

Soil Is an Important Pathway of Human Lead Exposure

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This review shows the equal or greater importance of leaded gasoline-contaminated dust compared to lead-based paint to the child lead problem, and that soil lead, resulting from leaded gasoline and pulverized lead-based paint, is at least or more important than lead-based paint (intact and not pulverized) as a pathway of human lead exposure. Because lead-based paint is a high-dose source, the biologically relevant dosage is similar to lead in soil. Both lead-based paint and soil lead are associated with severe lead poisoning. Leaded gasoline and lead in food, but not lead-based paint, are strongly associated with population blood lead levels in both young children and adults. Soil lead and house dust, but not lead-based paint, are associated with population blood lead levels in children. Most soil lead and house dust are associated with leaded gasoline. Lead-based paint dust is associated with cases of renovation of either exterior or interior environments in which the paint was pulverized. Based upon the limited data to date, abatement of soil lead is more effective than abatement of lead-based paint in reducing blood lead levels of young children. About equal numbers of children under 7 years of age are exposed to soil lead and lead-based paint. Seasonality studies point to soil lead as the main source of population blood lead levels. Soil lead is a greater risk factor than lead-based paint to children engaged in hand-to-mouth and pica behavior. In summary, soil lead is important for addressing the population of children at risk of lead poisoning. When soil lead is acknowledged by regulators and the public health community as an important pathway of human lead exposure, then more effective opportunities for improving primary lead prevention can become a reality. — *Environ Health Perspect* 106(Suppl 1):217–229 (1998). <http://ehpnet1.niehs.nih.gov/docs/1998/Suppl-1/217-229mielke/abstract.html>

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Introduction

Despite an impressive research effort over the last three decades, recognition that lead in soil is an important pathway of human lead exposure remains controversial. Some argue that lead-based paint is the most important source of lead exposure. Others argue that the evidence is insufficient to treat soil and paint as equally important pathways of human exposure. Hundreds of studies have investigated the sources of lead

exposure (principally leaded gasoline, industrial point sources, and lead-based paint), the movement of lead in the environment (from air to soil to dust to a child's hand to a child's mouth), and the effects of lead on human health. Clearly, there are many factors that influence the intensity of exposure experienced by an individual, including age, sex, season, hand-to-mouth behavior (pica), occupation, race,

socioeconomic status, diet, and cultural practices. Some of these factors will be weighed but only as they relate to the role of lead in soil as a contributor to the child lead problem. This review shows the greater importance of leaded gasoline compared to lead-based paint as a source of exposure, and that soil lead resulting from leaded gasoline, pulverized lead-based paint, and other sources is equally or more important than lead-based paint (intact or not pulverized) as a pathway of human lead exposure. When the role of leaded gasoline and lead-contaminated soil and dust are acknowledged as an important pathway of human lead exposure, more effective opportunities for improving primary lead prevention can become a reality.

Human beings no longer live in a natural setting. All around us are the artifacts of human existence. Our built environment, particularly the design of the modern industrial city, is a prime example of the synthetic character of our environment. To understand the flow of energy and materials within the built environment and its consequences for human existence, it is necessary to understand the geochemistry and the toxicity of trace metals in the environment at both a planetary and regional perspective (1). Nriagu and Pacyna (2) have argued that from a global perspective the toxicity of trace metals released into the environment exceeds that of all other radioactive and organic pollutants combined. Lead is a trace metal that has been associated with human civilization since the earliest practice of metallurgy. In the course of mining and concentrating the ore, smelting the ore to purify the metal, and manufacturing useful products from lead, there has been a geochemical transfer of lead from the mine to human habitats.

Two products have added massive quantities of lead to the built environment in modern times. These are lead-based paint and lead additives to gasoline. From a gross-tonnage perspective in the United States, about equal amounts of lead were used in white-lead paint pigment between 1884 and 1989 as in leaded gasoline between 1929 and 1989 (3,4) (Figure 1). The peak use of lead-based paint occurred in the 1920s when the U.S. economy was agrarian, rural, and relied mainly on rail transportation for moving goods and providing services. The lead-based paints were used as a protective coating on buildings and structures in both large and small

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Abbreviations used: ADVT, average daily vehicle traffic; ATSDR, Agency for Toxic Substances and Disease Registry; CDC, Centers for Disease Control and Prevention; dl, deciliter; HUD, Department of Housing and Urban Development; NHANES, National Health and Nutrition Examination Survey; Pb, lead; PbB, blood lead; ppm, parts per million = µg/g; µg, micrograms; µg/dl, micrograms per deciliter of whole blood; µgPb/dl, microgram of lead per deciliter of whole blood; µg/ft², microgram per square foot; µg/g, microgram per gram; µm, micrometer.

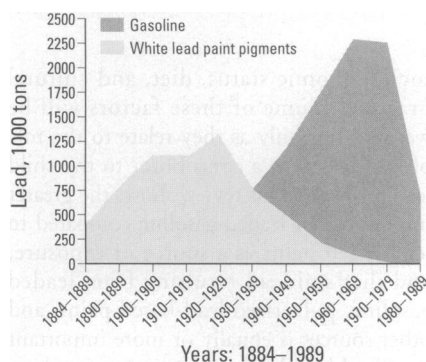


Figure 1. Lead used in gasoline and white lead paint pigments (3,4).

communities throughout the country. Most lead-based paint still exists as a thin mass on walls and structures. In contrast, the peak use of leaded gasoline occurred in the early 1970s at a time when the U.S. economy was industrial, urban, and relied on automobiles for transportation. About 75% of the gasoline lead was emitted from automobile exhaust pipes in the form of a fine lead dust (the remaining 25% of the lead ended up in the oil or was trapped on internal surfaces of the engine and exhaust system) (5). It is estimated that the use of leaded gasoline left a residue of 4 to 5 million metric tons in the environment (6,7).

The global distribution of lead used in gasoline was not even. Over 10 million metric tons of lead was transferred to the global environment via the motor vehicle fleet; about 5.9 million metric tons were dispersed into the United States alone (8). On a local scale, the flow of lead additives in gasoline into the built environment has also resulted in an uneven dispersal of lead. The modern industrial city has two features that contribute to the urban pattern of lead. First, the modern city contains a central business district, which is the daytime address for a large number of workers who commute on a daily basis from outlying areas. Second, the modern city has a ground transportation system dominated by privately owned automobiles and a highway network that concentrates traffic flows within the central business district. Add leaded gasoline to this picture and the result is a system for the inadvertent delivery and accumulation of lead in the densely populated areas surrounding the city center (1).

Soil studies conducted in Maryland, Minnesota, Louisiana, and elsewhere show a consistent pattern of lead geochemistry in urban environments based upon city size and community location (9-14). Specifically, large cities have median lead concentrations

1 or 2 orders of magnitude higher than those of small cities. The distance-soil concentration function from city center to suburbs/rural areas is curvilinear. For example, in Baltimore, Maryland (9), the highest garden soil contamination was so tightly clustered toward the city center that the probability that the concentration could be due to chance was 1 in 10^{23} . Median soil lead concentrations in the Twin Cities (Minneapolis and St. Paul, Minnesota) (11) were 10 times higher than those in adjacent suburbs with older housing where lead-based paint concentrations were higher. Similar results were found when comparing New Orleans, Louisiana with smaller towns (13). Mielke et al. (15) calculated estimates based on average daily vehicle traffic (ADVT) within 1-mile diameter areas within city cores. When the annual metric tons of lead emitted by New Orleans traffic (ADVT = 95,000) was compared to that for Thibodaux, Louisiana (ADVT = 10,000), New Orleans was found to be more than 10 times higher (5.15 metric tons) than Thibodaux (0.45 metric tons). Median soil lead concentrations were 300 to 1200 $\mu\text{g/g}$ in the high-traffic areas of New Orleans versus 60 $\mu\text{g/g}$ in the high-traffic areas of Thibodaux. In summary, the cultural use of metals has changed the pattern of planetary geochemistry, and the main locations of accumulation are in the built environment. There now exist "urban metal islands" analogous to "urban heat islands" meteorologists use to describe the modern industrial city (14). The geochemical reality of the urban environment results in enormous health and policy implications for society.

