ABSTRACT

There are approximately 57 million homes in the US with lead-based paint. The presence of this paint poses a particularly potent health threat to potentially millions of children. Their behavior and physiology significantly increases their intake, uptake, and adverse symptoms of lead exposure over most other segments of the population. Recent information on exposure, health effects, and toxicokinetics of lead have made lead-based paint dust ingestion by young children and the subsequent low-level exposure (resulting in potentially adverse behavioral and neurological effects) the focus of abatement strategies. Recent efforts on the part of the Department of Housing and Urban Development (HUD) have resulted in lead-based paint abatement techniques that hold greater promise of effectiveness in lead abatement than traditional deleading methodologies. Thus the question posed to public health authorities is how best to combine this biological information with the latest abatement technologies within today's regulatory and legal framework to create a mitigation strategy that is effective from both a public and economic perspectives? A number of strategies suggested in this paper include: the integration of lead-based paint dust related toxicokinetics into current legislation and regulation; the development of a profile for those individuals in the population most at risk for adverse health effects due to household lead exposure and targeting the majority of limited resources at them; further research on toxicokinetics, health effects and treatment for lead poisoning; further research and development for the testing and abatement of lead-based paint.
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I. INTRODUCTION

Public health officials consider lead the number one environmental health threat to children. The current risks posed by existing concentrations of lead in the environment are surprising given the long-standing ban on lead-based paint and the virtual absence of leaded gasoline at the gas pump. As Dr. Louis Sullivan, Secretary of the Health and Human Services says, "Lead poisoning is entirely preventable, yet it is the most common and societally devastating environmental disease of young children".

There are many possible sources of lead exposure for children. The government has effectively prevented new lead from pouring into the environment. One source of exposure, however, continues to be of most concern to officials - lead paint. Over 57 million private homes still contain lead-based paint and thus pose a serious health hazard to significant portions of the US populations. Lead poisoning in children exists primarily because lead paint persists on the walls, ceilings and other surfaces of much of US private and public housing. As the amount of research increases on the health hazard (especially adverse neurological effects) of even minute quantities of lead on young children, the impetus to confront this threat through further regulatory intervention grows.
With respect to the route of exposure, it is not the lead-based paint on the walls that directly constitutes the potential health hazard for children but rather the resulting lead-based paint dust and chips that accumulate as the painted surfaces deteriorate. The dust and chips can cause situations ranging from very low level lead exposure to severe cases of lead poisoning.

Lead-based paint was used most extensively in the first half of this century. The lead-based paint pigments were valued for their durability, adhesion and masking abilities. In the early 1950s, public health authorities were for the first time able to trace cases of lead poisoning to deteriorating lead-based paint surfaces. As a result, in 1955, the paint industry voluntarily set an industry standard of no more than 1% lead by weight of non-volatile solids in lead-based paint.

In 1971, the Lead-Based Paint Poisoning Prevention Act (LPPPA) was enacted by the federal government. This legislative act prohibited the use of lead-based paint (greater than 1% lead by weight) from being used in any residential structure constructed, rehabilitated or financed by the federal government in any form. It also authorized a mass screening program to identify children with lead poisoning, refer them for medical treatment, and to investigate and mitigate the sources of lead exposure in their residential environment. The LPPPA also successively lowered
the content of lead in paint necessary for it to be considered
'lead-based paint' from 1% to 0.06%. In 1973, amendments to
the LPPPA required that the US Department of Housing and Urban
Development (HUD) eliminate, to the extent practicable, the
hazard of lead-based paint poisoning in pre-1950 housing that
is federally owned and/or financially assisted by the federal
government. The construction cut-off date (housing built
after this date would not be scheduled for abatement
strategies) was later moved up to 1978. Finally amendments in
1988 required HUD to develop a "comprehensive and workable
plan for abatement in public and private housing" for all
homes built before 1979.

According to the Environmental Protection Agency, one out
of nine children under the age of 6 has enough lead in his/her
blood to place him/her in a condition scientists now consider
to represent a risk of adverse health effects. In the inner
city, the U.S. Public Health Service estimates one out of
every two children exceeds this limit of concentration'.

Lead poisoning through exposure to lead-based paint is a
serious problem primarily for infants and young children. The
young are at a greater risk because of their greater hand-to-
mouth activity, the vulnerability of their developing
neurological systems, the greater absorption and retention of
lead per unit body weight compared to adults, the greater
availability of lead in the blood and soft tissues to exert
toxic effects, and the more frequent presence of nutritional
deficiencies (i.e. copper, iron, calcium) that enhance uptake, absorption, and retention of lead\(^2\). Along with infants and children, fetuses are also at high risk, as lead may be transferred through the placenta of the woman to the fetus.

Only in the past decade or two has there been appreciable research interest in the potential risk posed by low levels of lead as measured on child IQ and development. Previously, emphasis was placed upon lead poisoning at higher levels where acute physiological distress such as anemia or stomach ailments was observed. But in 1979, Dr. Herbert Needleman in Boston, Massachusetts found that asymptomatic children who had higher lead residues in their teeth performed worse on IQ and development tests than those children with less lead\(^3\). Among these 158 first- and second-grade children, an IQ deficit of approximately 4 points was associated with poorer performance on auditory, verbal, attentional, and behavioral test with blood lead values in the range of 30 to 50 \(\mu g/dl\) range\(^4\). Since Needleman's study other researchers have continued observing the health effects of low-level lead on children. The Centers for Disease Control (CDC), in response to the recent research on the threat of low-level lead exposure in children, lowered the point at which the clinical diagnosis of lead poisoning is said to have occurred from 30 \(\mu g/dl\) in the 1970s to 10 \(\mu g/dl\) today.

As the problem of lead poisoning in the home becomes the focus of more scrutiny, the scope and dimension of lead
poisoning in our society is being gradually revealed. Lead poisoning was once considered the result of eating peeling lead paint but the importance of lead dust in the household is realized to pose the greatest health threat. A child can become severely ill with a blood lead level of 60-80 ug/dl by eating only a milligram of lead-based paint each day. Lead poisoning cuts across socio-economic lines - children in expensive townhouses in the process of being renovated can be as susceptible as children in inner city housing with peeling paint on the walls. Current research points to the previously unrecognized threat of low level exposure to lead on the well-being of young children, so that lead abatement efforts must be taken to a much greater level of stringency. Fortunately with this knowledge of the importance of a higher standard of abatement comes more information on the exposure, health effects, and toxicokinetics of lead with the human body. The question thus becomes how best to combine this biological information with the latest abatement technologies within today’s regulatory and legal framework to create a mitigation strategy that is effective from both a public health and economic perspective.
II. THE OCCURRENCE OF LEAD IN THE ENVIRONMENT

Lead is the most ubiquitous toxic metal. The blue-gray metal is found naturally in the earth's crust. Though lead is found naturally in the environment, the majority of the lead dispersed in the environment is due to human activity. It can be found in varying concentrations in the air, water, dust and soil as well as the plants and animals that humans consume. It exists either in solid forms, dust or particulates of lead dioxide or in the vapor forms like alkyl lead emitted from automobile exhaust. Lead is usually derived from mined ores or scrap metal. It is valued for its malleability and its corrosion resistance. Lead is used in batteries, piping, solder, gasoline and paint. In terms of public health efforts, the most important source is lead-based paint, with leaded gasoline and lead soldering in plumbing of less importance due to their lesser prevalence and exposure.

About a decade ago, most of the lead entered the environment via car exhaust. The burning of gasoline has been the largest source (approximately 90%) of lead in the atmosphere since the 1920s. EPA effort in the last fifteen years has resulted in only about 35% of the lead released in the air coming from gasoline. This reduction of lead in air correlates well with declines in childhood blood lead levels.
between 1976 and 1980 found by the National Health and Nutrition Survey (NHANES II). EPA reports that total atmospheric lead emissions have dropped 94% between 1978 and 1987 due to a phasing out of leaded gasoline and stricter regulations on industrial sources. Today, the release of lead from landfill to soil is a larger source of lead contamination than through airborne release by lead-emitting sources into the atmosphere. Lead usually enters water bodies via rain. Rain removes lead from the air and the rainwater then washes the lead attached to soil particles into rivers, lakes and streams.

The range in ambient airborne lead concentrations can encompass two to three orders of magnitude between remote, uninhabited regions of the globe to heavily industrialized cities. Values range from 0.000075 μg/m³ in the Antarctica to 10 μg/m³ near smelter compounds. However, even these isolated areas have considerably higher ambient lead concentrations than in pre-industrial times. Typically indoor airborne lead concentrations are around one half of exterior concentrations depending on the season and the presence of air conditioning in the household. The concentration of lead in ambient air varies significantly within the US. Urban areas have greater concentrations than rural regions and indoor concentrations are less than outdoor ones. The National Air Surveillance Network (NASN) has monitored ambient airborne lead levels since the 1960s. In
1988, the average concentration of 18 point source sites was 0.4 ug/m$^3$ and 0.1 ug/m$^3$ for urban sites. Both of these measurements are less than the National Ambient Air Quality Standard of 1.5 ug/m$^3$, and about one eighth of what they were in the 1970s - the decrease is primarily attributed to the phasing out of leaded gasoline$^{10}$.

In the atmosphere, lead is found in particulate form. It can be released from the atmosphere either by wet or dry deposition. Wet deposition involves the removal of lead from the atmosphere by wet fallout - it can account for 40 - 70% of lead deposition depending on geographic and emission considerations. Dry deposition involves particulate lead emissions settling near the source. Wet deposition is generally 1.5 to 2.5 times more significant quantitatively than dry deposition in terms of removing atmospheric lead$^{11}$. Lead removed from the atmosphere is retained primarily in the upper 2-5 cm of undisturbed soil, with the natural concentration usually under 30 ug/g soil$^{12}$. Near roadways, significant quantities of lead can be deposited by dry deposition up to about 25 meters away, thus leaving soil in the vicinity of the road with much higher lead soil concentrations than would otherwise be found - concentrations as high as 2 000 ug/g soil. Similarly, near lead smelter plants, lead in soil may be found in increased concentrations in a 5-10 km zone around the complex. Urban soil may also be contaminated with lead originating from the lead-based paint
chips from older houses. Levels of soil-lead concentrations over 10,000 ug/g have been measured around certain households.

Lead is found in water either dissolved or in particulate form. Lead may enter water bodies from industrial release, atmospheric deposition or runoff. Lead battery factories, lead smelters, automobile exhaust and waste landfills are prime examples of water-contaminating sources. Lead may also be leached from the lead solder used in pipes and thereby enter household drinking water. The phenomenon of leaching is particularly problematic if the water is acidic in nature. A combination of corrosive water and lead pipes or lead solder in the plumbing system can result in lead levels exceeding 0.50 mg/l\textsuperscript{13}. In surface and ground water, the levels of lead typically range from 5 to 30 ug/l with levels as high as 890 ug/l having been found in surveys\textsuperscript{14}. The variation in levels is generally due to properties of the individual water source such as proximity to lead emitting locations, lead content in sediments and other characteristics like temperature and pH. Lead concentrations in drinking water can range from 0.01 - 0.03 mg/l.

