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The Contribution of Lead-Contaminated House Dust and Residential Soil to Children's Blood Lead Levels

A Pooled Analysis of 12 Epidemiologic Studies[1]

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In 1992, the U.S. Congress passed the Residential Lead-Based Paint Hazard Reduction Act, which requires the promulgation of health-based dust lead and soil lead standards for residential dwellings to prevent undue lead exposure in children. Unfortunately, the levels of lead in house dust and soil that are associated with elevated blood lead levels among U.S. children remain poorly defined. This pooled analysis was done to estimate the contributions of lead-contaminated house dust and soil to children's blood lead levels. The results of this pooled analysis, the most comprehensive existing epidemiologic analysis of childhood lead exposure, confirm that lead-contaminated house dust is the major source of lead exposure for children. These analyses further demonstrate that a strong relationship between interior dust lead loading and children's blood lead levels persists at dust lead levels considerably below the U.S. Department of Housing and Urban Development's current postabatement standards and the Environmental Protection Agency's guidance levels. Finally, these analyses demonstrate that a child's age, race, mouthing behaviors, and study-site specific factors influence the predicted blood lead level at a given level of exposure. These data can be used to estimate the potential health impact of alternative health-based lead standards for residential sources of lead exposure.

Key Words: **blood lead; lead-contaminated house dust; soil; lead exposure; risk assessment; children; environmental exposure; prevention; standards; lead poisoning.**

INTRODUCTION

Levels of lead in house dust and soil that are hazardous to children remain poorly defined. The U.S. Environmental Protection Agency (EPA) recently adopted the U.S. Department of Housing and Urban Development postabatement clearance standards (by using a wipe method) for use as interim health-based guidance levels: 100 μg/ft^2 for floors; 500 μg/ft^2 for interior window sills; and 800 μg/ft^2 for window troughs (EPA, 1994a).[2] These dust levels were based on earlier standards set by the state of Maryland, which were promulgated in 1988 when blood lead levels of 25 μg/dL were considered acceptable (Code of Maryland, 1988). Moreover, these levels were based on limited data and do not appear to adequately protect children from undue lead exposure, which is currently defined as a blood lead

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[2] 1 square foot = 0.0929 square meter.

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level of 10 μg/dL or higher (Clark, 1995; Lanphear, 1996).

Setting health-based lead standards for house dust and residential soil is difficult. For example, some scientists disagree about whether an empirical or a mechanistic model should be used to develop health-based standards or whether dust lead loading (μg of lead per unit surface area) or dust lead concentration (μg of lead per gram of dust) should be used as the unit of measure for the dust standard. It has also been argued that epidemiologic studies are not useful for developing standards because the estimated contribution of lead from dust and soil often differed across studies. Since many of these studies often used different sampling methods or laboratory assays, sampled various surfaces, did not always adjust for other sources of lead, and included children of different ages, it is not surprising that the relationship between residential lead sources and children's blood lead levels varied across studies (Lanphear, 1996). Furthermore, variation across studies can itself be a useful source of information. Regardless, epidemiologic data provide the only direct measure of the relationship between lead-contaminated dust and soil with children's blood lead levels.

Earlier studies examining the relationship of lead-contaminated dust and soil with children's blood lead levels often consisted of a small group of children in a single community. It was therefore difficult to generalize from existing epidemiologic data to U.S. children. The purpose of this analysis was to provide estimates of the contribution of lead-contaminated house dust and residential soil to children's blood lead levels for setting health-based standards by conducting a pooled analysis of 12 epidemiologic studies in multiple communities.

METHODS

Inclusion criteria were developed for epidemiologic studies and, within each study, for individual children to be included in the pooled analysis. These inclusion criteria were:

- Well-defined sampling protocols for blood lead and environmental lead
- Measures of lead contamination of dust and soil were present
- Measures of paint lead content (X-ray fluorescence) and condition were available
- Dust samples collected with wipe or method which can be converted to wipe
- Dust lead samples must be taken within 3 months of the blood lead level
- Children were not selected on the basis of having a high blood lead level
- Ability to obtain and reanalyze original data sets
- Children were 6 to 36 months of age

Eighteen published and unpublished epidemiologic studies of lead-exposed children were identified (Tables 1 and 2). Because blood lead levels change in response to alterations in environmental lead, studies which attempted to alter the relationship of children's exposure to lead were not included, with the exception of their baseline results (i.e., at the time of entry into the study). Thus, only cross sectional data were included in the pooled analysis. Subjects and studies were both selected by using specific criteria to allow for inferences about the causal relationship between lead exposure and children's blood lead levels.

Each study included in the pooled analysis had to have a well-defined sampling protocol with respect to blood lead and principal lead sources (i.e., paint lead and soil lead). Data which were essential for inclusion into the pooled analysis were soil lead levels, paint lead levels, and paint condition (Tables 1 and 2). By contrast, water lead and air lead data were not considered essential. Water lead exposure is unlikely to strongly correlate with dust or soil lead and air lead contributes only a small amount to blood lead via direct inhalation (most of its impact is via ingestion of lead deposition in dust and soil). Therefore, failure to adjust for water and air lead levels is unlikely to bias the estimate of the slope for blood lead versus lead-contaminated house dust or residential soil.

Based on earlier research, cost, and ease of use, the U.S. Department of Housing and Urban Development has decided to use the wipe method to sample dust for clearance testing following residential lead hazard control work (U.S. HUD, 1995; Lanphear, 1995). It was therefore stipulated that *wipe* loading was to be used to establish the relationship of dust lead and children's blood lead levels. Thus, all studies in this pooled analysis collected dust by using either wipes or a dust sampling method that was able to be converted to estimates of lead loading as measured by wipe samples. Since reliable (i.e., side-by-side) data to convert vacuum methods to wipe loading were only available for the BRM (Baltimore Repair and Maintenance) sampler, a modified high-velocity cyclone sampler, and the DVM (or Microvac) sampler (Farfel, 1994; Lanphear,

TABLE 1

Epidemiologic Studies of Childhood Lead Exposure in Urban Settings

Reference (year of publication)	Age group (months)[a]	Blood sample[b]	Dust collection method	Sample	Study design	Composite samples[c]	Soil lead	Paint lead	Water lead	In pooled analysis
Bornschein (1985) Cincinnati Longitudinal	9 to 24	V	DVM	Convenience	Longitudinal	N	Y	Y	N	Y
Rabinowitz (1985) Boston Longitudinal	1 to 24	F	Wipe	Convenience	Longitudinal	N	Y	Y	Y	Y
Davies (1990) UK Study	24	V	Vacuum	Random	Cross section	Y	Y	Y	N	N
Weitzman (1993) Boston Soil Study	0–60	V	Sirchee-Spittler	Convenience	Longitudinal	Y	Y	Y	Y	N
Clark(1996) Cincinnati Soil Study	0–72	V	DVM	Convenience	Longitudinal	Y	Y	Y	Y	Y
Chisolm (1996) Baltimore Soil	0–72	V	Sirchee-Spittler	Convenience	Longitudinal	N	Y	Y	N	N
Lanphear (1996) LID Study	12–31	V	BRM Wipe DVM	Random	Cross section	N	Y	Y	Y	Y
Donovan (1996) National Survey	12–59	V	Wipe	Random	Cross section	N	Y	Y	Y	N
Farfel (1996) R & M Study	6–48	V	BRM	Stratified convenience	Longitudinal	Y	Y	Y	Y	N
Lanphear Rochester Longitudinal Study	6	V	Wipe	Convenience	Longitudinal	Y	Y	Y	Y	Y

Note. DVM, dust vacuum method or Microvac sampler; BRM, Baltimore Repair and Maintenance Sampling method.

[a] Age is described at baseline for intervention studies and as "_ to _" for longitudinal observational studies to indicate that various age cut-offs could be analyzed.

[b] V, veripuncture; and F, capillary fingerstick.

[c] Carpeted and hard floor dust samples were composited.

1995), studies using other sampling methods were excluded from this pooled analysis (Table 1).

