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 as important compared to lead based paint as a source of exposure and that soil lead resulting from leaded gasoline is more important than lead based paint in reducing blood lead levels of young children. While 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). Lead based paint is 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 renovating of exterior or interior environments in which the paint was pulverized (based upon the limited data to date abatement of lead soil 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/Suppl1/217-229mielke/abstract.html

Key words lead soil gasoline paint causality urban blood ecological abatement sources pathways

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 is 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 scale (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 pigments 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...
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 soil or was trapped on internal surfaces of the engine and exhaust system. It is estimated that the use of leaded gasoline left a residue of 4 to 5 million metric tons in the environment.

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. 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.

Soil studies conducted in Maryland, Northeast Louisiana, and elsewhere show a consistent pattern of lead geochemistry in urban environments based upon city size and community location. 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 suburban 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 10^25. 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 μg/g in the high traffic areas of New Orleans versus 60 μg/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 meteologists use to describe the modern industrial city. 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 (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 (μg/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 unconceivable 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. This is occurring despite the fact that HUD and other federal agencies (16-21) have concluded that lead in soil is an important source of lead. The Agency for Toxic Substances and Disease Registry (ATSDR) (19) specifically states 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 infants and young children surface dust and soil are important pathways. The U.S. Environmental Protection Agency (US 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 more 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, if effectively integrated, soil lead exposures in activities to reduce lead risk 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 — 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 historically is the source most closely linked to lead poisoning in children (25). Lead based paint is the most significant exposure factor for children (6). This lead based paint is widely regarded as the source of the most intensive and damaging exposure to lead and the prevalent cause of childhood lead poisoning in the United States (26). The convention 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 whether or not a substance is hazardous, then 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 yg of lead. The blood volume of that child is 100 ml. At 50% absorption the child would have a blood lead (PbB) level of 500 ug/dl. Lead can kill at PbB levels of 100 ug PbB/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 um in size with most of the remaining emissions between 10 and 20 um (3). In contrast the particle size of lead in paint dust/chips ranges from 200 to 300 um to the visible range. Hence large particles containing lead such as paint chips are less easily absorbed and therefore is bioavailable. It is well known that paint chips pass through the digestive system intact. This helps explain why a single 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. This research was done on paint chips which have a small particle size. Research shows that after a dose exceeds 500 ug (even of small particles) there is a dramatic flattening of the absorption capacity of lead in food soil dust drinking water and paint (17,32,33). As the dose increases beyond 500 ug 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 ug) 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 prevalence 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 as of the age of their housing units (34). The report concluded 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 underestimate the cumulative risk for a population that is posed by a uniquely persistent and ubiquitous presence of lead in lead based paint.
pollutants such as lead. This cumulative effect over extended time is of much greater magnitude than the prevalence of 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 subsecuent 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, 77% 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 III dataset. Rather they elected 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 (50.9%) of 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 µg/dl x 100 = 87% living in lead painted houses 52.9% > 10 µg/dl = 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 20 µg/dl (1.5% > 25 µg/dl x 100 = 12.8% living in unsound lead painted houses = 11.7% 100 - 883%) 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 housing and the number of children under 7 years of age

<table>
<thead>
<tr>
<th>Category</th>
<th>Houses no.</th>
<th>Houses / Base population</th>
<th>Population /</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total United States</td>
<td>80,390</td>
<td>100.0</td>
<td>100.0</td>
</tr>
<tr>
<td>Lead stained</td>
<td>41,954</td>
<td>52.2</td>
<td>67.0</td>
</tr>
<tr>
<td>Unsound lead painted</td>
<td>5,199</td>
<td>7.7</td>
<td>12.8</td>
</tr>
<tr>
<td>Total</td>
<td>6,772</td>
<td>8.7</td>
<td>14.0</td>
</tr>
<tr>
<td>Peeling paint</td>
<td>1,972</td>
<td>24.7</td>
<td>32.0</td>
</tr>
<tr>
<td>Broken plaster</td>
<td>1,594</td>
<td>20.3</td>
<td>26.4</td>
</tr>
<tr>
<td>Hole(s) in wall</td>
<td>2,032</td>
<td>26.0</td>
<td>33.2</td>
</tr>
</tbody>
</table>

Leads paint levels greater than 0.7 mgPb/cm². *U.S. white and black populations only. Data from ATSDR Tables VI 3 and VI 4 (18).

