EPA's POSITION ON THE HEALTH EFFECTS OF AIRBORNE LEAD

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I. INTRODUCTION

On February 23, 1972, the Environmental Protection Agency published fuel additive regulations which would result in the reduction of lead in gasoline by 60-65% beginning January 1, 1977.[1] The original health effects papers supporting this decision have been previously described.[2,3,4,5]

Following this announcement the Agency solicited public comment on the proposed regulation. A 90 day comment period was initiated and public hearings on this question were held in Washington, D.C. (April 11-12, 1972), Dallas, Texas (April 27-28, 1972), and Los Angeles, California (May 2-4, 1972). Additional comments were solicited in the form of questions which appeared in the Federal Register.[6]

Many opinions were expressed both by testimony at the hearings and by written submission immediately following the hearings and during this subsequent extended comment period. All comments received were read and evaluated. The entire hearing record and submitted comments are available for public inspection at the Environmental Protection Agency in Washington, D.C.

The purpose of this paper is to update the Agency's health position related to control of lead emissions from motor vehicle exhaust based upon the most recent information available to EPA, including the Public Hearing testimony, written comments which were received, and reevaluation of existing data. Since this document focuses primarily upon the possible direct and/or indirect effects of airborne lead and lead in gasoline upon man, it is recognized not to represent a balanced

comprehensive review of all that is known about the biophysiology of lead in relation to man. Further, although this paper is meant to be read primarily by members of the scientific community we have also endeavored to make it understandable to the lay public. This document will be considered by EPA in evaluating the health issues that pertain to its proposed fuel additive regulations.[7]

REFERENCES FOR SECTION I -INTRODUCTION

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[2]"Health Hazards of Lead," EPA, Research Triangle Park, N.C., February 23, 1972.

[3]"Health Hazards of Lead (Revised April 11, 1972)," EPA, Research Triangle Park, N.C., April 11, 1972.

[4]"Atmospheric Lead and Public Health," EPA, Research Triangle Park, N.C., April 11, 1972.

[5]"Corrections and Additions to Health Hazards of Lead (Revised April 11, 1972)," EPA, Research Triangle Park, N.C., April 27, 1972.

[6]Federal Register, Vol. 37, No. 115, pp. 11786-11787, June 14, 1972.

[7]Federal Register, Vol. 37, No. 36, pp. 3882-3884, February 23, 1972.

II. CLINICAL MANIFESTATIONS OF LEAD POISONING

Lead is a known toxic substance for which no beneficial biological role has yet been demonstrated. Effects of severe lead intoxication at high exposures have been recognized for centuries. These include death and irreversible neurological impairment.[1,2]

Symptoms of mild lead intoxication include loss of appetite, irritability, drowsiness, apathy, and abdominal pain. Since these symptoms are commonly found in many other diseases, the possible role that lead may have played in their origin is sometimes difficult to evaluate. These symptoms by themselves cannot be used to imply an effect caused by lead.

In view of the uncertainty in defining the presence of mild lead poisoning symptoms, some clinicians and health departments consider children with abnormally elevated blood leads as "asymptomatic" lead poisoning cases even if no symptoms or signs of lead intoxication are evident. Since subtle indications of lead poisoning are difficult to detect, perceptiveness of both parents and physicians is an important factor influencing whether symptomatic lead poisoning cases are identified. Hence, the distinction between excessive lead absorption and mild lead intoxication is sometimes unclear. For example, children considered initially to have no symptoms of lead poisoning have been found, on follow-up medical examinations, to be mentally retarded. In such instances, however, mental retardation may have been present before lead poisoning occurred.

In one large survey involving 425 children with lead poisoning, attributed primarily to ingestion of lead based paint, a large percentage (39%) showed evidence of nervous system damage during follow-up examinations.[3] Mental retardation and recurrent seizures were the most common and persistent findings. In this same study, of 232 children with symptoms of lead poisoning characterized initially by gastrointestinal complaints, but not by evidence of neurologic damage, 19% were later found to be mentally retarded and 13% to have convulsive disorders. Whether convulsions were observed only in children with mental retardation is uncertain from the article. Of 58 children treated for asymptomatic lead poisoning, five (approximately 10%) were found during follow-up studies to be mentally retarded. Again, one cannot rule out the possibility that mental retardation was present before these children were poisoned by lead.

In another study[4] eleven children who had been treated for lead poisoning attributed primarily to paint were reexamined 5-10 years later. Mental deterioration was not always obvious and physical and laboratory tests in general did not reveal abnormalities. However, specialized tests of visual motor performance indicated subtle brain damage in the majority of cases.

Several parents of children with blood leads of 50ug/100g and above have reported improvements in their child's behavior and language ability following treatment with drugs that removed lead from their bodies, even though the children had originally been considered asymptomatic cases of excessive lead exposure.[5] Although these were subjective findings which

were not compared to those from a matched control group, they do suggest the possibility that central nervous system damage was present, but previously undetected in these children.

The possibility that blood lead elevations even at levels generally not considered excessive (40ug/100g and below) may be associated with childhood behavioral disturbances such as hyperactivity has recently been reported.[6] Hyperactive children were observed to have higher blood lead levels as well as increased post-penicillamine urinary lead excretion levels compared to a reasonably matched control group. Both the hyperactive and the control group were similar with respect to age and sex. However, possible socioeconomic and racial differences between the groups may have partially confounded these results. Although blood leads in the hyperactive group were predominantly below 40ug/100g, urinary lead excretions were abnormally elevated in over 60% of these children compared to 21% in the control group. Hence, exposure to lead in quantities presently not considered to be toxic or even excessive may contribute to minimal brain damage as in the hyperactive syndrome. Since a history of lead exposure (but apparently not lead poisoning) was more frequent among the hyperactive children, increased lead exposure early in childhood may, in certain of these children, have contributed to the eventual development of hyperactivity problems.