Some studies were not included in the pooled analysis because they did not fulfill the inclusion criteria. Studies conducted in the 1970s typically examined children with blood lead levels usually higher than 40 μg/dL by using a case control design (Sayre, 1974; Charney, 1980). To minimize selection bias, these studies were not included in the pooled analysis since the children were selected on the basis of having a high blood lead level. Some studies were excluded because they used a dust sampling method other than the wipe, DVM, or BRM (Davies, 1990; Weitzman, 1993; Kimbrough, 1994). Finally, data from the Australian National Survey were not comparable with other data sets because there were extremely limited data on paint lead levels. Moreover, the paint lead values available from the Australian National Survey were based on paint chip samples. Paint chip analyses are reported in weight percentage and cannot be translated into units compatible with those obtained with the X-ray fluorescence analyzer (mg/cm^2), the instrument which was used in all the other studies (Donovan, 1996).

Since the analysis was to be achieved by conducting a pooled analysis of individuals, the ability to obtain the original data sets was a necessary condition for inclusion. This criterion did not exclude any studies which fit other inclusion criteria, with the exception of the Baltimore Repair and Maintenance Study (Farfel, 1996). All other identified data sets, including the Cincinnati Longitudinal Study (Bornschein, 1985), the Boston Longitudinal Study (Rabinowitz, 1985), the Cincinnati Soil Abatement Study (Clark, 1990), and the Rochester Lead-in-Dust Study (Lanphear, 1995) were available for the pooled analysis (Table 1). Data sets from numerous industrial (i.e., smelter, mining, or milling) communities and the ongoing lead exposure prevention trial in Rochester also were available. (Tables 1 and 2)

Children who are 6 to 36 months old are the most likely to demonstrate the clearest relationship between dust lead and blood lead and between soil lead and blood lead (i.e., their behavior places them at greatest risk for exposure to lead-contaminated dust and soil, and their blood lead levels are more likely to represent recent exposure). Older children obviously ingest lead-contaminated dust and soil, but their

TABLE 2

Epidemiologic Studies of Childhood Lead Exposure in Mining, and Smelting Communities

Reference Site (year of study)	Age group (months)	Blood sample[a]	Dust collection method	Location	Sample population	Study design	Composite dust samples	Soil lead	Paint lead	Water lead	In pooled analysis
Telluride, CO (1987)	0–72	V	DVM	Lead mill	Total population	Cross section	Y	Y	Y	Y	N
Midvale, UT (1989)	0–72	V	DVM	Mill & smelter	Stratified random	Cross section	Y	Y	Y	Y	Y
Butte, MT (1990)	0–72	V	DVM HVS Wipe	Mine, mill, & smelter	Total population	Cross section	Y	Y	Y	Y	Y
Leadville, CO (1991)	0–72	V	DVM	Mine, mill & smelter	Total population	Cross section	Y	Y	Y	Y	Y
Bingham Creek, UT (1993)	0–72	V	DVM	Copper & lead mills	Total population	Cross section	Y	Y	Y	Y	Y
Magna, UT (1994)	0–72	V	DVM	Copper smelter[b]	Stratified random	Cross section	Y	Y	Y	Y	Y
Sandly, UT (1994)	0–72	V	DVM	Lead smelters	Stratified random	Cross section	Y	Y	Y	Y	Y
Palmerton, PA (1994)	0–72	V	DVM	Zinc smelter	Total population	Cross section	Y	Y	Y	N	Y
Trail, BC (1992)	0–72	V	DVM	Lead & Zinc smelter*	Convenience	Cross section	Y	Y	Y	Y	N

[a] Age is described at baseline for intervention studies and as "_ to _" for longitudinal observational studies to indicate that various age cut-offs could be analyzed.

[b] Active smelter, all other sites are not active.

blood lead levels are largely influenced by past lead exposure (Clark 1991; R. Bornschein, unpublished data). Thus, attenuation of the association between lead exposures and older children's current blood lead levels resulting from more time spent away from their residence and bone lead stores outweighed any advantages of including older children in this pooled analysis. Furthermore, if promulgated lead standards protect children who are between 6 and 36 months of age, older children should also be protected.

Statistical Analyses

As previously noted, the U.S. Department of Housing and Urban Development has indicated a policy preference for dust lead loadings taken with a wipe over those taken with a vacuum. However, not all studies collected interior dust lead samples by using wipes; some used vacuum samplers. It was therefore necessary to convert the DVM values to "statistically equivalent" wipe values. Data for the wipe-DVM conversion were available from paired side-by-side wipe, DVM, and BRM dust-lead loading measurements on hard and carpeted floors from a study conducted in Butte, MT (NCLSH, 1994). The correlations among the three dust measurements were all greater than 0.76. A conversion equation was developed (J. Rogers *et al.*, in preparation) by using structural equation modeling (Bollen, 1989).

Because data from the 12 studies with different sampling and data collection procedures were combined, the definitions of some variables needed to be standardized. For studies with more than one child sampled per household, one child in each household was randomly selected. For the longitudinal studies, one set of measurements for each child was randomly selected from the repeat measures after accounting for inclusion criteria considerations, such as age and availability of environmental lead exposure measurements. The condition of the paint, the parents' socioeconomic status, and the child's mouthing behavior were standardized.

The contribution of lead-contaminated soil was estimated for the pooled analysis. In some studies soil samples were collected from the perimeter of the foundation, where lead levels are higher than midyard samples due to deposition of dust or chips from weathered exterior paint. Soil samples were also

sometimes collected from targeted "play" areas or at random locations in a yard. In some cases, soil was not present. The depth of sampling also varied across studies. For soil lead, differences in the following characteristics of soil samples were ignored in combining measurements from different studies: the depth of the sample taken (as long as it included surface soil), the type of surface on which the sample was taken (bare versus covered), the type of chemical treatment (e.g., acid digestion) used in preparing samples for analysis, and the number of locations and subsamples composited prior to measurement. Perimeter soil samples were preferentially used over mid-yard or play area samples because they were available from more studies. If more than one particle size fraction was analyzed the fine particle size soil lead level was used in the pooled analysis. Finally, when soil samples were not available, exterior dust was substituted for soil lead levels for the purposes of this pooled analysis.

Although water was not used as a selection criteria, water lead measurements were available for many observations. Water lead was included in the analyses and missing values were imputed as described below. The data sets for the Cincinnati Soil study and the study in Palmerton are both missing water lead for all observations. The proportion of water lead observations missing in the remaining studies ranges from 0 to 12%. Missing water lead observations were imputed with random values from a log normal distribution around the geometric mean for the respective study. For the Cincinnati Soil Program and Palmerton studies, average community water lead levels in Cincinnati, OH, and Palmerton, PA, were used to generate the respective log normal distributions.

All four of the environmental lead variables in the pooled analyses (dust, soil, paint, and water) had some values reported below the respective detection limit. These missing values were replaced with random values below the detection limit generated from a log normal distribution fit to the data values above the detection limit.

Several other variables that have previously been shown to modify the relationship of lead exposure and children's blood lead levels were examined. To address seasonal variation in blood lead levels, blood lead levels and dust samples were required to be taken within 3 months of each other. A variable indicating the season in which the blood, dust, and other lead measurements were taken was examined in the analysis. A variable for exterior dust was also included when, substituted for soil. Similarly, variables were included in the data set to "flag" industrial, mining, or smelter communities and year the study was conducted. Finally, age of child, race, sex, and socioeconomic variables were evaluated as potential effect modifiers.

Dust lead loading at both extremes was modified for the pooled analysis. Dust lead loadings collected with the DVM which were (1) collected from carpeted floors and greater than 100 $\mu g/ft^2$ or (2) collected from uncarpeted floors and less than 0.32 $\mu g/ft^2$ were not included in the DVM to wipe conversion equation. As a result, 9 (0.7%) of 1306 observations in the pooled data with dust lead loadings were excluded from the analysis. Dust lead levels below the detection limit were randomly assigned to a value below the detection limit based on a log-normal distribution.

