	20-50%	51-75%	>75%
--	--	40	31/196	16/47 (34.0%)	134/149 (89.9%)	16/31 (51.6%)	134/165 (81.2%)	256.8	6	9	13	7	2	15	8	4	4
--	--	25	58/196	26/47 (55.3%)	117/149 (78.5%)	26/58 (44.8%)	117/138 (84.8%)	263.5	5	8	7	4	2	12	6	2	1
--	--	20	84/196	31/47 (66.0%)	96/149 (64.4%)	31/84 (36.9%)	96/112 (85.7%)	253.0	3	6	6	3	1	7	6	2	1
--	--	10	150/196	44/47 (93.6%)	43/149 (28.9%)	44/150 (29.3%)	43/46 (93.5%)	245.3	0	0	3	0	0	2	1	0	0
--	--	5	179/196	45/47 (95.7%)	15/149 (10.1%)	45/179 (25.1%)	15/17 (88.2%)	219.2	0	0	2	0	0	2	0	0	0
400	250	--	147/190	41/44 (93.2%)	40/146 (27.4%)	41/147 (27.9%)	40/43 (93.0%)	241.5	1	1	1	0	1	2	0	0	1
1200	250	--	93/190	33/44 (75.0%)	86/146 (58.9%)	33/93 (35.5%)	86/97 (88.7%)	258.0	4	6	1	2	2	7	1	2	1
2000	250	--	81/190	27/44 (61.4%)	92/146 (63.0%)	27/81 (33.3%)	92/109 (84.4%)	242.1	5	8	4	3	2	10	2	2	3
5000	250	--	72/190	25/44 (56.8%)	99/146 (67.8%)	25/72 (34.7%)	99/118 (83.9%)	243.2	6	9	4	4	2	11	3	2	3
--	250	200	70/188	25/45 (55.6%)	98/143 (68.5%)	25/70 (35.7%)	98/118 (83.1%)	242.9	7	10	4	4	2	12	3	2	3
--	250	100	71/188	25/45 (55.6%)	97/143 (67.8%)	25/71 (35.2%)	97/117 (82.9%)	241.5	7	10	4	4	2	12	3	2	3

Table 6-8. (cont.)

Set of Candidate Standards for Lead In ...[1]			# Units At or Above At Least One Standard /Total # Units[2]	Performance Characteristics				Sum of the 4 Performance Characteristics (%)	# Units with EBL Children That Are At or Above No Standard and Have No Deteriorated LBP	# Units with EBL Children That Are At or Above No Standard, Where the % of Tested Interior Paint Surfaces Having Deteriorated LBP equals[7] ...				# Units with EBL Children That Are At or Above No Standard, Where the % of Tested Exterior Paint Surfaces Having Deteriorated LBP equals[7] ...			
				Sensitivity	Specificity	PPV	NPV										
Soil (ppm)	Window Sill Dust ($\mu g/ft^2$)	Floor Dust ($\mu g/ft^2$)		# (%) of Units with EBL Children That Are At or Above At Least One Standard[3]	# (%) of Units with No EBL Children That Are At or Above No Standard[4]	# (%) of Units At or Above At Least One Standard That Have EBL Children[5]	# (%) of Units At or Above No Standard That Do Not Have EBL Children[6]			0%	10-30%	31-50%	>50%	0%	20-50%	51-75%	>75%
--	250	50	75/188	27/45 (60.0%)	95/143 (66.4%)	27/75 (36.0%)	95/113 (84.1%)	246.5	6	9	4	3	2	11	3	1	3
--	250	40	80/188	30/45 (66.7%)	93/143 (65.0%)	30/80 (37.5%)	93/108 (86.1%)	255.3	5	7	4	3	1	9	2	1	3
--	250	25	93/188	34/45 (75.6%)	84/143 (58.7%)	34/93 (36.6%)	84/95 (88.4%)	259.3	4	6	2	2	1	7	2	1	1
--	250	20	106/188	36/45 (80.0%)	73/143 (51.0%)	36/106 (34.0%)	73/82 (89.0%)	254.0	2	4	2	2	1	5	2	1	1
--	250	10	150/188	44/45 (97.8%)	37/143 (25.9%)	44/150 (29.3%)	37/38 (97.4%)	250.4	0	0	1	0	0	1	0	0	0
--	250	5	175/188	44/45 (97.8%)	12/143 (8.4%)	44/175 (25.1%)	12/13 (92.3%)	223.6	0	0	1	0	0	1	0	0	0
400	250	200	144/184	41/44 (93.2%)	37/140 (26.4%)	41/144 (28.5%)	37/40 (92.5%)	240.6	1	1	1	0	1	2	0	0	1
400	250	100	144/184	41/44 (93.2%)	37/140 (26.4%)	41/144 (28.5%)	37/40 (92.5%)	240.6	1	1	1	0	1	2	0	0	1
400	250	50	144/184	41/44 (93.2%)	37/140 (26.4%)	41/144 (28.5%)	37/40 (92.5%)	240.6	1	1	1	0	1	2	0	0	1
400	250	40	145/184	42/44 (95.5%)	37/140 (26.4%)	42/145 (29.0%)	37/39 (94.9%)	245.7	0	0	1	0	1	1	0	0	1
400	250	25	146/184	42/44 (95.5%)	36/140 (25.7%)	42/146 (28.8%)	36/38 (94.7%)	244.7	0	0	1	0	1	1	0	0	1

Table 6-8. (cont.)

Set of Candidate Standards for Lead In ...[1]			# Units At or Above At Least One Standard /Total # Units[2]	Performance Characteristics				Sum of the 4 Performance Characteristics (%)	# Units with EBL Children That Are At or Above No Standard and Have No Deteriorated LBP	# Units with EBL Children That Are At or Above No Standard, Where the % of Tested Interior Paint Surfaces Having Deteriorated LBP equals[7] ...				# Units with EBL Children That Are At or Above No Standard, Where the % of Tested Exterior Paint Surfaces Having Deteriorated LBP equals[7] ...			
				Sensitivity # (%) of Units with EBL Children That Are At or Above At Least One Standard[3]	Specificity # (%) of Units with No EBL Children That Are At or Above No Standard[4]	PPV # (%) of Units At or Above At Least One Standard That Have EBL Children[5]	NPV # (%) of Units At or Above No Standard That Do Not Have EBL Children[6]			0%	10-30%	31-50%	>50%	0%	20-50%	51-75%	>75%
Soil (ppm)	Window Sill Dust ($\mu g/ft^2$)	Floor Dust ($\mu g/ft^2$)															
400	250	20	153/184	42/44 (95.5%)	29/140 (20.7%)	42/153 (27.5%)	29/31 (93.5%)	237.2	0	0	1	0	1	1	0	0	1
400	250	10	169/184	43/44 (97.7%)	14/140 (10.0%)	43/169 (25.4%)	14/15 (93.3%)	226.5	0	0	1	0	0	1	0	0	0
400	250	5	177/184	43/44 (97.7%)	6/140 (4.3%)	43/177 (24.3%)	6/7 (85.7%)	212.0	0	0	1	0	0	1	0	0	0
1200	250	200	91/184	33/44 (75.0%)	82/140 (58.6%)	33/91 (36.3%)	82/93 (88.2%)	258.0	4	6	1	2	2	7	1	2	1
1200	250	100	91/184	33/44 (75.0%)	82/140 (58.6%)	33/91 (36.3%)	82/93 (88.2%)	258.0	4	6	1	2	2	7	1	2	1
1200	250	50	95/184	35/44 (79.5%)	80/140 (57.1%)	35/95 (36.8%)	80/89 (89.9%)	263.4	3	5	1	1	2	6	1	1	1
1200	250	40	100/184	38/44 (86.4%)	78/140 (55.7%)	38/100 (38.0%)	78/84 (92.9%)	272.9	2	3	1	1	1	4	0	1	1
1200	250	25	107/184	38/44 (86.4%)	71/140 (50.7%)	38/107 (35.5%)	71/77 (92.2%)	264.8	2	3	1	1	1	4	0	1	1
1200	250	20	118/184	39/44 (88.6%)	61/140 (43.6%)	39/118 (33.1%)	61/66 (92.4%)	257.7	1	2	1	1	1	3	0	1	1
1200	250	10	155/184	43/44 (97.7%)	28/140 (20.0%)	43/155 (27.7%)	28/29 (96.6%)	242.0	0	0	1	0	0	1	0	0	0
1200	250	5	173/184	43/44 (97.7%)	10/140 (7.1%)	43/173 (24.9%)	10/11 (90.9%)	220.6	0	0	1	0	0	1	0	0	0

Table 6-8. (cont.)

Set of Candidate Standards for Lead in ...[1]			# Units At or Above At Least One Standard /Total # Units[2]	Performance Characteristics				Sum of the 4 Performance Characteristics (%)	# Units with EBL Children That Are At or Above No Standard and Have No Deteriorated LBP	# Units with EBL Children That Are At or Above No Standard, Where the % of Tested Interior Paint Surfaces Having Deteriorated LBP equals[7] ...				# Units with EBL Children That Are At or Above No Standard, Where the % of Tested Exterior Paint Surfaces Having Deteriorated LBP equals[7] ...			
				Sensitivity	Specificity	PPV	NPV										
Soil (ppm)	Window Sill Dust ($\mu g/ft^2$)	Floor Dust ($\mu g/ft^2$)		# (%) of Units with EBL Children That Are At or Above At Least One Standard[3]	# (%) of Units with No EBL Children That Are At or Above No Standard[4]	# (%) of Units At or Above At Least One Standard That Have EBL Children[5]	# (%) of Units At or Above No Standard That Do Not Have EBL Children[6]			0%	10-30%	31-50%	>50%	0%	20-50%	51-75%	>75%
2000	250	200	79/184	27/44 (61.4%)	88/140 (62.9%)	27/79 (34.2%)	88/105 (83.8%)	242.2	5	8	4	3	2	10	2	2	3
2000	250	100	79/184	27/44 (61.4%)	88/140 (62.9%)	27/79 (34.2%)	88/105 (83.8%)	242.2	5	8	4	3	2	10	2	2	3
2000	250	50	83/184	29/44 (65.9%)	86/140 (61.4%)	29/83 (34.9%)	86/101 (85.1%)	247.4	4	7	4	2	2	9	2	1	3
2000	250	40	88/184	32/44 (72.7%)	84/140 (60.0%)	32/88 (36.4%)	84/96 (87.5%)	256.6	3	5	4	2	1	7	1	1	3
2000	250	25	99/184	35/44 (79.5%)	76/140 (54.3%)	35/99 (35.4%)	76/85 (89.4%)	258.6	3	5	2	1	1	6	1	1	1
2000	250	20	112/184	37/44 (84.1%)	65/140 (46.4%)	37/112 (33.0%)	65/72 (90.3%)	253.8	1	3	2	1	1	4	1	1	1
2000	250	10	152/184	43/44 (97.7%)	31/140 (22.1%)	43/152 (28.3%)	31/32 (96.9%)	245.0	0	0	1	0	0	1	0	0	0
2000	250	5	172/184	43/44 (97.7%)	11/140 (7.9%)	43/172 (25.0%)	11/12 (91.7%)	222.3	0	0	1	0	0	1	0	0	0
5000	250	200	70/184	25/44 (56.8%)	95/140 (67.9%)	25/70 (35.7%)	95/114 (83.3%)	243.7	6	9	4	4	2	11	3	2	3
5000	250	100	71/184	25/44 (56.8%)	94/140 (67.1%)	25/71 (35.2%)	94/113 (83.2%)	242.4	6	9	4	4	2	11	3	2	3
5000	250	50	75/184	27/44 (61.4%)	92/140 (65.7%)	27/75 (36.0%)	92/109 (84.4%)	247.5	5	8	4	3	2	10	3	1	3

Table 6-8. (cont.)