Plants and animals may retain lead, but have not been shown to biomagnify. Not surprisingly, the majority of the highly contaminated animal and plant life has occurred near locations of high automobile traffic and/or industrial sources of lead emission.
Lead has also been measured in significant concentrations in food. The use of lead solder in sealing canned goods was once a large contributor of dietary lead. Typically, food can range in lead concentration from 0.002 ug/g to well over 0.6 ug/g of food. The lead enters foodstuff through bioconcentration or during its harvesting, transporting, processing, and preparation.
III. HUMAN EXPOSURE TO LEAD

The three possible routes of absorption for lead are from inhalation, oral or dermal exposure. The relative importance of each exposure route is dependent to a large degree on the location of exposure and the age of the individual being exposed. For instance, adults in occupational settings are exposed primarily by inhalation of airborne lead. Whereas children in a home setting are exposed primarily by the ingestion of lead-based paint dust contaminating their immediate environment or in some cases they may eat lead-based paint chips directly - a process termed the pica phenomenon.

Inhalation may occur via direct inhalation of lead-containing particles emitted from a variety of industrial sources such as smelters, battery factories and the combustion of gas, oil and coal. Oral exposure and the subsequent ingestion seems to be the primary route of exposure in non-occupational settings. Home refinishing or deteriorating lead paint in homes results in interior surface dust that coats interior surfaces and objects which are subsequently ingested by the frequent hand-to-mouth activity of children. Exterior soil may also be contaminated due to exterior lead-based paint, or from the
atmospheric fallout from combustion of gasoline, factory emissions, industrial solid waste etc. Contaminated soil can result in crop land and water sources having a high lead content. Lead intake can also occur through foods and beverages. The lead enters the food either through the environment or from the processing of food. Lead intake through consumption of food varies with age, sex and food habits. Dermal exposure and subsequent absorption is a much less significant route than the respiratory or GI routes for inorganic lead but not necessarily for lead alkyls. Dermal exposure has minimal significance as a public health threat and it is more important in specific occupational exposures.

Exposure to lead in a home environment varies greatly with individual circumstances e.g. presence and condition of lead-based paint and proximity to industrial emissions of lead. Lead exposure may also be increased significantly by other habits such as smoking (2.5-12.2 ug per cigarette), consumption of lead-containing wine, eating produce from a high lead soil family garden, or pica on the part of young children. As a result a child may be taking in as much as 1 300 ug lead/day (with adults normally exposed to much lower levels) in a household.

Older homes may have lead or copper pipes with lead solder which would leach lead into the drinking water supply if the water was sufficiently acidic or corrosive. In recognition of this potential health hazard, the Safe
Drinking Water Act in 1986 banned the use of lead solder with more than 0.2% lead content and lead pipes or fittings with more than 8% lead.

Occupational exposure accounts for the highest and most lengthy exposures to lead. The industries most likely to involve high occupational exposure to lead are lead smelting, refining and manufacture. In these work areas, lead exposure occurs primarily by inhalation and ingestion of lead-containing dusts and fumes. Lead dust may settle on clothes, food, water and other surfaces and eventually be transferred to the mouth. Alkyl leads used in the creation of gasoline additives may be absorbed dermally.

In monitoring lead exposure, biological testing of workers is used rather than measuring environmental lead concentrations. Within a lead related industry, the range in blood lead levels can vary greatly over a given time period as can the ambient lead concentrations. For example, in a particular electronics firm, workers who manufacture ceramic-coated components were exposed to airborne lead concentrations ranging from 61 to 1700 ug/m³ with blood lead levels from 16 to 135 ug/dl. As much as a third of the workforce was on medical leave at any one time for having had blood lead levels over 40 ug/dl.

The occupational safety and health guidelines for inorganic lead exposure vary depending on the organization.
The Occupational Safety and Health Administration (OSHA) has a permissible (airborne) exposure limit (PEL) for lead of 50 \( \text{ug/m}^3 \). The National Institute for Occupational Safety and Health (NIOSH) has a recommended (airborne) exposure limit (REL) of 100 \( \text{ug/m}^3 \). The American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit value (TLV) is 150 \( \text{ug/m}^3 \). In setting these airborne lead concentration limits, a time-weighted average (TWA) lead concentration is used with the assumption of an 8-10 hour workday and a 40 hour work-week\(^8\).

In decreasing lead exposure, proper housekeeping and adequate ventilation are crucial. Protective work clothing can also play a part in reducing exposure. The risk is considerable as even tiny amounts of dust (i.e. 10 mg) containing 100 000 \( \text{ug/lead/g} \) can account for up to a 1 000 \( \text{ug/day} \) intake of lead\(^9\).

Secondary occupational exposure can occur from the worker transporting lead on his/her clothes to the household from the workplace. In some cases, this secondary occupational exposure can be an important source of exposure to the worker and his/her family.

A number of studies have demonstrated the significance of dust lead as a pathway of exposure for children. The Food and Drug Administration 1980-1982 Diet Study, conducted in 13 locations across the US with infants and toddlers, estimates that children receive approximately 75\% of their
lead exposure through dust. Dr. E. Charney and colleagues in 1980, first demonstrated that dust lead and hand lead were significantly correlated with blood lead levels\(^2\). In their next study, Charney and his colleagues investigated the efficacy of dust control methods in lowering blood lead levels\(^3\). The results indicated that such methods could lower blood lead levels. Dr. D. Bellinger and colleagues collected data on a variety of variables concerning children with low to moderate blood lead levels in a metropolitan area\(^4\). The two characteristics that were most significantly associated with blood lead levels were the amount of environmental lead (i.e. in water, air, dust, paint) and mouthing behavior.

The Cincinnati study was the landmark study in determining the antecedent conditions which occur in most cases of high environmental exposure to lead by young children\(^5\). It is not solely the presence of lead-based paint that results in lead exposure but the condition of the painted surface. Deteriorating interior or exterior lead-based painted surfaces result in high levels of paint dust and chips in and around the home. The lead dust contaminates the interior and exterior surfaces, resulting in high amounts of dust on the child's hands. It is the hand contamination which ultimately leads to the subsequent ingestion of lead and to higher blood lead levels. The greater amount of lead-based paint dust in the air also
results in a greater intake via inhalation. Lead-based paint dust becomes of tantamount importance during dust-creating activities such as home refinishing, sanding, scraping and repainting\textsuperscript{24}. In the Cincinnati lead study, a variety of variables were examined - blood lead, hand dust lead, interior surface dust lead, exterior surface dust lead, and the lead content and condition of the paint. The final conclusion is that the lead paint content and condition of painted surfaces determines the amount of dust lead and impacts on the amount of hand lead which ultimately determines blood lead.

In defining which individuals are most likely to be exposed to lead (other than the occupationally exposed), research has shown that black children in the lowest family income stratum living in central cities of metropolitan areas of a million or more had the highest blood lead levels - 68% had levels greater than 15 ug/dl in 1984. In comparison, in the least affected group (white children in the highest family income stratum living outside central cities in metropolitan areas of a million or more) only 4.7% the children had above 15 ug/dl\textsuperscript{25}. Although the relative percentages of the various categories vary greatly, the absolute numbers in all categories indicate extensive exposure to lead.

The importance of race in determining incidence of lead exposure, assuming other factors are held constant, is not
easily explained. Differences in biology, behavior and/or nutritional patterns may account for the discrepancy.

Federal lead-based paint regulation and abatement is authorized primarily by the Lead-Based Paint Poisoning Prevention Act of 1971 (LPPPA) and its later amendments. In 1987, the LPPPA was comprehensively amended. One of the amendments required that HUD perform a national survey of lead-based paint in housing to estimate the actual extent of lead-based paint hazards in US residences.

The objective of the national survey of lead-based paint in housing was to determine the number of housing units with lead-based paint, the condition of the paint, the incidence of lead in the household dust as well as exterior soil, and the characteristics of those households which pose the greatest lead related health hazard to its occupants. Most lead poisoning prevention programs use X-ray fluorescence to detect the amount of lead in the paint of household surfaces. The detectors measure the amount of lead in paint surfaces in milligrams per square centimeter (mg/cm²). All the homes surveyed were built before 1980, as the Consumer Product Safety Commission banned the use of lead-based paint by consumers and its use in residences in 1978.

The national survey required by the LPPPA 1987 amendments was performed by Westat Inc. in 1989-90. Prior to this most recent national surveys, there were four
earlier surveys - three were local and one was national\cite{7}. Three of the four surveys had HUD sponsorship - only the Phoenix, Arizona Survey was not under their aegis. The Pittsburgh, Washington D.C. and Phoenix surveys were local in scope - examining urban housing units and conducted in the mid-seventies. In contrast, the Modernization Needs Study of Public Housing, was conducted in 1984-85, was national in scope. The study was based in 34 US cities and inspected 131 public housing projects.

Unfortunately, these surveys were limited in usefulness due to the imprecision of the particular XRF analyzers used and the questionable representativeness of most of these surveys. The incidence of lead in house dust and exterior soil were also not monitored.

A housing unit is considered to contain lead-based paint if any of the paint has a lead content of 1.0 mg/cm\(^2\) or greater. This lead paint concentration threshold is in accordance with the Federal standard for lead-based paint, established in Section 566 of the Housing and Community Development Act of 1987. However, two states have different standards - Maryland (0.7 mg/cm\(^2\)) and Massachusetts (1.2 mg/cm\(^2\)). Using the data from these surveys the Agency for Toxic Substances and Disease Registry (ATSDR) estimate that the total number of dwellings in the US with lead-based paint concentrations greater than or equal to 0.7 ug/cm\(^2\) is \(41\,964\,000\)\cite{28}. This estimate assumes lead based paint was
in 99% of residences built before 1940, 70% of the residences built between 1940 and 1959, and 20% of units built between 1960 and 1974. In addition, the ATSDR estimates that 1,972,000 of these units are in 'unsound' condition.

According to the survey data, an estimated 57.4 million homes or 74% of all occupied housing units built before 1980 have lead-based paint in the building. The age of the home seems to be a significant factor in determining the likelihood of the presence of lead-based paint. An estimated 90% of housing units before 1940 have lead-based paint, 80% of the homes built from 1940-1959 and 62% of the homes built from 1960-1979.

The concentration of lead in the painted surface of a particular household for it to be considered 'lead-based' varies on the threshold chosen. For the purpose of the survey, a lead content value of 1.0 mg/cm² or greater as measured by XRF constitutes 'lead-based paint'. Reducing the threshold to a lower value would significantly increase the number of housing units with lead-based paint increasing from 57.4 million to roughly 66.3 million homes.

Though the incidence of lead paint in new homes is drastically less than that of older homes, the overall number of newer homes with lead-based paint is larger due the larger overall base of new homes.
The Westat Survey also shows that the exterior of the home is more likely to be coated with lead-based paint than the interior but also to be in a worse condition and hence a greater health threat. Interestingly, the lead household dust seems to be generated primarily by exterior lead-based paint. The majority of that dust is located on window wells and sills where they can easily receive dust from the interior or exterior of the house. The exterior paint can also contaminate soil surrounding the home. Non-intact, lead-based paint surfaces pose a direct hazard due to the presence of paint flakes and resultant greater household lead-containing dust. Roughly 18% of the housing units showed evidence of non-intact lead-based paint.

