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STANDARDS FOR SOIL LEAD LIMITATIONS IN THE UNITED STATES

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ABSTRACT

Soil and dust are enriched in Pb by many sources (automotive emissions, paint residues, industrial emissions, etc.), such that the greatest quantities of Pb are usually found in inner-city urban areas. Soil Pb can reach children by 1) plant uptake of Pb from soil and transport into edible plant tissues; and 2) direct ingestion of soil and housedust, both inadvertently by hand-to-mouth play, and by pica. In general, soil ingestion provides a much greater potential pathway for soil Pb transfer to children than does growing plants on the same soil.

Expert groups have concluded that inadvertent soil and dust ingestion can increase blood Pb in children when soil exceeds 500-1000 mg Pb/kg soil. Recent research findings indicate that even lower soil Pb levels can increase blood-Pb, and that pica ingestion of soil with 500 mg Pb/kg strongly increases blood-Pb. Social, economic, behavioral, and nutritional factors strongly interact with exposure to Pb-rich soil such that some individuals may be at risk when most are not. Rat feeding studies indicate that soil Pb has greater bioavailability at higher soil Pb concentration, making the soil-Pb:blood-Pb relationship curvilinear. Some research has indicated that the slope of the increase in blood Pb decreases with increasing diet Pb. These curvilinear relationships are independent.

Research on Pb health effects in children now indicate that the maximum blood-Pb level children should be allowed to reach is 15 ug/dL if they are to avoid these ill effects of Pb. Prospective longitudinal studies of children in Pb-rich environments show the great importance of Pb in soil and dust to blood-Pb levels in children, and the importance of neurobehavioral impairment due to excessive blood-Pb.

Research is summarized which indicates that substantial areas of urban soil greatly exceed 500 mg Pb/kg and will need to be replaced to avoid health effects in children, especially in the inner-city and surrounding point sources of Pb. Pb-B of children is raised, in the average, about 5 ug Pb/dL for every 1000 mg Pb/kg soil/dust where the child lives or plays. The range of this response extends to at least 3-to-5 times higher than the mean response because of individual child behavior. Some evidence indicates that soil Pb > 150 mg/kg can cause excessive Pb-B levels in the "most-sensitive, most-exposed" children. Even this level does not include a deliberate safety factor required in other environmental risk analysis for Pb and other elements. Because Pb is immobile in the soil, pollutant-Pb will persist in surface soils until humans take corrective actions. Recent changes in Superfund will require that a study be made to evaluate the response of Pb-B to replacement of Pb-polluted urban soils in several US cities.

INTRODUCTION

The U.S. currently practices "secondary prevention" for undue Pb absorption. This means children with excessive blood-Pb (Pb-B) are found through screening programs, or because symptoms bring them to medical attention. Good Pb poisoning prevention programs then examine these children's environments to identify Pb sources, and require abatement of Pb available to the child before the child may return home, or help the family move to "Pb-free" housing. These programs have had less funds in recent years, and are less able to protect urban children (139). Unfortunately, children are still the sampling device our society uses to identify areas of Pb risks, and we continue to have many urban children with undue Pb absorption (2,18,23,24,51,112,139,153,161).

Depending on which limit one uses to define "undue Pb absorption" or "excessive Pb-B", the US has a large or moderate epidemic resulting from allowing children to inhabit in areas rich in environmental Pb. The recent National Health and Nutrition Examination Survey-II (NHANES-II) 1976-1980 survey of Pb-B in the U.S. found 4.0% of all children aged 0.5-to-5 years had Pb-B > 30 ug/dL, the then defined limit for normal Pb-B (2,96). The mean Pb-B level for all 0.5-to-5-year-old children was 16.0 ug/dL, although children's Pb-B appears to have declined during the survey in relation to the reduction in Pb use in gasoline (3). The survey found 4.0% > 30 ug/dL, 9.1% > 25 ug/dL (the current Center for Disease Control (CDC) Pb-B guideline for medical intervention), 24.5% > 20 ug/dL, 56.1% > 15 ug/dL, and 87.8% > 10 ug/dL. Because we will focus on 15 ug Pb/dL or lower as a likely safe level based on newer research findings, we note that 56.1% of children over 15 ug Pb/dL equals 9,350,000 U.S. children aged 0.5 to 5.

Standards for acceptable Pb-B levels have declined over the years as research gained new information about Pb risk to children's health. Initially, standards were set to prevent frank Pb encephalopathy, a severe disease which may cause death, mental retardation, etc. Subsequently, anemia induced by Pb was used in standard setting. Limits were later lowered to afford greater safety, until the 1985 CDC report indicating 25 ug/dL as the current guideline for medical intervention (18). In the last few years research on erythrocyte protoporphyrin (EP) accumulation in blood in response to increased Pb-B has shown that EP will accumulate (62,95,120,125) above a threshold of about 15 ug Pb/dL, . This is a undesirable biological effect of Pb-B, although the risk from small increases in EP are debated.

Other research has evaluated neurobehavioral impairment due to increased Pb-B. A number of surveys and, more recently, prospective longitudinal studies of children living in different Pb-enriched environments (154), have found reduction in IQ and other learning impairments at Pb-B levels common among children today (18,28,113,115,154,165,177). Although it may take many years before a new threshold for Pb-B effect on neurobehavioral impairment is defined and generally accepted, the threshold appears to be in the range of 10-15 ug Pb/dL (113,126,177). From this perspective, it is clear that over 50 % (2) of U.S. children may be at risk from their present avoidable exposures to environmental Pb.

ENVIRONMENTAL SOURCES OF Pb FOR CHILDREN

Children absorb Pb from many sources, including at least air, drinking water, food, housedust, play area soil and dust, interior and exterior paints, improperly glazed ceramics, and toys. Federal regulators and advisors worked many years to reduce childhood exposure to Pb from paint, air, water, and food. Limits were placed on Pb in new interior paints, and for paints used on toys (but not exterior or metal paint, or interior paint already in homes except in Federally supported programs). Great progress has been made in reducing new emissions of Pb from automobiles and stacks, and Pb use in gasoline is scheduled to end. A new, lower, drinking water Pb limit, and prohibition of Pb-based solders used on household plumbing, are reducing water-Pb. The Food and Drug Administration (FDA) achieved reduction of Pb in infant foods to one-tenth the levels prevalent in the early 1970's by reducing use of Pb solder in cans for infant foods (80). Reduction of Pb-rich automotive dust in food processing and home food preparation areas will further reduce food-Pb intakes in the next few years (3,141).

Unfortunately, too few regulations have been developed to prevent risk from Pb-rich interior and exterior paints in existing housing. Although Pb poisoning prevention programs made good progress in the 1970's, the problem did not end. Many effective programs were terminated as funds became unavailable. The Newark, NJ program (51,139), the Baltimore program (23), the Cincinnati program (24) etc., found many children with excessive Pb-B due to exposure to paint-Pb. Interestingly, paint chip ingestion has been reduced in most cases by public education programs. However, paint residues become part of housedust ingested during hand-to-mouth play allowing regular Pb intakes rather than the extreme episodic intakes previously seen with paint chip ingestion (63).

In the absence of Pb-pollution, Pb-B levels are very low. Since the early 1970's, a new appreciation has developed for the importance of Pb sources other than air, water, food, and interior house paint. As errors in Pb-B analysis were corrected, researchers found that children living in remote areas where very little environmental Pb pollution has occurred have quite low Pb-B values. Yanomamo Indians in Venezuela averaged 0.8 ug Pb/dL (range, 0.0 to 3.9) (72); Nepalese children averaged 5.2 ug Pb/dL (range, <1 to 16) (119); and Papua New Guinea children averaged 5.2 ug Pb/dL (range, 1.0-13) (121). Poole et al. (121) graphed the distribution of Pb-B concentrations in the Papua New Guinea children and a similar group of urban schoolchildren in Sydney, Australia. Nearly all the Australian children had Pb-B levels in excess of the maximum for the Papua New Guinea children. A study of Pb-B in Japanese farmers found the mean was 4.9 ug/dL for men and 3.2 ug/dL for women (169), far lower than urban dwellers or US citizens. These findings also strongly support the conclusion that environmental Pb sources (other than air, water, food, and paint chips) contribute much Pb to children in developed countries.

Behavior of Child and Caretaker Affect Child's Pb-B. The occurrence of excessive Pb-B in a child depends on many factors besides simple exposure to Pb. Behavioral, socioeconomic, family operational, nutritional, and demographic factors strongly influence exposure, ingestion, and absorption of environmental Pb (2,13,15,18,38,54,75,92,93,96,106,112,126,151,152,164,165). Analysis of the NHANES-II data on Pb-B levels in a random subset of U.S. citizens found that Pb-B was higher in blacks than whites. in central cities than in suburban or rural areas, and in poorer than in richer children (2,96). Even among children who inhabit Pb-rich environments, parental supervision, educational programs about urban Pb, improvement in house cleaning to reduce the amount of housedust, hand washing before eating, and improved nutrition allow some children to have low Pb-B while others in the same community have excessive Pb-B and require chelation therapy (13,21,24,75,112,152,165).