Findings such as these cause speculation, in the opinion of EPA, that children may be suffering subtle but unrecognized neurological impairments due to lead.

REFERENCES FOR SECTION II - CLINICAL MANIFESTATIONS OF LEAD POISONING

[1]McLaughlin, M. C.: "Lead Poisoning in Children in New York City, 1950-54: An Epidemiologic Study," NY State J Med 56:3711-3714, 1956.

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[6]Oliver,D.; Clark,J.; and Voeller, K.; "Lead and Hyperactivity," Lancet, pp. 900-903, Oct. 28, 1972.

III. LOW LEVEL METABOLIC EFFECTS OF LEAD

Lead is known to interfere with enzyme systems at blood lead levels lower than those generally associated with clinical symptoms of lead intoxication.[1] This is especially true for enzymes containing sulfhydryl groups which are particularly sensitive to lead.

Delta aminolevulinic acid dehydrase (ALAD), an enzyme involved in hemoglobin synthesis, is the best documented example of lead enzyme inhibition in man.[2] Measurable increases in urinary ALA resulting from ALAD inhibition are generally not found until blood lead levels have reached 40ug/100g. A panel of the National Academy of Sciences concluded that, at blood lead levels of 40ug/100g and above, inhibition of this enzyme is physiologically significant.[3]

The true significance of ALAD inhibition in man is at present unclear. Inhibition of ALAD by lead in peripheral blood by itself may not be clinically important, especially since these reports are based upon in vitro biochemical determinations which may not accurately reflect what is happening in man. However, lead induced inhibition of ALAD required for cytochrome synthesis in other tissues may reflect a more significant impairment.

For example this inhibition has been demonstrated to occur in brain, kidney, liver and spleen of suckling rats lead poisoned by maternal milk.[4] ALAD activity in the blood of the suckling rats was observed to correlate with ALAD activity in the brain. These results suggest that ALAD inhibition in peripheral blood of children, which occurs

at blood lead levels in the 20-40ug/100g range, may be associated with similar biochemical abnormalities in the brain. Whether this actually occurs in man, however, is at present unknown.

Inhibtion of enzymes involved in cellular energy production may partially explain the mechanism by which lead exerts its toxic effects. The central nervous system is especially sensitive to oxygen deprivation, and thus could conceivably be extremely sensitive to possible enzyme inhibition by lead. In this context even slight but sustained elevations of blood leads may cause subtle, though appreciable, impairment of central nervous system functions.

Lead has also been recently associated with the possible development of chromosomal abnormalities in man. Muro and Goyer first reported evidence of experimental chromosomal damage caused by lead in 1969.[5] In this study chromosomes derived from leukocyte cultures of mice fed 1% lead acetate in their diets demonstrated increased gap-break aberrations. The authors of the study concluded that similar aberrations in somatic cells would result in impaired growth. Should these disturbances be shown to occur in germ cells they would be of potential genetic significance. Since this finding, chromosomal abnormalities have been discovered in the lymphocytes of lead poisoned men[6] and in the lymphocytes of workers currently occupationally exposed to lead but not in former lead workers no longer occupationally exposed.[7] In a community located near a lead smelter, chromosomal abnormalities were found in 13 of 15 randomly selected exposed individuals.[8]

We do not know whether these effects are associated with low level chronic lead exposures among the general population. Although these studies are not by themselves conclusive, they indicate that we should be concerned about possible genetic implications resulting from general population exposures to lead.

REFERENCES TO SECTION III - LOW LEVEL METABOLIC EFFECTS OF LEAD

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IV. WHAT IS A SAFE BLOOD LEAD LEVEL?

Blood lead levels are frequently used and generally accepted as indices of lead exposure both in the general population and in occupational health situations. Much uncertainty still exists as to the precise relationship between any given blood lead level and the total amount of lead stored in the body. Blood lead levels are probably a function not only of the total body lead stores, but also of the degree of recent lead exposure from all sources including food, water and air. Although much emphasis in this paper is placed upon blood lead as an indicator of exposure, as well as associated risk due to lead, one must recognize that blood lead may not always be an accurate reflection of either situation. Our reason for employing blood lead as the primary exposure index in this document is that, in our opinion, it is presently the best available index that can be related to the possible development of clinical effects due to lead.

Establishment of a single safe blood lead level protective of all high risk groups in the general population is not possible with presently available data. A range of individual responsiveness to lead probably occurs in both children and adults.[1] Healthy adults usually do not demonstrate symptoms of lead intoxication until blood leads have reached 80ug/100g, although symptoms have been reported at blood lead levels in the 50-80ug/100g range.[2] Blood lead levels considered safe for adults may not always be safe for children. For example, clinical symptoms of lead intoxication often occur at lower blood lead levels in children than in adults.

Lead poisoning cases among children are most common in the 1-3 year old age category. This may reflect increased lead exposure among this group due to ingestion of non-food objects containing lead. This ingestion might occur during the normal developmental stage of oral exploration or as a result of an increased incidence of pica at this age. The borderline between abnormal non-food ingestion (pica) and routine oral exploration in children is difficult to define. The possibility that young children may absorb more lead from the oral route than older children and adults is an alternative explanation for the observation that more lead poisoning occurs in 1-3 year olds. Recent data suggest that healthy children may absorb as much as 50% of their oral lead intake compared to the commonly accepted figure of approximately 10% in adults.[3] The possibility that there may actually be an increased biological response to a given internal level of lead in young children must also be considered an alternative and/or contributing factor explaining why lead poisoning is more common in young children. The point of view that children and the young of any species may be more susceptible to lead has been supported by a number of workers in the field.[4,5]

In recognition of the possibility that young children may be more susceptible to lead than older children and adults, the newborn and the fetus would be expected to be especially vulnerable to lead. Exposure of the developing central nervous system in utero to lead, an established neurotoxic agent, should thus be kept at a minimum. The conservative point of view favors a reasonable safety factor between

what is considered an acceptable lead exposure among the fetus and newborn compared to older children and adults.