HUD has recommended clearance levels for lead dust after lead-based paint abatements. The levels are 200 ug/ft² for floors, 500 ug/ft² for window sills, and 800 ug/ft² for window wells. According to these guidelines, over 80% of homes with lead-based paint are under these guidelines.

Though there are approximately 57 million privately owned homes with lead-based paint, they differ in the degree of health hazard they pose. The three factors other than the presence of lead-based paint itself which determine the likelihood of lead poisoning are the presence of children under 7 years of age, the condition of the paint and the presence of lead in the household dust. Thus according to
the survey data less than 10 million of the 57 million homes containing lead-based paint contain children under the age of 7, and over 3.8 million of these homes have high dust lead levels or non-intact paint.

The CDC recommended mitigation if soil lead levels exist between 500 and a 1000 ppm. For the purposes of the survey, the guideline for soil lead was 500 ppm. Roughly, 18% of the housing units surveyed exceeded this guideline. Soil lead is a possible source of lead in household dust.

The lead in the soil has many possible environmental sources but the primary contributor to high lead content in the surrounding soil of a particular household is the presence of exterior lead-based paint especially in a non-intact state.

In the households surveyed, an average of about 580 square feet of the interior painted surfaces are covered with lead-based paint in those homes where lead-based paint was found. Surprisingly, though almost half of interior lead-based paint is found on walls and ceilings, only 6% of the paint on all walls and ceilings is lead-based. In fact, the lead-based paint is much more likely to be found on less obvious and smaller surfaces and components like radiators, window sills, stair trim etc. In terms of exterior lead-based paint, there is an average of about 900 square feet per a household and that there is more lead-based paint on the exterior of newer homes than in the interior.
Researchers are using the data from the Westat Survey to hypothesize the pathways by which lead-based paint results in dust which is ingested by young children\textsuperscript{31}. The hypothesized major pathways of lead from paint to dust can be two-way or one-way (see diagram i)\textsuperscript{32}. The values between pathways are the correlation coefficients between the natural logarithms of the pairs of survey measurements of lead associated with the pathways. All the correlations are significant to a 0.05 level and most are significant at the 0.001 level. Though correlations do not necessarily prove causation, regression analyses supports that the paint is the source of lead in the dust. Note that the wetness of the room affects the correlations between various outcomes.

Overall, the utility of the survey was unquestionable in confirming prior research and uncovering other aspects of the lead-based paint health hazard in US households; three-fourths of occupied housing units built before 1980 have lead-based paint; older homes (especially pre-WWII) are more likely to have lead-based paint, the presence of lead in household dust is strongly correlated with lead-based paint. Other aspects not fully realized previously were the prevalence of lead-based paint on the exterior contaminating soil eventually leads to interior lead-containing dust and the importance of window areas as the primary sources of lead dust from both exterior and interior origin.
MAJOR HYPOTHESESIZED PATHWAYS OF LEAD FROM PAINT TO DUST, AND CORRELATION COEFFICIENTS BETWEEN THE NATURAL LOGARITHMS OF SURVEY MEASUREMENTS OF LEAD FOR EACH PATHWAY
To determine a minimum or baseline exposure for humans, it is necessary to quantify the amount of lead consumption derived from the major sources of lead (air, water, soil, and food). The result is that baseline exposure has three main sources of lead: inhaled air; food, water and beverages; and dust. Atmospheric air would result in exposure via inhalation whereas food, water, beverages, and dust would result in exposure through ingestion. Based on information from EPA and the FDA Total Diet Study, the baseline exposure to lead for a 2 year-old child would be 0.5 ug/day by inhalation, 5.0 ug/day by food and drink, and 21 ug/day by dust for a total consumption of 26.5 ug/day. Similarly, an adult female would inhale 1.0 ug/day, eat and drink 8.0 ug/day, and ingest 4.5 ug/day of dust for a total consumption of 13.5 ug/day. The adult male would consume 1.0 ug/day by inhalation, 12.0 ug/day by food and drink, and 4.5 ug/day by dust for a total consumption of 17.5 ug/day. The baseline human exposure is achieved primarily by ingestion with 98% or more of the daily lead intake by this route.

Beyond the baseline level of human exposure, additional amounts of lead consumption can occur depending on individual circumstance. Possibly as much as 90% of the population is affected by one of these additional sources of exposure. As many as 11.7 million children less than 7 years old are exposed to lead in dust and soils from a
variety of sources: 5.9 million children are exposed from lead-based paint; 5.6 million children are exposed from leaded gasoline and lead emissions in cities; and 0.2 million children are exposed from stationary emission sources such as smelters and battery plants. Also, 6.6 million children may be at risk from exposure to lead in drinking water as a result of lead solder and pipes used in plumbing. The contribution of these sources may dwarf the average daily intake in the general population. For example, in young children, various exposure scenarios may greatly increase their total lead intake over the baseline: urban atmosphere (increase of 91 ug/day), interior lead paint (+110 ug/day), home close to lead smelter (+880 ug/day), presence of family garden (+48 ug/day), and/or secondary occupational exposure (+150 ug/day). In addition, the adult baseline exposure may be increased by occupational exposure (+1100 ug/day), smoking (+30 ug/day), urban atmosphere (+28 ug/day), interior lead paint (+17 ug/day), home close to a lead smelter (+100 ug/day), family garden (+120 ug/day) and/or heavy wine consumption (+100 ug/day). Note that these estimates are useful only to illustrate the relative significance of various exposure scenarios. Also, it is important to realize that the same source of additional lead exposure tends to result in a much greater lead intake in children than in adults. There are a number of assumptions made in the derivation of the amount of daily
lead intake attributable to additional exposure (beyond the baseline exposure) in an attempt to calculate their 'typical' contribution to daily lead intake e.g. distance from smelters, soil lead concentrations, amount of packs of cigarettes smoked, atmospheric lead concentration, lead content of dust, etc.

As the majority of the population in the US consumes more than the baseline exposure to lead due to additional exposure scenarios, it is worthwhile to examine which routes of exposure might be most effectively mitigated. It seems from the exposure data, that ingestion is the primary route of exposure. Mitigation should thus concentrate on reducing lead in our food and beverages and in the reduction of lead-containing dust in the environment or its consumption. This fact does not negate the importance of atmospheric lead as a contributor to lead intake. Atmospheric lead contaminates soil and water sources which in turn result in lead-containing food and beverages. And food and beverages are the single largest source of lead in baseline exposure.

In terms of additional exposure, public health authorities have stressed the importance of lead-based paint in the home as a source of exposure for lead. Lead-based paint may not be as concentrated a source of lead exposure as secondary occupational exposure or exposure due to close proximity to a lead smelter. Yet, it poses a more serious hazard because of its overall prevalence. Lead-based paint
and dust has the potential to affect millions of children from all socio-economic groups, and as such poses a serious health threat.
IV. EMPIRICAL MEASURES OF THE RELATIONSHIP BETWEEN EXPOSURE AND INTAKE

In determining the degree of exposure of an individual to lead, biological testing is used. A bioassay of exposure to lead is usually found by measuring the total lead levels in bodily tissues or fluids. Some examples of these bioassays are the measurement of lead in plasma, teeth, hair, and urine. Lead exposure may also be measured indirectly via physiological changes that are known to signify lead exposure and thus serve as useful biomarkers. Some examples of these biomarkers for lead exposure are the measurement of erythrocyte porphyrin, urinary coproporphyrin, delta-aminolevulinic acid dehydrase activity, delta-aminolevulinic acid in urine and plasma, pyrimidine-5'-nucleotidase activity and plasma 1,25-dihydroxyvitamin D.

Blood lead concentration is the most widely used bioassay for lead exposure. As the half-life of lead in blood is approximately 36 days, it is considered to be an indicator of relatively recent exposure. The problem with this biomarker is that lead is cycled between the blood and bone (see diagram ii). A given blood lead concentration could represent a short-term recent exposure or the result of chronic, long-term exposure - two very different exposure
DIAGRAM (ii)

Lead Metabolism Model*

DIET + AIR

\[ \sim 48 \frac{\mu g}{\text{Day}} \]

3 BONE?
\sim 200 \text{ mg}
\sim 10^4 \text{ Days}

\[ \lambda_{31} \sim 7 \frac{\mu g}{\text{Day}} \]

\[ \lambda_{13} \sim 7 \frac{\mu g}{\text{Day}} \]

1 BLOOD
\[ 1.9 \pm 0.1 \text{ mg} \]
\[ 36 \pm 5 \text{ Days} \]

\[ \lambda_{10} \]

\[ \lambda_{12} \sim 15 \frac{\mu g}{\text{Day}} \]

\[ \lambda_{21} \sim 2 \frac{\mu g}{\text{Day}} \]

2 SOFT TISSUE?
\sim 0.6 \text{ mg}
\sim 40 \text{ Days}

URINE
\[ 36 \pm 8 \frac{\mu g}{\text{Day}} \]

BILE, HAIR, SWEAT, NAILS...
\sim 12 \frac{\mu g}{\text{Day}}

* Source: Rabinowitz et al. 1976
scenarios. Also at low levels of lead exposure, blood lead concentration is more sensitive than at higher levels.

Attempts to correlate environmental lead levels in separate media such as air, water and soil with a given blood lead level have met with mixed success (see diagram iii). For ambient air levels, the overall relationship between blood lead and environmental exposure is nonlinear across the entire range of exposure. Yet, there is linearity in the blood lead – air relationship within normal ambient exposures (0.1 – 2.0 ug/m$^3$), assuming the subject’s blood lead level does not exceed 30 ug/dl$^3$ . At higher ambient air levels, especially above 10 ug/m$^3$, either nonlinear or linear relationships can be fitted (see diagram iv). For adults, a blood lead level of 1.64 $\pm$ 0.22 ug/dl per 1 ug/m$^3$ is a typical estimate for an adult$^4$. Over a wide range of water-lead concentrations, there is curvilinearity with blood lead levels. However at typical ambient water levels, there exists a linear relationship. At low water lead levels (<100 ug/l), an estimate of 0.06 ug/dl per 1 ug/dl has been calculated$^2$. In a soil medium, it is much harder to predict blood lead levels given a certain soil-lead concentration due to factors such as soil depth, particular hand-to-mouth activity, and age of children. However a median estimate of 2 ug/dl per 1 000 ug/g of soil has been estimated$^4$. 

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Figure 1-18. Geometric mean blood lead levels of New York City children (aged 25-36 months) by ethnic group, and ambient air lead concentrations versus quarterly sampling period, 1970-1976.