Set of Candidate Standards for Lead in ...[1]			# Units At or Above At Least One Standard /Total # Units[2]	Performance Characteristics				Sum of the 4 Performance Characteristics (%)	# Units with EBL Children That Are At or Above No Standard and Have No Deteriorated LBP	# Units with EBL Children That Are At or Above No Standard, Where the % of Tested Interior Paint Surfaces Having Deteriorated LBP equals[7] ...				# Units with EBL Children That Are At or Above No Standard, Where the % of Tested Exterior Paint Surfaces Having Deteriorated LBP equals[7] ...			
				Sensitivity	Specificity	PPV	NPV										
Soil (ppm)	Window Sill Dust ($\mu g/ft^2$)	Floor Dust ($\mu g/ft^2$)		# (%) of Units with EBL Children That Are At or Above At Least One Standard[3]	# (%) of Units with No EBL Children That Are At or Above No Standard[4]	# (%) of Units At or Above At Least One Standard That Have EBL Children[5]	# (%) of Units At or Above No Standard That Do Not Have EBL Children[6]			0%	10-30%	31-50%	>50%	0%	20-50%	51-75%	>75%
5000	250	40	80/184	30/44 (68.2%)	90/140 (64.3%)	30/80 (37.5%)	90/104 (86.5%)	256.5	4	6	4	3	1	8	2	1	3
5000	250	25	92/184	33/44 (75.0%)	81/140 (57.9%)	33/92 (35.9%)	81/92 (88.0%)	256.8	4	6	2	2	1	7	2	1	1
5000	250	20	105/184	35/44 (79.5%)	70/140 (50.0%)	35/105 (33.3%)	70/79 (88.6%)	251.5	2	4	2	2	1	5	2	1	1
5000	250	10	148/184	43/44 (97.7%)	35/140 (25.0%)	43/148 (29.1%)	35/36 (97.2%)	249.0	0	0	1	0	0	1	0	0	0
5000	250	5	172/184	43/44 (97.7%)	11/140 (7.9%)	43/172 (25.0%)	11/12 (91.7%)	222.3	0	0	1	0	0	1	0	0	0

[1] The data compared to these standards are average (wipe) floor dust-lead loading, average (wipe) window sill dust-lead loading, and average soil-lead concentration (across dripline and play areas, with only one of the two areas represented if no data existed for the other area). Units having no reported soil-lead concentration but with no bare soil reported were assumed to have a soil-lead concentration of 0 ppm.

[2] Total number of units having available data that could be compared to all specified candidate standards, as well as data on the percentage of tested interior lead-based paint that is deteriorated and the percentage of tested exterior lead-based paint that is deteriorated.

[3] Cell entries are(number of homes at or above at least one standard that have EBL children)/ number of homes containing EBL children), followed by the corresponding percentage (in parentheses).

[4] Cell entries are (number of homes not at or above at least one standard that do not have EBL children)/(total number of homes not containing EBL children), followed by the corresponding percentage (in parentheses).

[5] Cell entries are (number of homes at or above at least one standard that have EBL children)/(total number of homes at or above at least one standard), followed by the corresponding percentage (in parentheses).

[6] Cell entries are (number of homes not at or above at least one standard that do not have EBL children)/(total number of homes not at or above any standard), followed by the corresponding percentage (in parentheses).

[7] No housing units had between 0 and 10% deteriorated lead-based paint on interior tested surfaces or between or between 0 and 20% deteriorated lead-based paint on exterior tested surfaces.

6.1.4.3 Analysis Involving Only Dust-Lead Standards and a Standard on the Amount of Deteriorated Paint. In some cases, a risk assessment may involve only dust sampling (of floors and window sills) and a visual inspection of painted surfaces for deterioration. That is, no testing of painted surface for lead within the paint would be done, and no soil sampling would be done. In this setting, it was of interest to investigate the extent to which candidate dust-lead loading standards, with standards on the maximum percentage of surfaces with deteriorated paint, performed in the absence of soil standards, within a performance characteristics analysis. The combinations of standards considered in this analysis were the following:

- uncarpeted floor dust-lead loading: 5, 10, 20, 25, 40, 50, 100 $\mu g/ft^2$
- window sill dust-lead loading: 125, 250 $\mu g/ft^2$
- maximum amount of deteriorated paint on a tested surface: >5%, >15%.

The candidate paint standards were defined to coincide with the type of paint condition measurement made in the Rochester study. The following 63 combinations of these candidate standards were considered in this analysis:

- 7x2x2=28 combinations of the candidate floor-dust, sill-dust, and paint standards
- 7x2=14 combinations of only the candidate floor-dust and sill-dust standards
- 7x2=14 combinations of only the candidate floor-dust and paint standards
- 7 candidate floor-lead standards without the others.

Table 6-9 contains the results of the performance characteristics analysis, with each row of the table corresponding to one of the 63 combinations of candidate standards being considered. The following are examples of what can be concluded from Table 6-9:

- While, on their own, the higher candidate floor dust-lead standards trigger few units containing elevated blood-lead children, the number of these homes that are triggered with the addition of a deteriorated paint standard increases dramatically (e.g., from 10.6% to 70.2% at a floor dust-lead standard of 100 $\mu g/ft^2$, if the 15% paint standard is added).

- The performance characteristics do not appear to increase substantially with an increase in the sill standard from 125 to 250 $\mu g/ft^2$.

If the risk assessment does, in fact, do paint testing for lead, then the above standard for paint can be re-defined to represent the maximum amount of deteriorated lead-based paint on a tested surface. Table 6-10 contains the results of the performance characteristics analysis where the paint standard is modified in this manner.

Table 6-9. Results of Performance Characteristics Analysis Performed on Data for Housing Units in the Rochester Lead-in-Dust Study, for Specified Sets of Candidate Standards for Dust-Lead Loadings and Observed Amount of Damaged Paint on a Tested Surface

EBL = elevated blood-lead level (≥ 10 μg/dL)

Set of Candidate Standards			# Units At Or Above At Least One Standard / Total # Units[2]	Performance Characteristics			
Uncarpeted Floor Dust-Lead Loading (μg/ft²)	Window Sill Dust-Lead Loading (μg/ft²)	Max. Amt. Of Damaged Paint on a Tested Surface (%)[1]		Sensitivity # (%) of Units with EBL Children That Are At or Above At Least One Standard[3]	Specificity # (%) of Units with No EBL Children That Are At or Above No Standard[4]	PPV # (%) of Units At or Above At Least One Standard That Have EBL Children[5]	NPV # (%) of Units At or Above No Standard That Do Not Have EBL Children[6]
100	–	–	9/197	5/47 (10.6%)	146/150 (97.3%)	5/9 (55.6%)	146/188 (77.7%)
50	–	–	19/197	9/47 (19.1%)	140/150 (93.3%)	9/19 (47.4%)	140/178 (78.7%)
40	–	–	31/197	16/47 (34.0%)	135/150 (90.0%)	16/31 (51.6%)	135/166 (81.3%)
25	–	–	58/197	26/47 (55.3%)	118/150 (78.7%)	26/58 (44.8%)	118/139 (84.9%)
20	–	–	84/197	31/47 (66.0%)	97/150 (64.7%)	31/84 (36.9%)	97/113 (85.8%)
10	–	–	150/197	44/47 (93.6%)	44/150 (29.3%)	44/150 (29.3%)	44/47 (93.6%)
5	–	–	180/197	45/47 (95.7%)	15/150 (10.0%)	45/180 (25.0%)	15/17 (88.2%)
100	250	–	71/189	25/45 (55.6%)	98/144 (68.1%)	25/71 (35.2%)	98/118 (83.1%)
50	250	–	75/189	27/45 (60.0%)	96/144 (66.7%)	27/75 (36.0%)	96/114 (84.2%)
40	250	–	80/189	30/45 (66.7%)	94/144 (65.3%)	30/80 (37.5%)	94/109 (86.2%)
25	250	–	93/189	34/45 (75.6%)	85/144 (59.0%)	34/93 (36.6%)	85/96 (88.5%)
20	250	–	106/189	36/45 (80.0%)	74/144 (51.4%)	36/106 (34.0%)	74/83 (89.2%)
10	250	–	150/189	44/45 (97.8%)	38/144 (26.4%)	44/150 (29.3%)	38/39 (97.4%)
5	250	–	176/189	44/45 (97.8%)	12/144 (8.3%)	44/176 (25.0%)	12/13 (92.3%)
100	125	–	116/189	35/45 (77.8%)	63/144 (43.8%)	35/116 (30.2%)	63/73 (86.3%)
50	125	–	118/189	36/45 (80.0%)	62/144 (43.1%)	36/118 (30.5%)	62/71 (87.3%)
40	125	–	122/189	38/45 (84.4%)	60/144 (41.7%)	38/122 (31.1%)	60/67 (89.6%)
25	125	–	128/189	39/45 (86.7%)	55/144 (38.2%)	39/128 (30.5%)	55/61 (90.2%)
20	125	–	134/189	40/45 (88.9%)	50/144 (34.7%)	40/134 (29.9%)	50/55 (90.9%)
10	125	–	159/189	45/45 (100%)	30/144 (20.8%)	45/159 (28.3%)	30/30 (100%)
5	125	–	180/189	45/45 (100%)	9/144 (6.3%)	45/180 (25.0%)	9/9 (100%)
100	–	>15%	101/197	33/47 (70.2%)	82/150 (54.7%)	33/101 (32.7%)	82/96 (85.4%)
50	–	>15%	105/197	34/47 (72.3%)	79/150 (52.7%)	34/105 (32.4%)	79/92 (85.9%)
40	–	>15%	108/197	35/47 (74.5%)	77/150 (51.3%)	35/108 (32.4%)	77/89 (86.5%)
25	–	>15%	116/197	35/47 (74.5%)	69/150 (46.0%)	35/116 (30.2%)	69/81 (85.2%)
20	–	>15%	127/197	38/47 (80.9%)	61/150 (40.7%)	38/127 (29.9%)	61/70 (87.1%)
10	–	>15%	170/197	46/47 (97.9%)	26/150 (17.3%)	46/170 (27.1%)	26/27 (96.3%)

Table 6-9. (cont.)

Set of Candidate Standards			# Units At Or Above At Least One Standard / Total # Units[2]	Performance Characteristics			
Uncarpeted Floor Dust-Lead Loading ($\mu g/ft^2$)	Window Sill Dust-Lead Loading ($\mu g/ft^2$)	Max. Amt. Of Damaged Paint on a Tested Surface (%)[1]		Sensitivity # (%) of Units with EBL Children That Are At or Above At Least One Standard[3]	Specificity # (%) of Units with No EBL Children That Are At or Above No Standard[4]	PPV # (%) of Units At or Above At Least One Standard That Have EBL Children[5]	NPV # (%) of Units At or Above No Standard That Do Not Have EBL Children[6]
5	--	>15%	188/197	47/47 (100%)	9/150 (6.0%)	47/188 (25.0%)	9/9 (100%)
100	--	>5%	164/197	43/47 (91.5%)	29/150 (19.3%)	43/164 (26.2%)	29/33 (87.9%)
50	--	>5%	165/197	44/47 (93.6%)	29/150 (19.3%)	44/165 (26.7%)	29/32 (90.6%)
40	--	>5%	167/197	45/47 (95.7%)	28/150 (18.7%)	45/167 (26.9%)	28/30 (93.3%)
25	--	>5%	167/197	45/47 (95.7%)	28/150 (18.7%)	45/167 (26.9%)	28/30 (93.3%)
20	--	>5%	168/197	45/47 (95.7%)	27/150 (18.0%)	45/168 (26.8%)	27/29 (93.1%)
10	--	>5%	185/197	47/47 (100%)	12/150 (8.0%)	47/185 (25.4%)	12/12 (100%)
5	--	>5%	192/197	47/47 (100%)	5/150 (3.3%)	47/192 (24.5%)	5/5 (100%)
100	250	>15%	118/189	36/45 (80.0%)	62/144 (43.1%)	36/118 (30.5%)	62/71 (87.3%)
50	250	>15%	120/189	37/45 (82.2%)	61/144 (42.4%)	37/120 (30.8%)	61/69 (88.4%)
40	250	>15%	123/189	38/45 (84.4%)	59/144 (41.0%)	38/123 (30.9%)	59/66 (89.4%)
25	250	>15%	127/189	38/45 (84.4%)	55/144 (38.2%)	38/127 (29.9%)	55/62 (88.7%)
20	250	>15%	134/189	40/45 (88.9%)	50/144 (34.7%)	40/134 (29.9%)	50/55 (90.9%)
10	250	>15%	167/189	45/45 (100%)	22/144 (15.3%)	45/167 (26.9%)	22/22 (100%)
5	250	>15%	182/189	45/45 (100%)	7/144 (4.9%)	45/182 (24.7%)	7/7 (100%)
100	125	>15%	142/189	39/45 (86.7%)	41/144 (28.5%)	39/142 (27.5%)	41/47 (87.2%)
50	125	>15%	144/189	40/45 (88.9%)	40/144 (27.8%)	40/144 (27.8%)	40/45 (88.9%)
40	125	>15%	147/189	41/45 (91.1%)	38/144 (26.4%)	41/147 (27.9%)	38/42 (90.5%)
25	125	>15%	149/189	41/45 (91.1%)	36/144 (25.0%)	41/149 (27.5%)	36/40 (90.0%)
20	125	>15%	152/189	42/45 (93.3%)	34/144 (23.6%)	42/152 (27.6%)	34/37 (91.9%)
10	125	>15%	171/189	45/45 (100%)	18/144 (12.5%)	45/171 (26.3%)	18/18 (100%)
5	125	>15%	182/189	45/45 (100%)	7/144 (4.9%)	45/182 (24.7%)	7/7 (100%)
100	250	>5%	162/189	41/45 (91.1%)	23/144 (16.0%)	41/162 (25.3%)	23/27 (85.2%)
50	250	>5%	163/189	42/45 (93.3%)	23/144 (16.0%)	42/163 (25.8%)	23/26 (88.5%)
40	250	>5%	165/189	43/45 (95.6%)	22/144 (15.3%)	43/165 (26.1%)	22/24 (91.7%)
25	250	>5%	165/189	43/45 (95.6%)	22/144 (15.3%)	43/165 (26.1%)	22/24 (91.7%)
20	250	>5%	166/189	43/45 (95.6%)	21/144 (14.6%)	43/166 (25.9%)	21/23 (91.3%)
10	250	>5%	179/189	45/45 (100%)	10/144 (6.9%)	45/179 (25.1%)	10/10 (100%)
5	250	>5%	185/189	45/45 (100%)	4/144 (2.8%)	45/185 (24.3%)	4/4 (100%)

Table 6-9. (cont.)