Within the U.S. built environment, over 12 million children are exposed to the risk of adverse health effects from 10 million metric tons (10^{19} μg) of lead residues resulting from gasoline and paint use (6,16). The total tolerable daily intake of lead for children is about 6 μg lead per day. We measure lead in micrograms of lead per deciliter of whole blood ($\mu\text{g/dl}$). The mass of lead in our built environment potentially available for exposure to children is about 19 orders of magnitude greater than the quantity of lead relevant to a child. Clearly, there is an almost inconceivable amount of lead potentially available to children. The critical concern, then, is the amount of lead actually available to the child.

For most urban areas, the child lead problem is a function of previous paint and gasoline use and their accumulation into the soil pathway of exposure (17). The

immediate societal issue is prevention of exposure to those who are being excessively overexposed and maintaining the health status of those who are not. It is important for those who have power and influence over implementing lead prevention activities to understand the enormity of the soil lead contribution to the child lead problem. Many have claimed that lead in soil is nothing like the contribution of lead from paint. The Department of Housing and Urban Development (HUD), for example, minimizes the regulatory requirements for lead in soil compared to lead in paint in their rules recently proposed in the federal register (18). This is occurring despite the fact that HUD and other federal agencies (16,19-21) have concluded that lead in soil is an important source of lead. The Agency for Toxic Substances and Disease Registry (ATSDR) (19) specifically stated that lead in paint and dust/soil lead were the two major sources of lead. The Centers for Disease Control and Prevention (CDC) (20) states that "lead-based paint and lead-contaminated dusts and soil remain the primary sources..." HUD (21) states that "for infants and young children...surface dust and soil are important pathways..." The U.S. Environmental Protection Agency (U.S. EPA) (16) states that "the three major sources of elevated blood lead are lead-based paint, urban soil and dust... and lead in drinking water." In addition to these statements by government agencies, there are more than 20 other government reports that recognize soil/dust lead as a major contributor to lead in children (22).

An understanding of the relative risk of lead sources is important because Title X (the Residential Lead-Based Paint Hazard Reduction Act of 1992) focuses on lead hazards, not on the mere presence of lead-based paint, and hazard is defined to include lead in soil. Community- and site-specific responses to environmental lead must consider soil and dust to address the problem effectively (23). It is clear that soil is not being considered an equal threat to children. For example, only 9 of the 26 member countries of the Organization for Economic Cooperation and Development regulate lead in soil in contrast to 17 for lead in paint (24). Hence, to effectively integrate soil lead exposures in activities to reduce lead risk, it is necessary to contrast and compare lead in soil with the source commonly believed, perhaps mistakenly, to be the most important contributor to the child lead problem, i.e., lead-based paint.

The Relative Risks of Lead-Based Paint and Lead in Soil and Dust

Whenever one addresses the issue of lead in soil, the first statement one hears is that lead-based paint is the number one problem and any or all other lead sources must be a distant second. The implication is that lead in gasoline or its pathways of soil and house dust are trivial in comparison to direct exposure to lead-based paint itself. When reviewing the evidence, national studies usually frame the argument as follows: "Lead-based paint is the most concentrated source of lead to children and, historically, is the source most closely linked to lead poisoning in children" (25). "Lead-based paint is the largest source of high-dose lead exposure for children" (6). And "lead-based paint is widely regarded as the source of the most intensive and damaging exposures to lead and the preeminent cause of childhood lead poisoning in the United States" (26). The contention that lead-based paint is the number one problem uses the following reasoning: it is a high-dose source; it is closely linked to lead poisoning; and the principle source of lead in house dust and soil is lead paint. Let us examine these reasons as well as the additional risk assessment factors for lead in paint.

Does a High-Dose Source Mean Greater Risk?

Many argue that lead-based paint is the number one source of lead in children because it is a high-dose source. If a measurable amount of lead is a criterion used to determine delivered dose, then lead-acid batteries should be a larger hazard than lead-based paint. Is measurable lead the only factor considered when determining the level of risk of a hazardous material? Obviously, the issue is not just measurable lead but the accessibility and bioavailability of the lead. For example, lead-acid batteries are encapsulated and generally out of harm's way for children. Lead-based paint presents another type of problem. Imagine this scenario: a 2-year-old child eats a 1-g paint chip containing 2% or 20,000 µg of lead. The blood volume of that child is 100 dl. At 50% absorption, the child would have a blood lead (PbB) level of 100 µg/dl. Lead can kill at PbB levels of 100 µg Pb/dl or less. Why, then, are not young children dying in large numbers? The answer has to do with the bioavailability of lead, i.e., the likelihood of the child ingesting a sufficient dose of lead,

and the ability of the intestinal tract to absorb and retain lead.

Several key factors are at issue besides the total lead available from the source. It is known that about 50% of ingested dietary lead is absorbed by children less than 5 years of age (27). Experiments on lead in soil and paint show that 2 to 6 times as much lead can be biologically extracted from soil than from paint (28). Most studies use 30%, i.e., 3 times more lead is bioavailable from lead in soil than in paint (29,30). Moreover, human absorption and retention of lead is a function of both particle size and chemical species (31). The smaller the particle, the more easily it is absorbed by the digestive system. Nearly half the exhaust emitted from gasoline was less than 0.25 µm in size, with most of the remaining emissions between 10 and 20 µm (5). In contrast, the particle size of lead in paint dust/chips ranges from 200 to 300 µm to the visible range. Hence, large particles containing lead such as paint chips are less easily absorbed and, therefore, less bioavailable. It is well known that paint chips pass through the digestive system intact. This helps explain why a single lead paint chip does not kill a child.

Also, bioavailability is not simply a function of particle size. Research has shown that much lead is reabsorbed by food or other substances already in the digestive system, thereby limiting the availability of lead to membrane absorption sites. A child absorbs less lead just after eating than during the period between meals (31). Further, the capability of the digestive system to absorb lead is limited. Consequently, although the first increment of lead is absorbed, subsequent increments are less likely to be absorbed until some point when the receptor sites are saturated. Research shows that after a dose exceeds 500 µg (even of small particles) there is a dramatic flattening of the absorption capability of lead in food, soil, dust, drinking water, and paint (17,32,33). As the dose increases beyond 500 µg, the incremental effect of more lead decreases until it has zero effect upon absorption. Hence, it is the first incremental amount of lead (100–500 µg), not the total lead ingested, that poses the largest risk of lead absorption to young children.

For the above reasons, extremely high concentrations of lead in a paint chip do not translate into a linear increase in PbB levels. The fact that the amount of lead in a paint chip measures higher than the

amount of lead in soil is biologically irrelevant. Measurable lead does not equate with either the effective dose or the hazard that lead imposes. Potential dose does not equal hazard.

Is Lead-Based Paint the Primary Cause of Lead Poisoning?

Central to the argument that paint is the number one lead source is that lead-based paint is closely linked with lead poisoning. Here again, this evidence must be critically evaluated.

Nature & Extent Report to Congress.

In response to the 1986 Superfund reauthorization legislation, the ATSDR examined area-stratified lead exposure among U.S. preschool children (19). "This examination consisted of...both enumeration...and estimation methodologies...to yield prevalences of preselected blood lead criterion levels and those children whose environmental setting would be expected to provide a significantly elevated risk of systemic exposure despite the absence of specific blood lead prevalence data." The report estimated the number of black and white children with PbB levels above selected criterion values, actual counts of children identified through U.S. screening programs for 1984, and the number of children in 318 SMSAs (Standard Metropolitan Statistical Areas) who have the highest potential exposure to lead paint (34).