Source: Billick et al. (1980).
### Summary of Blood Inhalation Slopes (β)

<table>
<thead>
<tr>
<th>Population</th>
<th>Study</th>
<th>Study Type</th>
<th>N</th>
<th>Slope</th>
<th>Model Sensitivity* of Slope</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Children</strong></td>
<td>Angle and McIntire (1979) Omaha, NE</td>
<td>Population</td>
<td>1074</td>
<td>1.92</td>
<td>(1.40-4.40)*</td>
</tr>
<tr>
<td></td>
<td>Roels et al. (1980) Belgium</td>
<td>Population</td>
<td>148</td>
<td>2.46</td>
<td>(1.55-2.46)*</td>
</tr>
<tr>
<td></td>
<td>Yankel et al. (1977); Walter et al. (1980) Idaho</td>
<td>Population</td>
<td>879</td>
<td>1.52</td>
<td>(1.07-1.52)*</td>
</tr>
<tr>
<td><strong>Adult Male</strong></td>
<td>Azar et al. (1975). Five groups</td>
<td>Population</td>
<td>149</td>
<td>1.32</td>
<td>(1.08-1.59)*</td>
</tr>
<tr>
<td></td>
<td>Griffin et al. (1975) NY Prisoners</td>
<td>Experiment</td>
<td>43</td>
<td>1.75</td>
<td>(1.52-3.38)*</td>
</tr>
<tr>
<td></td>
<td>Gross (1979)</td>
<td>Experiment</td>
<td>6</td>
<td>1.25</td>
<td>(1.25-1.55)*</td>
</tr>
</tbody>
</table>

*Selected from among the most plausible statistically equivalent models. For nonlinear models, slope at 1.0 μg/m³.

*Sensitive to choice of other correlated predictors such as dust and soil lead.

Sensitive to linear versus nonlinear at low air lead.

Sensitive to age as a covariate.

Sensitive to baseline changes in controls.

Sensitive to assumed air lead exposure.
Urinary lead levels as a biomarker is questionable in its utility because of the relatively low levels of lead excreted in the urine and the tendency for these levels to fluctuate. Teeth, being a form of bone, are known to accumulate lead over time, and thus make a better indicator of cumulative lead exposure than current lead exposure. Hair is used as an indicator of lead exposure as well and is valued for its non-invasive measurement but can be altered due to various hair treatments such as bleaching or dyeing. Also it is difficult to differentiate between external and internal deposition of lead with hair samples.

Erythrocyte porphyrin (EP) or zinc protoporphyrin may be measured as a biochemical index of lead exposure. With lead exposure, EP IX has impaired placement of divalent iron to form heme with divalent zinc taking the place of the iron. The interference with heme synthesis that occurs with lead exposure also results in an increase of urinary coproporphyrin. Similarly, the reduction in heme synthesis results in decreased activity of the enzyme delta-aminolevulinic acid dehydrase (ALA-D). The decreased ALA-D activity results in the accumulation of its precursor, delta-aminolevulinic acid and its subsequent increase in concentration in the urine and plasma ALA-D is considered a more sensitive indicator of lead exposure than ALA^4. A less specific yet sensitive biomarker for lead exposure is serum 1,25-dihydroxyvitamin D concentrations. It seems that
lead, inhibits this precursor metabolite of vitamin D which is crucial in bone mineral metabolism.
V. TOXICOKINETICS OF LEAD IN THE BODY

Inhalation remains the primary route of exposure for persons involved in lead related industries, whereas gastrointestinal absorption through dietary lead sources remains the major route for the general population. Children due to their greater hand-to-mouth activity and greater physiological capacity for GI tract lead absorption remain the most at risk for exposure through ingestion.

The deposition fraction of particulate airborne lead is 30-50% depending on ventilation rate and particle size. And from the total inhaled lead mass, 30-37% will retained in the pulmonary alveoli and subsequently absorbed. It is thought that once the lead deposits in the alveolar region of the lower respiratory tract, it is almost completely absorbed into the bloodstream. For alveolar deposition, particle size has to be approximately less than 1 um. For example, studies of the size distribution of airborne lead particles in urban atmospheres indicate a mass median aerodynamic diameter (MMAD) of between 0.18 and 0.6 um. Respiratory uptake of lead in children appears to be greater than adults on a body-weight basis. Also the relative deposition fraction in children tends to be 1.6-2.7 times
higher than adults per a unit body-weight basis due to different relative airway dimensions.48

In children, the gastrointestinal system is the primary location for lead absorption following ingestion. Children are much more efficient then adults in their uptake as well as intake of lead. Dietary lead absorption in children is roughly 50% compared to 15% gastrointestinal lead absorption measured in adults49. Gastrointestinal absorption of lead is not only age dependent but related to food intake. Fasted mice seem to have significantly greater routes of gastrointestinal absorption with smaller particles more easily absorbed. The chemical form of lead also seems to have a slight effect on its absorption.

In the Rabinowitz model (see diagram v) absorbed lead distributes itself into three main compartments - blood, bone and soft tissues50. The lead burden or the total amount of lead in the body is not distributed homogeneously between these physiologically distinct compartments. The blood compartment is the first compartment for the lead to enter and the last one it occupies before excretion. It also acts as an intermediary between the bone and soft tissue compartments. It is the bone compartment which ultimately retains over 90% of the human body burden of lead51.

Compartment 1, the blood compartment, contains approximately 1% of the body lead burden. In the 1% of the
A three-compartmental model of human lead metabolism. This model is derived from tracer and balance data from five healthy men. The lead content and mean life of each pool and the rates of lead movement between pools (λ) are shown. Numerical values represent the mean values (± S.D.) for all subjects for whom data were available. Loss of lead from the body via pool two (λ_{20}) is from integumentary structures (hair, nails, sweat) and alimentary tract losses, such as salivary, biliary, gastric and pancreatic secretions.
lead burden in the blood, 90% is bound to the erythrocyte and 10% is in the plasma free to diffuse between the other two compartments. Blood lead levels can be affected by inhalation, ingestion or release of lead from deep tissue and/or bone compartments. In individuals in the early phase of lead exposure, blood lead levels are good indicators of current lead exposure, but in persons with long-term heavy exposure to lead, blood lead levels primarily reflect release of stored lead from skeletal lead stores.

In a lead metabolism study of 5 normal men exposed to an atmospheric lead concentration of about 2 ug/m³, 14+/-4 ug/day of lead were inhaled and about 34 ug/day were ingested. The experimental atmospheric lead concentration is in the high end of the range of normal values, resulting in a decrease in the differential between the relative contribution of the ingestion route over that of inhalation. All the values in the experiment are approximate except for air-lead intake, blood lead burden, blood lead mean half-life and urine excretion. The blood compartment was in a dynamic equilibrium with the bone compartment as 7 ug/day of lead was being absorbed by the skeletal structure and 7 ug/day were being resorbed back into the bloodstream from the bone. About 15 ug/day of lead was absorbed from the bloodstream into the soft tissue compartment, and 2 ug/day was redirected in the opposing direction. And 36+/-8 ug/day of lead was excreted from the blood into the urine. The
result was a blood lead burden of 91.9+/-1 mg in the blood compartment with a mean half-life of 36+/-5 days. Thus in total roughly 53+/-4 ug/day are entering the blood compartment and 58+/-8 ug/day are being removed from this compartment.

Compartment 2, the soft tissues, can exchange lead with the blood compartment or excrete it in bile, hair, sweat and nails. It has a net lead absorption of 13 ug/day from the bloodstream and a loss of 12 ug/day in excretion. The total soft tissue-lead burden is about 0.6 mg with a mean half-life of 40 days.

Compartment 3 consists mostly of the skeletal structure. Lead accumulates in this compartment throughout life. Over 90% of the total body burden of lead is located in the bones of human adults compared to only 73% in children due to their younger, more metabolically active skeletal systems. In fact, the bone-lead burden can result in an excessive recirculation of lead back into the bloodstream resulting in unhealthy high concentrations of lead in the blood.

Finally, 'chelatable lead' is the fraction of the lead body burden that is mobile and is potentially the most toxic to the human body due to its greater bioavailability. This portion of the lead burden can be chelated with a chelating agent such as CaNa2EDTA and thus be removed for therapeutic reasons. The origin of this chelatable fraction probably
occurs in the labile lead compartments within the bone or soft tissues.

Inorganic lead is not known to be metabolized or biotransformed (Phase I processes) but it does undergo Phase II processes. Conversely, alkyl lead compounds do undergo Phase I reactions. Lead that is ingested either through diet or by swallowing of air is eliminated from the gastrointestinal tract in the feces. That portion of lead that is eliminated from the bloodstream is excreted through the renal tract or the GI tract via biliary clearance. Overall, 75% of total lead absorbed is excreted, roughly 50% immediately and the other 25% over time.

The fact that young children are much more susceptible to lead exposure than children is well documented. To reiterate, children have more sensitive, developing neurological systems; greater hand-to-mouth activity bringing them in greater contact with lead in the environment; more efficient absorption and retention of ingested lead and a higher likelihood of nutritional deficiencies that can enhance absorption and retention of lead. Also well researched is that adverse neurological effects can occur at as low a blood lead level as 10 ug/dl.

The differing dosages resulting from a hypothetical daily intake of 1 gram of lead permit a comparison between the exposure by inhalation, ingestion and dermal routes.
The estimates are approximate and are based on toxicokinetic information already presented. Inhalation would result in a dosage of about 0.35 gram-days for adults and 0.80 gram-days for children. Ingestion would result in a dosage of 0.15 gram-days for adults and 0.50 gram-days in children. Based on a study using adult human volunteers and lead acetate, dermal absorption would result in 0.06 gram-days.

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VI. HEALTH EFFECTS OF LEAD

The organ systems that are most affected by exposure to lead are the red blood cells and their precursors, the nervous system, and the kidneys. Lead has also been shown to have adverse effects on the male and female reproductive system as well as being a potential human carcinogen and mutagen. Recently, studies have pointed towards evidence of the dangerous effect of lead on children’s development due to pre-natal lead exposure.

Lead has a strong disruptive effect on heme biosynthesis. Specifically, it inhibits delta-aminolevulinic acid dehydrase (ALA-D), ferrochelatase and coproporphyrinogen oxidase. Subsequently, heme activity is decreased resulting in a loss of feedback inhibition of delta-aminolevulinic acid synthetase (ALAS). Ultimately these changes result in increased urinary porphyrins, coporphyrins, delta-aminolevulinic acid (ALA), erythrocyte protoporphyrins (EP), free erythrocyte protoporphyrins (FEP), and erythrocyte zinc protoporphyrins (ZPP).  

The disruption of heme biosynthesis by lead exposure leads to a reduction in hemoglobin, finally resulting in anemia. Lead workers have shown a documented decline in hemoglobin levels at 40-60 ug/dl. With children the...
threshold in blood lead depression of hemoglobin may be even lower\textsuperscript{39}. There also seems to be a heme basis of lead neurotoxicity - the increase in ALA in the body due to the effect of lead on heme biosynthesis may inhibit the release of the neurotransmitter gamma aminobutyric acid (GABA) from presynaptic receptor adversely affecting neurotransmission\textsuperscript{60}. Disruption of neurological functioning may result in acute encephalopathy, neurobehavioral impairment, or central and peripheral nervous system dysfunction.