Most scientists in the field of lead including those from two leading manufacturers of lead additives, the Ethyl Corporation and DuPont, are in agreement with acceptance of 40ug/100g as the upper acceptable blood lead level for adults in the general population.[6,7] Blood lead levels above 40ug/100g in adults are thus usually considered evidence that excessive exposure to lead has occurred. In children, the blood lead level generally considered to be evidence of excessive lead exposure has also been established at 40ug/100g.[8,9,10] Since children are probably more susceptible to lead than adults, one must consider the desirability of revising this figure downward to some level below 40ug/100g. An upper acceptable individual blood lead level in children of 35ug/100g has in fact recently been proposed.[11]

Cases of lead poisoning have been reported among children with blood lead levels in the 40-50ug/100g range.[12,13,14,15] Relating the onset of clinical symptoms of lead poisoning to any specific blood lead level is, of course, recognized to be difficult. The possibility that blood lead is being measured at a point in time when the child is actually asymptomatic must be considered. Such a situation would result in attributing symptoms of lead poisoning to a blood lead level lower than had actually existed when symptoms had initially occurred. As a result, many workers in the field are reluctant to routinely attribute possible symptoms of lead poisoning in children to blood leads in the 40-50ug/100g

range. However, one should also recognize that since symptoms of mild lead poisoning in children are so difficult to identify, the possibility that clinical disease and/or borderline functional impairments may occur at blood lead levels in the 40-50ug/100g range and below has not yet been adequately ruled out. Should many children be shown to have blood lead levels in the 40-50ug/100g range, the need to reduce lead exposures wherever and whenever possible due to the large number of children conceivably affected may greatly outweigh any uncertainty in the observation itself.

As stated above the prudent position is to recommend establishment of a reasonable safety factor between what is considered an acceptable blood lead level in the fetus and newborn in comparison to older children and adults. On this basis, we suggest that umbilical cord blood lead levels of 30ug/100g and above for the newborn and the fetus be considered abnormally elevated. This reflects probable vulnerability of the developing central nervous system to lead, an established neurotoxic agent. This recommendation must, of course, be viewed as a judgement which has not yet been adequately validated by scientific studies.

Compared to adults, newborn babies generally have elevated hematocrits. Since approximately 90% of blood lead is believed bound to the red blood cell, in theory a blood lead level of 30ug/100g in a newborn is really equivalent to a lower blood lead in adults based upon this hematocrit correction. However, the fact that the newborn has a higher proportion of circulating blood volume in comparison to body mass than adults could compensate to some extent for this hematocrit difference.

In addition, the larger mass of central nervous system tissue and the smaller mass of skeletal tissue in the newborn per unit of body weight compared to adults, as well as the relative immaturity of the blood brain barrier at birth are additional factors favoring a margin of safety between what is considered an upper acceptable blood lead in the newborn as compared to older children and adults.

In the opinion of EPA, the available information supports the position that to provide adequate assurances of safety, upper acceptable blood lead guidelines for the general population should be defined as shown in Table IV-1. Blood lead levels above these guidelines in individuals do not necessarily indicate that clinical disease is actually present. These guidelines reflect a judgmental decision with regard to which levels of lead exposure may be associated with a greater possible occurrence of adverse clinical and/or subclinical effects.

For the fetus and the newborn the upper acceptable blood lead limit should be 30ug/100g; for children it should be no more than 40ug/100g (possibly 35ug/100g). In adults a blood lead level of 40ug/100g or above should be considered abnormal and evidence of excessive lead exposure. For expectant mothers a blood lead level of 30ug/100g or above may be a potential hazard to her newborn infant since blood lead levels in newborns are dependent upon and correlate well with maternal blood lead levels.[16]

TABLE IV-1

Blood Lead Guidelines in the General Population

Group	Upper Acceptable Blood Lead Level (ug/100g)
Fetus and Newborn	30
Children	No more than 40
Adults	40
Expectant Mothers	30

REFERENCES FOR SECTION IV - WHAT IS A SAFE BLOOD LEAD LEVEL?

[1]Lin-Fu, Jane S.: "Medical Progress - Undue Absorption of Lead Among Children - A New Look at an Old Problem," New Eng J Med, 286: 702-710, March, 1972.

[2]"Airborne Lead in Perspective," National Academy of Science, Washington, D. C., 1972, p.94.

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[4]Ibid.

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[6]Supplement, comment of Ethyl Corporation on EPA's Proposed Lead Regulation, Ethyl Corporation, July 13, 1972, p.12.

[7]Diggs, D.E: letter to the Deputy Assistant Administrator for Air Programs, EPA, in "Supplemental Statement by E.I. DuPont De Nemours and Company, Inc., Relative to EPA Request for Additional Information on the Health Effects of Airborne Lead," July 12, 1972.

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V. SOURCES OF LEAD EXPOSURE AMONG THE GENERAL POPULATION

Man is exposed to lead primarily through the food he eats, the water he drinks and the air he breathes. Children, especially those with pica, (habitual ingestion of non-food objects) are exposed to lead not only via air, food, and water, but may also be exposed through ingestion of lead contaminated paint, dirt, and dust. Lead contaminated dirt and dust are readily available for children to ingest. Possible exposure of children to lead contaminated paint, dirt and dust is particularly significant for all children, even those without pica, who may ingest these substances during the normal developmental phase of oral exploration.