The premise of the ATSDR report was that "...since the age of housing indicates the degree of exposure to lead in paint and plumbing, we analyzed the distribution of children living in SMSAs by the age of their housing units" (34). The report concluded that

...the counts...in terms of housing age and family income produced the unexpected finding that more children in older housing (high lead paint and plumbing lead levels) were also in noncentral city, nonpoverty families than were children associated in typical risk groups. This observation is consistent with the stratified distributions of the report's projected numbers of the nation's children with elevated PbB levels. (34)

The report goes on to conclude that

estimates of exposure and toxicity based on data gathered in isolated points of time, such as the estimates and enumerations given in the report to Congress, greatly understate the cumulative risk for a population that is posed by a uniquely persistent and ubiquitous

pollutant such as lead. This cumulative toll over extended time is of much greater magnitude than the prevalence or total exposure estimates for a given year. (34)

The effect of these conclusions dominated the creation of the 1992 Lead-Based Paint Hazard Reduction Act with all its subsequent mandates and problems.

Are the methodologies and conclusions of the ATSDR report valid, i.e., is age of housing a valid surrogate variable for lead exposure? ATSDR's own data, particularly in conjunction with National Health and Nutrition Examination Survey (NHANES) II and NHANES III data, can answer this question. Tables 1 and 2 summarize the extent of the problem as ATSDR found it. Over half the housing in the U.S. contains lead-based paint, based on age of housing. Further, 87% of children under 7 years of age live in housing with lead-based paint. In addition, 7.7% of U.S. lead-painted housing is in unsound or deteriorated condition. Further, 12.8% of U.S. children live in unsound, lead-painted housing. If one contrasts these figures with the NHANES II dataset (Table 3), one sees a close correspondence between the number of children

less than 6 years of age with PbB levels greater than 10 µg/dl (87.8%) and the number of children in lead-painted housing (87%). Further, the number of children with PbB levels greater than 25 µg/dl (14.3%) closely matches the number of children in unsound, lead-painted houses (12.8%), who presumably would have greater exposures to lead. Consequently, at first glance, the presence and deterioration of lead-based paint appears to explain population PbB levels in young children.

ATSDR did not, of course, base their conclusions on the NHANES II dataset. Rather, they selected 1984 lead-screening data, adjusted for NHANES II results, with census data for 318 SMSAs to determine prevalence rates for children in lead-painted houses. Their analysis revealed that 46% of children under 7 years of age had PbB levels greater than 10 µg/dl and only 1.5% had PbB levels greater than 25 µg/dl (Table 3). From these data they then calculated an estimate of the percent of children in unsound, lead-painted houses above selected PbB levels (Table 2). They calculated that about half (50.5%) the children with PbB levels greater than 15 µg/dl lived in unsound, lead painted houses and that

93.7% of the children with PbB levels greater than 25 µg/dl did so. Based on these data, they reached the conclusions noted above, i.e., that their estimates underestimated the risk of lead exposure in young children. Nothing in their analysis challenged their premise that living in lead-painted houses was the dominant risk factor for young children.

There are a number of indicators that the ATSDR conclusions require a careful review in light of NHANES III (35–37). First, even if we assume that all children with PbB levels greater than 10 µg/dl lived in lead-painted houses, over 47% of the children living in lead-painted houses had PbB levels below 10 µg/dl ($46\% > 10 \mu\text{g/dl} \times 100 \div 87\% \text{ living in lead-painted houses} = 52.9\%$; $100 - 52.9 = 47.1\%$). This is very close to chance and does not indicate that intact lead-based paint correlates with population PbB levels. Second, even if we assume that all children with PbB levels greater than 25 µg/dl lived in unsound, lead-painted houses, 88% of the children living in such houses had PbB levels below 25 µg/dl ($1.5\% > 25 \mu\text{g/dl} \times 100 \div 12.8\% \text{ living in unsound, lead-painted houses} = 11.7\%$; $100 - 11.7 = 88.3\%$). The ATSDR data indicate that living in unsound, lead-painted houses is a necessary condition to having PbB levels greater than 25 µg/dl. But with over 88% of children less than 7 years of age living in unsound, lead painted houses with PbB levels less than 25 µg/dl and nearly half with PbB levels less than 10 µg/dl, it is not a sufficient condition. Third, the ATSDR analysis predicts that the highest PbB levels will occur in noncentral city areas among the highest income groups. It was clear in the NHANES II dataset that the opposite was true, the highest prevalences were in central city areas among the poor. This indicates that perhaps the analysis is skewed and their premise faulty, i.e., that age of housing is a good predictor of PbB levels in the U.S. population. Fourth, contrary to the ATSDR conclusion that they may have underestimated the risk to the U.S. population, the NHANES III data clearly show a massive decrease in PbB levels within the U.S. population (Table 3). The NHANES III dataset continues to show the highest PbB levels in larger cities among people of color and the poor. Further, the steep decline in PbB levels took place in the absence of any significant effort to abate unsound, lead-painted houses (38). Consequently, the primary source of information used by Congress to derive lead

Table 1. ATSDR best estimate of pre-1980 lead painted^a houses and the number of children under 7 years of age by deterioration criteria in the United States.

Category	Houses, no. 1000	Houses, %	Base population, ^b 1000	Population, %
Total United States	80,390	100.0	13,840	100.0
Lead painted	41,964	52.2	12,043	87.0
Unsound lead painted				
Total	6,199	7.7	1,772	12.8
Peeling paint	1,972		567	
Broken plaster	1,594		458	
Hole(s) in wall	2,602		747	

^aLead paint levels greater than 0.7 mgPb/cm². ^bU.S. white and black populations only. Data from ATSDR, Tables VI-3 and VI-4 (19).

Table 2. ATSDR best estimate of the percent of children under 7 years of age above selected blood lead levels in unsound lead-painted housing.

Category	Total children, 1000	Percentage of children with PbB levels (µg/dl) greater than			Percentage of U.S. children
		15	20	25	
ATSDR base population ^a	13,840	17.2	5.2	1.5	100.0
Children in unsound lead-painted housing	1,772	67.8	30.8	10.6	12.8
Children ^b in unsound lead-painted housing selected PbB levels					
> 15 µg/dl	2,380.6	50.5	—	—	17.2
> 20 µg/dl	715.5	—	76.2	—	5.2
> 25 µg/dl	200.7	—	—	93.7	1.5
Children ^b in unsound lead-painted housing compared to total base population	13,840	8.7	4.0	1.4	100.0

^aU.S. white and black population only. ^bTranslated from actual numbers into percents. Data from ATSDR, Tables I-3 and VI-6 (19).

Table 3. Distribution of blood lead levels above selected values for children 6 months to 6 years of age in the United States.

Survey ^a	Reference	Mid-year	Mean PbB levels, $\mu\text{g}/\text{dl}$	Percentage of children with PbB levels ($\mu\text{g}/\text{dl}$) greater than		
				10	15	25
NHANES II	(16)	1978	15.6	87.8	56.2	14.3
ATSDR	(19)	1984	7.9	46.0 ^b	17.2	1.5
NHANES III						
Phase 1	(36)	1990	3.6	8.9	2.7	0.5
Phase 2	(37)	1993	2.7	4.4	1.3	<0.4 ^c

^aDifferences between values in text and tables reflect different numbers used by different sources. ^bData from Crocetti et al. (34). ^cPbB > 25 $\mu\text{g}/\text{dl}$ not provided; PbB > 20 $\mu\text{g}/\text{dl}$ = 0.4%.

abatement policy had overstated conclusions and was based on a faulty premise that the presence of lead paint or its deterioration, as measured by age of housing, is the best predictor of population PbB levels.