In terms of neurotoxic effects of lead, acute lead encephalopathy can result in a coma, convulsions, irreversible intellectual and behavioral impairment and possibly even death. In adults, acute lead encephalopathy generally occurs at blood lead levels of 120 ug/dl or higher, and with children as low as 80-100 ug/dl\textsuperscript{61}. Asymptomatic individuals with high blood lead levels may have severe neural damage without any obvious signs of acute encephalopathy. Recently, neurological dysfunction has been reported at blood lead levels previously considered comparatively safe i.e. 40-60 ug/dl.

Presently, nationwide there is a concern about the effect of low levels of lead exposure and irreversible deficits in intelligence and possible behavioral effects. Blood lead levels of 50-70 ug/dl in asymptomatic children have been associated with an average 5 point IQ decrement in
several studies. In the 30–50 ug/dl range, a 4 point decrement was observed. Below 30 ug/dl, the various studies have mixed results. Some studies show no significant difference and others estimate an average 1–2 point decrement in IQ. A shift in mean IQ of between 2 and 5 points may seem small but would result statistically in a tripling of the number of children with IQs below 80 and great reduction in the children with IQs above 125.

In adults, alterations in CNS function are evident at blood lead levels as low as 40 ug/dl – a blood lead concentration once considered to be relatively safe. Other subjective signs of neurotoxicity such as fatigue, headaches, weakness, decreased libido, lethargy were observed in lead workers at below 40 ug/dl. Deficits in oculomotor function were observed in lead workers with mean blood lead levels of 57–61 ug/dl and in hand-eye coordination/reaction tests at a mean level of 60.5 ug/dl. Other studies have shown decreased attention, concentration, memory and increased fatigue, psychological disturbances and psychomotor performances among those lead-exposed workers who had blood lead levels of 50–80 ug/dl. Blood lead levels in these ranges are not at all unusual in heavy lead using industries like battery plants or smelting operations where blood lead levels over 120 ug/dl can be found.

The exact causes and mechanisms of lead-induced nephropathy have not been fully revealed. But for over a
century, the implication of lead in kidney disease has been known. Most of the studies and cases of lead-induced nephropathy involve occupationally exposed workers, possibly due to the high levels and long duration of lead exposure necessary for adverse effects to occur. Early or acute effects of lead on the kidneys include cellular changes in the proximal tubules and increased sodium and decreased uric acid excretion. Chronic lead nephropathy may lead to interstitial fibrosis, dilation of tubules, cellular effects on tubular epithelial cells, and decreased glomerular filtration rate. Renal effects have been associated with blood lead levels as low as 30 ug/dl in occupationally exposed workers. In certain case studies there have been some evidence of excess mortality in lead battery and smelter workers of chronic renal disease.

Most of the data regarding the toxicity potential of lead on the human reproductive system is based on laboratory animals. Based on rodent data, fetotoxic effects have been observed by adding 600-800 ppm of inorganic lead in their diet. Slight effects are noticed at 5-10 ppm in drinking water or in ambient air with an air-lead concentration of 10 mg/m³. Teratogenic effects have only occurred upon a maternal dose by injection. There are reports of occupational exposure to lead resulting in an increase in spontaneous abortions among female lead smelter workers and to a lesser degree women in nearby towns. Studies also show
a marked decrease in fertility among workers exposed to lead. It has also been suggested that lead stored in maternal bones can be released during pregnancy and thereby transferred to the fetus. The potential danger of lead on fetuses is thought to occur mainly at blood lead levels of over 25 ug/dl. Occupational exposure of lead to men has caused a decrease in sperm count and motility and an increased proportion of abnormal spermatozoa at an average blood lead concentration of 42.5 ug/dl.

Animal data have shown that lead can act as a genotoxic and carcinogenic substance only at relatively high concentrations suggesting it is not a potent carcinogen. The International Agency for Research on Cancer (IARC) classifies lead as a 2B carcinogen - evidence for carcinogenicity is adequate for animals but inadequate in humans. There is a paucity of human data but a statistically significant rise in respiratory tract and digestive diseases in workers occupationally exposed to high levels of lead has been observed. Sister chromatid exchange has been observed in lead-exposed workers at a mean blood lead concentration of 48.7 ug/dl and in environmentally exposed children between 30-63 ug/dl. Some studies demonstrate that lead exposure may have certain adverse immunological effects such as decreases in leukocyte counts, circulating antibody and antibody forming cells. Lead has been shown to render animals more susceptible to endotoxins.
and infection. Adverse effects of lead have also been observed on cardiovascular, hepatic, gastrointestinal and other systems at high lead levels.

Occupational, general population and animal studies all show evidence of a small but positive association between blood lead levels and increases in blood pressure. Based on theses studies, the effect of blood lead concentration on blood pressure was estimated to be an increase of 7 mmHg at blood lead levels between 14 and 30 ug/dl.

Animal models have been used extensively in determining the potential health effects of environmental toxins such as lead. A scarcity of dose-response data is due to the impractical and/or unethical nature of using human subjects. Thus most information on lead toxicity on humans originates from cohort studies performed on occupationally exposed workers and to a lesser degree environmentally exposed children. Given differences between humans, rodents and primates in physiology and anatomy, it is difficult to determine what constitutes an equivalent internal exposure level between human and lab specimen e.g. does 50 ug/dl for a monkey equal 50 ug/dl in a human? Thus, the primary value for animal studies involves information of the basic mechanisms of neurotoxicity, genotoxicity, carcinogenicity etc.

The major problem in the dose-response data for lead exposure in children is that seldom is the concentration of
lead in all environmental media and the magnitude of intake by a subject per unit time quantified. Estimates for total lead intake have been made for broad demographic groups but not relationships between various doses of lead and corresponding blood lead levels.

The following study is a good example of most of the studies on lead exposure in children. Blood lead levels of 1 to 9 year-old children living within 1.6 km of a lead smelter for a duration of up to nine years resulted in an average blood lead level of over 60 ug/dl\(^3\). The children were exposed to an annual mean air lead concentration of 18 ug/m\(^3\), which is about 10-20 times the normal atmospheric concentration of lead. In comparison, at normal atmospheric lead concentrations, children in the same age range exhibit blood lead levels in the 12-15 ug/dl range\(^4\). Or another example - research shows that acute encephalopathy becomes apparent in a child population with a blood lead level over 80 ug/dl\(^5\). Note that in the studies the total intake of lead is not estimated, only the concentration of lead in one medium or the health effects observed at a certain internal concentration. The scarcity of information on total environmental media lead concentration and lead intake results in a difficulty in establishing a lowest-observed-(adverse-) effect level for children whom are environmentally exposed to lead. Thus the crucial links
between total environmental concentration, total intake and threshold dose for lead are not easily forged.

Using the Rabinowitz model, an estimate for the blood lead level of a 70 kg adult male with a blood volume of 3500 ml and a baseline lead intake of 17.5 ug/day can be made. In the Rabinowitz experiments, a 48 ug/day intake would result in a blood lead burden of 1.9+/−1 mg and thus a blood lead concentration of approximately 55 ug/dl. Assuming the same relationship for the lower baseline exposure, an estimate for blood lead concentration for an adult male would be approximately 18 ug/dl. Unfortunately, there is insufficient experimental data to estimate what the blood lead concentration would be for a child at a baseline exposure of lead of 26.5 ug/day. It is not unreasonable to assume a significantly higher blood lead concentration given that children have a greater intake of lead as well as more efficient physiological lead absorption.

The adult male, at baseline exposure, would probably not be afflicted with lead induced adverse health effects. Most of the adverse health effects do not become apparent until at least 30 ug/dl in the adult. But the child, being more vulnerable to the effects of lead exposure, could quite possibly show evidence of neurological effects at 18 ug/dl. The CDC uses 10 ug/dl as the point at which concern is warranted in children. It is important to realize that these are values are for baseline lead exposure and do not
include any abnormal exposure e.g. lead-based paint, proximity to lead smelter, etc. A safe daily lead intake for an adult male would be below 20 ug/day but for a child, daily lead intake should be considerably below 10 ug/day. This reduction would entail a gross reduction in the amount lead-containing dust that is ingested as this is the major contributor of lead intake for most children.

Given the range of lead concentrations in a variety of media and the toxicokinetic information already presented, a chart for the relative risks for lead can be devised (chart I). This chart allows public health authorities to be able to decide which environmental compartment poses the most risk for the theoretical average adult and child. The information then can be used to address a lead exposure related health problem at a community level (chart II).
# Chart 1

## Relative Risks for Lead in Various Media

<table>
<thead>
<tr>
<th>Compartment</th>
<th>Exposure</th>
<th>Range of Conc'n</th>
<th>Daily Intake Per Unit Conc'n (adult/child)</th>
<th>Daily Uptake Per Unit Conc'n (adult/child)</th>
<th>Relative Risk (adult/child)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air</td>
<td>Inhalation</td>
<td>0.000075-10 ug/m^3 (0.4 ug/m^3 average)</td>
<td>1.30-2.20ug/1.31-2.18ug</td>
<td>1.30-1.60ug/1.31-1.62ug</td>
<td>0.10/0.03</td>
</tr>
<tr>
<td>Water</td>
<td>Ingestion</td>
<td>10-500ug/l (20ug/l average)</td>
<td>40ug/20ug</td>
<td>6ug/10ug</td>
<td>0.40/0.19</td>
</tr>
<tr>
<td>Food</td>
<td>Ingestion</td>
<td>0.002-0.65ug/g (0.09ug/g average)</td>
<td>45ug/22.5ug</td>
<td>6.75ug/11.25ug</td>
<td>0.45/0.21</td>
</tr>
<tr>
<td>Soil</td>
<td>Ingestion</td>
<td>0.01-10000ug/g (30 ug/g average)</td>
<td>6ug/60ug</td>
<td>0.9ug/30ug</td>
<td>0.05/0.57</td>
</tr>
</tbody>
</table>

Assumptions: 500ml tidal volume, 15 breaths/min., 500g of food/day, 2 L of water/day, 0.2g of soil/day (adult)
250ml tidal volume, 20 breaths/min., 250g of food/day, 1 L of water/day, 2.0g of soil/day (child)
DECISION MAP FOR PUBLIC HEALTH OFFICIALS FOR MITIGATION OF LEAD POISONING IN A COMMUNITY

DECISION 1: Is it feasible to universally screen all children 10 years and younger?

Possibility A: Yes. Then proceed to screen all children.

Possibility B: No. Then target resources on those children most at risk e.g. ages 6 to 36 months who live in dilapidated buildings.

Rationale for Decision 1: Ideally, everyone should be tested for lead poisoning. Adults are less susceptible than children but may be exposed to high lead levels in occupational settings. But the physiology and behavior patterns of young children results in lead being a particularly potent health threat. Yet, logistically and/or financially, it may be unfeasible to test all children. Thus given current levels of knowledge, public health officials can identify those characteristics that put a child at an especially high risk of lead poisoning.

DECISION 2: Is there a significant number of children in the community with blood lead levels above 10 ug/dl?

Possibility A: Yes. Then proceed to activate a community-level intervention plan with continued surveillance, lead poisoning education and hazard abatement.