Research in two areas have provided convincing evidence that children living in environments with **unrecognized** Pb-rich soil or dusts cannot avoid ingesting increased amounts of Pb: 1) Smelter emissions slowly accumulate in soil and dust over years of operation. Although warning the public to reduce their children's exposure to Pb does reduce Pb-B (and presumably soil and dust Pb ingestion), Pb-B levels were found to be remarkably increased near many Pb smelters (1,4,15,87,100,105,107,110,114,127,132,133,138,165,167,173,-175). 2) Until the Occupational Safety and Health Administration regulations required changes in industrial practices, many workers went home in dirty "work" clothes and their homes had high levels of Pb-rich housedusts. Children living in these homes ingested Pb-rich soil and dust, and had high Pb-B (4,5,35,36,43,107,131,170,173). Even changing clothes and showering at work did not completely prevent increased Pb-B in several studies. Some smelter managements had workers wash dirty clothes at home (5,36), but workers could become recontaminated as they walked to their cars and carried Pb into their homes (131).

The importance of Pb in soil and dust in excessive Pb-B of children was first recognized in the early 1970's (20,25,38,88,135,166). Some children ingest soil and dust inadvertantly, by hand-to-mouth play which is a normal developmental process in children. Others have pica (ingest non-food objects) for soil, or soil is ingested without the knowledge of the parent. When soil-Pb exceeds a few hundred mg Pb/kg, pica for soil sharply increases Pb-B (6,117,142,143,172,174).

In our judgement, the highly significant increase in Pb-B due to these unrecognized Pb exposures in the domestic environment demonstrates the logical importance of preventing ingestion of Pb-rich soil and dust. Habitation in Pb-rich environments, in the absence of extra-ordinary parental care and supervision, can cause proportional increase in Pb-B. These findings are clearly relevant to assessing risks from Pb-rich urban soils and dusts contaminated largely by paint residues and automobile emissions.

SOURCES OF Pb IN URBAN SOIL AND DUSTS

Population densities of urban areas increase from the outskirts to the inner-city where the highest Pb exposure of children occurs. It is important to examine the processes which operate to cause the inner-city to supply the highest Pb exposure and generate the highest Pb-B of the general population excepting near smelters. We conclude that materials used in, and designs of modern industrial cities, cause Pb enrichment of urban areas. It is important to characterize the processes and sources of urban Pb pollution because different laws and regulations will protect against specific sources and ignore others. Some regulations will require abatement of paint-Pb polluted soil but not gasoline-Pb polluted soils, and other regulations will require the opposite. It is critical that we understand that urban soil is polluted by multiple sources, and that children ingest soil-Pb and soil-contaminated housedust-Pb, regardless of whether we can identify the predominant source of the Pb in urban soil and housedust, as might be required to satisfy the letter of some specific regulation.

Urban Soil Pb Comes From Many Contamination Sources. Many activities have been shown to cause Pb accumulation in soils, and to affect the depth and areal pattern of Pb concentrations. These range from contamination of soil by paint scrapings or sandblasting (which cause extreme local Pb enrichment), to burning of trash rich in Pb, to the residue of demolished structures, the dumping and burning of Pb batteries and their cases, the historic use of Pb pesticides, emissions of refuse incinerators and other high temperature processes, use of sewage sludge as fertilizer, and emissions of vehicles fueled with leaded gasoline (19,56,59,68,78,156). All these practices contaminate soil with Pb. Likewise, many activities disturb soils and remove Pb or reduce the concentration of Pb in the surface soil, such as erosion, road repair, building construction, gardening, and landscaping. These human activities add complexity to the detailed pattern of soil Pb in the urban environment. Sayre and Katzel (136) reported that exterior sources of Pb-rich dusts penetrate houses and accumulate unless removed by cleaning processes; leaky windows were an important route of aerosol particulate entry into houses.

Paint Can Be an Important Pb-Source For Soils. Exterior and interior paint rich in Pb can be an intense local source of Pb in soils. The first report on paint-Pb contamination of houseside soil we are aware of is Hardy et al. (65). Bertinuson and Clark (9) found houseside soils were very rich in Pb. TerHaar and Aronow (157) reported decreasing soil Pb with increasing distance from a rural barn, clearly showing the exterior paint was the Pb source. Shellshear (142) found a child with excessive Pb-B who had pica for a soil contaminated by exterior paint-Pb. Further study on the relationship of housing construction materials to soil Pb contamination confirmed the exterior paint Pb source (83) and showed that other children with pica for contaminated soils had excessive Pb-B (143). Solomon and Hartford (148) and Getz et al. (57) noted that both aerosol-Pb and paint-Pb could contaminate houseside soils. Linton et al. (89) used electron microscopy and x-ray microanalysis to identify the source of particles in houseside soils. They found Pb and titanium (Ti) co-occurred in particles of paint residue, but that aerosol Pb sources could also be identified in houseside soils. We reviewed many reports of paint-Pb contamination of urban soils (19); many scientists have studied paint contaminated soils (24,26,27,47,82,83,89,102,-103,112,122,142,143,148,149,153,156,157,174). A recent paper by Yaffe et al. (174) used stable isotopes of Pb to trace paint-Pb to soil, from there to housedust, and from there to blood-Pb of children. Sandblasting removal of paint from buildings or metal structures disperses fine powders which are extremely rich in Pb (e.g. 44,86). Building demolition releases interior paint rich in Pb to the soil if zoning codes do not require removal of the paint residue from the lot. Many of the urban gardens we studied in Baltimore had paint-Pb as an important Pb source (19). It is clear that paint can be an intense local Pb source for contamination of soil. However, it is also clear that both aerosol (gasoline) Pb and paint Pb contribute to the Pb in all urban soil and housedust.

Patterns of Pb Contamination of Urban Soils. Despite all the processes that cause variability, urban soil Pb patterns are highly predictable. Repeated samplings are remarkably similar for a given area of a city. Soils have been very reliable for assessing Pb pollution (38). Research by Mielke et al. (102,103,104) has focused on patterns of soil Pb in urban areas. In order to assess the various processes which can independently increase soil Pb at specific places in the city, major sources, geographic factors, and cultural variables must all be considered.

Our published study of soil Pb in Baltimore, MD established the scientific basis for asserting that the inner-city areas of our society are excessively contaminated with Pb (102). Table 1 lists the Pb levels which correspond to various percentiles in metropolitan Baltimore, MD, and in Saint Paul, MN. The Baltimore soils were sampled from vegetable gardens mixed to a depth of 20 to 30 cm, while the Saint Paul soils were collected as 2 cm surface scrapes from locations in residential neighborhoods where young children have a high probability of contact with bare soils (i.e., around front and back steps, and along sides of homes). Thus, it would be expected that the Saint Paul soil scrapes would have higher Pb levels than Baltimore vegetable gardens. However, both data sets support similar conclusions regarding patterns of urban Pb distributions.

Another means of describing soil Pb contamination is in terms of Pb levels found in various concentric zones around the city. The zonal results for Baltimore are shown in Table 2. It is clear that distance from the city center plays a major role in soil Pb enrichment. In Baltimore, the soils highest in Pb were so highly clustered toward the center of the city that the results could be explained by chance in one out of 10^{23} tries (102). This was a particularly striking finding for Baltimore because the most consistently high garden soil Pb levels were found in the area of the city that was predominantly unpainted brick buildings, while generally lower soil Pb levels were found in the part of the city in which old painted wooden structures were located. Subsequent studies showed that the Minneapolis-Saint Paul metropolitan area was contaminated in the same pattern as Baltimore (103). Further, size of city and geographic features were shown to be important variables in the degree of urban contamination (104). A

TABLE 1. DISTRIBUTION OF SOIL Pb CONCENTRATIONS FOR BALTIMORE, MD AND SAINT PAUL, MN

Percentile	Baltimore (N=422)	Saint Paul (N=1697)
	------------mg Pb/kg dry soil	------------
Min	1	2
10	14	69
20	24	100
30	35	132
40	56	174
50	100	228
60	167	308
70	259	424
80	421	636
90	778	1096
max.	10900	23760

Sample method differed between cities; see text.

decline in soil Pb concentration with distance from the city center was also found by Davies et al. (30) for London, who had previously showed that soil Pb increased with longevity of habitation (29). Gulson et al. (60) found that Pb isotope ratios were altered in soils downwind from a major city, indicating the aerosol Pb source reaches soils many kilometers from the city. Why is there such a strong relationship between soil Pb and distance from the city center, and what are the roles of paint-Pb and gasoline-Pb as sources of soil Pb contamination for the whole city?