Several model structures were considered for predicting blood lead concentration from environmental lead variables, including modeling the blood lead concentration as a linear function of the environmental lead levels (with an appropriate error structure) and modeling the log transformed blood lead concentrations as a linear function of the log transformed environmental lead variables. After review of the alternate approaches, the second model structure was selected for this analysis because it has been traditionally used by other researchers (Charney, 1980; Clark, 1985; Davies, 1990; Donovan, 1996; Farfel, 1990; Lanphear, 1996; Rabinowitz, 1985) and it provided a better prediction of blood lead levels, as judged by the correlation among the measured and predicted log transformed blood lead (Jiang, 1996; Rust, 1997). Correlations among the log transformed predicted blood lead concentrations from the different model structure were greater than 0.92. Comparison of the different model structures will be presented elsewhere.

The model used in the blood lead–environmental lead analyses in this current analysis predicts the log transformed child's blood lead as a linear function of log transformed measures of environmental exposure. The following variables were included in the pooled analysis:

- Interior floor dust lead loading ($\mu g/ft^2$)
- Exterior lead exposure from perimeter soil, play area soil, or exterior dust (ppm)
- Maximum interior paint lead content (mg/cm^2)
- Household water lead (ppb)
- Paint hazard: 1 if the paint is damaged, 0 otherwise
- Name of study: 1 of 12 studies (categorical)
- Child's race: White and Other (or Minority) (categorical)

- Child's age (months)
- Socioeconomic status of the child's family: 1 (low), 2, 3, 4, 5 (high) (categorical)
- Child's mouthing behavior: Often, Sometimes, Rarely, Unknown (categorical)

Estimates of the parameters were generated using the SAS procedure PROC GENMOD using normal errors and an identity link. For this model, multiple regression would generate the same results as PROC GENMOD. Because all independent environmental lead variables are accompanied by some error, the parameters obtained are biased estimates of the causal contribution of the true environmental exposures to the child's blood lead levels. Simulation extrapolation (SIMEX) was used to adjust the parameter estimates for the effect of measurement error (Carroll, 1995). For each environmental exposure measure, the variance among multiple measurements within the same home and between similar homes was used to estimate the measurement error variance used in the SIMEX procedure.

The model predictions vary according to the conditions to which a child is exposed. The median environmental lead levels in U.S. housing extrapolated from the 1989–1990 U.S. Department of Housing and Urban Development National Survey were 5.0 $\mu g/ft^2$ for dust lead loading (estimated by conversion of the Blue Nozzle sampling method to the wipe method (NCLSH, 1994)), 72 ppm for soil lead concentration, and 1.6 mg/cm^2 for maximum interior paint lead concentration. A median water lead concentration of 1 ppb was estimated from the pooled data and other sources and used to represent a national median for water lead concentration. For the purposes of illustrating the model predictions as a function of dust lead loading and soil or exterior dust lead exposure, predicted blood lead concentrations correspond to those for a 16-month-old child (i.e., the mean age of the child in the pooled analysis) and, unless otherwise indicated, exposed to median environmental lead exposures. The effects for categorical variables were set to the arithmetic mean effect across the population represented by the study data, except that we assumed children were exposed to undamaged paint and that perimeter soil samples were obtained.

RESULTS

Twelve of the 18 studies identified were included in the pooled analysis. Characteristics of the children and environmental exposures that were included in the analysis are shown in Table 3. The studies were conducted over a 15-year time period,

TABLE 3

Descriptive Statistics of the Key Variables in the Blood Lead–Environmental Lead Analysis

				Geometric Means for Observations used in Analyses						
Study	Study sample size	Number of observations used	Percentage blood lead ≥10 μg/dL	Blood lead (μg/dL)	Dust lead loading (μg/dL)	Exterior lead exposure (ppm)	Maximum paint XRF (mg/sm^2)	Percentage max XRF on damaged paint	Mean age (months)	Mean SES level
Boston Longitudinal Study	175	40	28%	4.29	2.29	247.01	0.83	30%	13.5	1.7
Cincinnati Longitudinal Study	285	250	54%	11.17	293.40	472.36	3.12	43%	13.6	1.7
Cincinnati Soil Study	99	52	62%	10.44	20.37	965.51	0.75	0%	20.0	1.9
Rochester Longitudinal Study	274	264	2%	2.86	8.30	914.19	5.36	10%	6.1	2.9
Rochester LID Study	205	195	22%	6.33	17.79	689.67	7.12	54%	20.4	2.4
Butte, MT Study	118	110	6%	3.60	2.50	519.61	2.45	1%	21.1	2.9
Bingham Creek, UT Study	335	100	2%	3.20	1.92	96.91	0.58	0%	23.7	3.4
Leadville, CO Study	108	84	12%	4.92	4.73	755.01	1.62	1%	20.2	2.8
Magna, UT Study	64	54	11%	4.45	8.87	247.43	2.97	4%	21.3	2.5
Sandy, UT Study	46	40	0%	3.15	6.11	415.97	1.58	3%	22.3	3.0
Midvale, UT Study	86	65	12%	4.62	3.68	326.96	0.99	0%	19.6	2.5
Palmerton, PA Study	45	43	7%	4.74	5.91	581.87	0.28	2%	20.8	3.0
All studies	1,861	1,297	20%	5.07	13.52	508.61	2.46	20%	16.3	2.5

beginning in 1982 and continuing until 1997. On average, 70% (range = 23 to 96%) of children were included from each of the 12 studies. There were a variety of reasons for the exclusion of specific children; the majority of exclusions were due to a child being outside the 6- to 36-month age range or missing data on key environmental variables.

The arithmetic mean age of children included in the pooled analysis was 16 months. The geometric mean blood lead level of children was 5.1 μg/dL, with 95% of the blood lead levels between 1.2 and 26 μg dL; 19% of children had a blood lead level of 10 μg dL or higher. The geometric mean floor dust lead loading and soil lead levels were 13.5 μg/ft^2 and 508 ppm, respectively (Table 3). Across all studies, 95% of floor dust loadings were between 1.0 and 4500 μg/ft^2 and 95% of exterior lead exposure concentrations were between 8 and 10,200 ppm. In general, children who lived in urban areas had higher blood lead levels and higher exposures to environmental lead levels than did children living in suburban or rural areas or in towns with nearby lead-related industries.

In the multivariate regression, floor dust lead loading was the most significant environmental predictor of children's blood lead levels (see Appendix for full model). To a lesser extent, lead-contaminated soil contributed to children's lead intake. Child's age, mouthing behaviors, and race were also significant predictors of children's blood lead levels (See the Appendix for details). The R^2 for this model, prior to adjusting for measurement error, was 0.53.

To examine the contribution of floor dust lead loading at levels below 10 μg/ft^2, we conducted some additional analyses. If we restricted the data to only include cases with floor dust lead loading below 10 μg/ft^2 (53% of the data), dust lead loading remained the most significant environmental predictor of children's blood lead levels, based on the model uncorrected for measurement error ($P < 0.0001$).

The estimated geometric mean blood lead levels and the proportion of children with a blood lead level of 10 or 15 μg/dL are shown (Tables 4–6 and Figs. 1–4). At a floor lead loading of 10 μg/ft^2 and soil lead exposure of 72 ppm, that is, a dust lead level that is 10-fold lower than the current EPA guidance level for floor dust at the estimated median soil lead level for residential housing in the United States, the geometric mean blood lead levels observed were 4.6 μg/dL and 7.4% of children had a blood lead

TABLE 4

Children's Predicted Blood Lead Levels for Floor Dust Lead Loading (μg/ft^2) and Exterior Lead Exposures (ppm)[a]