### 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

<table>
<thead>
<tr>
<th>Category</th>
<th>Total children</th>
<th>Percentage of children with PbB levels (µg/dl) greater than</th>
<th>Percentage of U.S. children</th>
</tr>
</thead>
<tbody>
<tr>
<td>ATSDR base population</td>
<td>13,840</td>
<td>17.2</td>
<td>100.0</td>
</tr>
<tr>
<td>Children in unsound lead painted housing selected PbB levels</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 15 µg/dl</td>
<td>2,303.6</td>
<td>50.5</td>
<td>17.2</td>
</tr>
<tr>
<td>&gt; 20 µg/dl</td>
<td>715.5</td>
<td>76.2</td>
<td>5.2</td>
</tr>
<tr>
<td>&gt; 25 µg/dl</td>
<td>200.7</td>
<td>93.7</td>
<td>1.5</td>
</tr>
<tr>
<td>Children in unsound lead painted housing compared to total base population</td>
<td>13,840</td>
<td>8.7</td>
<td>100.0</td>
</tr>
</tbody>
</table>

*U.S. white and black population only *Translated from actual numbers into percents. Data from ATSDR Tables I-3 and VI-6 (19).
abatement policy had overstated conclusions and was based on a faulty premise that the presence of lead paint or its deter
oration 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 fiscal years between 1974 and 1981 that out of 125 060 children with blood lead levels of 30 or 40 µg/dl in 40 percent of confirmed cases of elevated blood lead levels a possible source of lead paint hazard was not located (5). Furthermore, because a lead paint source was located about half the time does not mean isopox 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 5 years of age who were living less than 1 mile from the smelter had PbB levels greater than 40 µg/dl. Mean soil lead concentrations were 7500 µg/g. At 2.5 miles from the smelter mean soil lead concentrations had declined to 1400 µg/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 µg/dl. Similarly, studies in neighborhoods near El Paso, Texas smelter found 53% of the children living closest to the smelter had PbB levels greater than 40 µg/dl where mean soil lead levels were about 1800 µg/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 over winning PbB contribution was from lead dust via hand to mouth activity. Demonstration 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 µg/dl the Chicago Lead Clinic in its worst year (1969) found that only 8% of children had PbB levels greater than 50 µg/dl (with an average of 3.2% having levels greater than 50 µg/dl). A significant reduction in mean population PbB levels for the mid year of the NHANES II study (15.6 µg/dl in 1978) and the ATSDR study (7.9 µg/dl in 1984) based on screening data in 1984 and adjusted for the NHANES II model. This shift of 7.7 µg/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 µg/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 µg/dl (36, 37). A shift in the population mean of 7.7 µg/dl from 1978 to 1984 resulted in a decline of nearly half the cases with PbB levels greater than 10 µg/dl and a

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

<table>
<thead>
<tr>
<th>Survey</th>
<th>Mid Year</th>
<th>Mean PbB levels (µg/dl)</th>
<th>Percentage of children with PbB levels (µg/dl) greater than</th>
</tr>
</thead>
<tbody>
<tr>
<td>NHANES II (19)</td>
<td>1978</td>
<td>15.6</td>
<td>87.8</td>
</tr>
<tr>
<td>ATSDR (19)</td>
<td>1984</td>
<td>7.9</td>
<td>48.0</td>
</tr>
<tr>
<td>NHANES III</td>
<td>1990</td>
<td>3.6</td>
<td>8.9</td>
</tr>
<tr>
<td>Phase 1 (36)</td>
<td>1993</td>
<td>2.7</td>
<td>4.4</td>
</tr>
<tr>
<td>Phase 2 (37)</td>
<td>1993</td>
<td>2.7</td>
<td>4.4</td>
</tr>
</tbody>
</table>