TABLE 2. EFFECT OF DISTANCE FROM THE CENTER OF BALTIMORE ON Pb CONCENTRATION IN GARDEN SOILS COMPARED TO UNCONTAMINATED US CROPLAND (19,73)

Distance	N	Mean	Min.	Med.	90%-ile	Max.	>500
km		--------mg Pb/kg dry soil			----------		%
1-4	90	1020	48	664	1810	10900	61.1
4-6	92	414	11	314	892	2700	26.1
6-10	127	419	18	153	690	7820	15.7
10-20	169	269	6	48	324	10600	8.3
20-50	71	53	2	14	94	730	2.8
1-50	549	424	2	124	992	10900	20.9
US Cropland	3001	17.7	0.2	11	21	3500	1.0

Preliminary results are available from a study designed to evaluate processes which have contributed to the contamination of urban neighborhoods in Saint Paul, MN. One approach is to compare soil Pb characteristics of areas which have common or dissimilar ages and locations with respect to the metropolitan area. Table 3 shows results of three neighborhoods from this study. "A" was built as a subdivision in 1965, while "B" and "C" were constructed between the 1880's and 1940's; "B" is located in the inner-city while "C" is located at the outskirts of the metropolitan area. Surface 2 cm soil scrapes were collected within one meter of the front/back/side steps, and from within one meter of the side of the house. Twelve homes (6 brick or stucco, and 6 painted wood structures), and one roadside sample were collected per block in a 16 block grid for each neighborhood. Data on exterior finish, date of construction, distance from street, and x,y coordinates from the city center were collected. In order to place these urban soil Pb results in perspective, uncontaminated rural soils outside the metropolitan area were also examined, and found to contain 5 ppm median Pb.

TABLE 3. DISTRIBUTIONS OF SOIL Pb CONCENTRATIONS IN THREE NEIGHBORHOODS OF MINNEAPOLIS-SAINT PAUL, MN (Mielke et al., unpublished)

Percentile	A=Suburban (N=67)	B=Inner-city (N=63)	C=Metro-margin (N=66)
	-------mg Pb/kg dry soil-------		
Min	36	2	36
10	42	112	62
25	58	308	86
50	78	562	146
75	108	1020	260
90	160	1560	366
Max	452	6360	1480

Suburban homes were all built in 1965. Inner-city homes were built between 1886 and 1941. Metro-margin homes were built from 1890 to 1928, with two homes built in 1960; the 1960 homes have soil Pb similar to the older homes.

If the overwhelming source of soil Pb contamination were paint-Pb, it would be expected that painted homes of the same age would have similar soil Pb levels around them; painted homes of different ages should have substantially different soil Pb around them. The data in Table 3 support the following observations: 1) Median soil Pb around older homes in neighborhood "C" at the edge of the metro area were about 30 times higher than rural soils, suggesting that paint is a major contributor to soil Pb; 2) the median soil Pb around old homes of neighborhood "C" had soil Pb levels only twice those of new suburban tract homes of neighborhood "A", suggesting that paint-Pb may be rather limited in its impact on soil Pb contamination and that another source has contributed; 3) the old inner-city neighborhood "B" has soil Pb levels about 4 times higher than similar aged homes in neighborhood "C" located at the metro margin, and 8 times higher than the suburban tract homes of neighborhood "A"; and 4) soil Pb levels of parks (medians in the range of 50 ppm Pb) tended to be far lower than soil Pb around residential structures within the same part of the city.

These empirical observations suggest a complex relationship between Pb sources, urban processes, and soil Pb concentrations. Why are there major differences between soil Pb levels around old painted homes located in different parts of the city? Why are soil Pb levels so similar between homes of substantially different ages on the outskirts of the city? What is a source of Pb which has substantially increased soil Pb around buildings in the inner-city but does not readily accumulate in open spaces?

For many years, the major use of Pb, accounting for over 90% of U.S. use, was as an additive to gasoline (161). The physical behavior of lead particles both in the engine and in the exhaust are well described (161). About 75% of gasoline Pb is emitted from the exhaust pipe. About 40% of exhausted Pb is in large enough particle sizes to be deposited in the vicinity of the highway. The deposited Pb accumulates in a pattern dependent on distance from the highway, with most of the accumulated Pb found within the first 20 to 40 meters from the highway, although significantly higher soil Pb extends to at least 220 m from the roadway (67,68). Very small particles make up about 35% of the exhaust-Pb, and these can move large distances from the roadway. In urban areas, these small particles tend to waft through the city and adhere to surfaces they come in contact with. The small aerosol particulates are scrubbed from the air by surface tension on foliage and structures. The particle removal process and ease of washing adherent particles from surfaces with water have been described by several authors (16,57,91).

Studies have been conducted on the pattern of Pb deposition around unpainted buildings (57,148). Pb in soil and dust is high next to the roadway, decreases toward the center of the yard, and increases by the side of unpainted structures. In the inner-city, where "set-backs" are often 5 m or less, there is little or no reduction from the roadway to the middle of the yard. Paint-Pb makes a different pattern by contributing amounts of Pb to the mid-yard but not to the streetside soils (57,83,122,-148,156). These kinds of studies are particularly relevant because buildings and their associated roadways are the main elements that make up the modern industrial city.

Highway traffic is unevenly distributed in metro areas. At the outskirts of the city, only a small quantity of gasoline-Pb is emitted per unit area. As one moves toward the central business district, there is a rapid increase in traffic flow as road networks increase in size and as the distance between major roadways decreases. Near the center of a city, almost every road carries a high volume of traffic. In these inner-city areas, the quantity of gasoline-Pb emitted is highest. Geographic variations in gasoline-Pb deposition can explain much of the unusual neighborhood Pb characteristics in Table 3. Neighborhoods "A" and "C" are in areas of low gasoline-Pb emission, while neighborhood "B" has high gasoline-Pb emission. Park soils have lower Pb levels because they lack the surface areas which buildings present to adsorb particles from the air (57).

Some authors have alleged that, because bromine (Br) is not greatly enriched along with Pb in houseside soils and housedusts, gasoline-Pb must make little contribution to houseside soil pollution with Pb. This argument rests on a misunderstanding of the fate of gasoline-Br. Although Pbbromide-chloride ($PbBrCl$) and related compounds are formed in the engine, allowing Pb to be discharged and Br to label the emitted Pb-rich aerosols (10,45,85,89,-161), the Br is rapidly lost from the Pb-rich aerosol particulate (10,45,-161). $PbBrCl$ reacts with sulfates in airborne particulates (10), and $PbSO_4$ is the only crystalline Pb compound found in surface soil near highways (10,69,116). Actually, even $PbSO_4$ added to soil rapidly reacts with the soil and Pb occurs as $PbCO_3$ or Pb adsorbed on Fe-oxides or organic matter (69). Br is highly water soluble and rapidly leaches away from the deposited particulates. Thus Br is unequivocally an inappropriate tracer for gasoline-Pb for longer that a few hours or days, and certainly not after the particulate joins the soil.

It is instructive to calculate the quantity of Pb consumed in leaded gasoline. Assuming Baltimore accounted for 10% of all traffic in Maryland, motor vehicles emitted between 5000 and 10000 metric tons of Pb into the city during the past 40 to 50 years (102). In Saint Paul, assuming a traffic flow of 4.5% of all traffic in Minnesota, at least 2800 to 5600 metric tons of Pb were emitted in the same time period (104). The above figures do not include local street travel, and are conservative for both cities, perhaps as much as 2-fold. Baltimore is 268 km^2 in area, while Saint Paul is 136 km^2 in area. Using low estimates, 24 and 20 metric tons (Mg) of Pb were deposited per km^2 (or 240 and 200 kg Pb/ha) in Baltimore City and Saint Paul, respectively. However, emitted Pb is not evenly deposited in the city. Thus, it seems clear that every major US city has become burdened with soil Pb in a pattern that corresponds with the urban road network.

While gasoline-Pb establishes one overall urban pattern of soil Pb, paint-Pb is also a prominent part of the contamination process. In any given part of the city, the highest soil Pb levels are found around buildings with Pb-rich paint, or where such buildings were demolished. For example, in the inner-city of Minneapolis, soil Pb around painted structures was about twice the level as around unpainted brick and stucco structures (103). Thus, in a given sector of the city, gasoline-Pb has contributed a major quantity of Pb to soil, but where paint-Pb is present, because of its particle size and Pb concentration, it adds an incremental spike of Pb-enrichment to the soil (26,156).

In summary, both paint-Pb and gasoline-Pb (and other point sources such as smelters, incinerators, etc.) have each played a role in the soil Pb contamination of the modern industrial city. But gasoline-Pb quantities so large, and were deposited in a such an uneven center-city pattern, that gasoline-Pb contamination of urban soils cannot be neglected. Both gasoline-Pb and paint-Pb are important to understanding the urban soil Pb enrichment process. Other urban factors exacerbate the Pb problem for children. For example, although parks are far less contaminated, young children are more likely to be playing near their residence. Because play space is often limited on the small lots of the inner-city, play sites are heavily trampled and are often bare of vegetation such that soils are easily accessible to children.

Pb levels in housedust and soil are correlated. Some researchers have studied only soil or only housedust, and estimate Pb transfer from each source separately. However, soil is a major component of housedust, approximately 45-50% (47). "Source apportionment" studies always find soil in housedust (10,31,38,46,47,49,69,74,82,83,89,116,130,156,158,159).