The Presence of Lead Paint in Poisoning Cases. In data published or made available by the CDC, a U.S. EPA analysis found in 5 fiscal years between 1974 and 1981 that, out of 125,060 children with blood lead levels of 30 or 40 $\mu\text{g}/\text{dl}$ "in 40-50 percent of confirmed cases of elevated blood lead levels, a possible source of lead paint hazard was not located..." (5). Further, just because a lead paint source was located about half the time does not mean, ipso facto, that lead-based paint was the source of the child's lead. In cases of elevated PbB levels, the relative contribution from various sources cannot be determined with certainty—whether it be lead paint, leaded gasoline, industrial emissions, or diet—without conducting isotopic analyses and even this approach has limited utility (39). If lead-based paint were present only about half the time in the U.S. EPA analysis of 125,060 cases, then lead-based paint is the number one potentially contributing source to elevated PbB levels by a relatively slim margin. There is no question, however, that when paint is pulverized into a lead dust during renovation, or inadequately abated, or a child has pica for paint chips, severe lead poisoning in young children is bound to result.

Lead Poisoning from Smelter Emissions. It is important to recognize that lead in dust and soil can cause high PbB levels in the complete absence of lead-based paint. Studies of smelter communities have revealed that soil and dust alone can cause epidemics of lead poisoning. For example, Yankel et al. (40) found that 99% of children, 1 to 9 years of age, who were living less than 1 mile from the smelter, had PbB levels greater than 40 $\mu\text{g}/\text{dl}$. Mean soil lead concentrations were 7500 $\mu\text{g}/\text{g}$. At 2.5 miles from the smelter,

mean soil lead concentrations had declined to 1400 $\mu\text{g}/\text{g}$ —an amount comparable to inner-city areas in the U.S. (13). At this distance, 28% of the children had PbB levels greater than 40 $\mu\text{g}/\text{dl}$. Similarly, studies in neighborhoods near the El Paso, Texas, smelter found 53% of the children living closest to the smelter had PbB levels greater than 40 $\mu\text{g}/\text{dl}$ where mean soil lead levels were about 1800 $\mu\text{g}/\text{g}$ (41). It is important to note that the route of exposure in smelter studies is believed to be hand-to-mouth activity. Studies in Omaha, Nebraska (32), and in Belgium (42) showed that after air lead emissions were substantially reduced, children living in soil-dust areas containing high lead and who were closest to the lead industries experienced little, if any, decline in mean PbB levels. This indicates that the overwhelming PbB contribution was from lead dust via hand-to-mouth activity, demonstrating that soil and house dust can cause epidemics of lead poisoning. In contrast to these lead industry studies showing 50 to more than 90% of young children with PbB levels greater than 40 $\mu\text{g}/\text{dl}$, the Chicago Lead Clinic in its worst year (1969) found that only 8% of children had PbB levels greater than 50 $\mu\text{g}/\text{dl}$ (with an average of 3.2% having levels greater than 50 $\mu\text{g}/\text{dl}$ out of hundreds of thousands of children screened for the years 1967–1971) (43). These studies suggest that lead dust can be a major source of the lead contributing to population PbB levels in inner cities and are similar to those in smelter communities, albeit from a different source. The data also imply indirectly that the link between lead paint and population PbB levels is not absolute.

Population PbB Levels Decrease with Gasoline Lead Reduction. Another line of evidence that raises questions about the hypothesized link between paint and population PbB levels is the change in the distribution of population PbB levels as the lead content of gasoline was reduced. Data from

the NHANES II (5,19,44–46) and NHANES III (36,37) studies show a significant reduction in mean population PbB levels for very young children (1–5 years of age), from 15.8 $\mu\text{g}/\text{dl}$ in 1976, to 7.9 $\mu\text{g}/\text{dl}$ in 1984, to 4.4 $\mu\text{g}/\text{dl}$ in 1994. Data from the NHANES III Phase One study show a major and overwhelming reduction in mean population PbB levels in young children: 77% for white children and 72% for black children compared to the NHANES II Study (36). These studies indicated that the change in mean PbB was due to the decline in the lead content of gasoline (5,36,45,46) and a decrease in the lead content of foodstuffs and lead solder in canned food (47). The overwhelming source clearly was leaded gasoline (5,36,45). Further, analyses have been conducted of the changes in air lead concentrations during this time frame (5) and the dose-response relationship between air lead concentrations and PbB levels as a function of both direct inhalation and indirect ingestion of lead dust (19). A review of this evidence strongly supports the conclusion that it was the decline in the availability of fresh lead dust, via ingestion of lead-contaminated soil and house dust that resulted in the steep decline in population PbB levels in young children during this period (48).

During the NHANES II and III time frames (1976–1984 and 1988–1994), there was also a significant decline in the extreme upper range of the distribution. The distribution of PbB levels in the U.S. population of children less than 6 years of age was roughly along the lines of a Gaussian distribution. It is an intrinsic property of Gaussian distributions that small changes in the mean imply major changes in the extremes, i.e., the tails of the distribution. Indeed, a comparison can be made between mean population PbB levels for the mid-year of the NHANES II study (15.6 $\mu\text{g}/\text{dl}$ in 1978) and the ATSDR study (7.9 $\mu\text{g}/\text{dl}$ in 1984) based on screening data in 1984 and adjusted for the NHANES II model. This shift of 7.7 $\mu\text{g}/\text{dl}$ in mean population PbB levels shifted the distribution of population PbB levels, as shown in Table 3. In addition, a comparison of NHANES II with NHANES III shows a similar decrease of 12.9 $\mu\text{g}/\text{dl}$ in mean PbB levels of children 1 to 5 years of age with a decrease from 14.3% to less than 0.4% in PbB levels greater than 25 $\mu\text{g}/\text{dl}$ (36,37). A shift in the population mean of 7.7 $\mu\text{g}/\text{dl}$ from 1978 to 1984 resulted in a decline of nearly half the cases with PbB levels greater than 10 $\mu\text{g}/\text{dl}$ and a

reduction by more than 10 times in the number of cases greater than 25 µg/dl. Similarly, a mean shift of 12.9 µg/dl from 1978 to 1993 resulted in a decline of 95% of the PbB levels greater than 10 µg/dl and a decline of 97.2% of the PbB cases greater than 25 µg/dl. These data suggest that the relationship is very strong between leaded gasoline and population PbB levels. During the years when lead was being removed from gasoline, there was little action to remove lead-based paint from buildings (38,49).

Soil and Dust Lead Dominate the Pathway. Multimedia studies suggest that lead-based paint is not closely linked with population PbB levels. Many anecdotal cases of lead poisoning have been attributed to lead-based paint. Although the number of individual cases relative to the population at risk has never been very high, many investigators simply assumed that all lead poisonings and all exposures could be attributed to lead-based paint. Multimedia lead studies help to "tease out" the relationship between various exposures and sources to PbB levels. For example, Menton et al. (50) found that detailed structural equation models in a longitudinal study in Boston were consistent in showing that "blood-lead levels are significantly related to dust-lead and soil-lead, and the incidence of refinishing activities." Burgoon et al. (51), in a review of 11 studies found that "...these results reaffirm the soil-to-dust-to-blood pathway said to represent the dominant mechanism of childhood lead exposure." There are, of course, conditions that allow paint to overwhelm soil as a pathway, i.e., whenever housing is renovated with unsafe work practices that pulverize paint into a dust when subsequent cleaning is not conducted or is inadequate for the situation, or where lead-contaminated soil concentrations are low (52). Yet it must be noted that lead-contaminated bare soil can poison children when ingested via geophagia or hand-to-mouth activity.