	Geometric mean blood lead levels (μg/dL) with 90% Confidence Intervals[a] in parentheses							
	Exterior lead exposure (ppm)							
Dust lead loading (μg/ft^2)	10	72[b]	100	500	1000	1500	2000	4000
1	2.3 (0.9, 5.7)	2.8 (1.1, 7.0)	2.9 (1.2, 7.3)	3.5 (1.4, 8.7)	3.8 (1.5, 9.4)	4.0 (1.6, 9.8)	4.1 (1.6, 10.1)	4.4 (1.8, 11.0)
5	3.2 (1.3, 8.0)	4.0 (1.6, 9.8)	4.1 (1.7, 10.1)	4.9 (2.0, 12.0)	5.3 (2.1, 13.0)	5.5 (2.2, 13.6)	5.7 (2.3, 14.0)	6.1 (2.5, 15.2)
10	3.7 (1.5, 9.2)	4.6 (1.8, 11.3)	4.7 (1.9, 11.7)	5.6 (2.3, 13.9)	6.1 (2.5, 15.0)	6.3 (2.6, 15.7)	6.5 (2.7, 16.2)	7.1 (2.9, 17.5)
15	4.0 (1.6, 10.0)	5.0 (2.0, 12.3)	5.1 (2.1, 12.7)	6.1 (2.5, 15.1)	6.6 (2.7, 16.3)	6.9 (2.8, 17.0)	7.1 (2.9, 17.6)	7.7 (3.1, 19.0)
20	4.2 (1.7, 10.6)	5.3 (2.1, 13.0)	5.4 (2.2, 13.5)	6.5 (2.6, 16.0)	7.0 (2.8, 17.3)	7.3 (3.0, 18.0)	7.6 (3.1, 18.6)	8.1 (3.3, 20.1)
25	4.4 (1.8, 11.2)	5.5 (2.2, 13.6)	5.7 (2.3, 14.1)	6.8 (2.8, 16.8)	7.3 (3.0, 18.1)	7.7 (3.1, 18.9)	7.9 (3.2, 19.5)	8.5 (3.5, 21.1)
40	4.9 (1.9, 12.3)	6.1 (2.4, 15.0)	6.3 (2.5, 15.6)	7.5 (3.0, 18.5)	8.1 (3.3, 19.9)	8.4 (3.4, 20.8)	8.7 (3.5, 21.5)	9.4 (3.8, 23.2)
55	5.2 (2.1, 13.2)	6.5 (2.6, 16.1)	6.7 (2.7, 16.6)	8.0 (3.2, 19.7)	8.6 (3.5, 21.3)	9.0 (3.7, 22.2)	9.3 (3.8, 22.9)	10.0 (4.1, 24.8)
70	5.5 (2.2, 13.8)	6.8 (2.7, 16.9)	7.0 (2.8, 17.5)	8.4 (3.4, 20.7)	9.1 (3.7, 22.3)	9.5 (3.8, 23.4)	9.8 (4.0, 24.1)	10.5 (4.3, 26.0)
100	5.9 (2.3, 14.9)	7.3 (2.9, 18.2)	7.6 (3.1, 18.9)	9.0 (3.7, 22.3)	9.7 (3.9, 24.1)	10.2 (4.1, 25.2)	10.5 (4.3, 26.0)	11.3 (4.6, 28.0)

[a] Confidence interval is estimated to cover 90% of the observed blood lead levels with 5% above and 5% below the interval.
[b] Estimated median levels based on U.S. Housing and Urban Development national survey, 1989–1990

TABLE 5

Likelihood of a Child's Blood Lead ≥ 10 μg/dL for Floor Dust Lead Loadings and Exterior Exposure Levels (ppm)[a]

Dust lead loading (μg/ft²)	Probability of blood lead greater than 10 μg/dL							
	Exterior lead exposure (ppm)							
	10	72[b]	100	500	1000	1500	2000	4000
1	0.33% (0.05, 2.24)	1.0% (0.3, 3.8)	1.2% (0.3, 4.2)	2.7% (0.9, 7.4)	3.7% (1.3, 9.7)	4.4% (1.6, 11.5)	4.9% (1.8, 12.8)	6.5% (2.3, 16.9)
5	1.8% (0.4, 7.9)	4.4% (1.7, 11.0)	5.0% (2.0, 11.8)	9.3% (4.7, 17.6)	12% (6, 21)	14% (7, 24)	15% (8, 26)	18% (9, 32)
10	3.3% (0.8, 12.6)	7.4% (3.1, 16.5)	8.3% (3.8, 17.5)	14% (8, 24)	18% (10, 29)	20% (12, 32)	22% (13, 35)	26% (15, 41)
15	4.5% (1.2, 16.2)	9.8% (4.3, 20.7)	11% (5, 22)	18% (11, 29)	22% (14, 34)	25% (15, 37)	27% (16, 40)	31% (19, 47)
20	5.7% (1.5, 19.2)	12% (5, 24)	13% (6, 25)	21% (13, 33)	26% (16, 38)	28% (18, 41)	30% (19, 44)	35% (22, 51)
25	6.7% (1.8, 21.8)	14% (6, 27)	15% (7, 28)	24% (15, 36)	28% (18, 41)	31% (20, 45)	33% (22, 47)	38% (25, 54)
40	9.4% (2.7, 27.8)	18% (9, 33)	20% (10, 35)	30% (19, 43)	35% (23, 48)	38% (25, 52)	40% (27, 54)	45% (31, 61)
55	12% (3, 32)	21% (10, 38)	23% (12, 40)	34% (22, 48)	39% (27, 53)	42% (29, 57)	45% (31, 59)	50% (35, 65)
70	13% (4, 36)	24% (12, 42)	26% (14, 44)	37% (24, 52)	43% (29, 57)	46% (32, 60)	48% (34, 63)	54% (38, 69)
100	17% (5, 41)	28% (14, 48)	31% (16, 49)	43% (28, 58)	48% (34, 63)	51% (37, 66)	54% (39, 68)	59% (43, 73)

[a]All other variables held at their national median.

[b]Estimated median levels based on U.S. Housing and Urban Development national survey, 1989–1990.

level in excess of 10 μg/dL (Tables 4 and 5). At 100 μg/ft² and a soil lead exposure of 72 ppm, the geometric mean blood lead level was 7.3 μg/dL and 28% of children were estimated to have a blood lead level of 10 μg/dL or higher (Tables 4 and 5).

The contribution of lead-contaminated floor dust to children's blood lead level was greater than the contribution from lead-contaminated soil. That is, for the range of exposures observed in these studies, there was a greater increase in the proportion of children with an elevated blood lead level associated with floor dust lead loading compared with soil lead levels (Tables 4 and 5). The proportion of children with a blood lead level greater than 10 and 15 μg/dL by levels of lead-contaminated house dust and soil or exterior lead levels are illustrated graphically (Figs. 3–4)

After correcting for other effects, differences in blood lead levels among studies were statistically significant (Fig. 5). Given the same environmental and social conditions, the predicted blood lead levels in the Cincinnati Soil Study are higher than the weighted average across all studies by a factor of 1.6. In contrast, the predicted blood lead levels for the Sandy, UT, and Boston Longitudinal studies are lower than the weighted average by a factor of roughly 1.6. The geometric mean blood lead levels for the other studies are all within 17% of the weighted average across all studies. Although the Study effect was highly significant, removing Study from the model had a small effect on the r^2 (53 to 51%) and on the parameter estimates (relative to their standard errors uncorrected for measurement error). Regression was used to identify possibly significant predictors of the differences among studies, using year of study, urbanization of the study area, and type of dust sampler (wipe or DVM). Although the results were not conclusive because urbanization and type of sampler are confounded, study differences were most highly correlated with urbanization.