TABLE 4. URBAN FACTORS WHICH INFLUENCE URBAN SOIL Pb CONTAMINATION

Factor	Inner-City	Non-inner-city
<u>Population Density:</u>	High	Medium to Low
<u>Residence:</u>		
Paint-Pb	High Pb	Low Pb
Construction	Varied	Varied
Home age	Older	Newer
Maintenance	Often poor	Usually good
<u>Traffic (Gasoline-Pb):</u>		
Traffic Density	High due to population density and commuters	Low to Moderate
<u>Surface Soil (0-1 cm):</u>		
Soil Pb, mg/kg	Commonly 500 or higher	<100 to 250
Play Area	Small	Larger
Set Back From Roadway	2 to 10 m	usually > 10 m
Soil Cover	Often bare from use	Lawns, well kept
<u>Other Factors:</u>		
Socioeconomic	Lower Middle Less educated	Middle to Upper Middle Well educated
Nutrition	Poorer overall	Better

Summary: In all cases, the conditions which operate to increase Pb sources and transfer to children are at a maximum in the inner-city environment.

Further, the concentration of Pb in housedust is well correlated with that of yard or houseside soils, although housedust Pb is usually higher than soil Pb in locations where Pb air pollution has occurred, but lower than soil when only soil-Pb is a source (7,8,22,31,38,47,49,83,126,130,148,156, 159).

Soil usually has a larger average particle size than housedust or hand-dust. The concentration of Pb in soil and dust increases as particle size decreases, thus increasing the ease of Pb transfer to children's hands (38,39,48,123,149), and/or increase persistence on hands (39). Both smaller particle size and greater Pb concentration in the smaller particles increase the bioavailability of Pb in dust and soil.

For the inner-city child, all risk factors are unfavorable. They not only live in the environments most contaminated with all Pb sources, but they also have the highest probability of being in a socioeconomic and nutritional status which increase their likelihood of ingesting and absorbing Pb. Geographic factors, Pb sources, and cultural variables which operate to cause the excessive contamination of inner-city soils are summarized in Table 4.

BIOAVAILABILITY OF INGESTED Pb

When Pb in soil or dust is ingested by humans, the potential for adverse effects depends on absorption of the ingested soil/dust borne Pb. Research on laboratory animals over many years has characterized the effect of nutritional factors on Pb absorption and on Pb effects (93,94,-98). More recently, adult human Pb isotope absorption studies and Pb balance studies in infants have clarified much about human Pb absorption. In addition, several feeding studies using livestock and laboratory animals have directly tested Pb absorption from dietary soil/dust Pb.

Nutritional studies, mostly with rats, showed that deficiency of calcium (Ca) and iron (Fe) had very strong effects on Pb absorption. When dietary Ca fell below about 50% of the dietary levels recommended by the National Research Council (NRC), Pb absorption increased strongly (97). Not only was Pb absorption increased due to low dietary Ca, but the deposition of Pb in bone decreased, allowing Pb to reach higher levels in soft tissues than is usually observed for similar bone Pb levels. Many factors interact with Ca nutrition. A complex vitamin D interaction with Ca and Pb (147) did not explain seasonal variation in blood Pb in children (98). Mahaffey (93) has summarized these nutritional interactions in relation to known dietary limitations in urban poor children who comprise the "most-susceptible" group for excessive soil Pb.

Iron deficiency was also found to strongly affect Pb absorption in rats. Pb absorption declined with further increase in dietary Fe above the minimum dietary requirement (99). However, not all experiments came to this conclusion, although rat and chick feeding studies did find low Fe status increased Pb retention.

Independent research programs evaluated Pb absorption by adult humans (11,12,50,71,79,90,124,128,171). One of the most important findings of this research was that Pb absorption is greatly reduced by simultaneous ingestion of food (11,50,71,79,124) compared to Pb ingested during fasting. The effect of a meal on Pb absorption lasted about 2-3 hours after eating because of slow gastric emptying after a meal (79). This result may have important implications for absorption of soil/dust-Pb and especially water-Pb ingested between meals. Studies of which dietary components reduced Pb absorption identified minerals, then Ca, phosphorus (P), and phytate (inositol-hexaphosphate)/fiber (11,12,79). Combinations of Ca and P had more effect on Pb absorption than did Ca alone (12,71). Pb isotopes were incorporated into lamb liver and kidney, and into spinach to allow comparison of Pb intrinsic to a food with Pb isotope extrinsically added to a meal with that food. This research showed that "food" Pb was absorbed equal to Pb salts added to an equivalent meal (71). Pb in oysters was about 70% as bioavailable to Japanese quail as Pb acetate (155). James et al. (79) evaluated a number of meals and dietary components. Test meal components such as phytate (79) or ethylene-diamine-tetraacetate (EDTA) (50,79) reduced Pb absorption compared to the effect of an equal amount of Ca and P in a low phytate basal meal (more highly refined diet). On the other hand, milk in a meal increased Pb absorption compared to the expected effect of the Ca and P in the milk. Thus, phytate, fiber, and Ca in whole grain foods would tend to appreciably reduce Pb absorption compared to more highly refined grain products.

Although Ca reduction of Pb absorption by humans has been independently corroborated, and generally mimics results found in laboratory animals, opposite results have been reported for Fe effects. Watson et al. (171) reported that individuals with low ferritin, who absorb an increased fraction of dietary Fe, also absorbed increased amounts of carrier free Pb. However, Flanagan et al. (50) using ^{203}Pb with 200 ug carrier Pb, found no effect of Fe status (serum ferritin) or added dietary Fe on Pb absorption by humans. This was a direct test of the previous report, but used a better experimental design. One man who regularly used Fe supplements absorbed only 6% of dietary Fe but 75% of dietary Pb (fasting condition). Interestingly, the researchers (50) found no effect of dose on %-absorption within the range of 4-to-400 ug Pb/meal. The contradictory results of these researchers (50,171) are difficult to explain. However, the detailed design, proven methodologies, and numbers of subjects in the Flanagan et al. (50) study seemed convincing.

Another aspect of diet effect on Pb absorption was corroborated independently, that EDTA reduces Pb absorption (50,79). Chelation of an element can reduce its chemical activity and reduce absorption of some elements. The role of phytate (79) and of some fiber components (118) should be similar to EDTA, so that by chelating or adsorbing Pb in the small intestine (pH > 6), intestinal Pb absorption is reduced. Soil and dust should act like fiber in this regard, sorb Pb (70), and reduce Pb bioavailability .

Nutritional interactions usually depend on molar ratios of ions in the lumen of the intestine. Because typical dietary Pb may be only 20 ug/day for children, and < 100 ug/day for adults, testing 200 ug Pb in a single test meal now seems somewhat high for food Pb. Nevertheless, higher levels may be required to test for interactions relevant to pica levels of ingestion of Pb-rich soil.

Pb balance studies were conducted in babies fed experimental formula diets which differed in Pb concentration because of soldered cans used for some formulas and juice products (134,178). The very young infants received milk as the bulk of their diet, 41% of dietary Pb was absorbed when intake exceeded 5 ug Pb/kg body weight/day. Increased dietary Ca significantly reduced Pb retention in these babies (178). In a second study (134), infants ingesting milk from paper cartons got 16 ug Pb/day, while infants ingesting canned milk or formulas ingested 61 ug Pb/day. Blood Pb levels were significantly different, 7.2 vs. 14.4 ug/dL. These infants absorbed a much higher percentage of milk Pb than adults, confirming the effect of age on Pb absorption that was found in laboratory animal studies (98). These findings were very helpful in the Food and Drug Administration (FDA) attempt to replace Pb-soldered cans use for infant foods (80).

BIOAVAILABILITY OF PB IN INGESTED SOIL AND DUST

As discussed above, wildlife are unable to avoid exposure to soil/dust Pb in their habitat (42,76,77,137,176). These studies indicated appreciable bioavailability of soil/dust-Pb, but did not make specific comparison witn Pb added to control diets. Similarly, livestock grazing pastures on soils rich in Pb (mine wastes, smelter pollution, naturally Pb-rich soils) ingest soil-Pb. Toxicity has resulted in many cases (40,41,64,77,1[illegible], and Pb levels in body tissues show appreciable bioavailability of soil Pb.

Several studies were conducted to test the bioavailability of soil/dust-Pb. Stara et al. (150) reported studies of rats fed tunnel, highway, or smelter dusts. Accumulation of Pb in bone or kidney was non-linear with dose, with lower %-absorption as dose increased. They also showed that El Paso smelter dust (0.67 % Pb) had appreciably lower effect on blood Pb than Queens tunnel dust (2.22 % Pb) or Los Angeles freeway dust (1.04 % Pb) (fed 1 mg Pb/d in gelatin capsules, equal to approximately 100 mg Pb/kg diet). Bone and kidney were also lower for the lower Pb concentration smelter soil/dust than for the tunnel or freeway dusts. These tests did not compare Pb absorption from dusts with Pb acetate; they used a purified diet rather than a lab chow diet.