Inner-city Children Show Uniformly Higher PbB Levels. Several studies explain population-based PbB levels. Sayre et al. (53), who conducted pioneering work on the role of lead dust in the exposure of children to lead, questioned the hypothesis of paint chip pica for all lead exposures that prevailed within the medical community. The criticism of Sayre et al. was based on observations of uniform elevation of lead exposure by inner-city children. They noted that exposures to lead dust were the same regardless of the condition

of lead paint and reasoned that if paint chips were the major source of lead exposure, they should see high PbB levels in a few children and low levels in those not ingesting paint chips. Instead, they observed that elevated PbB levels tended to persist to 5 years of age, which is difficult to account for because pica behavior rarely persists beyond age 3. It is important to distinguish between pica and hand-to-mouth behaviors. Pica behavior is the deliberate ingestion of nonfood items including soil (geophagia). Children with soil pica routinely ingest 5 g of soil per day with 20 g not uncommon. In contrast, hand-to-mouth behavior is the inadvertent ingestion of lead dust (particle size < 50 µm) adhering to fingers, hands, or objects. The hand-to-mouth behavior pathway of exposure results in the ingestion of quantities of dust that rarely exceed 0.20 g per day. The ubiquitous occurrence of the behavior combined with the physical-chemical characteristics of small dust particles make hand-to-mouth behavior a potent pathway of lead exposure. After comparing inner-city and suburban children hand-dust lead levels and the environment and noting large differences based on community location relative to city core, they proposed lead dust as a major lead source in children. They did not propose leaded gasoline as an alternative, but it should be noted that lead additives in gasoline peaked between 1970 and 1972 when Sayre et al. were conducting their field research.

Charney et al. (54) compared two groups of high-risk, inner-city black children: group I had PbB levels greater than 40 µg/dl; group II had PbB levels less than 30 µg/dl. They found that four factors explained 40 to 91% of the variance between these two groups. They asserted that "hand lead level, house dust lead level, lead in outside soil, and a history of pica all appear to be multiplicative factors, contributing independently to the very high proportion of total variance explained" [*p* values < 0.005, 0.005, 0.04, 0.001, respectively]. Interior paint was not a strong independent factor in this study.

The Sachs (43) study and other similar studies seem to imply that children with PbB levels greater than 40 µg/dl and who live in deteriorating housing obtain their lead only through paint chip pica. In an effort to see if this was always true, Hammond et al. (55) examined young children with PbB levels in the 40 to 70 µg/dl range and who lived in houses with a lead paint hazard. He expected to find paint

chips in children's stools and fecal lead spikes indicating intermittent high source doses. Instead, he found relatively high continuous exposure to lead evenly mixed throughout the stool with no paint chips or high lead fecal spikes. He concluded that the lead exposure was due to ingestion of lead dust via hand-to-mouth activity. Further, it could not be established that lead paint was the source of the dust (56). Children moved to low lead dust housing experienced an immediate drop in fecal lead concentrations.

Note that, like Sayre et al. (53), some literature refutes the idea that deteriorating lead paint is correlated with population PbB levels. Angle et al. (57) examined the distribution of PbB levels based on the location of dilapidated housing with lead paint, high-traffic roads, and industrial point sources in Omaha. The distribution of PbB levels matched the locations of point sources and traffic but not dilapidated housing. This would appear to indicate that the presence of flaking, peeling paint is insufficient by itself to significantly raise PbB levels in a neighborhood relative to the contribution of other sources such as leaded gasoline or industrial/commercial point sources. Angle's study suggests that although the presence of deteriorated lead paint may be evidence of a hazard, it does not necessarily explain population PbB levels.

Mielke et al. (12) found that the concentration of Pb in children's blood varied in the same direction as the concentration of lead in soil but not with the age of housing (Table 4). A small older community with low traffic flows (Rochester, Minnesota) and an older inner-city community with low traffic flows (North Minneapolis, Minnesota) had statistically significant lower concentrations of lead in blood and soil compared to those in a relatively younger inner-city community with high traffic flows (South Minneapolis). The difference in PbB levels can be explained by soil lead concentrations, which reflected the historic pattern of traffic density, and, ultimately, the lead used in gasoline. These results are consistent with the NHANES II and III studies and the published literature.

It is important to note that research on geographic areas larger than a single residence has demonstrated a consistent central tendency of soil lead results in given neighborhoods or communities indicates the reliability of soil sampling for purposes of comparing geographic areas larger than a single residence. Median soil

Table 4. Comparison of selected variables between Rochester, Minnesota, and inner-city South and North Minneapolis.^a

Variable	Rochester	Inner-city North Minneapolis	Inner-city South Minneapolis
PbB levels, %			
> 10 µg/dl	0.0	26.4	46.7
> 15 µg/dl	0.0	7.6	29.3
> 25 µg/dl	0.0	1.4	6.4
Housing built before 1950, %	74.2	67.1	58.8
Soil lead > 150 µg/g, %			
Foundation samples	38.9	67.6	93.9
Streetside samples	11.1	53.3	78.2
Yard samples	0.0	62.3	81.9

^aMielke et al. (12)

lead concentrations reflecting a neighborhood, zone, community location, or city size can be readily compared (58). For example, teams collecting in the same neighborhoods by two different research groups in Minneapolis and St. Paul showed strong correlations of 0.66 for houseside samples (p value = 0.001) and 0.60 for streetside samples (p value = 0.01) (59). One neighborhood in Minneapolis was sampled by five different teams independent of each other, yet the distributions and measures of central tendency between groups were similar.

These studies and more like them indicate that PbB levels in the general population are closely linked to lead in soil and house dust and that only some unknown fraction is directly linked to lead-based paint. Both the U.S. EPA and the Royal Society of Canada have concluded that, at a minimum, 30 to 40% of children's elevated PbB levels is attributable to lead from gasoline (16,60). A comparison of the decline in children's PbB levels from NHANES II and NHANES III suggests that 75 to 95% of cases of children with PbB levels above 9 µg/dl assumed to be attributable to lead paint were, in fact, from leaded gasoline.

Is Lead-Based Paint the Principle Source of Lead in Soil and House Dust?

Some researchers have argued (26) that lead-based paint contributes lead to both interior house dust and exterior dust and soil, but that gasoline contributes lead only to exterior soil and dust. In other words, paint is said to be the sole source of interior house dust lead.

The primary argument in favor of the idea that lead-based paint is responsible for increased PbB levels is that very high PbB levels are often found in children living in older housing. Most of the lead-based paint used in the U.S. (92%) was manufactured prior to 1950 (Figure 1). Therefore, it is

concluded that the lead paint in the older housing caused the lead poisoning. Another way to view older housing is as lead traps; the older the house, the greater the amount of exterior lead trapped inside (61). Interior house dust lead concentrations often reflect exterior soil lead concentrations (33), which in turn generally reflect the historic use of lead in gasoline and its increase with traffic density rather than with the age of housing. Older housing associated with high PbB levels reflects exterior gasoline-contaminated soil lead that accumulated in the interior of the dwelling, when it was tracked in over time and became available to very young children through hand-to-mouth activity (14). In short, variations in the contributions of sources to house dust appear to be unrelated to the age of homes (62). The following information supports this idea.

Lead Tonnage Equivalent in Gasoline and Paint. From a gross tonnage perspective, approximately equal quantities of lead were used in leaded gasoline between 1929 and 1989 as were used in white-lead paint pigments between 1884 and 1989 (3,4) (Figure 1). All the lead emitted from automobile exhaust pipes was in the form of a fine lead dust. In contrast, most lead-based paint still exists as a thin mass on walls and structures and is not readily accessible to children. It is estimated that the use of leaded gasoline left a residue of 4 to 5 million metric tons of lead in the environment, which poses a risk to sensitive populations (6,7).

Geographic Pattern of Gasoline Lead Emissions and Blood Lead. The dispersion of lead from the combustion of leaded gasoline resulted in a distinct geographic pattern through the various environmental media (air to soil to house dust to blood lead). This pattern demonstrates the massive contribution of leaded gasoline to lead in the air; subsequent deposition of lead dust from the air onto soil; the tracking of

lead soil dust into structures to contaminate interior house dust; and, most importantly, subsequent uptake of lead dust from either or both interior and exterior environments by young children through hand-to-mouth activity. These processes are discussed below.