DISCUSSION

The findings of this pooled analysis demonstrate that lead-contaminated house dust is the major source of lead intake for children who have low to moderately elevated blood lead levels (i.e., blood lead levels between 10 and 25 μg/dL). This pooled analysis further indicates that children's mean blood lead

TABLE 6

Likelihood of a Child's Blood Lead ≥ 15 μg/dL for Floor Dust Lead Loadings and Exterior Exposure Levels (ppm)[a]

	Probability of blood lead greater than 15 μg/dL							
	Exterior lead exposure (ppm)							
Dust lead loading (μg/ft²)	10	72[b]	100	500	1000	1500	2000	4000
1	0.027% (0.002, 0.319)	0.11% (0.02, 0.63)	0.13% (0.02, 0.72)	0.37% (0.09, 1.52)	0.55% (0.14, 2.17)	0.70% (0.18, 2.70)	0.82% (0.21, 3.16)	1.2% (0.3, 4.6)
5	0.22% (0.03, 1.65)	0.70% (0.19, 2.60)	0.84% (0.24, 2.86)	1.9% (0.7, 4.9)	2.7% (1.1, 6.5)	3.2% (1.3, 7.7)	3.7% (1.5, 8.7)	4.9% (2.0, 11.8)
10	0.48% (0.07, 3.14)	1.4% (0.4, 4.6)	1.7% (0.5, 5.0)	3.5% (1.5, 7.9)	4.8% (2.2, 10.1)	5.6% (2.6, 11.7)	6.3% (3.0, 13.0)	8.2% (3.8, 17.0)
15	0.74% (0.12, 4.49)	2.1% (0.7, 6.3)	2.4% (0.8, 6.8)	4.9% (2.3, 10.3)	6.5% (3.2, 12.8)	7.6% (3.8, 14.7)	8.5% (4.2, 16.3)	11% (5, 21)
20	0.99% (0.17, 5.73)	2.7% (0.9, 7.8)	3.1% (1.1, 8.4)	6.1% (2.9, 12.4)	8.0% (4.1, 15.2)	9.3% (4.8, 17.2)	10% (5, 19)	13% (7, 24)
25	1.2% (0.2, 6.9)	3.2% (1.1, 9.2)	3.7% (1.4, 9.8)	7.2% (3.5, 14.2)	9.3% (4.9, 17.2)	11% (6, 19)	12% (6, 21)	15% (8, 26)
40	1.9% (0.4, 9.9)	4.7% (1.7, 12.8)	5.4% (2.1, 13.5)	10% (5, 19)	13% (7, 22)	14% (8, 25)	16% (9, 27)	19% (11, 32)
55	2.6% (0.5, 12.5)	6.1% (2.2, 15.8)	6.9% (2.7, 16.6)	12% (6, 22)	15% (9, 26)	17% (10, 29)	19% (11, 31)	23% (13, 37)
70	3.2% (0.6, 14.7)	7.2% (2.6, 18.3)	8.2% (3.3, 19.2)	14% (8, 25)	18% (10, 29)	20% (11, 32)	21% (13, 34)	26% (15, 40)
100	4.3% (0.9, 18.6)	9.3% (3.5, 22.6)	10% (4, 24)	18% (9, 30)	21% (12, 35)	24% (14, 37)	26% (15, 40)	30% (18, 46)

[a] All other variables held at their national median.
[b] Estimated median levels based on U.S. Housing and Urban Development national survey, 1989–1990

levels and the proportion of children who are estimated to have a blood lead level ≥10 μg/dL increase dramatically at floor dust lead levels considerably lower than the current HUD postabatement floor standard and the EPA floor guidance level.

A number of variables, including child's age, race, and mouthing behaviors, were significantly related with blood lead levels. These variables have consistently been found to be risk factors in other epidemiologic studies (Charney, 1980; Clark, 1985; Rabinowitz, 1985; Lanphear, 1996). Some characteristics, including socioeconomic status, age of child, and mouthing behaviors, were also effect modifiers of the relationship of lead-contaminated dust, soil, and water with children's blood lead levels (see Appendix).

Generalizability of Results

The estimated geometric mean blood lead levels at selected levels of lead-contaminated floor dust and soil are dependent on the relative weights for the 12 studies included in this analysis. The studies were not selected to represent the entire United States and the individual study sites, such as inner-city residents in Cincinnati, may not be typical of towns and cites across the nation. This pooled analysis attempted to quantify the factors which affect children's blood lead levels across study locations. Similar factors are expected to affect children's blood lead concentrations across the United States, but the geometric mean blood lead will differ for individual communities.

For median environmental lead levels (that is, dust lead loading of 5.0 μg/ft², soil lead concentration of 72 ppm, maximum interior paint lead concentration of 1.6 mg/cm², and water lead concentration of 1 ppb), the geometric mean predicted blood lead for children in our analysis is 4.0 μg/dL and the probability of having a blood lead above 10 μg/dL is 4%. The probability of having a blood lead above 15 μg/dL is 1%. These estimates correspond nicely with the estimated geometric mean blood lead level (3.1 μg/dL) and percentage of children with a blood lead level of 10 μg/dL or higher (5.9%) from the recent NHANES III, phase II data, collected from 1991–1994, for 12- to 36-month-old children (CDC, 1997).

The distribution of levels of lead-contaminated floor dust among housing units in the United States

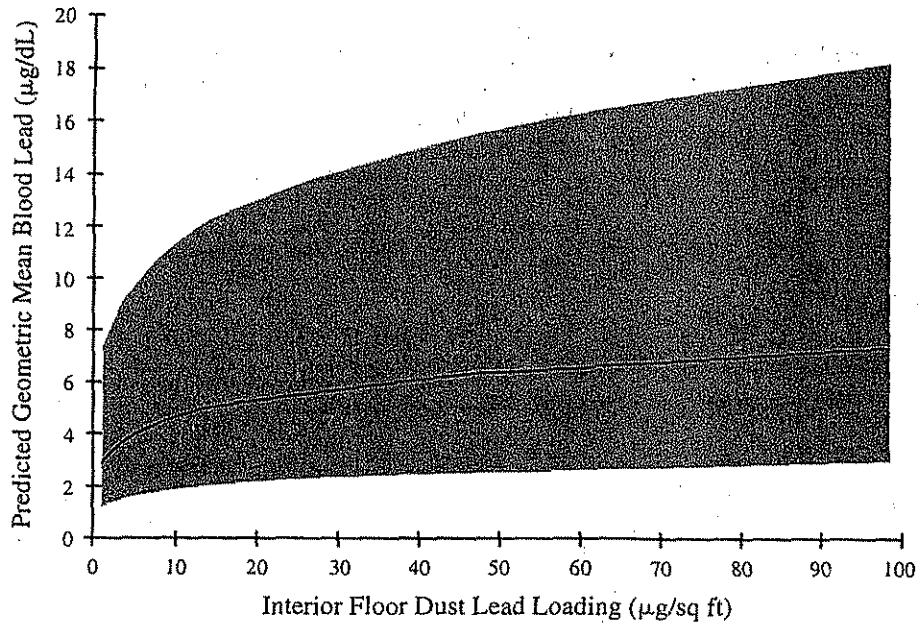

FIG. 1. Predicted geometric mean children's blood lead levels as a function of floor dust lead loading. Other variables are set to geometric means. Six percent of the dust lead loading data values are greater than 100 μg/ft^2). Shaded area covers 90% of children for the given dust lead exposure level, with other environmental exposures at their median level.

is uncertain. Compared with some earlier studies, floor dust lead levels in this pooled analysis *appear* to be low (Sayre, 1974; Charney, 1980). Direct comparison of the various studies is difficult, however, due to variation in dust sampling protocols and study design. For example, dust lead levels observed in the present study were lower than those found in a study done in Rochester in the early 1970s, but

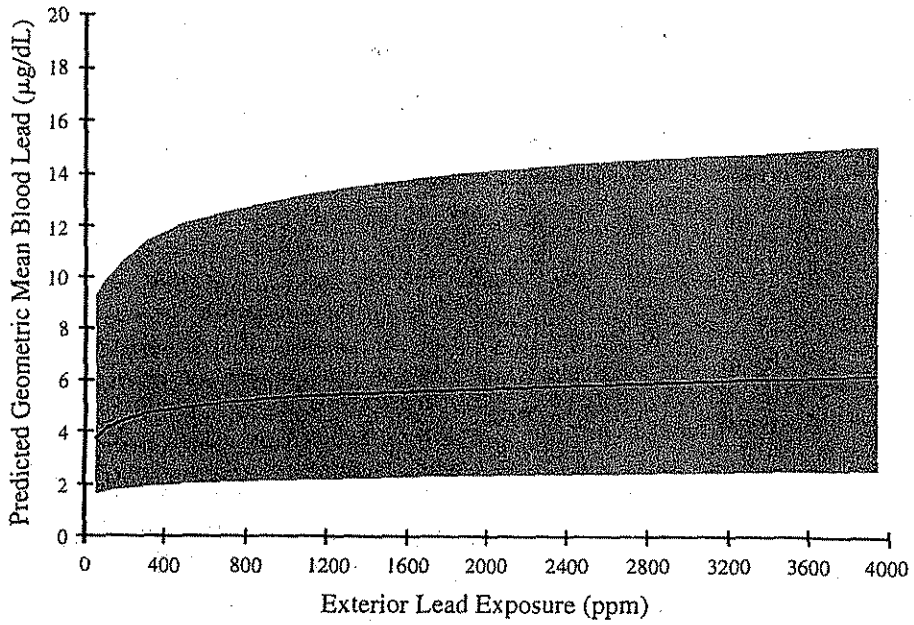

FIG. 2. Predicted average children's blood lead levels as a function of exterior lead exposure. Other variables are set to geometric means. Nineteen percent of the exterior lead exposure data values are greater than 2000 ppm and 9% of the exterior lead exposure data values are greater than 4000 ppm. Shaded area covers 90% of children for the exterior lead exposure level, with other environmental exposures at their median level.