Possibility B: No. Then see if there are any cases of individuals with blood lead levels above 10 ug/dl. These individuals would get more personalized attention to see what factors lead to their anomalous high blood lead levels and what mitigative efforts are best utilized in their situations (see following decisions).

Rationale for Decision 2: The CDC endorses the 10 ug/dl blood lead concentration is the point at which action must be undertaken due to the consensus in the scientific community that this is the LOAEL (Lowest Observed Adverse Effect Level). Thus this level is a good point at which to initiate public health programs.

DECISION 3: Is there any cases of individuals with a blood lead level of 15 ug/dl?

Possibility A: Yes. Then proceed do an individual case management type approach. At this level, education and nutritional changes may be sufficient to decrease the level of risk. If not, a more medically oriented approach in conjunction with a pediatrician may be warranted.

Possibility B: No. No further action is required until next routine follow-up.

Rationale for Decision 3: At this blood lead level, behavioral and slight physical symptoms may become more readily apparent. 15 ug/dl may not cause an extreme danger to the child's health but yet it is still too high to ignore.

DECISION 4: Is there any cases of individuals with a blood level of 20 ug/dl or more?

Possibility A: Yes. Then proceed immediately with a medical evaluation and an individual environmental assessment. Most of the monitoring and care will be provided by the child's pediatrician. Household lead abatement could enter as an option depending on the circumstances. Chelation or succimer treatment may be recommended if the blood lead level is considerably higher than 20 ug/dl. Above 80 ug/dl, the child could be considered to be suffering from acute lead poisoning and is in immediate danger.

Possibility B: No. No further action is required until next routine follow-up.

Rationale for Decision 4: At these higher blood lead levels, the danger of neurotoxicity, nephrotoxicity, and other systemic effects becomes a real possibility. As a result, personalized medical attention is necessary.
VII. LEAD-BASED PAINT ABATEMENT TECHNOLOGY

Lead-based paint abatement technology and application is still in the early stages of development. Much of the current knowledge about abatement techniques is the result of the LPPPA charging HUD to identify efficient and cost-effective methods for abatement in public housing. This requirement by the LPPPA took the form of an Abatement Demonstration Program in both HUD-owned single family and multi-family properties and in public housing. From this demonstration, estimates for costs associated with testing and abatement can be developed for the nation. HUD was then required to present to the Congress a "Comprehensive and Workable Plan for the Abatement of Lead-Based Paint in Privately Owned Housing".

The National Institute of Building Science (NIBS) identifies two broad lead-based paint abatement strategies - paint replacement/removal, and enclosure/encapsulation.

Replacement involves the removal of components such as windows, doors and trim with lead-based painted surfaces and installing new lead-free components. Removal of lead-based paint from a surface can be performed on-site or by transportation and stripping of it off-site. On-site paint stripping if performed by abrasive methods like sanding
requires the use of a high-efficiency particle accumulator (HEPA) filtered vacuum to capture the dust generated during the process. Removal can also be performed by chemicals or a heat gun. Regardless of the removal method, HEPA vacuums and a high phosphate wash should be used to meet dust lead concentration standards.

Encapsulating or enclosing involves somehow covering the lead-based paint surface rather than removing it. As the lead-based paint surface is not broken, less dust is created than in removal abatement methods. As a result, lower dust means less stringent worker protection, lower hazardous waste disposal and lower clean-up costs. Unfortunately in encapsulation, the lead-based paint hazard is postponed rather than removed. The durability and maintenance needed for the encapsulating method affects its suitability as an abatement method. Encapsulation involves sealing with a material that bonds to the surface such as acrylic or epoxy coating whereas enclosure uses a gypsum wall, plywood etc, to cover the lead-based paint surface. Encapsulation is also significantly cheaper than enclosure and all methods of paint removal as well.

The five abatement methodologies that were used in the FHA demonstration encapsulation, enclosure, chemical removal, hand-scraping with a heat gun and replacement.

Each of the units in the FHA demonstration were assigned one of these possible methodologies. In performing
the strategies, the NIBS published guidelines for worker and environmental protection. These guidelines included the use of polyethylene sheeting for dust containment, the use of protective clothing and respirators and disposal of hazardous waste in accordance with federal state and local regulations.

Another methodology that does not replace the previously mentioned lead-based paint abatement methods, seeks to manage the potential hazard by in-place management of the amount of lead dust present in the household. In-place management is oriented largely toward the maintenance of painted surfaces, clean-up of lead dust, and controlling further accumulation of lead dust. Due to the potentially high cost of lead-based paint abatement in certain households, lead dust control may provide an interim or even alternate strategy. The in-place management of lead is based on the assumption that lead dust provides the primary pathway of childhood exposure especially in cases of low-level exposure.

The demonstration by HUD was conducted in 7 metropolitan areas: Baltimore, Birmingham, Denver, Indianapolis, Seattle, Tacoma, and Washington D.C. About 300 units were tested initially using the XRF analyzers and consequently 173 properties were selected for lead-based paint abatement strategies. All of these units were vacant.
single-family housing units owned by the Federal Housing Administration (FHA).

The purpose of the FHA demonstration was in part to arrive at an estimate of the cost of lead-based paint abatement at the dwelling unit level. Though both lead-based paint abatement methodologies — encapsulation and enclosure make lead hazards less accessible, encapsulation has proven to be the less expensive strategy. Out of the replacement/removal strategy, hand scraping via heat gun is the most cost-effective for interior surfaces, chemical stripping is the most cost-effective for large exterior surfaces and replacement of components is the least expensive for door and window trim and baseboards. Abrasive methods of removal were generally not cost-effective due to the large amounts of paint dust created and the resulting greater worker protective measures and clean-up required.

Analyzing for lead content in a housing unit averaged $404 and $320 for single and multi-family dwellings respectively. The average cost of abatement per dwelling with lead-based paint using the encapsulation method was $5,453 and for the replacement/removal strategy — $7,704 (1989 dollars). Thus for units with lead-based paint, encapsulation is approximately 30% less expensive than removal. Estimates for dust abatement protocol including sampling, testing analysis, clean-up and follow-up vary.
greatly from a low end estimate of $3,380 to a high end estimate of $7,032 (1989 dollars). Note these figures are mean values as the median values are much lower than the mean. In other words, a small percentage of the homes require a very costly abatement strategy. For example, using the encapsulation strategy, 18.3% of households with lead-based paint would have a cost of abatement of over $10,000 and 4.7% over $25,000. Also the figure given for the average cost of encapsulation as the lead-based paint method for a household with lead-based paint present must also factor the useful life for the encapsulants used as they do not last indefinitely.

In a real-world situation, there are factors that were not considered in the FHA demonstration cost figures for lead-based paint abatement strategies. Most importantly, interior dust lead in these units was abated but not exterior soil and exterior surface dust lead, or dust lead in forced-air ducts (research indicates that exterior soil and lead may not only pose a direct hazard but that it may be transported indoors). The FHA demonstration was conducted on vacant households which eliminated the cost of relocating the household occupants and protecting household property for dust contamination. Also the cost of disposing of hazardous waste materials during lead abatement was not added to the final cost. But other factors may have resulted in higher costs during the demonstration. For
example, normally, lead-based paint abatement would often be performed in conjunction with rehabilitation/renovation work thus spreading the fixed costs and possibly integrating the abatement and rehabilitation tasks. Instead of relying on one strategy per housing unit, mixing strategies may result in greater cost-effectiveness. Also worker protection measures may be safely reduced in the future with the low dust creating strategies such as encapsulation and chemical removal. Finally, in all likelihood, economy of scale would eventually result in lower cost as abatement is performed on more and more housing units in the future and as contractors further streamline the abatement process.

In determining the national cost of lead-based paint abatement a rough cost estimate may be derived from combining data from the HUD sponsored national survey of lead-based paint in housing in the US and the FHA demonstration. A model has been derived which attempts to incorporate changes in housing stock and the population over a 10 year period of the hypothetical national lead-based paint abatement efforts. According to the chart, an estimated total annual cost of $36.3 billion dollars for removing all lead by enclosure/encapsulation and $49.9 billion by replacement/removal. These figures can be reduced depending on the definition chosen for what constitutes a lead paint hazard i.e. should we only attempt to abate lead paint in homes with small children? To put
these numbers in perspective, the estimated annual cost for abatement of all lead-based paint would equal about 3 times the total cost the total annual private expenditure on repainting and between one-half and one-third of the current total private expenditure on rehabilitation and remodelling for private housing43.

It is important to note that these cost are rough estimates and can vary depending on the comprehensiveness and extent that lead-based paint is removed or mitigated in the housing stock. For instance, if the government wishes to regulate at an abatement threshold of 1.5 mg/centimeters squared the estimated total cost may decrease as much as 60%44.

It is difficult to determine the efficacy of a dust abatement procedure in terms of a human health. Available data is not yet adequate to permit a confident estimate of the amount of reduction in childhood blood lead levels that occurs by the various abatement methods45. Thus comparing cost-effectiveness of the abatement methods is difficult. The quantity of lead dust per a unit area or per a unit volume of a particular household can be measured before and after abatement, as can the blood lead levels of the occupants. But a reduction in lead exposure does not necessarily correlate directly with blood lead concentrations. It is difficult to quantify the exact contribution of a lead-based paint source of lead exposure
among the multiplicity in the environment. Much depends on a given situation. In some cases, vehicular and industrial emissions might be the major source of lead exposure or in others it may be the lead content in the drinking water or the lead-based paint. To further complicate the situation a multiplicity of sources may share dust as an intermediary - the lead dust in the home environment may be from deteriorating painted surfaces (interior and/or exterior), vehicular and industrial emissions or from transportation from a workplace on the clothes and shoes of a family member. Even if the lead-based paint is the major source of lead dust in the environmental compartment of concern, the effect on blood lead levels of abatement procedures may not be apparent due to lead moving from skeletal stores into the circulatory system.

There are a few reported studies on the effects of lead-based paint abatement in the ability to reduce blood lead levels. In total, they give qualified support to the conclusion that removal or covering of lead-based paint in the childhood environment reduces the risk of lead poisoning. The qualifications arise on the efficacy of the abatement because the techniques used in the studies are not as comprehensive as those used in the FHA Lead-Based Paint Abatement Demonstration and potentially create more lead dust in the interim. In traditional deleading, interior nonintact and chewable surfaces within four feet from the
floor. Open flame heating, scraping and sanding were often used on wood trim. Stripped surfaces were often left unpainted. Exterior surfaces were not abated. Cleanup after abatement was minimal.

Dr. E. Charney and his colleagues found that traditional deleading did not reduce mean blood lead levels. Only when abatement was followed by a thorough clean-up and wet mopping twice a month did mean blood lead levels drop. A mean decrease of 6.9 ug/dl within a year was obtained, from 38.6 ug/dl to 31.7 ug/dl. Dr. M. Farfel compared the results of traditional deleading procedure with a more comprehensive modified abatement protocol. Neither method was successful in reducing blood lead levels. Farfel attributed these findings to the high dust level during and after abatement. His suggestions to improve abatement involved a number of procedures which he believed would reduce the high dust levels. These suggestions for a lead-based paint abatement protocol that focused on dust control as well as paint removal became the basis for the HUD Interim Guidelines which were eventually implemented into the HUD sponsored FHA Lead-Based Paint Abatement Demonstrations.