AIR. Air lead concentrations were highest where lead exhaust was greatest. According to the U.S. EPA (5), air lead levels were highest in the inner city, lower in the outer city, lower still in suburban areas, and lowest in rural areas. A distinct concentration gradient occurred in air lead concentrations away from the downtown areas of most major urban areas. Lead in the air settles to the ground and contaminates the soil.

SOIL. Numerous researchers have shown a decreasing pattern of soil lead concentrations similar to air lead concentrations, i.e., highest in the inner city, lower in the outer city, lower still in suburban areas, and lowest in rural areas (12). This pattern was clearly demonstrated in maps showing decreasing soil lead concentrations in foundation soils away from the downtowns of Minneapolis and Saint Paul, Minnesota (59), and in New Orleans, Louisiana (15), even though communities away from the inner city were as old as the inner-city communities. Foundation soils reflect the accumulated impact and wash-down of both air lead dust and exterior lead-based paint dust.

HOUSE DUST. Numerous studies have demonstrated that a large portion of interior house dust lead is due to leaded gasoline. Fergusson and Kim (61) demonstrated that house dust lead concentrations increase as a function of building age, indicating that structures act as traps for lead dust. They also found that house dust lead concentrations increase as a function of traffic density, i.e., decreasing house dust lead concentration gradients with increasing distances from areas of high lead traffic similar to geographic patterns found for air lead and soil lead concentrations. Bornschein et al. (33) found that soil lead concentrations and house dust lead concentrations are closely correlated [$r^2 = 0.57$]. Fergusson et al. (63) found that house dust is at least 50% soil dust. Research has demonstrated that soil dust lead enters a structure by being tracked in (64–66). Chemical composition studies of house dust have revealed that the source of lead in house dust is primarily leaded gasoline. Such studies consist of apportioning sources of house dust lead based on the ratio of chemical elements in the original dust sources (e.g., paint or soil)

or the likely contribution of lead from organic and inorganic sources, or gravimetric and microscopic measurement in fine fractions compared to possible sources. Sturges and Harrison (67), using gravimetric and microscopic measurement of fine fractions, reported that 85% of house dust lead was from leaded gasoline. Fergusson and Schroeder (68), after examination of the organic and nonorganic contribution of sources to house dust, reported that the source of 95% of house dust lead was leaded gasoline in newer housing and at least 50% was from leaded gasoline in older housing.

BLOOD LEAD. The geographic distribution of PbB levels follows the same patterns as lead in air, soil, and house dust and changes as a function of the availability of lead in gasoline. Lead in food, water, and paint do not exhibit specific geographic patterns. In the case of lead-based paint, old houses everywhere, old farm houses, small cities, and inner cities alike contain similar amounts of lead in paint. The NHANES II and III studies and the ATSDR study on 1984 lead screening data showed that PbB levels were highest in the inner city, lower in the outer city, lower in small communities, and lowest in rural areas (19,35,37,45). Numerous reports in the literature support this pattern (15).

Does Lead Abatement Affect PbB Levels?

Intervention does improve environmental conditions and lower PbB levels in exposed populations. The U.S. EPA (69), in a review of 16 studies addressing lead abatement effectiveness, found that "...intervention did reduce exposed children's blood lead concentrations...on the order of 18-34%...6-12 months following a variety of intervention strategies." Four studies that used PbB levels as a biologic marker concluded:

The Milwaukee Retrospective Educational Study...(70) results indicate a 13.6% decline 2 to 15 months following intervention as the effect of their in-home educational outreach efforts. Dust control measures, conducted in the Baltimore Dust Control Study...(54), were associated with a 16.1% effect 12 months following initiation. Soil abatements, performed in the Boston 3-City Soil Abatement Study...(71-72), exhibited an 11.5% effect by 11 months post-intervention. Finally, the 1990 St. Louis Paint Abatement Study...(73) also reported an 11.5% effect on the blood-lead levels of resident children 10 to 14

months following the abatement of damaged lead-based paint (recall that a multiple linear regression model predicted a 13% effect). Though the data are limited, these results suggest that these intervention strategies are comparable in their effect on blood-lead concentrations.

The Boston portion of the U.S. EPA 3-City Soil Abatement Study addressed soil lead abatement. This study consisted of three different groups involving children whose PbB levels were in the 7 to 24 µg/dl range: the study group that received abatement of soil, house dust, and loose paint; comparison group A, which received abatement of house dust and loose paint; and comparison group B, which only received abatement of loose paint. Only the study group that included soil abatement had a statistically significant reduction in PbB levels (2.44 µg/dl) 11 months post-abatement (71). In a follow-up study, PbB levels continued to decline (3.03 µg/dl) in the study group, indicating a persistent intervention effect at least over the short term (2 years) (72). Moreover, soil lead abatement performed in a subset of comparison groups A and B resulted in a reduction in PbB levels of 41 and 13%, respectively (69). The combined reduction in comparison groups A and B was 3.63 µg/dl as a consequence of the subsequent soil abatement (72). The U.S. EPA analysis of the Boston portion of the 3-City Study concluded that "blood lead were reduced by approximately 1.86 µg/dL at 10 mo[nths] after soil lead abatement... additional reductions in blood lead of about 2.0 µg/dL (relative to non-abated) were observed at 22 mo[nths] postabatement..." (74). One other soil abatement study is worth noting. Soil lead abatement in the smelter town of Rouyn-Noranda and the community of St-Jean-sur-Richelieu, Quebec, resulted in decreases in PbB levels of about 30% (3.2 µg/dl) and 50% (5.1 µg/dl), respectively (75).

The U.S. EPA Urban Soil Lead Demonstration Project (3-City Study) integrated conclusion was that "when soil is a significant source of lead in the child's environment, under certain conditions, the abatement of that soil will result in a reduction in exposure that will cause a reduction in childhood blood lead concentrations" (74). The U.S. EPA further concluded that

in the first year after soil abatement, at most 40 to 50 percent of a child's existing blood lead burden may be removed by

soil abatement or any other combination of abatements and interventions apart from medical treatment by chelation. There may be a much greater effect of lead abatement in preventing lead exposure in future residents.

Additional Risk Information on Lead-Based Paint and Other Sources/Pathways. A number of other factors should be considered in determining whether lead paint is the principal source of childhood lead poisoning. These factors include: the number of children at risk for lead exposure by lead source; the role of seasonality in the child lead problem; and the role of pica in the child lead problem.

First, ATSDR estimated that nearly 12 million children under 7 years of age are at risk from lead in paint and 12 million children are at risk from urban soil and dust (19). Clearly, since there are only 18 million children under age 7 in the United States, there exists considerable overlap between the two groups. The U.S. EPA (16) also concluded that about 12 million children were exposed to "lead-based paint + urban background", i.e., lead in soil. Hence, both lead in soil and paint pose a risk, separately or in combination to about an equal number of children, roughly two-thirds of all children in the United States under 7 years of age.

Second, one of the striking features about the distribution of lead in populations is that, if PbB levels are monitored temporally, they change as a function of the seasons of the year. The NHANES II survey showed that PbB levels in the summer were about 20 to 30% higher than in the winter (46). Hunter (76,77) has reported that the prevalence of lead poisoning cases (defined as a PbB level > 40 µg/dl at the time) was 5 to 10 times greater in the summer than the winter. At first it was thought that these seasonal differences were due to the effect of increased sunlight on 1,25-CC vitamin D metabolism and its effect on calcium transport. Subsequent research, however, showed that the seasonal change in vitamin D metabolism is too small to explain changes in PbB levels (78).