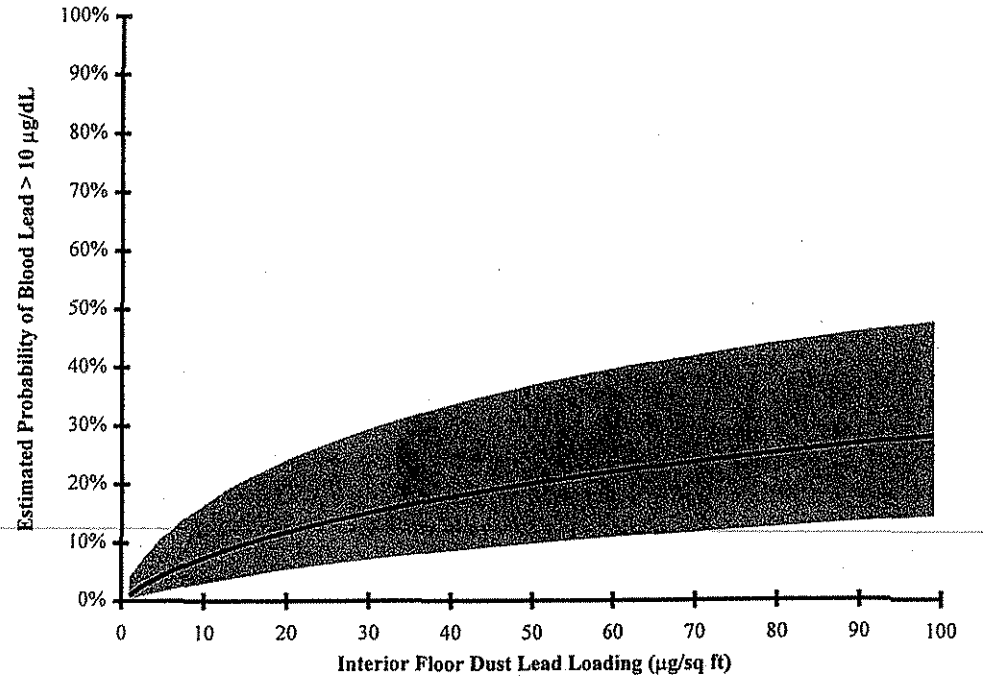

FIG. 3. Estimated probability of children's blood lead ≥ 10 µg/dL as a function of floor dust lead loading. Other variables are set to geometric means. Shaded area shows the 95% confidence interval.

that study included children with higher blood lead levels (Sayre, 1974; Charney, 1980). In contrast, dust lead levels found in middle-class housing in Boston during the early 1980s were similar to the levels observed in the current pooled analysis (Rabinowitz, 1985). In high-risk housing that was slated to undergo abatement, floor lead loading was only 35 µg/ft^2 in (Farfel, 1990). More recently,

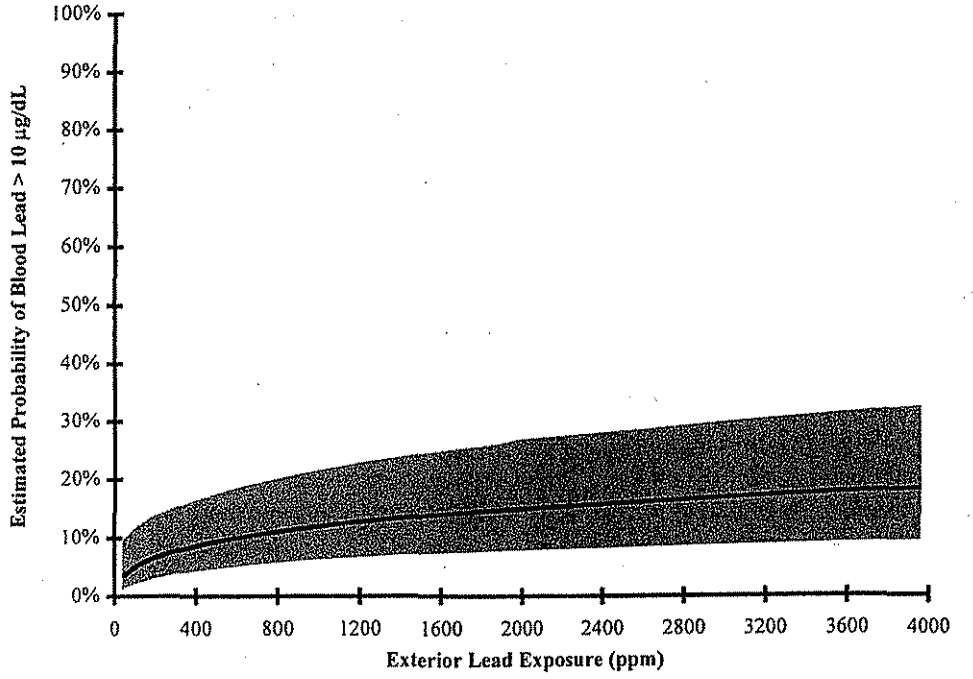

FIG. 4. Estimated probability of children's blood lead ≥ 10 µg/dL as a function of exterior lead exposure. Other variables are set to geometric means. Shaded area shows the 95% confidence interval.

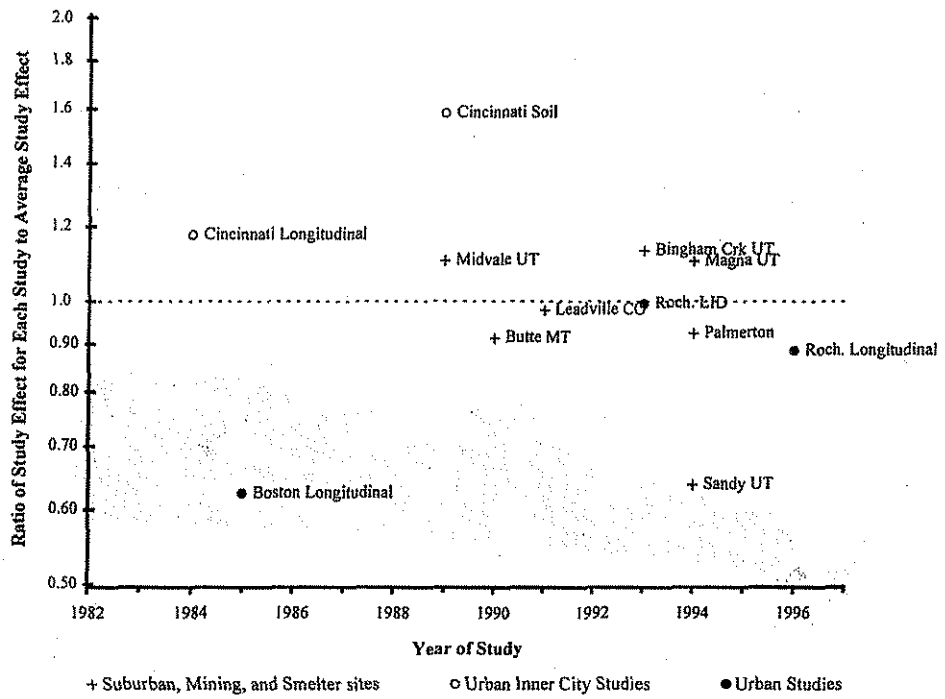

FIG. 5. Estimated effects of study after accounting for other effects, by year of investigation and urbanization of the study area. (+) Suburban, mining, or smelting sites; (●) urban studies; (○) urban, inner-city studies.

baseline median dust lead loading of high-risk, low-income housing from housing across the United States was reported to be 22 $\mu g/ft^2$ (NCLSH, 1997). Many of these studies were conducted when the concentration of lead in motor vehicle emissions was higher or specifically targeted high-risk, urban housing. It is therefore likely that current national dust lead levels are, in fact, lower than those observed in many of these studies.