Data from Crockett et al (43) PbB > 25 µg/dl not provided. PbB > 25 µg/dl 0.4/

The data also imply indirectly that the link to elevated PbB levels was not located (5). Further just because a lead paint source was located about half the time does not mean isopox 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 5 years of age who were living less than 1 mile from the smelter had PbB levels greater than 40 µg/dl. Mean soil lead concentrations were 7500 µg/g. At 2.5 miles from the smelter mean soil lead concentrations had declined to 1400 µg/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 µg/dl. Similarly, studies in neighborhoods near El Paso, Texas smelter found 53% of the children living closest to the lead industry had PbB levels greater than 40 µg/dl where mean soil lead levels were about 1800 µg/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. Demonstration 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 µg/dl the Chicago Lead Clinic in its worst year (1969) found that only 8% of children had PbB levels greater than 50 µg/dl (with an average of 3.2% having levels greater than 50 µg/dl). A significant reduction in mean population PbB levels for the mid year of the NHANES II study (15.6 µg/dl in 1978) and the ATSDR study (7.9 µg/dl in 1984) based on screening data in 1984 and adjusted for the NHANES II model. This shift of 7.7 µg/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 µg/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 µg/dl (36, 37). A shift in the population mean of 7.7 µg/dl from 1978 to 1984 resulted in a decline of nearly half the cases with PbB levels greater than 10 µg/dl and a

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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 35% of the PbB levels greater than 10 μg/dl and a decline of 97% 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 as 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 refurnishing activities. Burogno et al. (51) in a review of 11 studies found that these results reaffirm the soil to dust to lead 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., when housing is renovated with unsafe 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 dust, soil, or contaminated dust 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 in 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 for 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 50 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 Sichs (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 soil with no paint chips or high lead fecal spikes. Hammond 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. Children moved to low lead dust housing experienced an immediate drop in fecal lead concentrations.

Note that unlike 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. (58) 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 high 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 reflect 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 lead soil 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
lead concentrations reflecting a neighbor
hood 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 = 0.01) and 0.60 for streetside sam-
plies (p value = 0.01) (59). One neighbor
hood 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 indi-
cate that PbB levels in the general popu-
lation 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 a
minimum of 30% to 40% of children's elevated
PbB levels is attributable to lead from gaso-
line (16, 60). A comparison of the decline in
children's PbB levels from NHANES II and
NHANES III suggests that 57 to 59% 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 lev-
els reflects exterior gasoline contaminated
soil 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 contribu-
tions of sources to house dust appear to be
unrelated to the age of homes (62). The fol-
lowing information supports this idea.

Lead Tonnage Equivalent in Gasoline
and Paint From a gross tonnage perspec-
tive approximately equal quantities of lead
were used in leaded gasoline between 1929
and 1989 as were used in white lead paint
pigments between 1984 and 1989 (3, 4)
(Figure 1). All the lead emitted from auto-
mobile 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 mil-
lion metric tons of lead in the environ-
ment which poses a risk to sensitive
populations (67).

Geographic Pattern of Gasoline Lead
Emissions and Blood Lead The disper-
sion 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 mas-
sive 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 that contain
and form an important pathway of human lead exposure

Table 4 Comparison of select variables between Rochester Minnesota and inner city South and North
Minneapolis

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rochester</th>
<th>Inner city North Minneapolis</th>
<th>Inner city South Minneapolis</th>
</tr>
</thead>
<tbody>
<tr>
<td>PbB levels (µg/dl)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;10 µg/dl</td>
<td>0.0</td>
<td>26.4</td>
<td>46.7</td>
</tr>
<tr>
<td>&gt;15 µg/dl</td>
<td>0.0</td>
<td>7.6</td>
<td>29.3</td>
</tr>
<tr>
<td>&gt;25 µg/dl</td>
<td>0.0</td>
<td>1.4</td>
<td>6.4</td>
</tr>
<tr>
<td>Housing built before 1950%</td>
<td>74.2</td>
<td>57.1</td>
<td>58.8</td>
</tr>
<tr>
<td>Soil lead &gt;150 µg/gram</td>
<td>38.9</td>
<td>67.6</td>
<td>53.9</td>
</tr>
<tr>
<td>Foundation samples</td>
<td>11.1</td>
<td>53.3</td>
<td>78.2</td>
</tr>
<tr>
<td>Streetside samples</td>
<td>11.1</td>
<td>62.3</td>
<td>61.9</td>
</tr>
</tbody>
</table>

Mielke et al (12)