Set of Candidate Standards			# Units At Or Above At Least One Standard / Total # Units[2]	Performance Characteristics			
Uncarpeted Floor Dust-Lead Loading ($\mu g/ft^2$)	Window Sill Dust-Lead Loading ($\mu g/ft^2$)	Max. Amt. Of Damaged Paint on a Tested Surface (%)[1]		Sensitivity # (%) of Units with EBL Children That Are At or Above At Least One Standard[3]	Specificity # (%) of Units with No EBL Children That Are At or Above No Standard[4]	PPV # (%) of Units At or Above At Least One Standard That Have EBL Children[5]	NPV # (%) of Units At or Above No Standard That Do Not Have EBL Children[6]
100	125	>5%	166/189	42/45 (93.3%)	20/144 (13.9%)	42/166 (25.3%)	20/23 (87.0%)
50	125	>5%	167/189	43/45 (95.6%)	20/144 (13.9%)	43/167 (25.7%)	20/22 (90.9%)
40	125	>5%	169/189	44/45 (97.8%)	19/144 (13.2%)	44/169 (26.0%)	19/20 (95.0%)
25	125	>5%	169/189	44/45 (97.8%)	19/144 (13.2%)	44/169 (26.0%)	19/20 (95.0%)
20	125	>5%	170/189	44/45 (97.8%)	18/144 (12.5%)	44/170 (25.9%)	18/19 (94.7%)
10	125	>5%	179/189	45/45 (100%)	10/144 (6.9%)	45/179 (25.1%)	10/10 (100%)
5	125	>5%	185/189	45/45 (100%)	4/144 (2.8%)	45/185 (24.3%)	4/4 (100%)

[1] In the Rochester study, each measurement of lead in paint had the amount of damaged paint specified as "<5%" (good condition), "5-15%" (fair condition), or ">15%" (poor condition) of the tested surface, with no indication of total damaged surface area.
[2] Total number of units having available data that could be compared to all specified candidate standards.
[3] Cell entries are(number of homes at or above at least one standard that have EBL children)/ number of homes containing EBL children), followed by the corresponding percentage (in parentheses).
[4] Cell entries are (number of homes not at or above at least one standard that do not have EBL children)/(total number of homes not containing EBL children), followed by the corresponding percentage (in parentheses).
[5] Cell entries are (number of homes at or above at least one standard that have EBL children)/(total number of homes at or above at least one standard), followed by the corresponding percentage (in parentheses).
[6] Cell entries are (number of homes not at or above at least one standard that do not have EBL children)/(total number of homes not at or above any standard), followed by the corresponding percentage (in parentheses).

Table 6-10. Results of Performance Characteristics Analysis Performed on Data for Housing Units in the Rochester Lead-in-Dust Study, for Specified Sets of Candidate Standards for Dust-Lead Loadings and Observed Amount of Damaged Lead-Based Paint on a Tested Surface

EBL = elevated blood-lead level (≥10 μg/dL); LBP = Lead-Based Paint

Set of Candidate Standards			# Units At Or Above At Least One Standard / Total # Units[2]	Performance Characteristics			
Uncarpeted Floor Dust-Lead Loading (μg/ft²)	Window Sill Dust-Lead Loading (μg/ft²)	Max. Amt. Of Damaged LBP on a Tested Surface (%)[1]		Sensitivity # (%) of Units with EBL Children That Are At or Above At Least One Standard[3]	Specificity # (%) of Units with No EBL Children That Are At or Above No Standard[4]	PPV # (%) of Units At or Above At Least One Standard That Have EBL Children[5]	NPV # (%) of Units At or Above No Standard That Do Not Have EBL Children[6]
100	--	--	9/197	5/47 (10.6%)	146/150 (97.3%)	5/9 (55.6%)	146/188 (77.7%)
50	--	--	19/197	9/47 (19.1%)	140/150 (93.3%)	9/19 (47.4%)	140/178 (78.7%)
40	--	--	31/197	16/47 (34.0%)	135/150 (90.0%)	16/31 (51.6%)	135/166 (81.3%)
25	--	--	58/197	26/47 (55.3%)	118/150 (78.7%)	26/58 (44.8%)	118/139 (84.9%)
20	--	--	84/197	31/47 (66.0%)	97/150 (64.7%)	31/84 (36.9%)	97/113 (85.8%)
10	--	--	150/197	44/47 (93.6%)	44/150 (29.3%)	44/150 (29.3%)	44/47 (93.6%)
5	--	--	180/197	45/47 (95.7%)	15/150 (10.0%)	45/180 (25.0%)	15/17 (88.2%)
100	250	--	71/189	25/45 (55.6%)	98/144 (68.1%)	25/71 (35.2%)	98/118 (83.1%)
50	250	--	75/189	27/45 (60.0%)	96/144 (66.7%)	27/75 (36.0%)	96/114 (84.2%)
40	250	--	80/189	30/45 (66.7%)	94/144 (65.3%)	30/80 (37.5%)	94/109 (86.2%)
25	250	--	93/189	34/45 (75.6%)	85/144 (59.0%)	34/93 (36.6%)	85/96 (88.5%)
20	250	--	106/189	36/45 (80.0%)	74/144 (51.4%)	36/106 (34.0%)	74/83 (89.2%)
10	250	--	150/189	44/45 (97.8%)	38/144 (26.4%)	44/150 (29.3%)	38/39 (97.4%)
5	250	--	176/189	44/45 (97.8%)	12/144 (8.3%)	44/176 (25.0%)	12/13 (92.3%)
100	125	--	116/189	35/45 (77.8%)	63/144 (43.8%)	35/116 (30.2%)	63/73 (86.3%)
50	125	--	118/189	36/45 (80.0%)	62/144 (43.1%)	36/118 (30.5%)	62/71 (87.3%)
40	125	--	122/189	38/45 (84.4%)	60/144 (41.7%)	38/122 (31.1%)	60/67 (89.6%)
25	125	--	128/189	39/45 (86.7%)	55/144 (38.2%)	39/128 (30.5%)	55/61 (90.2%)
20	125	--	134/189	40/45 (88.9%)	50/144 (34.7%)	40/134 (29.9%)	50/55 (90.9%)
10	125	--	159/189	45/45 (100%)	30/144 (20.8%)	45/159 (28.3%)	30/30 (100%)
5	125	--	180/189	45/45 (100%)	9/144 (6.3%)	45/180 (25.0%)	9/9 (100%)
100	--	>15%	84/197	27/47 (57.4%)	93/150 (62.0%)	27/84 (32.1%)	93/113 (82.3%)
50	--	>15%	88/197	28/47 (59.6%)	90/150 (60.0%)	28/88 (31.8%)	90/109 (82.6%)
40	--	>15%	94/197	31/47 (66.0%)	87/150 (58.0%)	31/94 (33.0%)	87/103 (84.5%)
25	--	>15%	104/197	33/47 (70.2%)	79/150 (52.7%)	33/104 (31.7%)	79/93 (84.9%)
20	--	>15%	115/197	36/47 (76.6%)	71/150 (47.3%)	36/115 (31.3%)	71/82 (86.6%)
10	--	>15%	162/197	44/47 (93.6%)	32/150 (21.3%)	44/162 (27.2%)	32/35 (91.4%)

Table 6-10. (cont.)

Set of Candidate Standards			# Units At Or Above At Least One Standard / Total # Units[2]	Performance Characteristics			
Uncarpeted Floor Dust-Lead Loading ($\mu g/ft^2$)	Window Sill Dust-Lead Loading ($\mu g/ft^2$)	Max. Amt. Of Damaged LBP on a Tested Surface (%)[1]		Sensitivity # (%) of Units with EBL Children That Are At or Above At Least One Standard[3]	Specificity # (%) of Units with No EBL Children That Are At or Above No Standard[4]	PPV # (%) of Units At or Above At Least One Standard That Have EBL Children[5]	NPV # (%) of Units At or Above No Standard That Do Not Have EBL Children[6]
5	--	>15%	183/197	45/47 (95.7%)	12/150 (8.0%)	45/183 (24.6%)	12/14 (85.7%)
100	--	>5%	146/197	39/47 (83.0%)	43/150 (28.7%)	39/146 (26.7%)	43/51 (84.3%)
50	--	>5%	147/197	40/47 (85.1%)	43/150 (28.7%)	40/147 (27.2%)	43/50 (86.0%)
40	--	>5%	149/197	41/47 (87.2%)	42/150 (28.0%)	41/149 (27.5%)	42/48 (87.5%)
25	--	>5%	150/197	42/47 (89.4%)	42/150 (28.0%)	42/150 (28.0%)	42/47 (89.4%)
20	--	>5%	155/197	44/47 (93.6%)	39/150 (26.0%)	44/155 (28.4%)	39/42 (92.9%)
10	--	>5%	181/197	47/47 (100%)	16/150 (10.7%)	47/181 (26.0%)	16/16 (100%)
5	--	>5%	189/197	47/47 (100%)	8/150 (5.3%)	47/189 (24.9%)	8/8 (100%)
100	250	>15%	107/189	32/45 (71.1%)	69/144 (47.9%)	32/107.(29.9%)	69/82 (84.1%)
50	250	>15%	109/189	33/45 (73.3%)	68/144 (47.2%)	33/109 (30.3%)	68/80 (85.0%)
40	250	>15%	114/189	36/45 (80.0%)	66/144 (45.8%)	36/114 (31.6%)	66/75 (88.0%)
25	250	>15%	119/189	37/45 (82.2%)	62/144 (43.1%)	37/119 (31.1%)	62/70 (88.6%)
20	250	>15%	126/189	39/45 (86.7%)	57/144 (39.6%)	39/126 (31.0%)	57/63 (90.5%)
10	250	>15%	160/189	44/45 (97.8%)	28/144 (19.4%)	44/160 (27.5%)	28/29 (96.6%)
5	250	>15%	178/189	44/45 (97.8%)	10/144 (6.9%)	44/178 (24.7%)	10/11 (90.9%)
100	125	>15%	135/189	37/45 (82.2%)	46/144 (31.9%)	37/135 (27.4%)	46/54 (85.2%)
50	125	>15%	137/189	38/45 (84.4%)	45/144 (31.3%)	38/137 (27.7%)	45/52 (86.5%)
40	125	>15%	141/189	40/45 (88.9%)	43/144 (29.9%)	40/141 (28.4%)	43/48 (89.6%)
25	125	>15%	144/189	41/45 (91.1%)	41/144 (28.5%)	41/144 (28.5%)	41/45 (91.1%)
20	125	>15%	147/189	42/45 (93.3%)	39/144 (27.1%)	42/147 (28.6%)	39/42 (92.9%)
10	125	>15%	167/189	45/45 (100%)	22/144 (15.3%)	45/167 (26.9%)	22/22 (100%)
5	125	>15%	181/189	45/45 (100%)	8/144 (5.6%)	45/181 (24.9%)	8/8 (100%)
100	250	>5%	147/189	38/45 (84.4%)	35/144 (24.3%)	38/147 (25.9%)	35/42 (83.3%)
50	250	>5%	148/189	39/45 (86.7%)	35/144 (24.3%)	39/148 (26.4%)	35/41 (85.4%)
40	250	>5%	150/189	40/45 (88.9%)	34/144 (23.6%)	40/150 (26.7%)	34/39 (87.2%)
25	250	>5%	151/189	41/45 (91.1%)	34/144 (23.6%)	41/151 (27.2%)	34/38 (89.5%)
20	250	>5%	156/189	43/45 (95.6%)	31/144 (21.5%)	43/156 (27.6%)	31/33 (93.9%)
10	250	>5%	175/189	45/45 (100%)	14/144 (9.7%)	45/175 (25.7%)	14/14 (100%)
5	250	>5%	182/189	45/45 (100%)	7/144 (4.9%)	45/182 (24.7%)	7/7 (100%)

Table 6-10. (cont.)