Although there are data on lead exposure from the HUD National Survey of Lead-Based Paint in Housing (Weitz, 1990; EPA, 1995), limited data are available to convert the Blue Nozzle vacuum method to wipe lead loading (Farfel, 1994). Moreover, it has been almost 10 years since that survey was done and a number of important changes occurred to affect levels of lead in house dust and residential soil, such as the phase-out of leaded gasoline. The finding of this and other analyses, that dust lead loadings 5- to 10-fold lower than current standards and guidance levels are associated with approximately 5% of children having undue lead exposure, emphasizes the importance of conducting a national survey to measure levels of lead-contaminated dust in U.S. housing stock (Lanphear, 1996). If a dust standard is set too low, it may be difficult to ensure that children have access to affordable housing; if it is set too high, it will not adequately protect children from undue lead exposure.

Dust lead loading rather than dust lead concentration was selected as the unit of measure for dust lead in this pooled analysis. Although concentration has traditionally been the unit of measure for quantitation of environmental toxicants, recent studies suggest that lead loading is a more predictive measure of children's blood lead levels than dust lead concentration (Davies, 1990; Lanphear, 1996). In a random sample of 97 children in the U.K., dust lead loading explained a higher percentage of variation in children's blood lead levels than did dust lead concentration (Davies, 1990). Similarly, in a side-by-side comparison of three dust sampling methods, dust lead loading was found to be a better predictor of children's blood lead levels (Lanphear, 1995). In other studies, both dust lead concentration and loading were highly correlated with children's blood lead levels (Clark, 1991; Farfel, 1997). Other considerations, including cost of sampling, ease of use, and respondent burden were taken into account for the decision to use dust lead loading (HUD, 1995).

Alternative Models

Empirically based statistical modeling was used to estimate the relationship between lead in house dust and soil with children's blood lead levels. A second approach that has been previously used to estimate

exposure–response relationships between levels of lead contamination in one or more environmental media and blood lead levels is the mechanistic model, such as the U.S. EPA's Integrated Exposure Uptake Biokinetic (IEUBK) model (EPA, 1994b).

In environmental risk assessment, mechanistic models are often employed where a direct empirical assessment of risk is not feasible, for example, because populations available for study are not large enough or lack a sufficient latent period since first exposed (e.g., the estimation of cancer risk from low-dose exposures or from exposures to newly introduced substances). In these situations, a mechanistic model cannot be validated in entirety; only specific steps in the causal pathway can be tested, usually through laboratory investigations.

In the case of pediatric lead exposure, the mechanistic approach attempts to reflect the current understanding of the intermediate steps from exposure to a given blood lead level, including ingestion, absorption, distribution, storage, and excretion. While empirical models can currently only assess the impact of variation in lead exposure levels, mechanistic models allow for the theoretical possibility of assessing the impact of variation in other factors, like lead absorption or release of lead from bone stores. This theoretical advantage is offset by the difficulty in direct measurement of some of the parameters in the field, such as dust ingestion rates, that are employed in a mechanistic model. In contrast, empirical models typically rely upon readily obtainable field measurements (e.g., mass of lead in house dust per unit surface area of floor) as their independent variables. Of course, these field measurements are imperfect proxies for the causal variables of interest (e.g., mass of house dust ingested each day and mass concentration of lead in house dust). We have attempted to address one of the principal limitations of empirically based statistical models (bias due to measurement error) by employing statistical methods designed to compensate for measurement error. Of course, measurement error can also be a source of bias in applying any mechanistic model.

Limitations

There are several limitations of this pooled analysis. Not surprisingly, there were some significant differences in the predicted blood lead among studies. These study differences may be due to different field conditions, different measurement procedures, or differences in other unrecognized factors that both affect blood-lead levels and differ among studies. To the extent that data are available to explain the differences, the predominant factor accounting for these differences appeared to be degree of urbanization and, to a lesser extent, the year in which a study was conducted. These study effects may also be due, in part, to temporal trends in lead exposure. A second limitation is that the analytic model is believed to most reliably describe the relationship between blood lead and environmental lead exposures in the vicinity of the median or typical values. Consequently, the relationship between environmental lead exposures and blood lead for exposures at extreme ranges is less certain. Finally, although the model is constructed with causal relationships in mind, the parameters may not describe cause and effect relationships.

The focus of this pooled analysis was to provide an estimate of the contribution of lead-contaminated floor dust loading and residential soil with children's blood lead levels. In this pooled analysis, we only estimated the relationship of lead-contaminated floor dust with children's blood lead levels—other data must be examined to estimate the contribution of lead from interior window sills or window troughs to children's blood lead levels. A number of childhood lead exposure studies have been conducted over the past three decades, but the objectives and design of these studies were often different, and the type of dust sampling method and the protocol for selecting locations or surfaces from which to collect dust samples varied considerably across studies (Duggan, 1985, Que Hee, 1985). For example, although most investigators sampled floors and some earlier researchers sampled dust from under a bed, over a door jamb, or on upholstered furniture, many investigators did not examine lead-contaminated dust from interior window sills and window troughs (Stark, 1982; Rabinowitz, 1985). Dust sampling protocols are still often difficult to compare. Some investigators collected floor samples from the perimeter of a room, whereas others sampled the mid-point of a room; lead levels are often higher at the perimeter, especially if the samples are collected underneath a window (Sayre, 1979).

Conversion of the DVM sampling method to the wipe method was necessary to conduct this pooled analysis. Although it is desirable to have a number of studies which use a standard sampling protocol, the results of this pooled analysis were essential to provide a timely estimate of the relationship of blood lead levels with lead-contaminated house dust and soil to assist in the development of residential standards. Fortunately, the estimates obtained in this pooled analysis are strikingly similar with another recent report which indicated that exposure to floor

dust lead levels of 5 μg/ft² were associated with about 5% of children having a blood lead level of 10 μg/dL or higher (Lanphear, 1996).

Conclusion

In 1904, Lockhart Gibson recognized that lead-contaminated house dust was the cause of lead poisoning among children via hand-to-mouth activity (Gibson, 1904). Since then, there have been important advances in our understanding of the risks and sources of childhood lead exposure. Still, almost one century after lead-contaminated dust was first identified as a cause of lead poisoning, lead standards for house dust and residential soil have not yet been promulgated and many children continue to be unduly exposed to lead because they live in housing which is in disrepair or has undergone renovation (Clark, 1991; Lanphear, 1996). In addition, lead hazard control may inadvertently increase children's blood lead levels (Farfel, 1990; Aschengrau, 1997).

The results of this pooled analysis confirm that the proportion of children with an elevated blood lead level increases dramatically at floor dust lead loadings of 5 to 10 μg/ft²—levels that are 10 to 20 times lower than the U.S. Department of Housing and Urban Development's current postabatement standard and the Environmental Protection Agency's guidance level. This finding emphasizes the importance of conducting a national survey to measure levels of lead-contaminated dust in U.S. housing stock and identify what proportion of housing would fail specific dust and soil lead standards. If a standard is set so low that an unacceptably high proportion of housing is considered unsafe for children, it may be difficult to ensure access to housing; if it is set too high, it will not adequately protect children. Finally, it is imperative to identify and evaluate lead hazard controls for their ability to attain and sustain safe levels of lead in residential house dust without placing children at undue risk for lead exposure.