Fallout of lead from the air is a significant contributor to the lead present in dirt and dust found in urban streets, parks, and homes. Airborne lead is in turn directly related to the use of lead as a gasoline additive. Over 90% of airborne lead emissions in the United States are a result of leaded gasoline combustion.[1] Hence, levels of lead in dust and dirt, especially in urban areas are a function of the use of lead additives. This position is supported by the observation that average soil lead levels collected in front yards of homes in urban areas are two to three times greater than soil lead concentrations in back yards which are located further away from roadways.[2] Preliminary data also indicate that levels of lead in housedust from middle class homes in urban areas are roughly double those found in housedust from middle

class homes in suburban areas.[3] Levels of lead in housedust exceeding 0.06% were reported in these urban homes. Use of lead as a gasoline additive is believed to be a significant factor contributing to this difference and is consistent with the established decrease of lead fallout from the air with increased distances from roadways[4] as well as the higher levels of lead fallout observed in commercial areas compared to residential areas.[5]

The potential contribution of air lead exposures to blood lead in the general population remains a significant issue of debate within the scientific community. While many investigators continue not to support the position that community air lead exposures especially at or below $2ug/m^3$ are capable of affecting blood lead levels in adults residing in the general population, EPA believes that recently completed studies indicate that blood leads are affected. The frequently discussed "Seven City Lead Study"[6] has often been cited as evidence in support of the view that low air lead levels (around $2ug/m^3$) do not have an effect upon blood lead. EPA is not in agreement with this interpretation since depending upon the method of data analysis, effects of air lead at these exposures on blood lead levels can be demonstrated. For example, although a good correlation between air lead and blood lead was not obtained when all geographic areas were considered together, females residing in urban areas and exposed to higher air lead levels were consistently found to have higher blood lead levels than females residing in suburban areas. These issues are discussed more fully in Appendix B. This finding may

be most significant in terms of the potential impact that higher blood lead levels in mothers may have upon blood lead levels among their newborn babies.

One can never be completely certain that increased blood lead levels in female urban residents are entirely due to increased air lead exposures. Other factors such as dietary lead differences as well as the possibility that lead content of water is higher in urban areas may also be contributing. Nevertheless, the consistent blood lead increments found in areas of greater air lead exposure suggests that air lead may be a factor and a factor which may be easily controlled.

Recent reports of significantly increased blood lead levels among women residing in homes in close proximity to a well traveled roadway compared to those living greater distances from that roadway add support to the possibility that air lead resulting from combustion of gasoline containing lead additives is a factor capable of increasing blood lead levels in urban communities.[7] Blood lead level differences among groups reported in this study were probably not significantly affected by either dietary or water lead differences since all women studied presumably had reasonably common sources for both diet and water. Further, the air lead exposures in this investigation were measured inside the homes and on the front porches of the homes of the women studied. This represents a much more accurate determination of true air lead exposure than those from the "Seven City Study" where measurements were made at air sampling stations much further distances away (as great as one mile) and sometimes at heights far above ground level.

Of particular note is the fact that, in the study near the well traveled road, higher blood lead levels were found in the high exposure group despite air lead measurements in the homes and in the vicinity of the homes of this group which were only 1-2ug/m^3 greater than those from the low exposure groups. Further, the high exposure group was characterized not only by statistically significant increases in average blood lead levels, but also by more women with blood lead levels at or above 30ug/100g compared to those in the low exposure categories. Although more women residing in homes 400 feet from the roadway had blood leads above 29ug/100g than women residing in homes 125 feet from this road, this difference was not statistically significant based upon a Chi square analysis ($0.10 < p < 0.20$). However, the increase in number of women with blood leads greater than 29ug/100g in the group living closest to the roadway compared to the groups living 125 and 400 feet away was statistically significant in each instance ($p < 0.01$). These results, considered by EPA to be even more reliable than those from the "Seven City Study" generally confirm similar observations derived from the "Seven City Study." The following table summarizes these results.

Comparisons between values obtained from the Seven City Study with those from the Roadway Study must be made with caution since the groups may not be matched closely enough with regard to all variables generally recognized to influence blood lead levels (such as diet, age, race, hematocrit and smoking). However, the comparisons within the Roadway Study and those within specific urban-suburban areas from the "Seven City Study" are more valid so that causal inferences appear reasonably justified

TABLE V-1

Summary of Data Relating Blood Lead Levels in Women to Place of Residence

Population	Number Studied	Average Air Lead Exposure (ug/m^3) Front Porch	Average Air Lead Exposure (ug/m^3) In Home	Average Blood Lead ug/100g	%Blood Leads Above 29ug/100g	%Blood Leads 40 & Above	%Blood Leads Above 50
From Roadway Study[7]							
Living Near Roadway (12 feet away)	55	4.60	2.30	23.1	25.4	1.8	1.8
Living Away From Roadway							
(a) 125 feet away	34	2.41	1.50	17.4	0	0	0
(b) 400 feet away	61	2.24	1.57	17.6	6.6	1.6	0

	Number Studied	Average Air Lead Exposure ug/m^3 (geometric mean)	Average Blood Lead ug/100g (geometric mean)	%Blood Leads Above 29ug/100g	%Blood Leads 40 and Above	%Blood Leads Above 50
From Seven City Study[6]						
New York Urban	140	2.08	16.6	1.4	0	0
New York Suburban	198	1.13	15.3	0.5	0	0
Chicago Urban	147	1.76	17.6	3.4	0.7	0
Chicago Suburban	208	1.18	13.9	0.5	0	0
Philadelphia Urban	136	1.67	20.5	11.0	1.5	0
Philadelphia Suburban	150	1.15	18.0	4.7	0	0

with respect to the possible impact of air lead upon blood lead.

Although recent theoretical predictions derived from microscopic analyses of lead particulate matter suggest that airborne lead in the general urban environment might not be appreciably absorbed via the respiratory route[8] these above data would seem to contradict this position. These data suggest that even small increments in community air lead exposures have a definite effect upon blood lead levels among women residing in urban areas.