Dacre and TerHaar (27) conducted an evaluation of the bioavailability of Pb in houseside soil (990 mg Pb/kg) and roadside soil (2300 mg Pb/kg) compared to Pb acetate. Equal Pb concentrations were added from the 3 sources, 50 mg Pb/kg diet. A high fiber, high nutrient rat chow was used as the basal diet. Much lower blood and bone Pb levels were reached in their experiment than in that of Stara et al. (150). Bone and kidney Pb concentrations after 90 days feeding showed that soil Pb had significantly lower effect than Pb acetate. Bone results indicate that soil Pb was about 70% as bioavailable as Pb acetate.

We (19) reported data from a more detailed evaluation of the effect of soil on dietary Pb absorption. This research was conducted in collaboration with Drs. S. Welsh and O.A. Levander at Beltsville, MD. Rats were fed purified complete casein-sucrose diets with and without 5% uncontaminated soil, and with and without 50 mg Pb/kg diet as Pb acetate to test the effect of dietary soil on Pb absorption. Rats were also fed five Baltimore urban garden soils to compare bioavailability of real Pb-rich urban soils with that of Pb acetate. All Pb was added at 50 mg Pb/kg dry diet (unequal soil amounts). The results are shown in Table 5. Bone Pb concentrations were used to evaluate diet Pb bioavailability. The addition of 5% uncontaminated soil to the diet reduced Pb absorption to 53% of the Pb acetate control. Four soils with about 1000 mg Pb/kg yielded bone Pb about 25% of Pb acetate, while a garden soil with 10240 mg Pb/kg yielded bone 70% of the Pb acetate control. The general trend showed increased soil Pb bioavailability at higher soil Pb concentration (when soil Pb concentrations were higher, %-soil in the diet was correspondingly lower). We believe this would be expected because soil acts like a fiber and a Ca and P source in the diet and should adsorb heavy metals (70) in the lumen of the intestine and reduce Pb absorption.

Our use of purified diets (19) yielded much greater Pb absorption from dietary soil than that found by Dacre and TerHaar (27). The experimental protocols were similar, and dietary Pb was fed at the same level. Tibia Pb reached 247 mg Pb/kg in rats fed 50 mg Pb (Pb acetate)/kg purified diet, but femur Pb reached only 5-7 mg Pb/kg in rats fed the same level of Pb in a lab chow diet (27). Mahaffey and Michaelson (98) discussed this phenomenon, and stated it resulted from the much higher levels of Ca, Fe, and fiber in chow diets compared to "NRC" purified complete diets. A similar effect of diet was observed by Mylroie et al. (111) in a direct comparison of diet type on the absorption of paint-Pb. Very much higher bone and kidney Pb were reached using the purified complete rat diet normally recommended for toxicology studies. In many ways, these purified diets are similar to US diets because of low fiber and mineral levels (93,98).

TABLE 5. EFFECT OF SOIL ON BIOAVAILABILITY OF Pb TO RATS, AND BIOAVAILABILITY OF Pb IN URBAN GARDEN SOILS (19)

Diet1/	Pb in Tibia mg/kg tibia ash mean ± std. err.		Pb absorption compared to that of Pb acetate, %
Basal	0.3 ± 0.3	e2/	-
Basal + 5% Soil3/	0.0 ±	e	-
Basal + Pb Acetate	247. ± 10.1	a	100
Basal + Pb Acetate + 5% Soil3/	130. ± 29.5	bc	53
Basal + 706 ppm Pb Soil	40.0 ± 6.1	de	16
Basal + 995 ppm Pb Soil	108. ± 26.3	c	44
Basal + 1078 ppm Pb Soil	37.1 ± 7.3	de	15
Basal + 1265 ppm Pb Soil	53.6 ± 7.4	d	22
Basal + 10240 ppm Pb Soil	173. ± 21.8	b	70

1/ A purified casein-based complete diet was fed to Fisher rats for 30 days. Pb acetate and garden soils were added to supply 50 mg Pb/kg dry diet. The experimental garden soils comprised 7.08, 5.02, 4.64, 3.95, and 0.488 % of the dry diet, respectively.

2/ Means followed by the same letter are not significantly different ($P < 0.05$) according to Duncan's Multiple Range Test.

3/ Unpolluted farm soil (11 mg Pb/kg) near Beltsville, MD, similar to original soil in the urban gardens used in this experiment.

Research from another area provides other data relevant to the question of bioavailability of urban soil Pb. Sewage sludge has been added to usual, or practical, diets of livestock (comprised of normal bulky feed ingredients fed in commercial production practices) to evaluate food chain transfer (bioavailability) of Pb and other potentially toxic materials in the sludges. Substantial percentages of sludge in diets were used to simulate poor livestock management (worst case) situations in which cattle ingest up to 14% soil (52). Sludges used in these studies contained varied levels of Pb and other elements such as Fe and Ca known to interact with Pb. Studies reported by Kienholz et al. (84) and Johnson et al. (81) in which cattle ingested sludge with 780 or 466 mg Pb/kg, respectively, found increased bone, liver, and kidney Pb. Response was curvilinear, with the slope of increase in tissue Pb decreasing at higher sludge dose. In other studies, cattle grazing pastures amended with sludges or sludge compost containing lower amounts of Pb (380 mg/kg) had no significant change in bone or liver Pb concentration (34). Livestock have been poisoned by Pb when they grazed pastures growing on Pb-polluted soils under poor management conditions where forage was limited and usually had adhering soil (41,64,168). Most guidance on sludge utilization indicates that sludges must be below 500 mg Pb/kg dry weight for use on pastures, based on the above research results (33,163) although sludge Pb is not yet regulated (162).

Many scientists have considered the bioavailability of Pb in ingested soil and dust (19,32,38,46,58,69,158). Some have conducted chemical extractions to simulate conditions of the stomach, and found soil Pb was very soluble (19,32,58,69). Although some argue that solubility means availability, the above research shows that soil components may adsorb Pb at the pH of the intestine and thereby reduce Pb absorption. This effect would cause increased Pb absorption at higher soil/dust Pb concentration at any particular Pb dose. Further, the above research showed that a decreasing response slope results when increasing amounts of a soil are fed.

These responses are different characteristics of the bioavailability of soil Pb in the diet. However, the human isotope feeding study evaluated dose-response; they compared absorption of 4, 40, and 400 ug Pb fed to a fasting individual, found no effect of dose (i.e. a linear dose effect on absorption) (50). Other studies of children ingesting Pb-rich water contaminated by soft water and Pb water pipes, blood-Pb increased with the logarithm or the cube root of daily Pb intake, or a decreasing slope of response with increasing dose (144,146). Whether this effect can be explained by the food vs. fasting effect on Pb absorption is unclear. More research is needed to characterize soil/dust-Pb bioavailability. The infants of animal species close to humans should be studied using Pb isotopes. Deliberate Pb absorption studies with human infants are unethical whether radioactive or stable Pb isotopes are used.

ABATEMENT OF Pb-POLLUTION REDUCES BLOOD-Pb

It is important to identify whether abatement or removal of a Pb-source for children actually reduces the Pb-B of children exposed to that source. This type of finding would confirm the importance of the source, and help to define limits required to avoid excessive Pb-B from that source. Reduction in Pb-B has been found with abatement of many Pb sources!

Moving children from homes with high levels of paint Pb to homes with low paint Pb lowers Pb-B in every case (23,24,51). Addition of chemicals (e.g. lime) to reduce corrosiveness of soft waters reduced Pb-B in children and adults (109,144,146). Replacing Pb pipes may further reduce Pb-B, at least in some water systems, after proper addition of lime has been achieved (109,146). Reduction of Pb in infant foods (80) clearly reduced Pb-B in infants (134). Reducing Pb in gasoline reduced Pb-B (e.g. 3), while temporarily shifting stable isotopes of Pb in gasoline shifted Pb isotopes in blood of children and adults, and in dusts. Studies around smelters showed that stopping the Pb emissions reduced Pb-B in older children, but not in infants and young children (132,133). Replacement of soil, or covering soil (the alternatives to relocation of the exposed children) does lower Pb-B (110,132). When housedust is rich in Pb, reducing the amount of housedust per unit area of surface available to children, or reducing the concentration of Pb in the housedust lowered Pb-B of children (21,105). Detergent alone was not as successful in removing Pb-rich housedust from carpets as was washing with "Calgon" followed by detergent (105). Regular washing of children's hands before eating has also been shown to reduce Pb-B (39,145,173), although young children don't wash well or recontaminate their hands quickly after washing (39).

SETTING A MAXIMUM SOIL PB LIMIT FOR INHABITED AREAS

The most generally cited recommended limit on soil-Pb is that of the CDC (18). This document notes "In general, lead in soil and dust appears to be responsible for blood lead levels in children increasing above background levels when the concentration in the soil or dust exceeds 500-1000 ppm." This refers to the surface 1 cm of soil (V. Houk, CDC; personal communication). The CDC (18) advice specifically notes "In severely contaminated residential areas, unless an effective barrier can be established between the children and soil, surface soil must be removed and replaced with soil having a low lead content." CDC advises that remediation of excessive soil Pb should follow source control for air or paint Pb pollution. Best action is removal of Pb-rich soil and replacement, or relocation of populations. It must be noted that this suggested limit came originally from the 1977 EPA report on Pb (161). However, this consensus was reached at a time when the acceptable Pb-B was still 30-40 ug/dL, far above current acceptable safe levels, and that lower limits will be needed on soil Pb if children are to attain the current desired levels. Perhaps the most conservative view has been that of Patterson (112) who concluded that urban areas are so Pb polluted that they will need to be abandoned to protect the health of US children.