Set of Candidate Standards			# Units At Or Above At Least One Standard / Total # Units[2]	Performance Characteristics			
Uncarpeted Floor Dust-Lead Loading ($\mu g/ft^2$)	Window Sill Dust-Lead Loading ($\mu g/ft^2$)	Max. Amt. Of Damaged LBP on a Tested Surface (%)[1]		Sensitivity # (%) of Units with EBL Children That Are At or Above At Least One Standard[3]	Specificity # (%) of Units with No EBL Children That Are At or Above No Standard[4]	PPV # (%) of Units At or Above At Least One Standard That Have EBL Children[5]	NPV # (%) of Units At or Above No Standard That Do Not Have EBL Children[6]
100	125	>5%	156/189	40/45 (88.9%)	28/144 (19.4%)	40/156 (25.6%)	28/33 (84.8%)
50	125	>5%	157/189	41/45 (91.1%)	28/144 (19.4%)	41/157 (26.1%)	28/32 (87.5%)
40	125	>5%	159/189	42/45 (93.3%)	27/144 (18.8%)	42/159 (26.4%)	27/30 (90.0%)
25	125	>5%	160/189	43/45 (95.6%)	27/144 (18.8%)	43/160 (26.9%)	27/29 (93.1%)
20	125	>5%	163/189	44/45 (97.8%)	25/144 (17.4%)	44/163 (27.0%)	25/26 (96.2%)
10	125	>5%	176/189	45/45 (100%)	13/144 (9.0%)	45/176 (25.6%)	13/13 (100%)
5	125	>5%	183/189	45/45 (100%)	6/144 (4.2%)	45/183 (24.6%)	6/6 (100%)

[1] In the Rochester study, each measurement of lead in paint had the amount of damaged paint specified as "<5%" (good condition), "5-15%" (fair condition), or ">15%" (poor condition) of the tested surface, with no indication of total damaged surface area.
[2] Total number of units having available data that could be compared to all specified candidate standards.
[3] Cell entries are(number of homes at or above at least one standard that have EBL children)/ number of homes containing EBL children), followed by the corresponding percentage (in parentheses).
[4] Cell entries are (number of homes not at or above at least one standard that do not have EBL children)/(total number of homes not containing EBL children), followed by the corresponding percentage (in parentheses).
[5] Cell entries are (number of homes at or above at least one standard that have EBL children)/(total number of homes at or above at least one standard), followed by the corresponding percentage (in parentheses).
[6] Cell entries are (number of homes not at or above at least one standard that do not have EBL children)/(total number of homes not at or above any standard), followed by the corresponding percentage (in parentheses).

6.2 INVESTIGATING INCIDENCE OF ELEVATED BLOOD-LEAD CONCENTRATION IN HOUSING UNITS MEETING ALL EXAMPLE OPTIONS FOR STANDARDS

An alternative to the performance characteristics analysis approach (Section 6.1) to evaluating a set of candidate standards is to use statistical modeling techniques to predict a distribution of blood-lead concentration as a function of environmental-lead levels found in homes which do not exceed any of the candidate standards, then estimate the percentage of children residing in these homes that are expected to have elevated blood-lead levels (i.e., at or above 10 μg/dL). It is desired to select a set of candidate standards so that the likelihood of children with elevated blood-lead concentration residing in homes that do not exceed any of the candidate standards would be very low. This section presents a modeling approach to estimate this likelihood, using the alternative Rochester multimedia model presented in Section 4.2 of this report ("Model A" in Table 4-1), and applies this approach to data from the Rochester study.

Recall from Section 4.2 that the reason for developing the alternative Rochester multimedia model was to have the risk estimates from model-based analyses be more comparable to the results of the performance characteristics analysis presented in the §403 proposed rule (Section 6.1.3) and the results of the follow-up performance characteristics analyses (Section 6.1.4). In particular, both the performance characteristics analysis and the model-based approach involving the alternative Rochester multimedia model use the following types of data as input when characterizing risk:

- household average (wipe) dust-lead loading from uncarpeted floors
- household average (wipe) dust-lead loading from window sills
- yard-wide average soil-lead concentration
- the larger of the following two percentages: % of interior tested surfaces that contain deteriorated lead-based paint (LBP), and % of exterior tested surfaces that contain deteriorated LBP

In the model-based analysis approach presented below, the candidate standards were used to identify a subset of homes in the Rochester study that were below all of the candidate standards, calculate the average (across homes) of the above three measures of lead levels in dust and soil, and fit the multimedia model to these average lead levels in order to predict a distribution of blood-lead concentrations for children residing in these homes. For simplicity, this analysis assumes that the homes do not contain deteriorated lead-based paint. Because the slope estimate for the paint variable in the alternative Rochester multimedia model is nearly zero (Table 4-1 of Section 4.2), making the assumption that no deteriorated lead-based paint exists in these homes should have a very minor impact on the resulting risk estimates.

6.2.1 The Model-Based Approach

This model-based approach had the following four steps:

1. For a given set of candidate standards for floor dust-lead loading, window sill dust-lead loading, and soil-lead concentration, identify those homes in the Rochester study that exceed none of the candidate standards in this set.

2. For each of the following three household measures, calculate the average across the homes identified in step #1: the household average floor dust-lead loadings, household average window sill dust-lead loading and for yard-wide average soil-lead concentration. These three averages are assumed to represent lead levels in housing represented by the Rochester study homes in step #1 (i.e., homes not exceeding any of the candidate dust and soil standards).

3. Use the three averages calculated in step #2 as input to the alternative Rochester multimedia model from Section 4.2 (assuming no deteriorated lead-based paint exists in the units).

4. Assume that log-transformed blood-lead concentration for children residing in the homes identified in step #1 is normally distributed with mean equal to the predicted log-transformed blood-lead concentration that is output from the model fitting in step #3, and standard deviation equal to ln(1.6). (Recall that this assumption on variability was made throughout the §403 risk analysis.) Using normal distribution theory, determine the percentage of children represented by this blood-lead distribution that have log-transformed blood-lead concentration or above log(10), or equivalently, that have blood-lead concentration at or above 10 μg/dL.

6.2.2 Examples of Applying the Model-Based Approach

To illustrate how the approach in Section 6.2.1 is applied to data from the Rochester study, the following combinations of candidate dust-lead and soil-lead standards are considered:

- (uncarpeted) floor dust-lead loading: either 40 or 50 $\mu g/ft^2$
- window sill dust-lead loading: 250 $\mu g/ft^2$
- yard-wide soil-lead concentration: 400 $\mu g/g$.

When the candidate floor dust-lead loading standard is 40 $\mu g/ft^2$, then the performance characteristics analyses documented in Table 6-8 of Section 6.1 (i.e., the row of Table 6-8 corresponding to these three candidate standards) indicates that 39 of the 184 Rochester study homes having measurements for dust-lead, soil-lead, and deteriorated lead-based paint do not exceed any of the three candidate standards. Across these 39 homes, the following averages were calculated from the Rochester study data:

- household average (uncarpeted) floor dust-lead loading: 12.7 $\mu g/ft^2$
- household average window sill dust-lead loading: 87.0 $\mu g/ft^2$

- yard-wide average soil-lead concentration: 125.3 µg/g.

When fitting the alternative Rochester multimedia model to these three averages (assuming no deteriorated lead-based paint), the model predicts a geometric mean blood-lead concentration of 4.68 µg/dL. If the standard deviation of log-transformed data is assumed to be 1.6 and normal distribution theory is applied as described above, then the estimated percentage of children with blood-lead concentration at or above 10 ug/dL in homes that do not exceed any of the candidate standards is 5.30%. This matches closely with the estimate of 5.1%, or 2 of these 39 homes in the Rochester study dataset, which the performance characteristics analysis (Table 6-8) indicated contained children with elevated blood-lead concentrations.

If the candidate floor dust-lead loading standard is increased to 50 µg/ft^2, then the number of Rochester study homes having measurements for dust-lead, soil-lead, and deteriorated lead-based paint and that do not exceed any of the three candidate standards increases by one home, to 40 total homes. Across these 40 homes, the following averages were calculated from the Rochester study data:

- household average (uncarpeted) floor dust-lead loading: 13.4 µg/ft^2
- household average window sill dust-lead loading: 85.6 µg/ft^2
- yard-wide average soil-lead concentration: 122.2 µg/g.

The predicted geometric mean blood-lead concentration under these assumed dust-lead and soil-lead levels (assuming no deteriorated lead-based paint) is 4.69 µg/dL, and the estimated percentage of children with blood-lead concentration at or above 10 ug/dL is 5.34%. This is a very slight increase from the estimate generated under the candidate floor dust-lead loading standard of 40 µg/ft^2. The performance characteristics analysis (Table 6-8) indicated that under these candidate standards, 7.5% of homes not exceeding any of the standards (i.e., 3 of these 40 homes in the Rochester study dataset) contained children with elevated blood-lead concentrations.

While these examples illustrate the estimation process, they also show that the number of homes in the given dataset whose lead levels fall below all specified candidate standards can be quite small, especially when at least one of the candidate standards is set at the low end of the distribution of lead levels (i.e., most homes have data that fall above the candidate standard). Therefore, as the set of candidate standards becomes more stringent, and as the size of the sample from which the environmental-lead data originate becomes smaller as a result, the variability associated with the estimated risk increases. Furthermore, as the set of candidate standards becomes less stringent (i.e., as the standards increase), the group of homes not exceeding any of the candidate standards is more likely to remain the same, and as a result, the estimated risk eventually reaches a plateau. This occurs in the above examples, as increasing the candidate floor dust-lead loading standard from 40 to 50 µg/ft^2 does little, if any, to increase the estimated risk beyond 5.3% under this approach and under the given set of data, assuming the candidate standards for the other media (window sill dust, soil) remain fixed.

The Rochester study data were used in this analysis as the multimedia model was fitted based on the Rochester data. If data from other studies were used instead, it would be necessary to verify that the model parameter estimates adequately reflect the underlying variability in these data in the same manner that they reflect variability in the Rochester study data.

While the approach presented in this section is relatively easy to implement, it could be modified even further in an attempt to achieve more accurate risk estimates. Such a modification could reduce the level of simplicity associated with applying the approach. For example, rather than calculate average environmental-lead levels across all homes and fit the model once to these averages, a simulation approach could be applied in an attempt to more accurately represent the entire distribution of environmental-lead levels in these homes and the resulting blood-lead distribution associated with exposure across the entire distribution of environmental-lead levels.

6.3 REVIEW OF PUBLISHED INFORMATION ON POST-INTERVENTION DUST-LEAD LOADINGS

This section summarizes published information on lead loadings (amount of lead per unit surface area) in dust samples collected by wipe techniques, as reported by earlier lead intervention studies. This information is used to evaluate assumptions made on post-intervention dust-lead loadings (40 $\mu g/ft^2$ for floors, 100 $\mu g/ft^2$ for window sills) within the §403 risk analysis. Details to supplement the summaries in this section are presented in Appendix H.

The following seven studies have been identified in which some type of paint or dust intervention was performed, dust samples were collected using wipes or some other technique (e.g., BRM vacuum) whose results could be converted to wipe-equivalent dust-lead loadings, and post-intervention dust-lead loadings on floors and/or window sills were reported (references for these studies are included in Appendix H):

- Baltimore Experimental Paint Abatement Studies
- Baltimore Follow-up Paint Abatement Study
- Baltimore Repair & Maintenance (R&M) Study
- Boston Interim Dust Intervention Study
- HUD Grantees Evaluation (data available through September 1997)
- Denver Comprehensive Abatement Performance (CAP) Study
- Jersey City Children's Lead Exposure and Reduction (CLEAR) Study

These studies employed a variety of intervention strategies, including single or repeated dust cleanings and interim control or complete abatement of lead-based paint. Dust-lead loadings were measured at varying intervals following intervention. Post-intervention dust-lead loadings were summarized for 19 groups of housing units across these seven studies. These study groups are defined in Appendix H.