Further, these results cast doubt on the adequacy of the previous position taken by EPA that achievement of a 2ug/m^3 air lead goal would assure a reasonably complete degree of public health protection. This is especially true in view of the possibility that blood lead levels at or above 30ug/100g in mothers might cause similar blood lead elevations among their newborn babies. A statistically significant correlation has been reported between blood lead levels in mothers and those in their newborn infants indicating that the concentration of lead in newborns is dependent upon levels of blood lead in the mother.[9] These observations are supported by additional studies showing that residence in urban environments is associated with elevated blood lead levels[10,11] and that persons living near highways generally tend to have higher blood lead levels than those living away from highways.[12] This latter study again implicates lead in gasoline as a factor contributing to these blood lead elevations.

An investigation of blood lead levels in taxi drivers and other occupational groups, not all occupationally exposed to automobile exhausts, using personal monitors to measure air lead exposure, is also reasonably consistent with these above observations.[13] Statistically significant

correlations between air lead and blood lead were not obtained within each group studied (30 subjects per group). However, when all groups were combined and adjusted statistically to account for dietary lead differences among groups, a significant correlation between air lead and blood lead was obtained. In this latter analysis, variation in air lead exposure accounted for 44% of the variation in blood lead levels which is reasonable considering the variation due to dietary lead sources that still existed within groups. Blood lead levels of 40ug/100g were generally not observed even in the high exposure group in which air lead measurements reached 9ug/m^3. This may seem to be somewhat contradictory at first. However, when one considers the small number of individuals studied within each group (30) the failure to detect blood lead levels of 40ug/100g and above only rules out the possible occurrence of these high blood lead levels at a rate of approximately 10% within each group.* Among general urban

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*What sample size N is needed to find at least one case where the prevalence rate is P, with probability 1 - a?

The probability of at least one case is one minus the probability of no cases:

$$1 - (1 - P)^N$$

Setting this equal to 1 - a gives

$$1 - (1 - P)^N = 1 - a$$
$$N = (\log a)/\log (1 - P)$$

for a = 0.05 we have:

p = 0.01	N = 299
p = 0.03	N = 99
p = 0.05	N = 59
p = 0.095	N = 30
p = 0.10	N = 28.4

adult populations, blood lead levels of 40ug/100g do not usually occur more frequently than 5%. These points are discussed in greater detail in the following section.

Carefully controlled chamber exposures to airborne lead at approximately 3ug/m^3 in human volunteers also demonstrate that blood lead increments can be expected at these air lead levels.[14] For example, after 12 weeks of nearly continuous exposure to air lead at 3.2ug/m^3, average blood leads rose more than 30% (from 18.0 to 24.1 ug/100g) among a group of 6 prisoner volunteers who had not been appreciably exposed to airborne lead since arriving at the prison which was located in a rural environment. During this study all men had common sources of dietary lead intake. Although smoking habits may have differed among the men, each man was his own control and presumably continued to smoke at the same rate during this study as before. Blood leads returned to pre-exposure levels following cessation of the experiment. Hence, air lead exposures at approximately 3ug/m^3 appeared responsible for the observed blood lead elevations. These blood lead increases were comparable to those found among the women residing near a well traveled road. (See Table V-1).

With respect to children, the possible effect of direct air lead exposures conceivably is of secondary importance when compared to the role air lead may play in contaminating dirt and dust which could then be ingested by young children. Elevated blood lead levels have been found among children attending school in higher air lead areas compared to those in lower air lead areas.[15] The difference in air lead exposure between areas (1.69ug/m^3 vs 1.48ug/m^3) may have been too small to singly account

for the entire difference in average blood lead levels which was observed, (34.1 ±9.7ug/100g, mean ± standard deviation, in high air lead area compared to 26.3 ±7.1ug/100g in low air lead area). In this case air lead levels below 2.0ug/m^3 were associated with blood lead levels of 40ug/100g and above in approximately 20% of tested children in the high exposure area. Since peeling lead paint was not believed to be a significant problem in this community, these findings suggest the possible role that the dustfall lead exposure mechanism may have played in contributing to these blood lead elevations. Several industrial sources of lead emissions were present in the vicinity of the homes where these children lived and could well have contributed to significant fallout of lead from the air to contaminate dirt and dust.

Previous epidemiologic studies of the lead poisoning problem among children have consistently associated lead poisoning and excessive lead exposure with residence in homes containing lead based peeling paint. Lead based peeling paint is a problem that must be vigorously attacked. However, associating residence in deteriorating housing containing peeling lead based paint with lead poisoning does not mean that other environmental lead sources, such as lead contaminated dirt and dust are not also contributing to excessive lead exposure among the children residing in these homes.

Analysis of exising data indicates that environmental exposures to lead contaminated dirt and dust can contribute significantly to excessive lead exposure in children.[16] Samples of dirt and dust collected from the streets of urban areas reveal concentrations of lead far greater

than those considered safe in paint by the Food and Drug Administration (0.06%).[17,18,19] These surveys of urban environments in Boston and Washington demonstrate elevated concentrations of lead in street dirt at times exceeding 0.5%. Levels of lead in dust were also found inside homes in the Boston area predominantly in the range between 0.1-0.2%.[20] Although lead from peeling paint may have partially accounted for this observation in older homes, this factor was not a reasonable explanation for the elevated housedust lead concentrations often found in homes built after 1950. Concentrations of lead in street dust of 0.2% and lead in dust on window frames near busy roadways of 0.175% have also been observed in European cities.[21] Based upon this study, lead concentrations up to 0.5% are believed common in fine fractions of street dust collected from busy roadways.