Because Pb in soil and dust contributes to a health hazard for children, it is essential that a soil Pb standard be established for child-accessible areas in order to begin dealing with this problem (19,38,61). Citizens need a soil Pb standard to make it possible to limit Pb sources and to reduce soil Pb levels in the urban environment (129). A standard sets in motion societal mechanisms which should make it possible to prevent Pb disease and protect children from this obvious environmental hazard both now and in the future. The fundamental question is "What limit on soil Pb will prevent excessive Pb absorption in exposed children?"

If we were preparing a new regulation for addition of Pb to unpolluted soils as a pesticide or as a component of sewage sludge or fertilizer, the evaluation would be straight forward. The "worst case" would be identified, and the draft regulation would have to protect the most-exposed, most susceptible population. As noted above, children's behavior and socioeconomic status are very important factors in estimating the effects of soil/dust-Pb ingestion. Thus, in the soil/dust-Pb case, the "most-exposed, most susceptible" population would be children with pica for soil/dust. The regulation would allow soil/dust-Pb concentration to be limited to such a level that the most susceptible child with pica for soil could not exceed acceptable blood Pb levels. Any regulation would have to take into consideration the background level of Pb consumption and background blood Pb levels for the most-exposed most-susceptible children, and the bioavailability of Pb in soil/dust.

As described in the introduction, the mean Pb-B of children 0.5-to-5-years-old in 1976-1980 was greater than 15 ug Pb/dL, the level currently believed to be the threshold for adverse effects of ingested Pb. Hence, there is room for no more environmental Pb, and present urban Pb pollution must be abated to correct existing adverse effects.

Duggan (38) has extensively evaluated the relationship of Pb-B to Pb in soil/dust. He used data from many published studies to estimate the slope of the Pb-B/Pb-Soil relationship in terms of increased Pb-B (ug/dL) with increase of soil Pb of 1000 mg/kg increments. However, he did not focus on the "most-exposed most-susceptible" individuals described above, but on "an average child playing in a normal dirty way." This is clearly not the worst case requiring protection usually used in US advisories and

TABLE 6. RELATIONSHIP OF Pb-B TO Pb CONCENTRATION IN SOIL AND DUST

Author(s)	Source of Pb	Change in Pb-B (ug/dL) / 1000 mg Pb/kg soil
Angle and McIntire (1)	Smelter/urban-suburban	4.0
Baker et al. (5)	Smelter worker-housedust	8.6
Barltrop et al. (7,8)	Housedust from mine wastes	4.0
Galke et al. (53)	High/Low urban soil	3.3
Gallacher et al. (54)	Housedust from mine wastes	4.7
Milar and Mushak (105)	Smelter worker-housedust	9.0
Port Pirie (4,100,173)	Smelter-soil	10-15
Reeves et al. (130)	Old/New housing	5.5
Roberts et al. (132)	Smelter-housedust	9.0
Roels et al. (133)	Smelter-playground dust	3.5
Schmitt et al. (138)	Smelter-soil; 1-3 and 6 year olds	7.8
Shellshear et al. (143)	Soil-houseside	3.9
Watson et al. (170)	Smelter worker-housedust	6.8
Yankel et al. (167,175)	Smelter-soil; 2-3 year olds	2.5

regulations. He does note that the Pb-B/Pb-Soil relationship does depend on many factors including at least: 1)age of person; 2)type of source; 3)form of Pb contamination (soil, housedust, paint chips); 4)time of year (higher in summer); and 5)geographic/demographic/socioeconomic factors such as race, diet/nutrition, personal hygiene, and social habits. Table 6 is our modified and updated version of Duggan's (38) summary of slopes based on data reported by many researchers. For these population average response slopes, the range was 2.5 to 9.0 ug Pb/dL per 1000 mg Pg/kg soil/dust. The range for many of these cases is 3-to-5-times the mean change in Pb-B, clearly showing that a 1000 mg Pb/kg standard will not protect the most-exposed most-susceptible population against excessive soil-Pb. Many other research studies support the general findings of Duggan, but lack the full data required to make a slope estimate (e.g. 14,22).

The study of excessive Pb-B in children in Port Pirie, Australia offers new and important data on what soil Pb level may be too high. A study was undertaken because possible Pb health effects on fetal development were noted in a city with long term and extensive Pb pollution from a primary Pb smelter (140). This was a prospective, longitudinal study, in which children were sampled repeatedly from birth to age 5. Thus, peak levels of Pb-B were observed for each child, taking into account age and season dependent processes and behaviors. This avoids limitations of the cross-sectional one-time sample where peak Pb-B levels are seldom observed for individual children. In this study, Pb-B was significantly raised (4,100,165) when soil Pb was in the range of 150-500 mg Pb/kg, for a 0-to-10 cm soil depth sample (17,101,160). Further, the method used to analyze soil Pb extracted about 85% of the total Pb (173). A separate cross-sectional survey of children's Pb-B was made after the severity of the problem was demonstrated. Soil was confirmed as a highly significant factor in Pb-B variation, as were bare soil percentage of lot, fingernail biting, dirty clothes and hands at school, mouthing behavior, and housedust Pb concentration.

Other research offers strong evidence that soil/dust Pb contributes to Pb-B well below average levels of 500 mg Pb/kg. These include studies by Angle (1), Brunekreef (15), and Cohen (25). Further, research results from the "middle class" prospective blood Pb study in Boston show a strong relationship of hand-Pb and soil/dust-Pb with Pb-B in infants with relatively low Pb-B levels (126).

Thus, we conclude that soils with 500 mg Pb/kg contribute to increased Pb-B in children, but seldom cause Pb-B to exceed 25 ug/dL in the absence of pica. Soil/dust-Pb especially contribute to the urban disadvantaged child who is the most susceptible, most exposed individual for Pb. If a 15 ug Pb/dL Pb-B standard is to be attained for all children, special care would be required to reduce inadvertant soil/dust ingestion even at 500 mg Pb/kg. Such special care is not available in the homes of many urban children. Further, children with pica are not protected by a 500 mg Pb/kg soil limit.

<u>Variability of Soil Pb Complicates Setting a Soil Pb Limit</u>. Another factor which must be considered in enforcing or setting a limit on soil Pb in areas accessible to children is the areal variability of soil Pb concentration. Both source factors which unevenly distribute Pb pollution, and human activities which disturb surface soil, affect the concentration of Pb in the surface 1 cm of soil most accessible to children. Two studies (19,37) show wide variation in soil Pb concentration in playgrounds and gardens. Table 7 shows results of detailed surveys of Pb concentration in three Baltimore urban gardens. Each garden was sampled in a grid pattern with 3 meters between point samples (bucket auger soil samples, 0-20 cm soil depth). Extreme variation occurred between sample locations, especially in garden C where it appeared that paint used to make a mural on the adjacent building may have been dumped on the soil. Both studies showed that surface soil Pb concentrations extend to at least 10-fold the mean value for an area, or the expected level for a composite sample. Duggan et al. (39) also showed the extreme variability of hand-Pb among children playing together.

Again, the child with highest hand Pb exceeded the group mean by over 3-fold, and the range exceeded 10-fold.

Table 7. VARIABILITY OF Pb CONCENTRATION WITHIN INDIVIDUAL URBAN GARDENS

	Garden A	Garden B	Garden C
	mg Pb/kg dry soil		
No. Samples	111	72	63
mean	740	626	261
C.V., %	56.2	70.3	207
geo. mean Pb	626	508	171
min. Pb	100	70	57
median Pb	640	525	152
max. Pb	2040	2440	4080
% over 500 Pb	66.4	51.4	4.8
% over 1000 Pb	25.2	16.7	1.6

If a mean of 500 mg Pb/kg soil were used as a soil-Pb limit, some specific points in a playground or garden/yard might exceed 5000 mg Pb/kg. If a mean of 100 mg Pb/kg were used, some points might exceed 1000 mg/kg due to specific human activities which created the polluted soil. Thus, formal limits on soil Pb concentration must take into account the uneven areal distribution of soil Pb.

Rapid analytical methods such as x-ray fluorescence could be used at a site where soil removal/replacement is being undertaken in order to determine Pb levels in many soil samples and aid in selecting soil areas which must be removed to be sure no remaining soil exceeds the desired level. Even in this case, some level below the actual regulated limit will have to be used to have any assurance that all excessive soil Pb has been abated.