For both floors and window sills, geometric mean and median dust-lead loadings were observed below the post-intervention assumptions established in the §403 risk analysis in a

majority of the study groups. However, this does not preclude results for individual housing units from being above the assumed levels. Furthermore, the extent to which results for these studies represent the nation's housing stock has not been determined. Results are now presented separately for floors and window sills (with more detailed presentations found in Appendix H).

6.3.1 Post-Intervention Floor Dust-Lead Loadings

Summaries of post-intervention floor (wipe) dust-lead loadings are presented in Table 6-11 according to housing group within each study. According to Table 6-11, all but two of the 19 study groups reported geometric mean or median floor dust-lead loadings at or below 41 $\mu g/ft^2$ from 6 months to 6 years post-intervention. The other two study groups were from the Baltimore Experimental Paint Abatement Study, where pre-intervention geometric mean dust-lead loadings were much greater (556 $\mu g/ft^2$ and 1261 $\mu g/ft^2$) than any other study group (at most 58.6 $\mu g/ft^2$). Eleven study groups reported geometric mean or median floor dust-lead loadings at or below 21 $\mu g/ft^2$ at follow-up periods ranging from 12 months to 2 years. Of these 11 groups, four of the HUD Grantees study groups reported median floor dust-lead loadings at or below 10 $\mu g/ft^2$ at 12 months post-intervention. Median pre-intervention floor dust-lead loadings in these four groups ranged from 9 to 26 $\mu g/ft^2$.

In the HUD Grantees evaluation, seven of the eight largest grantees have median floor dust-lead loadings at or below 21 $\mu g/ft^2$ at 12 months post-intervention, compared to a median of 14 $\mu g/ft^2$ across all grantees. Although pre-intervention floor dust-lead loadings were lower in the HUD Grantees evaluation compared to other studies, these preliminary results suggest that floor dust-lead loadings can be maintained at levels below 40 $\mu g/ft^2$ for at least 12 months post-intervention.

Results from the Denver CAP study, the Baltimore Follow-up Paint Abatement study, the Baltimore R&M study, the Boston Interim Dust Intervention study, and the Jersey City CLEAR study suggest that geometric mean floor dust-lead loadings of below 40 $\mu g/ft^2$ can be observed even beyond 12 months post-intervention and up to six years post-intervention, under the same conditions experienced by the housing units in these studies.

6.3.2 Post-Intervention Window Sill Dust-Lead Loadings

Summaries of post-intervention window sill wipe dust-lead loadings are presented in Table 6-12 according to housing group. Post-intervention geometric means or medians range from 24 $\mu g/ft^2$ to 958 $\mu g/ft^2$, which are considerably higher than the summaries for floors. Eleven study groups had geometric mean or median post-intervention window sill dust-lead loadings below 100 $\mu g/ft^2$, 6 groups were at or below 51 $\mu g/ft^2$, and 3 groups were at or below 41 $\mu g/ft^2$.

All but one of the HUD Grantees study groups (the Milwaukee grantee) had median window sill dust-lead loadings below 100 $\mu g/ft^2$ at 12 months post-intervention. As the intervention strategy for homes in the HUD Grantees evaluation frequently included partial or

Table 6-11. Summaries of Pre- and Post-Intervention Floor Wipe Dust-Lead Loadings for Housing Groups Within Seven Studies

Study	Study Group	Pre-Intervention Floor Dust-Lead Loadings[1] ($\mu g/ft^2$)	Post-Intervention Floor Dust-Lead Loadings[1]	
			Time Following Intervention (Months)	Summary Value ($\mu g/ft^2$)
Baltimore Experimental Paint Abatement Studies[2]	Study 1	1261	6-9	99
	Study 2	556	1.5 - 3.5 Years	69
Baltimore Follow-up Paint Abatement Study[2]	12-Month Follow-up	NA	10-14	20
	19-Month Follow-up	NA	14-24	36
Baltimore R&M Study[3]	Previously-Abated Units	45.6	4 - 6 Years	33.0
	Units Slated for R&M Intervention	58.6	24	35.0
Boston Interim Dust Intervention Study[2]	Automatic Intervention	33.2	6	23.9
	Randomized Intervention	37.3	6	31.4
HUD Grantees[4]	All Grantees	19	12	14
	Baltimore	41	12	41
	Boston	24	12	18
	Massachusetts	24	12	9
	Milwaukee	14	12	10
	Minnesota	18	12	18
	Rhode Island	26	12	6
	Vermont	28	12	21
	Wisconsin	9	12	5
Denver CAP Study[5]	Abated Units	NA	2 Years	21.0
Jersey City CLEAR Study	Intervention Group	22	12	15

[1] Values are geometric means except for the HUD Grantees studies, where values are medians. "NA" indicates not available.

[2] Results are adjusted to reflect total dust-lead loadings by exponentiating the "bioavailable" dust-lead loadings as reported in the study to the 1.1416 power.

[3] Results for the Baltimore R&M Study are converted from BRM dust-lead loadings to wipe-equivalent loadings.

[4] Data collected through September, 1997

[5] Results for the Denver CAP study are converted from CAP cyclone dust-lead loadings to wipe-equivalent loadings.

Table 6-12. Summaries of Pre- and Post-Intervention Window Sill Wipe Dust-Lead Loadings for Housing Groups Within Seven Studies

Study	Study Group	Pre-Intervention Sill Dust-Lead Loadings[1] ($\mu g/ft^2$)	Post-Intervention Sill Dust-Lead Loadings[1]	
			Time Following Intervention	Summary Value ($\mu g/ft^2$)
Baltimore Experimental Paint Abatement Studies[2]	Study 1	15215	6-9	958
	Study 2	2784	1.5 - 3.5 Years	199
Baltimore Follow-up Paint Abatement Study[2]	12-Month Follow-up	NA	10-14	41
	19-Month Follow-up	NA	14-24	147
Baltimore R&M Study[3]	Previously-Abated Units	163.5	4 - 6 Years	97.6
	Units Slated for R&M Intervention	778.4	24	204.9
Boston Interim Dust Intervention Study[2]	Automatic Intervention	787	6	210
	Randomized Intervention	205	6	110
HUD Grantees[4]	All Grantees	258	12	90
	Baltimore	1191	12	68
	Boston	174	12	49
	Massachusetts	328	12	50
	Milwaukee	264	12	217
	Minnesota	266	12	77
	Rhode Island	314	12	85
	Vermont	147	12	40
	Wisconsin	150	12	51
Denver CAP Study[5]	Abated Units	NA	2 Years	66.4
Jersey City CLEAR Study	Intervention Group	75	12	24

[1] Values are geometric means except for the HUD Grantees studies, where values are medians. "NA" indicates not available.

[2] Results are adjusted to reflect total dust-lead loadings by exponentiating the "bioavailable" dust-lead loadings as reported in the study to the 1.1416 power.

[3] Results for the Baltimore R&M Study are converted from BRM dust-lead loadings to wipe-equivalent loadings.

[4] Data collected through September, 1997

[5] Results for the Denver CAP study are converted from CAP cyclone dust-lead loadings to wipe-equivalent loadings.

complete window replacement, these results may not be representative of the outcomes of interventions prompted by the §403 rule.

Geometric mean window sill dust-lead loadings were below 100 $\mu g/ft^2$ for up to two years post-intervention in the Baltimore Follow-up Paint Abatement study, Denver CAP study, and Jersey City CLEAR study. However, in the Baltimore R&M study, Baltimore Experimental Paint Abatement studies, and Boston Interim Dust Intervention study, geometric mean dust-lead loadings remain above 100 $\mu g/ft^2$ over time. In addition, the 19-month follow-up study group within the Baltimore Follow-up Paint Abatement study and study group #2 of the Baltimore Experimental Paint Abatement studies suggest that geometric mean dust-lead loadings can dip below 100 $\mu g/ft^2$ immediately after intervention, but then exceed this level after one year or so.

6.4 SENSITIVITY AND UNCERTAINTY ANALYSES FOR RISK MANAGEMENT ANALYSES

The following subsections present the results of additional sensitivity and uncertainty analyses performed to gauge the level of uncertainty in the post-§403 risk estimates (and the associated decline from baseline estimates) associated with methodological assumptions. These results should be considered with those presented in the sensitivity and uncertainty analyses in Section 6.4 of the §403 risk analysis report to characterize overall uncertainty associated with the methods and assumptions taken in the risk management.

6.4.1 Considering How Baseline Environmental-Lead Levels May Have Changed Since the HUD National Survey

Section 5.1.4 of this report addressed the sensitivity of the pre-§403 model-based blood-lead distribution and the resulting health effects and blood-lead concentration endpoint estimates under the IEUBK and empirical models under different assumptions on how the national distribution of baseline environmental-lead levels as estimated using HUD National Survey data may have changed since the time of the survey (1989-1990). The same five sets of adjustments (i.e., percentage changes) made to the average baseline dust-lead loadings, dust-lead concentrations, and soil-lead concentrations for each housing unit in the HUD National Survey were considered in this sensitivity analysis to observe the impact on post-§403 risk estimates under the following set of example options for standards:

- Average floor dust-lead loading = 100 $\mu g/ft^2$
- Average window sill dust-lead loading = 500 $\mu g/ft^2$
- Average soil-lead concentration = 2,000 $\mu g/g$
- Amount of deteriorated lead-based paint requiring paint maintenance = 5 ft^2
- Amount of deteriorated lead-based paint requiring paint abatement = 20 ft^2

This set of options was the primary set considered in the sensitivity analyses within Section 6.4 of the §403 risk analysis report.

Table 6-13 presents the post-§403 estimates for the health effect and blood-lead concentration endpoints under both the IEUBK and empirical models, for each of the five sets of adjustments to the post-§403 environmental-lead levels in housing units within the HUD National Survey and under the above assumption on example standards. Also included in this table are the percentage of homes exceeding the various example standards, which will be lower than in the §403 risk analysis when declines in the appropriate environmental-lead levels are considered and higher when increases are considered. The table also lists the baseline risk estimates for comparison purposes.

Effect on risk analysis: Under the five sets of assumptions involving lower assumed baseline environmental-lead levels, the percentage of houses that exceed at least one of the example standards declined by at most about three percentage points (from 21.8% to 18.7%; Table 6-13), or about three million homes. The assumption that baseline environmental-lead levels are 25% higher than assumed in the §403 risk analysis results in an increase in the percentage of homes exceeding at least one standard from 21.8% to 24.1%, an increase of about 2.3 million homes (Table 6-13).

As would be expected, Table 6-13 shows that all assumptions on baseline environmental-lead levels result in post-§403 estimates of the predicted health effect and blood-lead concentration endpoints that are lower than baseline (the last column of the table). However, as the assumed baseline environmental-lead levels become lower in magnitude, the predicted post-§403 risks actually increase, converging to the baseline estimates. For example, as seen in Table 6-3, baseline lead levels that are 20% below what was assumed in the §403 risk analysis resulted in an estimated percentage of children with blood-lead concentrations at or above 10 µg/dL of 4.85%, compared to the §403 risk analysis estimate of 4.70%. When baseline lead levels are 50% below the §403 risk analysis estimates, the estimate of this percentage increases to 5.10%. Such a finding appears counter-intuitive when first reviewing the table. However, the alternative assumptions being considered in this sensitivity analysis are to baseline (i.e., pre-§403) environmental-lead levels. As assumptions on these baseline levels move lower, fewer homes are triggered by the §403 standards, and the post-§403 distribution of environmental-lead levels becomes less removed from the baseline distribution. As a result, post-§403 estimates of predicted health effects and blood-lead concentration are not as different from pre-§403 estimates. In contrast, as assumed baseline environmental-lead levels increase, more homes are triggered by the §403 standards and, therefore, have their environmental-lead levels drop as a result of interventions, and lower post-§403 risk estimates relative to baseline are observed.