The accepted explanation for seasonal differences in PbB levels and the prevalence of lead poisoning cases are that in the summer there is a greater risk of geophagia (pica for soil); increased access and resuspension of soil dust lead; increased deposition of lead in air through open windows; and most importantly, increased tracking in of lead-laden dust into dwellings from

the exterior by people and pets. Both interior house dust lead and exterior soil dust lead concentrations are associated with increases in PbB levels by season. If PbB levels were associated with interior lead-based paint, then PbB levels should rise in the winter when children are more often confined indoors. Instead, PbB levels are higher when children have access to exterior sources of lead, i.e., soil and, therefore, exterior lead dust.

Finally, what about pica children? If children are categorized into three groups (those that never mouth fingers or objects, those that do, and those that eat nonfood items [pica]), then the key to lead intake is accessibility to lead sources. The mere presence of lead will not affect those children who never engage in mouthing or pica behavior. For those children who do engage in mouthing behavior but not pica, the key variables are the frequency of mouthing behavior and the size of the lead dust particle. Rabinowitz and Bellinger (79) have shown that children who mouth more than others have 2 to 3 times higher PbB levels. The U.S. EPA (5) concluded that "dust sources are important because of children's hand-to-mouth activities and because a single gram of dust can contain 10 times more lead than the total diet of a child." Finally, children who have pica for soil or paint run the risk of ingesting high doses of lead. Some data suggest that pica children are about five times more likely to eat soil than paint chips (80). Lead paint chips easily contain 5000 to 20,000 μg of lead. Children with pica for soil may ingest 5 g or more per day. At a soil concentration of 1000 $\mu\text{g/g}$ (typical of the inner city), a child could ingest 5000 μg of lead in 1 day. Either source is more than enough to cause lead poisoning. As Houk (44) noted, a child who ingests as little as 1/6 g of soil daily can be lead poisoned [PbB > 30 $\mu\text{g/dl}$] in a few months. Indeed, smelter communities have suffered epidemics of lead poisoning in the absence of lead paint, with soil and house dust concentrations comparable to those of inner-city neighborhoods (40,41).

Biological and Ecological Causality: Soil Lead to Blood Lead

The evidence presented above argues that lead-contaminated soil is a pathway of human lead that is equally as important as exposure to lead-based paint. Critics of the role of lead-contaminated soil may assert that causality has not been proven. How is causality determined? Two centuries ago

David Hume stated that causality is a concept not susceptible to empirical demonstration. Epidemiologists and scientists contribute to the incremental accretion of data that one hopes can be assembled into a coherent picture, and from which lawfulness can be inferred (81).

If causality is not susceptible to empirical demonstration, how then do we know when causality is likely? Hill (82) delineated a series of parameters that are important in determining whether causality is likely in a biological sense. These parameters include: consistency of effect; biological gradients of effect; biological plausibility of effects; consistency of biological function; and strength and specificity of association. To determine causality, one must first frame the question, as was done by Rutter (83), on whether low-level lead exposure exerts adverse health effects.

In the discussion prior to this section, the case was made that the overwhelming contributor to lead in soil was deposition due to the combustion of leaded gasoline. Schwartz (49) argued for the causal relationship between gasoline lead and PbB by citing the following factors:

- Experimental evidence found in the investigation of the contribution of gasoline lead to PbB in isotopic studies indicated a magnitude similar to that found in the NHANES II dataset, i.e., that "in the late 1970s about 9 $\mu\text{g/dl}$ of blood lead resulted from lead in gasoline." (49)
- Cause preceded effect because given that the half-life of lead in blood is 30 days, the NHANES II dataset revealed that a 1-month lag between PbB levels and gasoline air lead concentrations was most significant on PbB, with current or 2-month lag period being less significant.
- The analysis was repeated in other localities by other investigators and the same patterns of gasoline lead emissions were found to be significantly related to PbB levels; this provides replicability and consistency.
- Additional analyses revealed a linear dose-response relationship between gasoline lead and PbB.
- Given that gasoline lead produced 90% of U.S. air emissions in the 1970s and was, therefore, a major source of contamination in the environment, air inhalation and ingestion of street dust, house dust, and soil contamination by hand-to-mouth activity demonstrate

that absorption from the lung and gut is biologically possible.

- To avoid Type I errors (accepting a spurious relationship as real) confounding factors were controlled for in various analyses; these included age, race, sex, income, season, degree of urbanization, and region of the county.
- Other sources of lead exposure did not change during the NHANES II examination period in any significant way; this externally validates the conclusion.

The following argument extends the causal argument of Schwartz (49) by examining the predominant intermediate pathway between gasoline lead and PbB, i.e., lead-contaminated soil. It must be remembered that soil is the sink for lead of all sources. The essential causal question is this: Is exposure to lead-contaminated soil that is accessible to young children a significant and important contributor to children's PbB levels?

Consistency of Effect

Causal inference can be concluded if the association has been observed in different investigations using different research strategies. A review of the literature as a whole (5,84,85) has consistently shown that exposure to lead in soil has an effect on PbB levels.

Biological Gradients of Effect

With regard to the effects of biological gradients, i.e., dose-response relationships, most investigations do show a dose-response relationship within the study, but scaling difficulties obscure the true dose-response relationship in many studies (7,85). A reanalysis by Burgoon et al. (51) of 11 studies estimated a dose-response relationship between soil lead and PbB of 6.8 $\mu\text{g/dl}$ per 1000 $\mu\text{g/g}$.

Biological Plausibility of Effects

The coherency between exposure and health effect is a necessary criterion for causality (86). It is well established that gasoline emissions resulting in increasing soil lead concentrations beyond background are strongly associated with PbB levels (a surrogate measure of health effects). There is no biological difference between soil lead exposure and exposures by different pathways; once lead is absorbed, after adjusting for relevant bioavailability issues, it exerts its effects.

Consistency of Biological Function

Causality occurs if the association makes biological sense, i.e., that a likely biological

mechanism exists by which the causal effect can be mediated. Paint and soil lead are absorbed through the gut after ingestion due to pica or mouthing behavior. There is no difference in biological effect, after adjusting for relevant bioavailability issues, because of the pathway of exposure.

Strength and Specificity of Association

Are the associations statistically strong and specific? Lead in soil is strongly associated with PbB levels (the specific effect) because it can occur in the absence of lead paint or other sources (5).

Ecological Causality: Relative Role of Lead-Based Paint and Leaded Gasoline

The central issue is whether the most important lead source is paint (intact or peeling), or soil and dust. The causality question then is this: What is the relative contribution of gasoline-contaminated soil and lead-based paint exposures to the child lead problem? To answer the causal question noted above, we first rewrite Hill's (82) delineation of biological parameters of causality into ecological parameters of causality. These parameters would include: consistency of exposure, ecological gradients of exposure, ecological plausibility of exposure, consistency of ecological function, and strength and specificity of exposure.

Consistency of Exposure

Does exposure to lead in soil/dust and/or paint correlate with population PbB levels? Exposure to lead-contaminated soil, house dust lead, or street dust lead has consistently shown a positive correlation between soil/dust lead concentrations and population PbB levels (Table 5). In contrast, exposure to lead paint is inconsistently correlated with population PbB levels.

Ecological Gradients of Exposure

Do population PbB level studies show a geographic gradient of effect and does lead in any pathway show this same effect? Both the NHANES II (45) study (a survey of 64 U.S. cities) and the ATSDR (19) study (a survey of 318 SMSAs) clearly and strongly showed that PbB levels vary as a function of distance. The larger the city or the closer to the center of the city, the greater the number and percent of children above selected PbB levels. Does any lead pathway match the pattern found in these large scale PbB surveys? Again, Table 5 shows that soil, house dust, street dust, air, and atmospheric deposition exhibit a distance gradient in

concentration similar to that found with lead in children's blood. In contrast, food, water, and paint pathways exhibit no such distance relationship. It appears, therefore, that exposure to lead in dust is an important predictor of lead in children's blood.