In Massachusetts, Dr. Y. Amatai and colleagues found a 29 percent decrease in blood lead levels 8 months after abatement. However, they did notice a significant, transient blood lead elevation during and after deleading.
This elevation was most obvious when torches, sanding, and dry scraping were used. Their research contributed to Massachusetts statutes banning the use of dry abrasive blasting, on-site use of methylene chloride and the use of propane torches.

Cost-effectiveness, as stated earlier, is difficult to measure in terms of dollar per a unit of blood lead reduction. Though currently effectiveness can not be measured in human health terms, what can be measured is the efficacy of a particular abatement methodology in achieving the NIBS Guidelines - 200 ug/ft² for floors, 500 ug/ft² for window sills and 800 ug/ft² for window wells. These were the standards used in the FHA demonstration. The testing was performed in all abated areas by surface wipe sampling with commercial wipes moistened with a non-alcoholic wetting agent. The wipe tests allow us to compare the five main abatement procedures used in the demonstration in their ability to meet the NIBS clearance standards (see diagram vi). In order of decreasing ability to meet the NIBS clearance standards, the abatement methods are - replacement, encapsulation, enclosure, chemical and finally hand-scraping with heat gun.

Encapsulation and enclosure are considered the components of the enclosure/encapsulation strategy and replacement, chemical removal, and hand-scraping with a heat gun are considered part of the replacement/removal strategy.
## DISTRIBUTION OF WIPE SAMPLE VALUES ON FLOORS BY CLEARANCE STANDARD (PASS/FAIL) ON INITIAL WIPE TEST BY UNIT ABATEMENT STRATEGY

### Unit Abatement Strategy

<table>
<thead>
<tr>
<th>Wipe Sample Value (ug/l)</th>
<th>Encapsulation</th>
<th>Enclosure</th>
<th>Chemical</th>
<th>Hand-Scrape w/1reat Gun</th>
<th>Replacement</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 200</td>
<td>188 (86.2%)</td>
<td>96 (30.0%)</td>
<td>276 (77.3%)</td>
<td>163 (71.2%)</td>
<td>203 (87.5%)</td>
</tr>
<tr>
<td>≥ 200</td>
<td>30 (13.8%)</td>
<td>24 (20.0%)</td>
<td>81 (22.7%)</td>
<td>66 (28.8%)</td>
<td>29 (12.5%)</td>
</tr>
<tr>
<td>ALL</td>
<td>218 (100.0%)</td>
<td>120 (100.0%)</td>
<td>35 (100.0%)</td>
<td>229 (100.0%)</td>
<td>232 (100.0%)</td>
</tr>
</tbody>
</table>

Chi-square: 25.1 Degrees of Freedom: 4 Significance 0.0000

## DISTRIBUTION OF WIPE SAMPLE VALUES ON WINDOW SILLS BY CLEARANCE STANDARD (PASS/FAIL) ON INITIAL WIPE TEST BY UNIT ABATEMENT STRATEGY

<table>
<thead>
<tr>
<th>Wipe Sample Value (ug/l)</th>
<th>Encapsulation</th>
<th>Enclosure</th>
<th>Chemical</th>
<th>Hand-Scrape w/1reat Gun</th>
<th>Replacement</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 500</td>
<td>157 (95.2%)</td>
<td>78 (91.8%)</td>
<td>173 (75.9%)</td>
<td>124 (75.6%)</td>
<td>137 (92.6%)</td>
</tr>
<tr>
<td>≥ 500</td>
<td>8 (4.8%)</td>
<td>7 (8.2%)</td>
<td>55 (24.1%)</td>
<td>40 (24.4%)</td>
<td>11 (7.4%)</td>
</tr>
<tr>
<td>ALL</td>
<td>165 (100.0%)</td>
<td>85 (100.0%)</td>
<td>228 (100.0%)</td>
<td>164 (100.0%)</td>
<td>148 (100.0%)</td>
</tr>
</tbody>
</table>

Chi-square: 39.5 Degrees of Freedom: 4 Significance 0.0000

## DISTRIBUTION OF WIPE SAMPLE VALUES ON WINDOW WELLS BY CLEARANCE STANDARD (PASS/FAIL) ON INITIAL WIPE TEST BY UNIT ABATEMENT STRATEGY

### Unit Abatement Strategy

<table>
<thead>
<tr>
<th>Wipe Sample Value (ug/l)</th>
<th>Encapsulation</th>
<th>Enclosure</th>
<th>Chemical</th>
<th>Hand-Scrape w/1reat Gun</th>
<th>Replacement</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 800</td>
<td>75 (74.3%)</td>
<td>45 (76.3%)</td>
<td>95 (34.3%)</td>
<td>61 (55.5%)</td>
<td>79 (79.0%)</td>
</tr>
<tr>
<td>≥ 800</td>
<td>26 (25.7%)</td>
<td>14 (23.7%)</td>
<td>80 (45.7%)</td>
<td>49 (42.5%)</td>
<td>21 (21.0%)</td>
</tr>
<tr>
<td>ALL</td>
<td>101 (100.0%)</td>
<td>59 (100.0%)</td>
<td>175 (100.0%)</td>
<td>110 (109.0%)</td>
<td>100 (100.0%)</td>
</tr>
</tbody>
</table>

Chi-square: 19.3 Degrees of Freedom: 4 Significance 0.0007
Comparing costs is difficult as different methodologies work best for a particular housing unit or fixture. But if one confines a mixture of methodologies to one of the two broad groups - as stated earlier enclosure/encapsulation strategy are on average considerably cheaper at $5,453 per unit compared with $7,703 per unit for replacement/removal (1989 dollars).

The best lead-based paint abatement for the theoretical average housing unit with lead-based paint in terms of cost-effectiveness (e.g. attaining the NIBS clearance standards) is the enclosure/encapsulation strategies (specifically the encapsulation method over enclosure method). To further bolster the strategy, the replacement methodology could be used on interior doors and windows where it is cheaper and more effective respectively. The hand-scraping and chemical removal have a high failure rate in meeting NIBS clearance standards and can be disregarded for most abatement purposes.
VIII. A MITIGATION STRATEGY

Much of the health threat lead poses in the environment is targeted towards young children. Their behavior and physiology significantly increases their intake, uptake, and adverse symptoms of lead exposure over most other segments of the population. As a consequence, most efforts at mitigation have been directed at decreasing lead exposure by children in the home environment.

With about 57 million homes in the US with lead-based paint and roughly 3 to 4 million children under the age of 6 with a blood lead level of 15 ug/dl or more - past and present mitigation strategies have not been fully effective in eliminating the lead-based paint hazard.

Much of the inaction may be due to ignorance within the appropriate regulatory agencies and other groups that could bring pressure to bear. Though the risks of occupational exposure and acute lead poisoning are well documented and known, the adverse health effects of low-level lead exposure found with lead-based paint are not.

Generally, the state and local governments have primary responsibility for regulating housing conditions in the US. In response to the enactment of the LPPPA in 1971, most programs concentrated on identifying and responding to
children with elevated blood levels. Few states (with Maryland and Massachusetts being notable exceptions) had the type of comprehensive plan where prevention was the focus. Lead poisoning in children was considered primarily a health problem, so there had been little input from housing authorities. Blood screening due to budget restraints has been limited to high-risk groups with medical intervention falling upon the child’s pediatrician. And if an individual was found to have unusually high blood lead levels, effective tracking of the lead-poisoning case was often not performed. In the case of a mitigation of lead exposure in the home of a child identified as suffering from lead poisoning, abatement options can be limited due to a lack of funds and qualified contractors.

Drs. Bellinger and Needleman, two pioneer researchers on the effects of low-level lead exposure on the neurological functioning of children have their own theories on the slow removal of lead in the environment. They believe foremost that lead-poisoning is perceived as a disease that is limited to poor minorities and is thus somehow the result of poor mothering and hygiene rather than environmental factors. In other words, a ‘blame-the-victim’ mentality predominates. This belief contradicts research which demonstrates that lead-based paint poisoning cuts across socio-economic lines and that there are significant numbers of affected children at all strata of society.
(though poor, inner-city minorities are affected in greater proportion). The erroneous belief persists that recent restrictions on lead content in gasoline and paint have solved the lead-poisoning problem. True, a large reduction in the amount of lead entering the environment has been realized through these measures, much lead still remains — i.e. lead-based paint in millions of homes. Finally, Bellinger and Needleman feel that the medical community considers lead-poisoning a low technology disease and as such does not capture their interest. As a result, the myriad of effects of low-level lead-poisoning are often overlooked in diagnosing pediatric ailments.

If lead-poisoning is to be eliminated from society, a more targeted approach must be developed. The research on toxicokinetics, health effects, socio-epidemiology, and lead-based paint testing/abatement technology can be translated into more effective policy.

Strategy 1: Integration of lead-based paint dust related toxicokinetics into current legislation, and regulations. Researchers in the last decade have determined repeatedly the significance of lead-based dust in the lead poisoning of children in the home environment. Yet, traditional abatement prescribed in regulations does not deal adequately with the contribution of dust levels in maintaining a lead-free environment. The research from the FHA abatement
demonstrations should be used to devise an abatement protocol which involves a careful clean-up as well as repair to defective paint surfaces. In the interim, until a better standard is developed, the NIBS clearance standards for abated dwelling units for floors, window wells, and window sills could be used to measure the efficacy in reducing household dust levels. Eventually with HUD sponsorship, these clearance standards could be mandatory for abatement efforts for all housing units.

Strategy 2: Development of a profile of those individuals in the population most at risk for adverse health effects due to household lead exposure and targeting the majority of limited resources at them.

The Agency for Toxic Substances and Disease Registry has found that childhood blood lead levels were associated with race, family income, residence inside or outside of a metropolitan central city, and the size of the metropolitan area. Thus the most affected group (black, lowest family income group, living in central cities of metropolitan areas of 1 million or more) may be 14 times more likely to be have elevated blood lead than the least affected group (white, highest family income group, living outside central cities in metropolitan areas of less than 1 million). But most importantly, although the percentage of children with elevated blood lead levels may be relatively low for some
population groups, the number of affected children is substantial in all groups\(^3\). Thus certain socio-demographic groups may need greater surveillance and medical intervention but no group can be completely neglected.

The answer lies in increasing the amount of medical and public health intervention proportionately with the levels of blood lead found in a given population. The Advisory Committee on Childhood Lead Poisoning Prevention of the CDC endorses the 10 ug/dl blood lead concentration as the point at which action must be undertaken because there is a consensus that at that level, adverse effects are detectable. Mass screening ideally would occur for children in all communities, with those with elevated blood lead levels resulting in further investigation. But realizing the present impossibility of universal screening, the highest risk groups (defined as children 6 to 36 months of age who live in dilapidated older buildings) would be targeted for the most thorough screening. A multi-tiered approach would then be established in lead poisoning intervention. If a community has a large proportion of children with 10 ug/dl, a community level-intervention should be activated with a surveillance, education and hazard abatement. At 15 ug/dl, individual case management should start with the emphasis on education and improved nutrition, and at 20 ug/dl, a medical evaluation and an environmental assessment should be performed for the
affected individuals. Thus, there is shift in focus from public health to medical solutions or from community to the individual as higher blood lead levels are found.