Emissions and Blood Lead

AIR Air lead concentrations were highest
where lead exhaust was greatest. According
to the U.S. EPA, air lead levels were
highest in the inner city lower in the outer
city lower still in suburban areas and low-
est 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
demonstrated a cleaning pattern of soil lead
communities similar to air lead concen-
trations where highest in the inner city
lower in the outer city lower still in subur-
ban areas and lowest in rural areas (12). This
pattern was clearly demonstrated in maps
showing decreasing soil lead concentra-
tions 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 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 concentra-
tions increase as a function of traffic density
i.e., decreasing house dust lead concentra-
tions with increasing distances from areas
of high lead traffic similar to geo-
graphic patterns found for air lead and soil
lead concentrations. Borsheim et al (33)
found that soil lead concentrations and
house dust lead concentrations are closely
correlated (r² = 0.57). Fergusson et al (65)
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 lead based paint
based on the ratio of chemical elements in
the original dust sources (e.g., paint or soil).
of 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. Ferguson 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 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 that the Milwaukee Retrospective Educational Study (70) results indicate a 13% decline 2 to 15 months following intervention as the effect of their home educational outreach efforts. Dust control measures conducted in the Baltimore Dust Control Study (54) were associated with a 16% effect 12 months following intervention. Soil abatement performed in the Boston 3 City Soil Abatement Study (71-72) exhibited an 11% effect by 11 months post intervention. Finally the 1990 St Louis Paint Abatement Study (73) also reported an 11% effect on the blood lead levels of resident children 10 to 14 months following the abatement of dam aged 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 months post abatement (74). Other soil abatement studies are 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 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 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 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 the environmental risk for 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 and urban background soil lead 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 20-30% 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 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 resuspenion of soil dust lead increased deposition of lead in air through open windows and most importantly increased tracking of lead laden dust into dwellings from...
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 acception 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:

1. Experimental evidence found in the investigation of the contribution of gasoline lead to PbB in isotope studies indicated in magnitude similar to that found in the NHANES II dataset that in the late 1970s about 9 μg/dl of blood lead resulted from lead in gasoline (49).
2. 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.
3. 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.
4. Additional analyses revealed a linear dose-response relationship between gasoline lead and PbB.
5. Given that gasoline lead produced 90% of US 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 demonstrates 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 country.

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 in 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 0.8 μg/dl per 1000 μg/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—the 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 biavailability 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 this gradient vary in any pathway show the same effect? Both the NHANES II (45) study (a survey of 64 US 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 lead 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, while most lead paint is still intact as a thin mass on interior surfaces. Hence, gasoline contaminated soil/dust provides a coherent explanation for population PbB level patterns.

Schwarz (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 in even low PbB groups showed major declines. This would not occur if paint lead were the major determinant. Furthermore, the铅 decline in PbB also occurred in suburban which has a low percentage of pre-1950 housing and therefore less lead paint yet both cities and suburbs 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.

<table>
<thead>
<tr>
<th>Source/Pathway</th>
<th>Number of study areas</th>
<th>Positive correlation with PbB levels</th>
<th>Positive correlation with PbB levels</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soil</td>
<td>46</td>
<td>42</td>
<td>30 of 30</td>
</tr>
<tr>
<td>House dust</td>
<td>45</td>
<td>40</td>
<td>17 of 18</td>
</tr>
<tr>
<td>Street dust</td>
<td>15</td>
<td>14</td>
<td>8 of 8</td>
</tr>
<tr>
<td>Air</td>
<td>50</td>
<td>28</td>
<td>27 of 27</td>
</tr>
<tr>
<td>Air deposition</td>
<td>12</td>
<td>12</td>
<td>9 of 9</td>
</tr>
<tr>
<td>Food</td>
<td>13</td>
<td>3</td>
<td>0 of 0</td>
</tr>
<tr>
<td>Water</td>
<td>28</td>
<td>2</td>
<td>0 of 0</td>
</tr>
<tr>
<td>Paint</td>
<td>30</td>
<td>14</td>
<td>0 of 0</td>
</tr>
</tbody>
</table>

Data from Reagan (9). In 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 μg/ft² have been of concern. Soil dust loadings of 100 ppm contain over 139,000 μg/ft² 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 μg/ft² 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 (63–65/67/90). 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, where 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 child PbB levels—beyond what could be attributed to chance. Both biologically and ecologically, this association can be interpreted causally. In the words of Needleman and Bellinger (81), we are well aware that making causal inferences in the real world is not a cut-and-dried problem. Nevertheless, it is reasonable to draw the causal conclusion above.
MIELKE AND RAFUAN

SOIL AN IMPORTANT PATHWAY OF HUMAN LEAD EXPOSURE

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