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LITERATURE CITED

1. Angle, C.R. et al. 1979. J. Toxicol. Environ. Health 5:855-870. See also Angle, C.R. et al. 1985. Adv. Pediatr. 29:3-31.
2. Annest, J.L., and K.R. Mahaffey. 1984. Blood lead levels for persons ages 6 months - 74 years, United States, 1976-1980. Vital and Health Statistics, Series 11, No. 233. DHHS Pub. No. 84-1683, Publ. Health Serv. U.S. Gov. Print. Off., Washington. 61 pp.
3. Annest, J.L., et al. 1983. N. Engl. J. Med. 308:1373-1377.
4. Baghurst, P., et al. 1985. Environ. Res. 38:24-30.
5. Baker, E.L., Jr., et al. 1977. New Engl. J. Med. 296:260-261.
6. Berg, J.M., and M. Zappella. 1964. J. Ment. Defic. Res. 8:44-53.

7. Barltrop, D., et al. 1974. Environ. Health Perspect. 7:75-82.
8. Barltrop, D., et al. 1975. Postgrad. Med. J. 51:801-804.
9. Bertinuson, J.R., and C.S. Clark. 1973. Interface 2:6.
10. Biggins, P.D.E., et al. 1979. Environ. Sci. Technol. 13:558-565.
11. Blake, K.C.H., et al. 1983. Environ. Res. 30:182-187.
12. Blake, K.C.H., and M. Mann. 1983. Environ. Res. 30:188-194.
13. Bornschein, R.L., et al. 1985. Environ. Res. 38:108-118.
14. Bourcier, D.R., et al. 1985. Trace Subst. Environ. Health 19:160-172.
15. Brunekreef, B., et al. 1981. Environ. Res. 25:441-448.
16. Carlson, R.W., et al. 1976. Environ. Sci. Technol. 10:1139-1142.
17. Cartwright, B., et al. 1976. Aust. J. Soil Res. 15:69-81.
18. Centers for Disease Control. 1985. Preventing lead poisoning in young children: A statement by the Centers for Disease Control -- January 1985. US-DHHS No. 99-2230. 35 pp.
19. Chaney, R.L., S.B. Sterrett, and H.W. Mielke. 1984. pp. 37-84. In J.R. Preer (ed.). Proc. Symp. Heavy Metals in Urban Gardens. Agric. Exp. Sta., Univ. Dist. Columbia., Washington.
20. Charney, E. 1982. pp 79-88. In J.J. Chisolm, Jr., and D.M. O'Hara (ed)- Lead Absorption in Children. Urban and Schwartzenberg, Baltimore.
21. Charney, E., et al. 1983. N. Engl. J. Med. 309:1089-1093.
22. Charney, E., et al. 1980. Pediatrics 65:226-231.
23. Chisolm, J.J., Jr., et al. 1985. Environ. Res. 38:31-45.
24. Clark, C.S., et al. 1985. Environ. Res. 38:46:53.
25. Cohen, C.J., et al. 1973. J. Amer. Med. Assoc. 226:1430-1433.
26. Cooney, P.A., and P.G. Blake. 1982. Environ. Health 90:3-5.
27. Dacre, J.C., et al. 1977. Arch. Environ. Contam. Toxicol. 6:111-119.
28. David, O.J., et al. 1985. Arch. Environ. Health 40:109-113.
29. Davies, B.E. 1978. Sci. Total Environ. 9:243-262.
30. Davies, B.E., D. Conway, and S. Holt. 1979. J. Agr. Sci. 93:749-752.
31. Davies, B.E., et al. 1985. Environ. Pollut. B9:255-266.
32. Day, J.P., et al. 1979. Bull. Environ. Contam. Toxicol. 23:497-502.
33. Dean, R.B., and M.J. Suess (eds.). 1985. Waste Manag. Res. 3:251-278.
34. Decker, A. M., et al. 1980. pp 37-41. In Proc. Nat. Conf. Munic. Ind. Sludge Util. and Disp. Inf. Trans. Inc., Silver Spring, MD.
35. Dolcourt, J.L., et al. 1981. Pediatrics 68:225-230.
36. Dolcourt, J.L., et al. 1978. Pediatrics 62:563-566.
37. Duggan, M.J. 1984. Sci. Total Environ. 33:37-48.
38. Duggan, M.J., and M.J. Inskip. 1985. Public Health Rev. 13:1-54.
39. Duggan, M.J., et al. 1985. Sci. total. Environ. 44:65-79.
40. Edwards, W.C., and B.R. Clay. 1977. Vet. Human Toxicol. 19:247-249.
41. Egan, D. A., and T. O'Cuill. 1970. Vet. Rec. 86:736-738.
42. Elfving, D.C., et al. 1978. Arch. Environ. Health 33:95-99.
43. Elwood, W.J., et al. 1977. Br. J. Prevent. Med. 31:154-163.
44. Every, R.R., et al. 1981. J. Am. Vet. Med. Assoc. 178:1277-1278.
45. Farmer, J.G., and J.D. Cross. 1978. Water, Air, Soil Pollut. 9:193-198.
46. Fergusson, J.E. 1986. Sci. Total Environ. 50:1-54.
47. Fergusson, J.E., et al. 1986. Sci. Total Environ. 50:217-221.
48. Fergusson, J.E., and D.E. Ryan. 1984. Sci. Total Environ. 34:101-116.
49. Fergusson, J.E., and R.J. Schroeder. 1985. Sci Total Environ. 46:61-72.
50. Flanagan, P.R., et al. 1982. Am. J. Clin. Nutr. 36:823-829.
51. Foster, J.D., et al. 1979. Arch. Environ. Health 34:368-371.
52. Fries, G.F., et al. 1982. J. Dairy Sci. 65:611-618.
53. Galke, W.A., et al. 1977. Proc. Int. Conf. Heavy Metals in the Environment. III:53-74.
54. Gallacher, J.E.J., et al. 1984. Arch. Dis. Child. 59:40-44.
55. Gallacher, J.E.J., et al. 1984. J. Epidem. Commun. Health 38:173-176.
56. Galloway, J.N., et al. 1982. Atmos. Environ. 16:1677-1700.
57. Getz, L.L., et al. 1977. Chapter 6. pp. 105-134. In W.R. Bogges and B.G. Wixson (eds.). Lead in the Environment. NTIS Report PB-278278.
58. Gibson, M.J., and J.G. Farmer. 1984. Sci. Total Environ. 33:49-57.

59. Greenberg, R.R., et al. 1978. Environ. Sci. Technol. 12:566-573.
60. Gulson, B.L., et al. 1981. Environ. Sci. Technol. 15:691-696.
61. Haines, R.C. 1984. Trace Subst. Environ. Health 18:450-460.
62. Hammond, P.B., et al. 1985. Environ. Res. 38:187-196.
63. Hammond, P.B., et al. 1980. Intern. Arch. Occup. Env. Health 46:191-202.
64. Harbourne, J. F., et al. 1968. Vet. Rec. 83:515-517.
65. Hardy, H.L., et al. 1971. Clin. Pharmacol. Therap. 12:982-1002.
66. Harrison, R.M. 1979. Sci. Total Environ. 11:89-97.
67. Harrison, R.M., et al. 1985. Sci. Total Environ. 46:121-135.
68. Harrison, R.M., et al. 1985. Sci. Total Environ. 46:137-145.
69. Harrison, R.M., et al. 1981. Environ. Sci. Technol. 15:1378-1383.
70. Hassett, J.J. 1974. Commun. Soil Sci. Plant Anal. 5:499-505.
71. Heard, M.J., et al. 1983. Sci. Total Environ. 30:245-253.
72. Hecker, L.H., et al. 1974. Arch. Environ. Health 29:181-185.
73. Holmgren, G.G.S., et al. 1983. Agron. Abstr. 1983:33.
74. Hopke, P.K., et al. 1980. Environ. Sci. Technol. 14:164-172.
75. Hunt, T. et al. 1982. Amer. J. Dis. Child. 136:538-542.
76. Hutton, M., and G.T. Goodman. 1980. Environ. Pollut. A22:207-217.
77. Ireland, M.P. 1977. Environ. Pollut. 12:85-92.
78. Jackson, J.W., et al. 1977. J. Environ. Sci. Health A12:465-473.
79. James, H.M., et al. 1985. Human Toxicol. 4:401-407.
80. Jelinek, C.F. 1982. J. Assoc. Offic. Anal. Chem. 65:942-946.
81. Johnson, D.E., et al. 1981. J. Anim. Sci. 52:108-114.
82. Johnson, D.L., et al. 1982. Trace Subst. Environ. Health 16:116-123.
83. Jordan, L.D., and D.J. Hogan. 1975. N.Z. J. Sci. 18:253-260.
84. Kienholz, E., et al. 1979. J. Anim. Sci. 48:735-741.
85. Kowalczyk, G.S., et al. 1978. Atmos. Environ. 12:1143-1153.
86. Landrigan, P.J., et al. 1982. New Engl. J. Med. 306:673-676.
87. Landrigan, P.J., et al. 1975. New Engl. J. Med. 292:123-129.
88. Lepow, M.L., et al. 1975. Environ. Res. 10:415-426.
89. Linton, R.W., et al. 1980. Environ. Sci. Technol. 14:159-164.
90. Little, P., et al. 1981. Sci. Total Environ. 17:111-131.
91. Little, P., and R.D. Wiffen. 1977. Atmos. Environ. 11:437-447.
92. Madden, N.A. et al. 1980. J. Pediatr. Psychol. 5:207-216.
93. Mahaffey, K.R. 1982. pp 63-78. In J.J. Chisolm, Jr., and D.M. O'Hara (eds.) Lead Absorption in Children. Urban and Schwarzenberg, Baltimore.
94. Mahaffey, K.R. 1985. pp. 373-419. In K.R. Mahaffey (ed.) Dietary and Environmental Lead: Human Health Effects. Elsevier Press, Amsterdam.
95. Mahaffey, K.R., and J.L. Annest. 1986. Environ. Res. 41:327-338.
96. Mahaffey, K.R., et al. 1982. N. Engl. J. Med. 307:575-579.
97. Mahaffey, K.R., et al. 1977. Proc. Intern. Conf. Heavy Metals in the Environ. 3:155-164.
98. Mahaffey, K.R., and I.A. Michaelson. 1980. pp. 159-200. In H.L. Needleman (ed.) Low Level Lead Exposure: The Clinical Implications of Current Research. Raven Press, Boston.
99. Mahaffey, K.R., et al. 1978. Proc. Int. Symp. Trace Elements Metabolism in Man and Animals 3:584-588.
100. McMichael, A.J., et al. 1985. Med. J. Aust. 143:499-503.
101. Merry, R.H., et al. 1981. Environ. Pollut. B2:37-48.
102. Mielke, H.W., et al. 1983. Am. J. Public Health 73:1366-1369.
103. Mielke, H.W., et al. 1984. Environ. Res. 34:64-76.
104. Mielke, H.W., et al. 1985. J. Minn. Acad. Sci. :19-24.
105. Milar, C.R., and P. Mushak. 1982. pp 143-152. In J.J. Chisolm, and D.M. O'Hara (eds.) Lead Absorption in Children. Urban and Schwartzenberg, Baltimore.
106. Milar, C.R., et al. 1980. Amer. J. Ment. Defic. 84:339-344.
107. Millar, I.B. 1978. J. Epidemiol. Community Health 32:111-116.
108. Millar, I.B., and P.A. Cooney. 1982. Atmos. Environ. 16:615-620.
109. Moore, M.R., et al. 1985. Environ. Res. 38:67-76.
110. Morse, D.L., et al. 1979. J. Am. Med. Assoc. 242:739-741.