As lead poisoning affects a broad demographic cross-section of the US population, the seriousness of lead exposure and information on lead poisoning prevention should be made widely known to the public as well as the real estate industry and the medical community. This dissemination of information can happen at many levels via brochures, primary care physicians, the news media, public service announcements and information hotlines.

Strategy 3: Further research on toxicokinetics, health effects and treatment for lead poisoning.
As stated earlier, there is an urgent need to be able to quantify lead intake and uptake. And to determine if a what blood lead concentration can be attributed to a particular exposure level. Equally important is the ability to discern individual variance in response to a given exposure level. Eventually, a safe exposure level may be able to be determined for a particular individual, thus giving lead-based abatement efforts a precise goal to achieve.

Perhaps research can develop the toxicokinetics of lead poisoning to an individual level, so that the particular balance of interior/exterior lead-based paint dust, pica, soil lead, atmospheric fallout, nutrition and mouthing
behavior may be more easily assessed. Mitigation could thus result in modification of other factors other than uniformly performing lead-based paint abatement in all households with lead poisoned children.

In the fight against lead poisoning, much promise lies within medical science. Though blood measurements are currently used in determining lead poisoning, the test has its disadvantages due to the potential for blood lead levels to either reflect indiscriminately more recent lead exposure or bone lead leaching back into the bloodstream. L-line x-ray fluorescence, a new radiological technique, quantitatively measures the total accumulation of lead over an individual’s lifetime. It is a quick and non-invasive method that aims partially polarized photons at the cortical bone of the tibia and a lead peak in the spectrum is measured. The only drawback is that it requires sophisticated equipment. Chelation with CaNa2EDTA is standard therapy for higher blood lead levels. Unfortunately, chelation therapy has its detractors whom point to the method’s non-specificity, potential renal toxicity and its tendency to redistribute lead from bone to other tissues including the brain. Recently, succimer has been approved by the FDA as an oral medication for treatment of lead poisoning and has shown to be more effective than CaNa2EDTA and easier to use as it is not administered intravenously.
Strategy 4: Further research and development for the testing and abatement of lead-based paint.
Research shows that testing for lead-based paint in homes is cost-effective but only if done with reliable equipment and trained personnel. Thus the improvement of portable XRF analyzers and accredited XRF inspector training should be implemented by the federal government. To further ensure accuracy, laboratory evaluation should verify onsite measurements using federally approved standards and protocol.

Although there has been significant progress lead-based paint abatement technology, there are still many aspects that need to be addressed - the abatement of exterior soil lead and interior lead dust in carpets, furniture, and forced-air ducts, standards and procedures for worker protection, proper handling of waste produced from abatement activities, the long-term efficacy of the various abatement strategies and the development of other less costly or interim abatement methods (i.e. in-place management of lead dust)\textsuperscript{96}.

Ultimately, the most important research to be performed is on the health effects of abatement. Though research indicates that abatement of lead-based paint if performed properly will reduce childhood lead poisoning, there is inadequate data to predict the amount of reduction or if the reduction is long-term by any of the various abatement
methods. As stated in Strategy 3, alternatives to abatement may be more closely examined in individual homes if other factors may be more easily controlled or more cost-effective in reducing blood lead levels.

Strategy 5: Greater funding and financial assistance at federal, state, community and individual level for lead poisoning programs, surveillance, lead-based paint abatement and other mitigation efforts.

The federal government needs to increase its support for state and local screening programs to ensure that a larger proportion of the children in the US are checked for lead poisoning. The federal government must also increase its grant, loan and insurance programs for house rehabilitation to ensure monies are available for the anticipated increase volume of abatement that will be performed with an increase public awareness of the hazards of lead exposure.

Since state and localities have primary responsibility for housing concerns in the US, it is important that the federal government sponsor their efforts to develop local capacity (i.e. lead-based paint abatement contractors) in abatement efforts and to develop their own technical expertise. The federal government would also sponsor the development of accredited training curricula for courses in inspection, abatement supervision, abatement project design
and construction to minimize potentially inadequate and hazardous abatement efforts.

In the last decade, there has been a dramatic decrease in the lead intake of most members of the population. The decrease in use of leaded gas and the absence of lead in the canning process are the primary reasons for this reduction. For the non-occupationally exposed, ingestion is becoming the route of lead exposure of the greatest concern. Exposure via ingestion, based on baseline lead exposure data, contributes 15 to 25 times more lead to the daily intake than inhalation. Exposure by inhalation can make significant contributions in certain situations e.g. if atmospheric lead concentrations are unusually high due to a local smelter. Exposure to lead via dermal exposure, occurring mainly in occupational settings, can be considered less significant from a public health stance. For adults, lead inhaled is absorbed about twice as efficiently as it is absorbed through ingestion. With children, lead inhaled is absorbed about 1.5 times more efficiently than ingested.

In terms of mitigation of excessive lead exposure, the route of ingestion is of particular importance as it by ingestion that lead-containing dust (whether from lead-based paint or industrial sources) enters the body. As socio-epidemiological studies have shown the ubiquity of lead-based paint in the various strata of the population, most
efforts to decrease lead exposure in the sensitive child population has focused on lead-based paint abatement. In individual cases, there may be other more potent sources of lead exposure but not from a general public health viewpoint.

Assuming the above mitigation strategies are implemented and that lead-based paint abatement is performed to the current limits of technology, a reduction in daily lead intake from dust would be expected. And since lead-containing dust is the largest single source of lead exposure for most children, a large concomitant decrease in daily lead intake could be expected. If lead intake via ingestion of lead-based paint dust was brought down to adult exposure levels, a reduction of approximately 80% in daily lead intake could be realized for the hypothetical lead-based exposed 2 year-old child (a reduction in daily lead intake from 136.5 ug/day to 43.5 ug/day). Unfortunately, there is not enough data to accurately predict what this new daily lead intake would represent in terms of a blood lead concentration.

The increasing body of knowledge on lead poisoning presents a strong argument for a comprehensive mitigation strategy under the aegis of the various levels of government. Yet, there still lies a host of factors which could impede such an approach. First and foremost, there is the problem of budgetary constraints. Even the most modest
program of lead abatement in only the most affected households runs into billions of dollars. Similarly universal testing and lead testing might also be prohibitively expensive. Secondly, the nature of lead abatement involves the need for a very meticulous methodology. Lead dust levels must be closely monitored after abatement and children with high blood lead levels must be tracked for further fluctuations. This meticulous methodology also requires trained personnel in lead abatement techniques and research. At present, this capacity has not been developed to the level necessary for a large scale effort in reducing lead exposure. Thirdly, the effects of low lead level exposure on children is not necessarily obvious. Thus, the difficulties in convincing policy-makers that lead based paint abatement is cost-beneficial could be difficult. Some might argue that the money would be better spent in areas with a seemingly greater (and thus perhaps more politically expedient) return?

To counter arguments on the high estimated costs of a comprehensive lead abatement plan, the relative not the absolute cost compared to other household maintenance activities must be examined. There is also sufficient epidemiological information so that limited resources can be targeted for those individuals suffering the most from household lead exposure. The smallest annual abatement cost
estimates, those for families with young children and non-intact lead-based paint, are about two-thirds of the current total expenditure on repainting. And lead-based paint abatement, depending on the method, would have to be done much less frequently than repainting and in some cases, it might have to be performed only once. Also, interim in-place lead abatement methods may be sufficient to lessen the health threats in certain households without incurring the costs of a full scale household lead abatement. The dissemination of information on the potential hazards of lead-based paint in the home might also be an excellent preventive measure for relatively little cost. If the cost-benefit of a large scale household lead abatement plan must be assessed then a number of indicators of success may be used. Attentional, behavioral, intelligence tests could be used to demonstrate before and after progress. Less abstractly, blood lead measures should immediately reflect the efficacy of a lead based paint abatement program. A lowered blood lead level could be shown to reduce the probability of any of a number of health or psychological end-points in the chosen sub-population. Epidemiological studies would ideally be able to compare incidences of various adverse effects before and after abatement and thereby demonstrate the utility of such a program.

2. Ibid.


6. ATSDR, Toxicological Profile for Lead, p. 3.


8. HUD, Comprehensive and Workable Plan, p. 2-11.


10. ATSDR, Toxicological Profile for Lead, p. 168.

11. ATSDR, Toxicological Profile for Lead, p. 164.


15. ATSDR, Toxicological Profile for Lead, p. 170.
16. EPA, Air Quality, I-47.
17. ATSDR, Toxicological Profile for Lead, p. 171.
26. HUD, Comprehensive and Workable Plan, p. 3-5.
27. HUD, Comprehensive and Workable Plan, p. 3-1.
29. HUD, Comprehensive and Workable Plan, p. 3-17.
30. HUD, *Comprehensive and Workable Plan*, p. 3-25.
31. HUD, *Comprehensive and Workable Plan*, p. 3-25.
37. 
42. ATSDR, *Toxicological Profile of Lead*, p. 123.
44. ATSDR, *Toxicological Profile for Lead*, p. 126.
49. ATSDR, *Toxicological Profile for Lead*, p. 96.
Journal of Laboratory and Clinical Medicine, 90(2): pp. 239-248.


52. Mahaffey, Dietary and Environmental Lead, p. 423.


54. EPA, Air Quality, p. I-70.


60. EPA, Air Quality, p. I-115.


64. ATSDR, Toxicological Profile for Lead, p. 39.
66. ATSDR, Toxicological Profile of Lead, p. 31,32
67. EPA, Air Quality, p. I-123.
69. ATSDR, Toxicological Profile for Lead, p. 53.
70. ATSDR, Toxicological Profile for Lead, p. 54.
71. ATSDR, Toxicological Profile for Lead, p. 56.
72. ATSDR, Toxicological Profile for Lead, p. 25.
73. Mahaffey, Dietary and Environmental Lead, p. 423.
75. ATSDR, Toxicological Profile for Lead, p. 42.
77. HUD, Comprehensive and Workable Plan, p. 4-16.
78. HUD, Comprehensive and Workable Plan, p. 4-10.
79. HUD, Comprehensive and Workable Plan, p. 4-4.
80. HUD, Comprehensive and Workable Plan, p. 4-11.
81. HUD, Comprehensive and Workable Plan, p. 4-18.
82. HUD, Comprehensive and Workable Plan, p. 4-12.
83. HUD, Comprehensive and Workable Plan, p. 4-22.
85. HUD, Comprehensive and Workable Plan, p. 6-10.
86. HUD, Comprehensive and Workable Plan, p. 2-21.
90. HUD, Comprehensive and Workable Plan, p. 2-24.
91. HUD, Comprehensive and Workable Plan, p. 5-2,3.
93. HUD, Comprehensive and Workable Plan, p. 2-7.
96. HUD, Comprehensive and Workable Plan, p. 6-8.