As indicated above, lead contaminated dirt and dust may be ingested by children. The prevalence of pica (habitual ingestion of non-food items) among children, is high, perhaps exceeding 50%.[22] While high concentrations of lead in individual paint chips are considered especially hazardous, cases of lead poisoning in children have been associated with paint surfaces containing less than 1% lead.[23,24] Data from the City of New York Lead Poisoning Control Bureau indicate that among children with blood leads betwen 35 and 44ug/100g, only half can be associated with peeling paint containing 1% lead or greater.[25] Further, nearly 20% of cases in this blood lead category lived in homes in which peeling paint was not identified.

It is well recognized that children may contact peeling lead based paint in homes other than their own and that peeling paint containing 1% or more lead may often be found upon re-inspection of homes initially not considered to contain this hazard. However, the presence of lead contaminated dirt and dust (even well below 1% lead) is also believed to be potentially harmful to children. Continued ingestion of only fractions of a teaspoon per day of the lead contaminated dirt and dust presently routinely found in urban areas by children would easily exceed the well recognized daily permissible intake of lead for children (DPI) established at 300ug lead per day.[26]

For example, lead emitted from automobiles is known to be absorbed from the gastrointestinal tract under experimental conditions. Rats fed samples of lead contaminated dirt collected from the Queens Midtown Tunnel in New York (at 5mg lead per day in their diet), demonstrate 3-4 fold increases in blood lead levels compared to controls not fed this material.[27] Combined ingestion of lead based paint and lead in dirt and dust thus could be responsible for the large number of urban children found to have abnormally elevated blood leads.

A report by the National Academy of Sciences is in agreement with this conclusion and states that, "the swallowing of lead contaminated dusts may well account in large part for the higher mean blood lead content in urban children and the rather large fraction whose blood lead content falls in the range of 40-60ug/100g."[28]

The possible role that calcium deficient diets may play in enhancing gastrointestinal lead absorption is another potentially important factor that must be considered. Low calcium diets greatly increased lead absorption and lead body burdens as well as associated lead pathologic changes in rats fed 200ppm lead in their drinking water compared to controls on normal calcium diets.[29] Calcium deficient diets also appeared to alter the partitioning of lead between fixed bone and the more readily diffusable lead found in soft tissues. These results suggest that at least under certain metabolic conditions (such as low calcium diets), lead stored in bone is more readily available for movement to other tissues. Conditions such as pregnancy, where there is a requirement for more calcium than usual, might also be associated with a similar movement of lead from bone unless adequate calcium is supplied. Such a situation, if it were shown to occur, should be considered potentially harmful to the fetus.

Since children living in high risk urban areas are often members of low socioeconomic groups, they might be at risk not only from exposure to lead based paint and lead contaminated dirt and dust but also by the possible coexistence of dietary calcium and/or iron deficiencies. In this context, experimental iron deficiency has also been shown to produce greater concentrations of lead in tissues of rats given subtoxic levels of lead compared to a similar group of rats without iron deficiency.[30]

Additional studies further support the possibility that the dust-fall lead exposure mechanism may represent a potential hazard to children.

In one investigation, 230 rural children and 272 children from an urban poverty area were tested for excessive lead exposure in the summer of 1971.[31] Nearly all of the rural children (18 out of 19) with excessive lead body burdens lived in homes containing at least one accessible surface with 1% lead paint or greater. However, this paint hazard could be found on accessible indoor and exterior surfaces in homes of only 60% of urban children found to have excessive lead exposure. Further, approximately one quarter of the urban children tested had abnormally elevated blood leads (40ug/100g and above) compared to less than 10% of the suburban children. Hence, young children living in urban areas appear to be more excessively exposed to lead than those residing in rural areas. These findings are consistent with the possibility that excessive lead exposures are caused not only by lead in paint, but also by lead in other urban environmental sources including lead in the air, and in the dust and dirt which settles out from the air. The Department of Health, Education and Welfare, in commenting upon EPA's position regarding removal of lead from gasoline notes: "For those children with pica who eat dirt, the danger from exposure to lead containing dust and dirt is great."[32]

Demonstration of excessive lead exposures among children residing near a smelter in El Paso, Texas, further emphasizes the potential importance of the dustfall lead exposure mechanism.[33] Approximately 90% of the 1-5 year old children sampled who were living near the smelter had blood leads of 40ug/100g or above. Information available to EPA indicates that lead paint was not a significant factor in the etiology of abnormally elevated blood lead levels found among children residing near the smelter.[34] Soil lead levels in the vicinity of the

smelter averaged 0.4%-0.5% lead with a range of 0.15% to just over 1%. These are not significantly different from levels of lead in dirt and soils found in many urban streets and parks.

Though air lead levels were also significantly elevated near the smelter (100-300ug/m^3), most of the airborne lead (approximately 75%) was judged to be in the nonrespirable range. A larger percentage (89.2%) of the 1-5 year old children living in the vicinity of the smelter had abnormally elevated blood leads compared to a 6-17 year old group residing in the same area (64.7%). This suggests that exposure to airborne lead as well as ingestion of lead contaminated dusts was contributing to excessive exposures in the group more likely to ingest non-food items, the 1-5 year olds. Hence, levels of lead in street dirt of this magnitude (averaging 0.4-0.5%) found near the smelter represent a potential hazard for children with pica. A more in depth study supervised by HEW, with EPA participation, is currently planned to further clarify the etiology and extent of this problem.

One recent reevaluation of lead sources in the environment, including factors to account for lead concentration as well as availability of the source considers lead in dirt (but not airborne lead directly) to represent a potential source of lead approximately one half the magnitude of that from lead in paint.[35]

In summary, blood lead should be considered a function of all exposure routes. Available evidence indicates that living in urban environments where lead exposures are generally elevated, is associated

with higher blood lead levels in adults. Even air lead levels around $2ug/m^3$ appear to contribute to blood lead levels among adults and may possibly contribute to elevated blood lead levels among babies born to mothers living in urban environments. Especially for children who are known to ingest non-food items, lead falling out from the air and in turn contaminating dirt and dust should be considered a potential hazard. Over 90% of airborne lead emissions are a result of leaded gasoline combustion. Consequently, lead in the air as well as in street dirt and household dust are preventable exposures which can be readily decreased by regulating the use of lead as a gasoline additive.