As seen in Table 6-13, the effect that different assumptions on baseline environmental-lead levels have on the risk estimates is considerably greater under the IEUBK model than the empirical model. The percentage of children with blood-lead concentrations at or above 20 µg/dL more than triples under the IEUBK model approach when 50% declines in both dust-lead and soil-lead levels were assumed (from 0.054% to 0.166%), compared to a 16% increase under the empirical model (from 0.406% to 0.469%). Smaller percentage differences are observed for the other endpoints for both models.

Table 6-13. Sensitivity Analysis on How Changes in Household Average Baseline Dust-Lead Loadings/Concentrations and Soil-Lead Concentration Impact Post-§403 Estimates of Health Effect and Blood-Lead Concentration Endpoints for Children Aged 1-2 Years Under a Specified Set of Example Standards[1]

Assumed Percentage Change in Average Dust-Lead Loadings and Concentrations (Both Floor and Window Sill) and in Yard-wide Average Soil-Lead Concentration							Baseline Estimate (from Table 5-1 of the §403 risk analysis report)
Dust:	No change	20% decrease	50% decrease	50% decrease	No change	25% increase	
Soil:	No change	20% decrease	50% decrease	No change	50% decrease	25% increase	
Percentage of Homes Exceeding Example Standards/Triggers							
Floor Dust	4.04	2.34	0.694	0.694	4.04	5.68	
Window Sill Dust	12.5	10.8	9.10	9.10	12.5	14.3	
Soil	2.49	1.52	0.746	2.49	0.746	3.27	
Interior Paint Maintenance	2.92	2.92	2.92	2.92	2.92	2.92	
Exterior Paint Maintenance	3.49	3.49	3.49	3.49	3.49	3.49	
Interior Paint Abatement	2.43	2.43	2.43	2.43	2.43	2.43	
Exterior Paint Abatement	5.77	5.77	5.77	5.77	5.77	5.77	
Any Standard/Trigger	21.8	20.6	18.7	18.9	21.6	24.1	
Predicted Health Effect And Blood-Lead Concentration Endpoints (Based on Empirical Model)							
PbB ≥20 (%)	0.406	0.429	0.469	0.445	0.427	0.378	0.588
PbB ≥10 (%)	4.70	4.85	5.10	4.95	4.84	4.52	5.75
IQ < 70 (%)	0.110	0.111	0.112	0.111	0.111	0.110	0.115
IQ decrement ≥1 (%)	36.3	36.7	37.3	36.9	36.7	35.9	38.5
IQ decrement ≥2 (%)	9.30	9.53	9.90	9.69	9.51	9.02	10.8
IQ decrement ≥3 (%)	2.93	3.04	3.21	3.11	3.03	2.80	3.70
Avg. IQ decrement	1.00	1.01	1.03	1.02	1.01	0.995	1.06
Predicted Health Effect And Blood-Lead Concentration Endpoints (Based on IEUBK Model)							
PbB ≥20 (%)	0.0539	0.117	0.166	0.121	0.0681	0.0542	0.588
PbB ≥10 (%)	1.66	2.48	2.98	2.55	1.86	1.64	5.75
IQ < 70 (%)	0.0984	0.102	0.104	0.102	0.0992	0.0982	0.115
IQ decrement ≥1 (%)	28.3	31.0	32.7	31.6	28.8	27.7	38.5
IQ decrement ≥2 (%)	4.31	5.77	6.65	5.94	4.67	4.22	10.8
IQ decrement ≥3 (%)	0.858	1.37	1.71	1.42	0.983	0.847	3.70
Avg. IQ decrement	0.848	0.894	0.924	0.904	0.857	0.839	1.06

[1] Example dust and soil standards were set at: 100 $\mu g/ft^2$ for floor dust-lead loading, 500 $\mu g/ft^2$ for window sill dust-lead loading, and 2,000 $\mu g/g$ for soil-lead concentration. Paint maintenance is performed if more than 5 ft^2, but less than 20 ft^2 of deteriorated lead-based paint exists. Paint abatement is performed if more than 20 ft^2 of deteriorated lead-based paint exists.

6.4.2 Impact on the Estimated Incidence of IQ Point Decrement Assuming Certain Thresholds on the IQ/Blood-Lead Relationship

The sensitivity of baseline and pre-§403 model-based estimates of IQ decrements greater than 1, 2, or 3, and of the average and standard deviation of the distribution of IQ point decrements was addressed in Section 5.1.5 of this report for various assumptions of a non-zero threshold of blood-lead concentration on the IQ/blood-lead relationship. The following thresholds were considered: 1, 2, 3, 5, 8 and 10 µg/dL. In this section, post-§403 estimates of these health effect endpoints are estimated (under the same set of options presented in Section 6.4.1, using both the IEUBK and empirical models) under these same alternative blood-lead concentration thresholds. These estimates are presented in Table 6-14.

Effect on risk analysis: As was also seen in Table 5-7 of this report, Table 6-14 shows that the post-§403 risk estimates decrease as the assumed blood-lead concentration threshold increases (i.e., smaller percentages of children experience IQ score decrements under larger threshold assumptions). The IEUBK model is more sensitive than the empirical model to the threshold level. For example, the probability of a child experiencing an IQ decrement of at least 1 point decreases by 63% under the IEUBK model (from 28.3% to 10.4%) when the threshold increases from 0 to 2 µg/dL, compared to only a 52% decrease under the empirical model (from 36.3% to 17.6%). As the assumed threshold increases, the likelihood of experiencing an IQ decrement of at least 1 point as a result of lead exposure decreases to very low values under both models, and the average IQ score decrement in the population declines to small fractions of points.

6.4.3 Considering Alternative Assumptions on Post-Intervention Dust-Lead Loadings

In the risk management portion (Chapter 6) of the §403 risk analysis report, it was necessary to make assumptions on predicted post-intervention lead levels when characterizing the blood-lead concentration and health effect endpoints in a post-§403 environment. These assumptions were documented in Table 6-2 of the §403 risk analysis report. Among these assumptions were that dust cleaning activities impacted interior dust-lead loadings in the following way:

- Post-intervention household average floor (wipe) dust-lead loadings equaled the minimum of 40 µg/ft^2 and the pre-intervention value.

- Post-intervention household average window sill (wipe) dust-lead loadings equaled the minimum of 100 µg/ft^2 and the pre-intervention value.

A dust cleaning was assumed to be included among the interventions performed when either the floor-dust, window sill-dust, soil, or interior paint abatement standards were exceeded within a home. These two assumptions on post-intervention dust-lead loadings were made within the §403 risk analysis based on data reported in EPA's Comprehensive Abatement Performance

Table 6-14. Sensitivity Analysis on the Assumed Blood-Lead Concentration Threshold on IQ Decrement and Its Impact on the Post-§403 Estimates of IQ Decrement Endpoints for Children Aged 1-2 Years, Under a Specified Set of Example Standards[1]

Assumed Threshold (μg/dL)	% of Children Aged 1-2 Years with a Specified IQ Decrement Due to Lead Exposure[2]			Average IQ Decrement (# points)[3]	Standard Deviation of IQ Decrement[3]
	IQ Decrement ≥ 1	IQ Decrement ≥ 2	IQ Decrement ≥ 3		
Baseline Estimates (Section 5.1.1 of §403 risk analysis report)					
0	38.5	10.8	3.70	1.06	0.895
1	27.3	8.08	2.88	0.804	0.891
2	19.6	6.10	2.26	0.588	0.860
3	14.2	4.66	1.80	0.428	0.802
5	7.83	2.80	1.16	0.233	0.666
8	3.50	1.40	0.627	0.103	0.494
10	2.15	0.915	0.429	0.0638	0.408
Post-§403 Estimates Based on IEUBK Model-Generated PbB Distribution					
0	28.3	4.31	0.858	0.848	0.567
1	17.1	2.78	0.589	0.594	0.564
2	10.4	1.82	0.410	0.379	0.529
3	6.48	1.21	0.289	0.234	0.462
5	2.65	0.566	0.149	0.0907	0.325
8	0.790	0.199	0.0593	0.0250	0.188
10	0.380	0.105	0.0335	0.0116	0.134
Post-§403 Estimates Based on Empirical Model-Generated PbB Distribution					
0	36.3	9.30	2.93	1.00	0.817
1	25.1	6.79	2.24	0.752	0.814
2	17.6	5.02	1.73	0.537	0.781
3	12.5	3.75	1.35	0.380	0.721
5	6.56	2.18	0.838	0.197	0.584
8	2.76	1.03	0.434	0.0812	0.417
10	1.64	0.653	0.289	0.0480	0.337

[1] Example dust and soil standards were set at: 100 μg/ft² for floor dust-lead loading, 500 μg/ft² for window sill dust-lead loading, and 2,000 μg/g for soil-lead concentration. Paint maintenance is performed if more than 5 ft², but less than 20 ft² of deteriorated lead-based paint exists. Paint abatement is performed if more than 20 ft² of deteriorated lead-based paint exists.

[2] A 0.257 IQ decrement is assumed for each 1.0 μg/dL increase in PbB above the assumed threshold (see Section 4.4.1 of the §403 risk analysis report). Thus, the following hold:

- P[IQ ≥ 1] = P[PbB ≥ (threshold + 3.9 μg/dL)]
- P[IQ ≥ 2] = P[PbB ≥ (threshold + 7.8 μg/dL)]
- P[IQ ≥ 3] = P[PbB ≥ (threshold + 11.7 μg/dL)]

[3] Average and standard deviation of IQ decrement are calculated assuming no IQ decrement occurs below the assumed threshold, and a 0.257 IQ decrement is assumed for each 1.0 μg/dL increase in PbB above the threshold.

study and in the Baltimore Experimental Paint Abatement study (see Section 6.1.2 of the §403 risk analysis report and Section H2.0 of Appendix H of this report).

Tables 6-11 and 6-12 within Section 6.3 of this report presented additional information on household average (wipe) dust-lead loading at pre- and post-intervention for floors and window sills, respectively, from several recent lead intervention studies. This information, some of which was received after the §403 risk analysis report was completed, suggests that it may be common in some instances to observe household average post-intervention dust-lead loadings below the assumptions made above, even from 12 months to six years post-intervention. These findings prompted a sensitivity analysis to investigate how setting assumptions on post-intervention household average dust-lead loadings to below the 40 $\mu g/ft^2$ and 100 $\mu g/ft^2$ specifications would impact the outcome of the risk management analyses.

In this sensitivity analysis, two alternative assumptions on household average post-intervention floor dust-lead loadings were made: 10 $\mu g/ft^2$ and 25 $\mu g/ft^2$. As the geometric mean (12-month) post-intervention floor dust-lead loading in the HUD Grantees evaluation was 14 $\mu g/ft^2$ (Table 6-8) and was even lower for certain grantees, an alternative of 10 $\mu g/ft^2$ was selected. The alternative of 25 $\mu g/ft^2$ for floors was selected as it fell halfway between the assumptions of 10 and 40 $\mu g/ft^2$ and was within the range of expected variability in the summaries for several of the studies in Section 6.3.1.

Similarly, two alternative assumptions on household average post-intervention window sill dust-lead loadings were made: 50 $\mu g/ft^2$ and 75 $\mu g/ft^2$. Evidence from Table 6-12 indicates that average window sill dust-lead loadings following intervention could approach 50 $\mu g/ft^2$ in some instances, especially when floor dust-lead loadings are low. The alternative of 75 $\mu g/ft^2$ was selected as it fell halfway between the assumptions of 50 and 100 $\mu g/ft^2$, and it was similar to the average levels observed by grantees within the HUD Grantees evaluation (although the HUD Grantees evaluation included window replacement, which was not among the assumed interventions in the §403 risk analysis).

In the sensitivity analysis, if a given household's pre-intervention average floor dust-lead loading fell below the given post-intervention assumption, its post-intervention household average floor dust-lead loading was assumed to be equal to its pre-intervention average (as was done in Chapter 6 of the §403 risk analysis report). Second, this sensitivity analysis considers predictions made only by the empirical model, as the IEUBK model does not accept dust-lead loading as input. Finally, the assumptions made in determining post-intervention soil-lead concentrations (150 $\mu g/g$ following soil removal) and amount of deteriorated lead-based paint (none is present following paint intervention) remained the same as specified in Table 6-2 of the §403 risk analysis report.