111. Mylroie, A.A., et al. 1978. Environ. Res. 15:57-64.
112. National Research Council. 1980. Lead in the Human Environment. National Academy of Sciences, Washington, D.C. 525 pp.
113. Needleman, H. L. 1980. pp. 43-51. In H. L. Needleman (ed.) Low Level Lead Exposure. Raven Press, New York.
114. Needleman, H.L., et al. 1974. N. Engl. J. Med. 290:245-248.
115. Needleman, H. L., et al. 1979 N. Engl. J. Med. 300:689-695.
116. Olsen, K.W., et al. 1975. Environ. Sci. Technol. 9:227-230.
117. Orton, W.T. 1970. The Medical Officer 123:147-152.
118. Paskins-Hurlburt, A.J., et al. 1977. Environ. Res. 14:128-140.
119. Piomelli, S., et al. 1980. Science 210:1135-1137.
120. Piomelli, S., et al. 1982. Proc. Natl. Acad. Sci. 79:3335-3339.
121. Poole, C., et al. 1980. Sci. Total Environ. 15:17-24.
122. Preer, J.R., et al. 1984. Biol. Trace Element Res. 6:79-91.
123. Que Hee, S.S., et al. 1985. Environ. Res. 38:77-95.
124. Rabinowitz, M.B., et al. 1980. Am. J. Clin. Nutr. 33:1784-1788.
125. Rabinowitz, M.B., et al. 1986. Environ. Res. 39:253-257.
126. Rabinowitz, M., et al. 1985. Environ. Res. 38:96-107.
127. Rabinowitz, M. B., et al. 1972. Environ. Sci. Technol. 6:705-709.
128. Rabinowitz, M.B., et al. 1976. J. Clin. Invest. 58:260-270.
129. Reagen, P.L. 1986. Protecting young children in Minneapolis: A proposal to reduce lead (Pb) exposure. 71 pp. The Lead Coalition, St. Paul,MN
130. Reeves, R., et al. 1982. N.Z. J. Sci. 25:221-227.
131. Rice, C., et al. 1978. Environ. Res. 15:375-380.
132. Roberts, T.M., et al. 1974. Science 186:1120-1122.
133. Roels, H. A., et al. 1980. Environ. Res. 22:81-94.
134. Ryu, J.E., et al. 1985. pp. 187-209. In K.R. Mahaffey (ed.) Dietary and Environmental Lead: Human Health Effects. Elsevier Press, Amsterdam.
135. Sayre, J.W., et al. 1974. Am. J. Dis. Child. 127:167-170.
136. Sayre, J.W., et al. 1979. Environ. Health Perspect. 29:179-182.
137. Scanlon, P.F., et al. 1983. Environ. Pollut. B6:157-160.
138. Schmitt, N., et al. 1979. Can. Med. Assoc. J. 121:1474-1478.
139. Schneider, D.J., et al. 1986. Am. J. Public Health 76:242-244.
140. Scragg, R.K.R., et al. 1977. Med. J. Aust. 2:68-
141. Settle, D.M., and C.C. Patterson. 1980. Science 207:1167-1176.
142. Shellshear, I.D. 1973. N. Z. Med. J. 78:251-254.
143. Shellshear, I.D., et al. 1975. N. Z. Med. J. 81:382-386.
144. Sherlock, J.C., et al. 1984. Human Toxicol. 3:383-392.
145. Sherlock, J.C., et al. 1985. Human Toxicol. 4:513-519.
146. Sherlock, J., et al. 1982. Human Toxicol. 1:115-122.
147. Smith, C.M., et al. 1978. J. Nutr. 108:843-847.
148. Solomon, R.L., et al. 1976. Environ. Sci. Technol. 10:773-777.
149. Spittler, T.M., et al. 1979. Commun. Soil Sci. Plant Anal. 10:1195-1210.
150. Stara, J., et al. 1973. pp. 28-29 In EPA Environmental Health Effects Research Series A-670/1-73-036.
151. Stark, A.D., et al. 1978. Arch. Environ. Health 33:222-226.
152. Stark, A.D., et al. 1982. J. Epidemiol. Community Health 36:133-139.
153. Stark, A.D., et al. 1982. Environ. Res. 27:372-383.
154. Starr, R.H., Jr. 1985. Environ. Res. 38:197-210.
155. Stone, C.L., et al. 1981. Environ. Res. 26:409-421.
156. Sturges, W.T., and R.M. Harrison. 1985. Sci. Total Environ. 44:225-234.
157. TerHaar, G., and R. Aronow. 1974. Environ. Health Perspect. 7:83-89.
158. Thornton, I. 1986. Proc. Canadian lead commission. In press.
159. Thornton, I., et al. 1985. Environ. Technol. Lett. 6:137-144.
160. Tiller, K.G., et al. 1976. CSIRO Div. Soils Report No. 15. 18 pp.
161. USEPA. 1977. Air Quality Criteria for Lead. EPA-600/8-77-017.
162. USEPA. 1979. 40 CFR 257. Federal Register 44(179):53438-53468.
163. USEPA, USFDA, and USDA. 1981. Land application of municipal sewage sludge for the production of fruits and vegetables. A statement of Federal policy and guidance. U.S. EPA Joint Policy Statement SW-905.

2 pp.
164. Vimpani, G., et al. 1985. Environ. Res. 38:19-23.
165. Vimpani, G.V., et al. 1986. Med. J. Aust. In press.
166. Vostal, J.J., et al. 1974. Environ. Health Perspect. 7:91-97.
167. Walter, S., et al. 1980. Arch. Environ. Health 35:53-58.
168. Wardrope, D.D., and J. Graham. 1982. Vet. Rec. 111:457-459.
169. Watanabe, T., et al. 1895. Arch. Environ. Health 40:170-176.
170. Watson, W.N., et al. 1978. J. Occup. Med. 20:759-761.
171. Watson, W.S., et al. 1980. Lancet 2:236-237.
172. Wedeen, R.P., et al. 1978. Environ. Res. 17:409-415.
173. Wilson, D., et al. 1986. Arch. Environ. Health 41:245-250.
174. Yaffe, Y., et al. 1983. Arch. Environ. Health 38:237-245.
175. Yankel, A.J., et al. 1977. J. Air Pollut. Contr. Assoc. 27:763-767.
176. Young, R.W., et al. 1986. J. Toxicol. Environ. Health 19:91-96.
177. Yule, W., and M. Rutter. 1985. pp. 211-259. In K.R. Mahaffey (ed.) Dietary and Environmental Lead: Human Health Effects. Elsevier Biomedical Press, Amsterdam.
178. Ziegler, E.E., et al. 1978. Pediatr. Res. 12:29-34.

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