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SOURCES OF LEAD EXPOSURE AMONG THE GENERAL POPULATION

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VI. EXTENT OF ABNORMAL LEAD EXPOSURE AMONG THE GENERAL POPULATION

Individuals within groups may often be excessively exposed to lead even though average lead exposures for the group are well within normal limits. Thus, although average blood lead levels among urban populations are well within normal limits, considerable numbers of individual urban residents have blood lead levels exceeding 40ug/100g.

Abnormal blood level elevations have been documented among adults. They are usually associated with residence in urban areas where air lead levels tend to be greatest. At present, it is unclear whether increased air lead exposures in urban areas are solely responsible for these blood lead elevations. Other sources, such as increased dietary lead and water lead, might be alternative explanations. Ingestion of water containing lead immediately above the PHS standard of 50ug/l would not generally increase ingested lead intake much above that expected as a result of normal dietary variability (100-500ug/day of lead).[1] Data such as those previously presented (Section V - Sources of Lead Exposure Among the General Population) suggest that increased air lead exposures in urban communities are contributing to the extent of excessive lead exposures among urban adults as summarized in Table VI-1. Since over 90% of airborne lead is due to lead automotive emissions,[2] these emissions are believed to be contributing significantly to this problem.

Extrapolation from the evidence in Table VI-1 indicates that approximately 1-2% of adult females and 3-5% of adult males residing in urban areas have abnormally elevated blood leads (40ug/100g and above). This observation reflects the probable existence of excessive lead exposures among millions of urban adults. In selected sub-groups such as

garage mechanics and parking attendants, this proportion is markedly higher, approaching 50% and above. Although these exposures are occupationally related and could possibly be controlled by better industrial hygiene practices, the source of lead is primarily from gasoline containing lead additives.

Within each city in Table VI-1 the percentages of individuals with abnormally elevated blood leads are generally consistent with the expected gradients according to exposure category. However, especially when specific exposure categories are compared from city to city inconsistencies become evident. This may reflect exposure to different levels of atmospheric lead in combination with differing amounts from dietary lead sources.[3]

Table VI-2 summarizes existing data which demonstrate that abnormally elevated blood lead levels among adults are found predominantly in urban areas where greater exposures to airborne lead are more likely to occur. When considering the data in this table, one must be aware that populations being compared may not be appropriately matched for all of the pertinent covariates recognized to influence blood lead levels. The people in the individual geographic areas which were combined in this first comparison were all women who were not always equally matched with respect to age, smoking habits, and dietary lead exposures. However, consistent urban-suburban differences for both smokers and non-smokers with higher blood lead levels recorded in urban areas were found in each area.[4] Since specific urban-suburban comparisons also tend to minimize dietary differences, this first comparison seems reasonably valid. (Individual urban-suburban breakdowns were previously presented in Table V-1).

The validity of the second and third comparisons are somewhat more suspect than the first. In the second comparison the large differences in numbers studied might in theory be important since, if only one additional person in the suburban group were studied who happened to have a blood lead level of 40ug/100g or above, the frequency of this occurrence would have been approximately equal in both groups. However, the fact that only 13% of the suburban group studied had blood leads of 20ug/100g or more compared to 23% of the urban residents, suggests that a real difference existed between the groups. The third comparison is least reliable since many individuals of unknown history and residence are combined in this instance; nevertheless there was a definite increase in the blood leads from the urban when compared to the suburban category.

Among children, extensive surveys (see Table VI-3) have demonstrated that excessive lead exposures have approached what many consider an "epidemic" proportion. Approximately one quarter of the children tested showed elevated blood leads of 40ug/100g and above. Although these excessively exposed children are often residents of homes coated with lead based paints, lead in the air, and consequently lead in dust and dirt may be contributing to and aggravating this problem. For example, in some of the children a history of exposure to lead based paint cannot be elicited and housing investigations fail to reveal the presence of peeling lead based paint (traditionally defined as paint containing 1% lead or greater). In such cases other potential sources of lead exposure such as lead from the air and lead which settles out from the air to contaminate dirt and

dust must be considered as possible contributing causes. This is discussed in more detail in the previous section.

Recent preliminary data indicate that excessive lead exposure may already be occurring before birth among babies born to mothers living in urban environments. This is based upon reported umbilical cord blood lead levels of 30ug/100g and above in these newborns. This is not much below the levels at which clinical symptoms of lead poisoning in children have been observed. Increased exposures to airborne lead in these urban environments should be considered a possible factor contributing to this problem. In Boston, where excessive drinking water contamination by lead is recognized, one might also suspect this source to be a potential contributor. For example, in a study conducted in the Boston area, umbilical cord blood lead values of 30ug/100g or above were present in 3 of 13 (23%) of babies tested who were born to urban mothers.[5] Cord blood leads of 37 and 39ug/100 were observed in two of these babies, but cord bloods of 30ug/100g and above were not found among any of the suburban babies studied. Since less than 23% of mothers in urban areas are believed to have blood leads of 30ug/100g or more, the Boston study probably reflects the role played by lead in water as well as lead in air.

A second study examined umbilical cord blood lead levels among babies born to mothers living only in New York City.[6] Of 100 urban newborns sampled, 6 were found to have umbilical cord blood lead levels in the range of 25-34 ug/100g. This is reasonably consistent with the reported occurrence of blood lead levels in this range among women residing in New York City (see Table V-1). Several of these babies were probably

born with cord blood leads of 30ug/100g or above, again a level considered to be evidence of excessive exposure.