Table 6-15 presents the estimated post-§403 health effect and blood-lead concentration endpoints associated with the set of example options for standards specified in Section 6.4.1 above, for the alternative assumptions on post-intervention floor and window sill dust-lead loadings specified above. Note that each alternative assumption is evaluated on its own (i.e., it is

Table 6-15. Sensitivity Analysis on How Changing the Assumption on the Post-Intervention Household Average (Wipe) Dust-Lead Loadings on Floors and Window Sills Impact Post-§403 Estimates (Based on the Empirical Model) of the Health Effect and Blood-Lead Concentration Endpoints for Children Aged 1-2 Years Under a Specified Set of Example Standards[1]

Health Effect And Blood-Lead Concentration Endpoint	Predicted Estimates of the Endpoint (Based on the Empirical Model)							Baseline Estimate (From Table 5-1 of the §403 Risk Analysis Report)
	Assumed Post-Intervention Household Average Dust-Lead Loading for Floors and Window Sills[2]							
	Floors = 40 μg/ft² Sills = 100 μg/ft²	Floors = 10 μg/ft² Sills = 100 μg/ft²	Floors = 25 μg/ft² Sills = 100 μg/ft²	Floors = 40 μg/ft² Sills = 50 μg/ft²	Floors = 40 μg/ft² Sills = 75 μg/ft²	Floors = 10 μg/ft² Sills = 50 μg/ft²	Floors = 25 μg/ft² Sills = 75 μg/ft²	
PbB ≥20 (%)	0.406	0.389	0.401	0.396	0.402	0.380	0.397	0.588
PbB ≥10 (%)	4.70	4.59	4.67	4.64	4.68	4.53	4.64	5.75
IQ < 70 (%)	0.110	0.110	0.110	0.110	0.110	0.110	0.110	0.115
IQ decrement ≥1 (%)	36.3	36.1	36.3	36.2	36.3	35.9	36.2	38.5
IQ decrement ≥2 (%)	9.30	9.13	9.25	9.20	9.26	9.03	9.21	10.8
IQ decrement ≥3 (%)	2.93	2.85	2.90	2.88	2.91	2.81	2.89	3.70
Avg. IQ decrement	1.00	0.999	1.00	1.00	1.00	0.995	1.00	1.06

[1] Example dust and soil standards were set at 100 μg/ft² for floor dust-lead loading, 500 μg/ft² for window sill dust-lead loading, and 2,000 μg/g for soil-lead concentration. Paint maintenance is performed if more than 5 ft², but less than 20 ft² of deteriorated lead-based paint exists. Paint abatement is performed if more than 20 ft² of deteriorated lead-based paint exists. This analysis follows the same approach conducted in Section 6.3.4 of the §403 risk analysis report. Assumptions on post-intervention soil-lead concentrations and amounts of deteriorated lead-based paint are unchanged from those specified in Table 6-2 of the §403 risk analysis report.

[2] Within a housing unit, the assumed post-intervention average floor dust-lead loading is the minimum of its pre-intervention average and the value for floors specified in the column heading. Similarly, the unit's assumed post-intervention average window sill dust-lead loading is the minimum of its pre-intervention average and the value for sills specified in the column heading.

the only change from the §403 risk analysis assumptions). In addition, considering the high correlation in dust-lead loadings between floors and window sills, the two lower alternatives (10 $\mu g/ft^2$ for floors and 50 $\mu g/ft^2$ for window sills) and the two higher alternatives (25 $\mu g/ft^2$ for floors and 75 $\mu g/ft^2$ for window sills) are evaluated together. For comparison purposes, post-intervention estimates under the §403 risk analysis (i.e., assuming 40 $\mu g/ft^2$ for floors and 100 $\mu g/ft^2$ for window sills) and the estimates generated under baseline (pre-§403) conditions (both presented in Table 6-7 of the §403 risk analysis report) are also included in Table 6-15.

Effect on risk analysis. Relative to the results reported in the §403 risk analysis report (column 2 of Table 6-15), the greatest deviation occurs with the most substantial change in the assumptions, i.e., the assumptions of 10 $\mu g/ft^2$ for floors and 50 $\mu g/ft^2$ for window sills (column 7 of Table 6-15). Under this particular set of alternative assumptions, the percentage of the nation's children aged 1-2 years that are anticipated to have blood-lead concentration at or above 10 $\mu g/dL$ following interventions conducted in response to the §403 rule (given the example standards specified in the footnote to this table) is reduced from 4.70% to 4.53% (a 3.7% decline, equivalent to approximately 13,700 children[12]). The corresponding reduction in the percentage of children with blood-lead concentration at or above 20 $\mu g/dL$ is from 0.406% to 0.380% (a 6.3% decline, equivalent to approximately 2,000 children).

Under the assumptions of 25 $\mu g/ft^2$ for floors and 75 $\mu g/ft^2$ for window sills (column 8 of Table 6-15), the percentage of the nation's children aged 1-2 years that are anticipated to have blood-lead concentration at or above 10 $\mu g/dL$ is reduced from 4.70% to 4.64% (a 1.2% decline, equivalent to approximately 4,800 children). The corresponding reduction in the percentage of children with blood-lead concentration at or above 20 $\mu g/dL$ is from 0.406% to 0.397% (a 2.3% decline, equivalent to approximately 750 children).

Generally, even lower percentage declines occur for the IQ endpoints compared to the blood-lead concentration endpoints. The exception occurs with the percentage of children with IQ decline of at least 3 points, where a 4.2% decline from the §403 risk analysis assumptions was observed under assumptions of 10 $\mu g/ft^2$ for floors and 50 $\mu g/ft^2$ for window sills.

This sensitivity analysis indicates that while more housing units may achieve reductions in average dust-lead levels on floors and window sills following a dust cleaning if the assumed post-intervention floor dust-lead loadings are lowered from those made in the §403 risk analysis, the corresponding reduction in the estimated blood-lead concentration and health effect endpoints appears to be modest, especially when compared to the reduction observed from pre- to post-§403 conditions.

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[12] Assuming that 7.96 million children aged 1-2 years reside in the U.S. housing stock (Table 3-35 of the §403 risk analysis report).

6.4.4 Characterizing the Post-Intervention Blood-Lead Distribution Based on Relative Change from Baseline in the Geometric Mean and the Probability of a Child's Blood-Lead Concentration Exceeding 10 μg/dL

As discussed in Section 4.3.1 above and in Appendix F1 of the §403 risk analysis report, a "scaling algorithm" was used in the §403 risk analysis to characterize the distribution of blood-lead concentration in the nation's children following interventions that would be performed as a result of implementing the §403 rule (where the algorithm was applied under a specified set of example options for the standards, using a specified blood-lead prediction model, and under assumptions made on the changes in environmental-lead levels that result from the interventions). This distribution is labeled the "post-§403" distribution. This approach calculated the geometric mean (GM) and geometric standard deviation (GSD) of the post-§403 blood-lead distribution in the following manner:

$$GM_{post\text{-}403} = GM_{baseline} * (GM_{model\text{-}based\ post\text{-}403} / GM_{model\text{-}based\ pre\text{-}403}) \quad (1)$$

$$GSD_{post\text{-}403} = GSD_{baseline} * (GSD_{model\text{-}based\ post\text{-}403} / GSD_{model\text{-}based\ pre\text{-}403}) \quad (2)$$

where the subscripts indicate the blood-lead distribution which either the GM or the GSD represents. See Section 4.3.1 for additional information on this approach.

One comment received on the §403 risk analysis was that because the blood-lead concentration endpoints utilized in the risk analysis were exceedance probabilities (i.e., the likelihood of a child's blood-lead concentration exceeding a specified value), it was more important to accurately characterize the right tail of the post-§403 distribution compared to the remainder of the distribution, especially at blood-lead levels beyond 10 μg/dL. Therefore, a variant of the scaling approach was considered that involved scaling the probability of a child's blood-lead concentration exceeding 10 μg/dL rather than the GSD. If P10 was used to represent this probability, then the alternative scaling algorithm would involve scaling the geometric mean as in (1) above, but replacing (2) above with the following calculation:

$$P10_{post\text{-}403} = P10_{baseline} * (P10_{model\text{-}based\ post\text{-}403} / P10_{model\text{-}based\ pre\text{-}403}) \quad (3)$$

The resulting value is the estimate of the probability of a child's blood-lead concentration exceeding 10 μg/dL in a post-§403 environment. It is calculated by multiplying the probability as calculated in the baseline distribution by the relative change in the probability from the pre-§403 to post-§403 environment as estimated from model-based blood-lead distributions. Then, in order to calculate the other blood-lead concentration and health effect endpoints, the GSD of the post-§403 distribution would be calculated by assuming that this distribution is lognormal. Therefore,

$$GSD_{post\text{-}403} = \exp\{(\log(10) - \log(GM_{post\text{-}403}))/\Phi^{-1}(1 - P10_{post\text{-}403})\} \quad (4)$$

where Φ^{-1} denotes the inverse of the standard normal distribution function.

Table 6-16 presents the estimated blood-lead concentration and health effect endpoints that result when applying this alternative scaling algorithm, under both the IEUBK and empirical models. The example options for standards that are assumed in this analysis are the same as those considered in Section 6.4.1 above and are specified in a footnote to Table 6-16. For comparison purposes, this table also contains the estimates under the original version of the scaling approach that was utilized in the §403 risk analysis.

Table 6-16. Estimated Post-§403 Health and Blood-Lead Concentration Endpoints Under the Original and Alternative Scaling Algorithms for Characterizing the Post-§403 Blood-Lead Distribution

Original scaling algorithm: Geometric mean and GSD are scaled.
Alternative scaling algorithm: Geometric mean and the probability of PbB exceeding 10 μg/dL are scaled.

Health Effect and Blood-Lead Concentration Endpoints	Post-§403 Estimates Under the Risk Management Analysis (Original Scaling Algorithm)		Post-§403 Estimates Under the Alternative Scaling Algorithm	
	IEUBK Model	Empirical Model	IEUBK Model	Empirical Model
% of Children with PbB ≥ 20 μg/dL	0.0539	0.406	0.156	0.249
% of Children with PbB ≥ 10 μg/dL	1.66	4.70	2.72	3.78
%of Children with IQ < 70 due to lead exposure	0.0984	0.110	0.102	0.107
% of Children with IQ decrement ≥ 1 due to lead exposure	28.3	36.3	30.1	35.5
% of Children with IQ decrement ≥ 2 due to lead exposure	4.31	9.30	6.05	8.03
% of Children with IQ decrement ≥ 3 due to lead exposure	0.858	2.93	1.56	2.24
Avg. IQ decrement due to lead exposure	0.848	1.00	0.884	0.977
Geometric Mean PbB (GSD)	2.74 (1.84)	3.03 (2.04)	2.74 (1.96)	3.03 (1.96)

Note: Example dust and soil standards were set at: 100 μg/ft² for floor dust-lead loading, 500 μg/ft² for window sill dust-lead loading, and 2,000 μg/g for soil-lead concentration. Paint maintenance is performed if more than 5 ft², but less than 20 ft², of deteriorated lead-based paint exists. Paint abatement is performed if more than 20 ft² of deteriorated lead-based paint exists. GSD = geometric standard deviation. PbB = blood-lead concentration

Effect on risk analysis. As indicated in Table 6-16, when the probability of exceeding 10 µg/dL is scaled instead of the GSD, the estimated probability is approximately 64% higher under the IEUBK model (1.66% to 2.72%), but nearly 20% lower under the empirical model (4.70% to 3.78%). Note that under the alternative approach, estimates based on the IEUBK and empirical models are more similar to each other than under the original scaling algorithm. In the alternative approach, the estimated post-§403 GSD is the same under both models: 1.96. Note that there was no change in the manner in which the geometric mean blood-lead concentrations were determined, and therefore, no change is noted between the two approaches.

The above results indicate that the alternative scaling approach has a more significant impact on the IEUBK model-based estimates compared to the empirical model-based estimates. The impact of the approach on the empirical model-based estimates is a reduction in the risk estimates due to a 4% reduction in the estimated GSD, while the impact on IEUBK model-based estimates is an increase in the risk estimates due to a 6.5% increase in the estimated GSD. However, because the two approaches did not differ in how the post-§403 geometric mean blood-lead level was calculated, the empirical model estimates remain higher than the IEUBK model estimates.