Ecological Plausibility of Exposure

Has the lead-based paint or gasoline been used in a manner that would explain the observed PbB level pattern? Environmental health issues can be analyzed through the ecological method (87). The ecological approach has many advantages. *a)* Because exposure and health are analyzed on a group basis, very large populations, orders of magnitude larger than the typical prospective cohort design of a few hundred, can be analyzed in a cost-effective manner. *b)* This approach has the practical advantage of using existing databases. *c)* Studies can be completed in a relatively short time. *d)* Because large databases are used, the studies can measure relatively small increases in risk. *e)* These types of studies are useful in investigating suspicious clusters of disease in relatively small geographic locations. When the ecological method is used in conjunction with other types of research (case-control investigations, animal research, prospective epidemiological studies) and there is consistency of evidence between the studies of different designs, it adds to the plausibility of health hazards suggested by the ecological data (88). The advantages of the ecological method is that it lends itself to the discussion of the causal nature of the subject being investigated. An ecological approach has often been used to observe that lead paint exposure is often found in older, deteriorated, or recently renovated housing. Because of the way cities grow and renew themselves, this pattern of lead paint exposure reflects the nature of a neighborhood, with older deteriorated

neighborhoods providing greater access to lead paint chips and lead paint dust. When one moves beyond an individualized case-control investigation and examines the PbB level patterns observed in populations during the NHANES II and ATSDR studies, one finds that it reflects a pattern of the city or metropolitan area as a whole (19,45). That pattern reflects an incidence rate based upon city size or community location. Similar to this city or metropolitan pattern are the patterns of traffic flow and leaded gasoline usage. Lead concentrations observed in soil and house dust also match traffic flow patterns (12). Nearly equal amounts of lead were used in gasoline and white-lead paint pigment (Figure 1). Most gasoline lead was emitted as a dust, yet most lead paint is still intact as a thin mass on structures. Hence, gasoline-contaminated soil/dust provides a coherent explanation for population PbB level patterns.

Schwartz (49) argued that the citywide pattern does not point to lead-based paint as having an effect on PbB levels because the adult decrease in PbB levels (37%) during the NHANES II study was similar to that for children (42%), and adults do not eat paint. In addition, ingestion of lead paint causes large increases in PbB levels. If there were a drop in lead paint exposure, it would only affect people whose PbB level is above the mean. However, the decrease in PbB during the NHANES II study shifted the entire distribution dramatically; even low PbB groups showed major declines. This would not occur if paint lead were the major determinant. Furthermore, the decline in PbB also occurred in suburbia, which has a low percentage of pre-1950 housing and, therefore, less lead paint, yet both cities and suburbia showed the same drop in PbB and the same gas lead coefficient. Finally, only 0.2% of the housing stock were included in lead paint

Table 5. Summary of the relationship between sources and pathways of lead exposure with blood lead levels and distance.^a

Source / Pathway	Number of study areas	Positive correlation with PbB levels	Positive correlation with distance ^b
Soil	46	42	30 of 30
House dust	45	40	17 of 18
Street dust	16	14	8 of 8
Air	50	28	27 of 27
Air deposition	12	12	9 of 9
Food	13	3	0 of 0
Water	28	2	0 of 0
Paint	39	14	0 of 0

^aData from Reagan (91). ^bIn this column, the first number represents how many studies were positively associated with distance, i.e., had a decreasing concentration gradient with distance. The second number indicates how many studies attempted to correlate the source/pathway with distance.

removal programs during this period, so paint exposure rates were unlikely to change during this period.

Consistency of Ecological Function

Does the deterioration of paint or the combustion of gasoline occur in a manner that best explains observed PbB patterns? When paint deteriorates, it presents a lead dust that settles onto the floor and elsewhere. House dust floor loadings of more than 200 $\mu\text{g}/\text{ft}^2$ have been of concern. Soil dust loadings of 100 ppm contain over 139,000 $\mu\text{g}/\text{ft}^2$ in the upper centimeter and soil lead concentrations often exceed 1000 ppm in inner-city areas, resulting in loadings of more than 1,000,000 $\mu\text{g}/\text{ft}^2$ in the upper centimeter. Foundation soil lead can be found at such concentrations around brick or stone buildings in the absence of lead paint (59,89). Many studies show that soil lead can be tracked into the house and result in severe contamination (63,65,67,68,90). Hence, leaded gasoline-contaminated soil/dust provides a consistent ecological explanation for observed patterns of human PbB levels.

Strength and Specificity of Exposure

Have studies that considered exposure to lead in soil/dust and paint together, found that one or more pathways consistently explain PbB results? Of the 161 studies summarized in Table 5, 26 considered lead both in soil and paint. Of these, PbB levels were positively associated with lead in soil in 22 studies, whereas paint was only positively correlated in 9 studies. When one pathway was positive and the other negative, 14 were positive for soil and not paint, and only 1 was positive for paint and not soil (91). Generally, then, lead in soil is strongly associated with population-based PbB levels.

The discussion above clearly reveals an association between two variables—soil lead

concentrations and childhood PbB levels—beyond what could be attributed to chance. Both biologically and ecologically, this association can be interpreted as causal. In the words of Needleman and Bellinger (81), we are well aware that “making causal connections in the real world is not a pure, value-free enterprise.” Nevertheless, it is reasonable to draw the causal conclusion above. In our view there is sufficient evidence to act on the conclusion that soil is equally important as a pathway for lead as paint. The main task remaining for regulatory agencies and others is to take this conclusion seriously.

Conclusion

The purpose of the above discussion is 2-fold: to evaluate the question of whether lead-based paint (intact or deteriorating in place) is a more important pathway for lead accumulation in young children than lead in soil from leaded gasoline and lead-based paint (sanded or sandblasted); and to set the stage for determining appropriate lead abatement policy. As philosopher Karl Popper noted, the way of science does not consist of any proof of a hypothesis; rather it consists of a series of failures to disprove the hypothesis. By this standard, it is clear that research has failed to disprove the hypothesis that soil lead exposure in young children is at least as important as lead paint exposure. Even if one argues that the work is incomplete, “that does not confer upon us a freedom to ignore the knowledge we already have, or to postpone the action it appears to demand at a given time” (92). If HUD or any other regulatory agency rejects the need to treat soil as an equally important pathway for lead as equally important as paint, they may be making a Type II error (rejecting a valid association as spurious) in judgment or interpretation, i.e., in this case, rejecting as spurious the importance of regulating lead in soil as well

as lead paint. We must not continue to rely on the false causal model that lead-based paint is the only significant source of lead exposure in young children.

Based on the arguments above, lead-based paint is not a greater risk to young children than lead in soil. While lead-based paint is a high-dose source, the biologically relevant dosage is not much, if any, greater than lead available in soil. While lead-based paint is clearly associated with severe lead poisoning, so too is lead in soil. Lead in gasoline and lead in food, but not lead in paint, are strongly associated with population PbB levels in both young children and adults. Further, lead in soil and house dust, but not lead-based paint, is associated with population PbB levels in young children. The overwhelming majority of lead in soil and house dust is associated with lead from gasoline. Lead-based paint dust is associated with cases of renovation of either exterior or interior environments where the paint was pulverized into a lead dust, or where lead-contaminated bare soil is low. Abatement of lead-contaminated soil may be more effective than abatement of lead paint in reducing PbB levels of young children, based upon the limited data available. Approximately equal numbers of children under 7 years of age are exposed to lead in soil dust and intact or deteriorating lead-based paint. Seasonality studies strongly point to lead in soil as a significant source of population PbB levels. Studies of pica children suggest that lead in soil is a greater risk factor than lead in paint. In summary, lead in soil may well be the primary causative agent for concern in addressing the population of children at risk of lead poisoning. If so, what does this mean for public policy? It means that equal regulatory attention must be given to lead-contaminated soil as to lead-based paint to solve the child lead problem.

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