APPENDIX: DETAILS OF STATISTICAL MODELING

A linear model was used to predict log transformed blood lead concentration as a function of continuous variables (child's age, log transformed dust lead loading, exterior lead exposure, water lead concentration, and maximum interior paint lead content) and discrete variables (study, mouthing behavior, SES, paint condition, race, location and type of exterior lead exposure measurement). The terms in the model and the parameter values are presented below. The modeling used data extracted from 12 epidemiological studies. The final data file has one record per household for those households with at least one child between 6 and 36 months of age. If there was more than one child less than 36 months old, one child was randomly selected to represent the household. Because different measurement methods were used in different studies, some values were transformed to obtain comparable measurements. Blood, dust, paint, and water lead measurements below the detection limit were randomly assigned a value between zero and the detection limit based on a log normal distribution.

Variables in the final model:

Blood lead	Child's blood lead concentration (μg/dL) measured from a blood sample.
Dust lead	Interior floor dust lead loading (μg/sq ft) measured using the HUD wipe sampling protocol.
Exterior lead	Exterior lead exposure (ppm) was measured either by a soil sample collected at the perimeter of the property or in the child's play area or by an exterior dust sample. Other variables were used to indicate the location and type of exterior lead exposure measurement.
Water	This variable provides the best available estimate of the water lead concentration (ppb) in the home. In some cases samples were taken within each home. Some samples were first draw, and some were taken after flushing the pipes. Community (or within study average) water lead measurements were used to impute missing water lead levels.
Maximum paint lead content	Maximum paint XRF reading within the home (mg/sq cm).
Age	Age of the child in months.
Study	Twelve levels, one for each study contributing data to the final analysis.

SES	Socioeconomic status, pseudo-Hollingshead measure of SES, with integers ranging from 1 for low SES to 5 for high SES. The information for assessing SES varied by study.
Mouthing behavior	Mouthing behavior, coded based on the best available information as Often, Rarely, Sometimes, and Unknown.
Race	Race of the child, coded as White and Other.
Exterior sample location	Exterior lead exposure sample location: coded as 0 for a perimeter soil sample and 1 for a play area soil sample.
Soil or exterior dust	Exterior lead exposure sample type: coded as 0 for a soil sample and 1 for an exterior dust sample.
Paint condition	Condition of the paint at the location of the maximum XRF measurement: coded as 0 for intact paint and 1 for damaged paint.

The model development considered the theoretical relevance and statistical significance of individual terms and the structure of the model. In addition to the independent variables in the final model, the initial set of candidate independent variables included the gender of the child and the season in which blood and environmental lead samples were collected.
The final model was obtained in accordance with the following:

1. All environmental lead variables including the exterior sample type and location and interior paint condition were included in the model regardless of their levels of significance as main effects;
2. Covariates were included as main effects if either (1) the covariate was significant as a main effect at the 5% level under either structure or (2) the covariate was significant as an interaction term at the 5% level.
3. The Study variable was included as a main effect *only* and was not a candidate interaction term.
4. Interactions (environmental lead-by-covariate or covariate-by-covariate) which were significant at the 5% level were included in the model.

The final model is shown below with interactions indicated by "*." The child's age was centered and included in the model using orthogonal polynomials for linear, quadratic, and cubic effects and symbolized by CAge, CAge2, and CAge3, respectively. The equations for the quadratic and cubic orthogonal polynomials for centered age are:

$$\text{CAge2} = \text{CAge}^2 - (85.55 + 4.82*\text{CAge})$$

$$\text{CAge3} = \text{CAge}^3 - (-490.71 + 10.32*\text{CAge}^2 + 122.30*\text{CAge}).$$

The environmental lead variables were log transformed and centered for the analysis, as symbolized by Cln. The values used to center the environmental variables were the log transformed geometric means from all complete observations and are 2.605 for dust lead loading, 6.232 for exterior lead exposure, 0.921 for maximum XRF reading, and 0.785 for water lead concentration.

$$\begin{aligned}\ln(\text{Blood Lead}) = {} & \text{Intercept} + \text{Ext Sample Type} + \text{Ext Sample Loc} + \text{Paint Condition} + \text{Study} + \text{Race} + \text{CAge} \\ & + \text{CAge2} + \text{CAge3} + \text{SES} + \text{Mouth Behavr} + \text{CAge*Race} + \text{CAge*SES} \\ & + \text{Cln(Dust Lead)*(1} + \text{CAge} + \text{CAge2} + \text{CAge3)} + \text{Cln(Ext Exposure)*(1} + \text{Ext Sample Type} \\ & + \text{Ext Sample Loc} + \text{Mouth Behavr)} + \text{Cln(Max XRF)*(1} + \text{Paint Condition)} \\ & + \text{Cln(Water Lead)*(1} + \text{SES).}\end{aligned}$$

The model was fit using the GENMOD procedure in the Statistical Analysis System (SAS). Measurement error corrections were performed using the SIMEX procedure with the following assumed variances for measurement error in the log transformed units: dust lead loading, 1.00; exterior lead exposure, 1.00; water lead, 0.75; and maximum XRF, 0.75. The tests of significance for the effects in the model were based on the covariance matrix of the error corrected parameters. The SIMEX procedure was applied to 10 bootstrap samples to

estimate the covariance matrix of the corrected parameters. The model parameters were generally, but not always, less significant after the SIMEX adjustment than before.

The parameter estimates and their significance as measured by an F test for each effect, corrected for measurement error, are shown in Table 7. The parameter estimates reflect the default parameterization in GENMOD.

TABLE 7

Summary Results from the Multivariate Regression Model

Parameter	Level	Estimate	P-value
Intercept		1.496	
Dust lead loading ($\mu g/ft^2$)		0.183	<0.0001
Water lead (ppb)		0.01398	0.2067
Soil or exterior dust lead (ppm)		0.02116	0.0025
Soil or exterior exposure dust lead * type of sample		0.005787	0.9247
Soil or exterior exposure dust lead ** type of sample location		0.4802	0.0409
Type of exterior exposure sample		−0.1336	0.2805
Soil or exterior exposure dust location		0.5858	0.0455
Paint lead content (mg/cm^3)		−0.02199	0.3402
CLN(MAX XRF)* paint condition		0.03811	0.3888
Paint condition		−0.0808	0.1685
Age		0.02126	<0.0001
Age 2		−0.001399	0.0044
Age 3		0.00007854	0.0022
Study	Boston	−0.3932	<0.0001[a]
	Butte	−0.01167	
	Bingham Creek	0.2027	
	Cincinnati Program	0.2392	
	Cincinnati Soil	0.5383	
	Leadville	0.05717	
	Magna	0.1761	
	Rochester Longitudinal	−0.04209	
	Rochester LID Study	0.07257	
	Sandy	−0.3712	
	Midvale	0.1777	
	Palmerton	0	
Race	Other	0.123	0.0079[a]
	White	0	
Socioeconomic status (SES)	1	0.3175	0.1081[a]
	2	0.2138	
	3	0.1799	
	4	0.1691	
	5	0	
Mouthing behavior	Often	−0.03233	0.0004[a]
	Rarely	−0.2454	
	Sometimes	−0.1397	
	Unknown	0	
Dust lead loading * Age		0.002649	0.1860
Dust lead loading * Age 2		−0.0003381	0.0573
Dust lead loading * Age 3		−0.00001281	0.6185
Exterior lead exposure* mouthing behavior	Often	0.2212	0.0419[a]
	Rarely	0.07892	
	Sometime	0.1663	
	Unknown	0	
Water lead levels (ppb) *SES	1	0.5305	0.0998[a]
	2	−0.0136	
	3	0.1033	
	4	−0.09098	
	5	0	

TABLE 7—*Continued*

Parameter	Level	Estimate	*P*-value
Age * race	Other	0.01192	0.0129[a]
	White	0	
Age * SES	1	− 0.01023	0.0061[a]
	2	0.003849	
	3	0.00008468	
	4	− 0.01679	
	5	0	
Standard deviation of the prediction error		0.5425	

Note. Interactions are indicated by asterisks
[a]Overall factor significance.

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