6.5 LEAD EXPOSURE ASSOCIATED WITH CARPETED FLOOR-DUST

While the §403 proposed rule included a proposed lead hazard standard for dust on uncarpeted floors, EPA determined that sufficient technical data were not available to direct how the rule should address lead-contaminated dust on carpeted floors. Based upon public comments on the proposed rule, EPA is revisiting that determination. This section summarizes the key findings of statistical analyses on dust-lead loading data for carpeted floors. The analysis had the following three objectives:

1. Assess the need to have dust-lead on carpeted floors addressed by the §403 rule:

    a. Characterize the relationship between floor dust-lead levels and blood-lead concentration in young children and how this relationship differs for carpeted and uncarpeted floors (with and without adjusting for the effects of key demographic variables and for lead levels in other media in which standards have been proposed in the §403 rule).

    b. Determine the added value of including a carpet dust-lead standard given the proposed §403 standards for soil, window sills and uncarpeted floors, or expanding the definition of floors in the rule to include carpeted as well as uncarpeted floors.

2. Identify appropriate candidates for carpeted floor dust-lead standards and, in particular, whether one candidate standard should correspond to 50 µg/ft^2, the uncarpeted floor dust-lead standard from the §403 proposed rule.

3. Determine whether the wipe technique is acceptable for sampling dust from carpeted floors for evaluating the risk of lead exposure associated with carpet-dust, or whether alternative vacuum methods are more appropriate.

A more detailed presentation of the statistical analyses that address these three objectives is found in Appendix I of this report.

The carpet dust-lead measurement data used in this analysis originated from two lead exposure studies: the Rochester (NY) Lead-in-Dust study, and the pre-intervention, evaluation phase of the HUD Lead-Based Paint Hazard Control Grant ("HUD Grantees") Program (data collected through September, 1997). Both studies were introduced in Section 3.3.1 of the §403 risk analysis report; additional details on these studies that are relevant to this analysis is presented in Section I3.1 of Appendix I. The results of this analysis, along with relevant findings documented in EPA's recent literature review report on lead exposure associated with carpets, furniture, and air ducts (USEPA, 1997b), were used to address the above objectives.

The summary of the analysis results now follows. It is formatted according to the above three objectives. References to statistical significance are made at the 0.05 level. Unless otherwise indicated, references to dust-lead loadings are assumed to be for samples collected using wipe techniques. Section numbers within Appendix I are specified in parentheses where additional information can be found.

Objective #1: Is there a need to have dust-lead on carpeted floors addressed by the §403 rule?

- Using data collected in the 1997 American Housing Survey, EPA estimates that approximately 54 million housing units built prior to 1978 contain some wall-to-wall carpeting. Of these units, wall-to-wall carpeting is found in a living room in approximately 47 million units and in a bedroom in approximately 46 million units (i.e., rooms in which children reside and play most frequently, and therefore, would be targeted in a risk assessment).

- While the §403 proposed rule indicates that lead from floor dust is an important exposure source for children, the proposed floor dust-lead loading standard was only relevant for uncarpeted floors. In homes with wall-to-wall carpeting, it is expected that floor-dust samples in certain rooms can come only from carpeted floors. While no guidance was given in the §403 proposed rule on a standard to which risk assessors should compare the results of lead analyses for carpet dust samples, EPA recognizes (and many commenters on the §403 proposed rule have noted) that some recommendation for a carpet dust-lead loading standard, based on using wipe collection techniques, is necessary.

- Because children come in frequent direct contact with carpeting when it is present in their homes, any lead that may be present in carpet dust is likely to be bioavailable to children.

Objective #1a: Is there any association between carpeted floor dust-lead loadings and blood-lead concentration?

- For both carpeted and uncarpeted floors in the two studies, the correlation between household average floor (wipe) dust-lead loading and children's blood-lead concentration was positive and significantly different from zero. (Sections I4.1.1.1 and I5.1.1.1 of Appendix I)

- No evidence was found in these analyses to suggest that wipe dust-lead loadings from uncarpeted floors are a better predictor of children's blood-lead concentration than wipe dust-lead loadings from carpeted floors. (Sections I4.1.1.2, I4.1.1.4, I5.1.1.2 and I5.1.1.4 of Appendix I)

- No significant difference in the statistical relationship between average floor dust-lead loading and blood-lead concentration was found between homes with floor dust sampling conducted from mostly carpeted floors and homes with sampling from mostly uncarpeted floors. (Sections I4.1.1.3 and I5.1.1.3 of Appendix I)

- Mixed results were found when investigating whether the effect of average carpeted floor dust-lead loading on blood-lead concentration remained significant after adjusting for the effects of lead levels in soil, window sill dust, and uncarpeted floor dust (i.e., other environmental media addressed by the proposed §403 standards). The carpet dust-lead loading effect was no longer statistically significant after adjusting for these other effects when analyzing data from the Rochester study, while the effect remained statistically significant when analyzing data from the HUD Grantees program evaluation. (Sections I4.1.2 and I5.1.2 of Appendix I)

- When interpreted as a whole, these findings provide a powerful argument for expanding the floor dust-lead standard in the §403 rule to include carpeted floors.

Objective #1b: Is there any added benefit to adding a carpeted floor dust-lead loading standard to the proposed §403 standards for lead in soil, window sill dust, and dust from uncarpeted floors, or to expanding the definition of floors in the rule to include carpeted floors? (Sections I4.1.3 and I5.1.3 of Appendix I)

- The extent of any added benefit is dependent on the value of the carpet dust-lead loading standard and the particular criteria being considered in evaluating performance. Adding a new standard to a set of existing standards will not reduce sensitivity (i.e., the proportion of homes with elevated blood-lead children that are

triggered by the set of standards), but it also will not increase specificity (i.e., the proportion of homes with no elevated blood-lead children that are not triggered for an intervention by the standards).

- If the uncarpeted floor dust-lead loading standard of 50 $\mu g/ft^2$ that was proposed in the §403 proposed rule was extended to included carpeted floors as well, the resulting performance of the§403 proposed standards (based on the outcome of performance characteristics analysis) changed little, if any. If a carpeted floor standard of 40 $\mu g/ft^2$ was added to the §403 proposed standards, slight improvements in the performance characteristics were noticed. These findings were observed regardless of whether or not uncarpeted floors were available to sample (i.e., whether or not the uncarpeted floor standard was considered).

- Analyses of the Rochester study data indicated that adding a carpet dust-lead loading standard of approximately 17 $\mu g/ft^2$ to the proposed §403 standards considerably improved certain performance characteristics, particularly sensitivity, without a large decrease in specificity.

- Analysis of the HUD Grantees evaluation data indicated that adding a carpet dust-lead loading standard of approximately 5 $\mu g/ft^2$ improved sensitivity and negative predictive value (NPV, equal to the proportion of homes not triggered for intervention by the standards that do not contain elevated blood-lead concentration), but was accompanied by a considerable decrease in specificity. If the proposed carpet dust-lead loading standard was increased to approximately 13 $\mu g/ft^2$, this loss of specificity relative to the gains in sensitivity and NPV was reduced.

- In general, these analyses concluded that expanding the proposed §403 floor dust-lead standard (of 50 $\mu g/ft^2$) to encompass both carpeted and uncarpeted floors, or setting this standard slightly lower at 40 $\mu g/ft^2$, would not lead to a large decrease in specificity, but it would tend to result in only minor increases in sensitivity from what was observed when carpeted floor standards were not being considered.

Objective #2: If a carpeted floor standard is needed, what should it be? Should it be different from the proposed uncarpeted floor dust-lead loading standard of 50 $\mu g/ft^2$?

- The findings listed above for Objective #1b suggest that it may provide an advantage to have a standard for carpeted floors that is lower than the standard for uncarpeted floors. (Sections I4.1.1.2, I4.2.1, I5.1.1.2 and I5.2.1 of Appendix I)

- Having a floor dust-lead loading standard of 40 to 50 $\mu g/ft^2$ that is expanded to represent carpeted floors as well as uncarpeted floors would be at least as protective of children (in terms of the predicted blood-lead concentration at which

95% of children exposed at the standard level would be expected to fall below) than if the standard represented only uncarpeted floors. (Sections I4.2.2 and I5.2.2 of Appendix I)

- When the Rochester study data was used in a performance characteristics analysis that considered only standards for either carpeted or uncarpeted floors (Sections I4.2.3 and I5.2.3 of Appendix I), a carpeted floor dust-lead loading standard in the range of 15 to 20 $\mu g/ft^2$ maximized the total of the four performance characteristics. In contrast, a standard of 50 $\mu g/ft^2$ resulted in considerably lower performance when the standard was for carpeted floors versus uncarpeted floors. The level of sensitivity achieved by an uncarpeted floor dust-lead loading standard of 50 $\mu g/ft^2$ was achieved for carpeted floor dust-lead loading standards below approximately 33 $\mu g/ft^2$. However, the uncertainty associated with these estimates may suggest that these lower levels may not actually differ from a practical standpoint from the uncarpeted floor dust-lead loading standard in the §403 rule.

Objective #3: What dust sampling method should be used on carpeted floors? (Sections I4.3 and I5.3 of Appendix I)

- The HUD Guidelines (USHUD, 1995) support the use of wipe methods to sample carpet dust. Participants in the §403 Dialogue Group meetings raised concerns that requiring widespread use of vacuum techniques for collecting dust samples in typical risk assessments would be impractical. Therefore, it would be preferable to allow wipe sampling as an option for collecting dust samples from carpets in a risk assessment unless wipe techniques were totally unacceptable.

- Different types of dust collection methods can collect different amounts of lead within a dust sample, especially when sampling from carpets where surface dust is easier to sample than dust that is deep within the carpet fibers. A laboratory study done in conjunction with the Rochester study (Emond et al., 1997) concluded that lead recovery from carpet dust was highest with the BRM vacuum (95.2%) compared to the wipe (24.4%) and the DVM vacuum (31.4%). For this reason, different dust collection methods for collecting carpet dust would require different lead standards to which to compare the results.

- When the wipe method is used on carpets, it tends to collect only dust on the carpet surface that can readily be removed by the method. This surface dust is also that which is most likely to come into direct contact with children (USEPA, 1997b).

- Blood-lead concentration tends to be more highly associated with dust-lead loading than with dust-lead concentration in carpets. (Only dust-lead loadings can be measured under wipe techniques, while loadings or concentrations can be measured under vacuum methods.) This contributes to the technical justification

that a carpet dust-lead standard would be better conveyed as a loading than as a concentration.

- Each of the three dust collection methods considered in the Rochester study (BRM vacuum, DVM vacuum, wipe) collected carpet dust samples whose dust-lead loadings were statistically associated with blood-lead concentration, with the level of association being similar for each method.

- On both carpeted and uncarpeted floors, dust-lead loading measurements from different dust collection methods were significantly positively correlated. This suggests that using any of the three methods (including wipe) would portray the extent of a carpet dust-lead hazard in a similar fashion.

- As wipe sampling is currently the method of choice for uncarpeted floors and all three methods have significant correlations with blood-lead concentration for carpeted and uncarpeted floors, it is reasonable to develop a carpeted floor dust-lead loading standard for the wipe sampling method. As this standard would not apply to vacuum sampled dust-lead loadings, measurements for samples collected using vacuum techniques could not be directly used in risk assessment via the §403 rule without first being converted to wipe-equivalent loadings using methods such as those documented in Section 4.3 of the §403 risk analysis report.

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15. Supplementary Notes

Support staff involved in the risk analysis and the production of this report included Halsey Boyd, Ying-Liang Chou, and Kensuke Shirakawa.

16. Abstract (Limit 200 words)

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. As part of Title X, the Toxic Substances Control Act (TSCA) was amended to include Title IV, "Lead Exposure Reduction". Section 403 of TSCA requires EPA to define standards for lead in paint, dust, and soil. Federal, state, and local public health agencies, as well as private property owners and other private sector interests, will use these standards to determine in which homes actions should be taken to reduce or prevent the threat of childhood lead poisoning.

This report is a supplement to EPA 747-R-97-006 (June 1998), which documented the outcome of a risk analysis that supported EPA's efforts to propose standards in response to Section 403. The additional literature reviews, data summaries, and data analyses presented in this report focus on selected topics raised in comments on the risk analysis that were made by EPA's Science Advisory Board and the general public. EPA has cited the findings of this report when preparing responses to these comments.

17. Document Analysis

a. Descriptors

Blood-lead concentration, childhood health risks, adverse health effects, dust-lead loading, soil-lead concentration, lead hazards, lead-based paint, risk assessment, risk characterization, risk management

b. Identifiers/Open-Ended Terms

abatement, intervention, hazard identification, exposure assessment, dose-response assessment, HUD National Survey, Rochester Lead-in-Dust Study, Evaluation of the HUD Lead-Based Paint Hazard Control Grant Program, NHANES III, bioavailability, modeling, performance characteristics analysis, sensitivity and uncertainty analysis, Section 403, Title X

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