A third study failed to demonstrate any difference in umbilical cord blood lead levels between babies born to mothers living in urban compared to suburban environments.[7] Since only a small number of babies were sampled (24 in total) this minimized the chances of detecting a significant difference between the groups, should a real difference have existed.

The relationship between blood leads in mothers and those in their newborn babies as measured via umbilical cord sampling would be expected to be dependent upon lead exposures to the mother. Findings such as these above confirm what has already been well established, that lead can readily cross the placenta from the mother to the baby. Possible concentration of lead by fetal blood compared to maternal blood has also been reported which may be of potential significance with regard to what is considered a safe blood lead level in an expectant mother.[8]

In summary, considered as a group, there studies indicate the probable existence of abnormally elevated umbilical cord blood lead levels among babies born in urban environments. If these studies are at all applicable to the general urban population, then significant percentages of babies born in urban environments are probably exposed to excessive amounts of lead even before birth.

TABLE VI - 1

Extent of Abnormally Elevated Blood Leads
Among Urban Adults

City	Exposure Category	Number Studied	% of Blood Leads Equal to or Greater than 40ug/100g
Cincinnati	Post Office Employees[1]	140	2.9
	Firemen[1]	191	3.0
	Service Station Attendants[1]	130	12.3
	Police[1]	40	12.5
	Drivers of Cars[1]	59	15.0
	Parking Attendants[1]	48	44.0
	Garage Mechanics[1]	152	67.0
Los Angeles Area	L.A. Police[1]	155	0.6
	Pasadena Male City Employees[1]	88	3.3
	L. A. Female Aircraft Employees[1]	87	3.3
	General L.A. Clinic Population[5]	45	4.4
	L.A. Male Aircraft Employees[1]	291	5.2
Oakland	Female Clinic Patients[5]	53	1.9
	Male Clinic Patients[5]	36	5.5
Philadelphia			
	Male Commuters [1]	43	2.3
	Police[1]	113	3.5
	Downtown Male Residents[1]	66	4.5
Camden, New Jersey	Women Living Near Freeways[4]	55	1.8
Composite Urban Samples	Females from New York, Phila., and Chicago[2]	423	0.7
	Males and Females from 6 Cities[3]	833	2.7*

* Only those above 40.

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TABLE VI-2

Urban--Suburban Blood Lead Comparisons in Adults

Group Studied	Number Studied	% Blood Leads Equal to or Greater than 40ug/100g
Urban Females[2]	423	0.7
Suburban Females	556	0
Philadelphia Males[1]		
Urban	66	4.5
Suburban	23	0
Composite[3]		
Urban	833	2.7*
Suburban	162	0

*Only those above 40.

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TABLE VI-3

Percentages of Children with Abnormally Elevated Blood Leads

City	Years Tested	Numbers Tested	% Blodd Leads Equal to or Greater than 40ug/100g
Baltimore[1]	1968	665	25.3
	1969	746	27.9
	1970	939	31.5
Chicago[1]	1967-70	120,000	20.0
New Haven[1]	1969-70	1,897	29.8
Newark[1]	1970	594	38.9
New York[1]	1969	2,648	45.5
	1970	84,368	28.7
New York[2]	1971	81,626	20.2
Philadelphia[1]	1970	3,496	34.0
Washington[1]	1970	808 (all ages)	5.8
	1970	1,152 (2 years)	22.0
Many Cities[3]	1971	2,309	9.1
Aurora, Ill.[4]	1971	449	24.3
Springfield, Ill.[4]	1971	670	30.1
Peoria, Ill.[4]	1971	387	31.3
E. St. Louis, Ill[4]	1971	376	24.7
Decatur, Ill.[4]	1971	793	12.2
Joliet, Ill.[4]	1971	383	24.3
Rock Island, Ill[4]	1971	285	21.1
E. Moline, Ill.[4]	1971	298	11.4
Robbins, Ill.[4]	1971	103	12.6
Harvey, Ill.[4]	1971	226	16.4
Carbondale, Ill.[4]	1971	264	17.0
Norfolk, Va.[4]	1971	1,225	22.7
New Haven, Conn[4]	NA	1,339	23.7
Washington, DC[4]	1971	1,821	39.2
Rockford, Ill.[4]	NA	1,200	19.5

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VII. FINDINGS AND RECOMMENDATIONS

Findings

1. Lead is a known toxic substance for which no beneficial biological role has yet been demonstrated.

2. Experimental evidence suggests that the least measurable quantities of lead within cells are capable of affecting cellular metabolism and that these effects are a function of lead concentration. For example, inhibition of the enzyme delta aminolevulinic acid dehydrase in the peripheral blood of man is a function of blood lead concentration even at blood lead levels well below those generally considered excessive (40ug/100g and above). Inhibition of this enzyme is not believed to be physiologically significant until blood leads have reached 40ug/100g. However, this effect has been noted in children as well as adults, although its true significance is at present unknown. Since ALAD inhibition by lead in peripheral blood of suckling rats correlates well with ALAD inhibition in the brains of these animals, this suggests that a similar phenomenon might also occur in young children. Recent associations of behavioral disturbances among children with increased lead exposure, but at blood lead levels presently not believed excessive (below 40ug/100g), raises the question whether lead inhibition of enzymes in the central nervous system of children might be a possible contributing factor in the etiology of these disturbances.

3. Susceptibility to lead may possibly be increased among young children as compared to adults. New born babies conceivably are potentially most vulnerable to lead. Exposure of the developing central nervous system in utero, to lead, an established neurotoxic agent, should be

kept at a minimum. The conservative position favors a reasonable safety factor between what is considered a safe blood lead level in children and what is considered an acceptable exposure among the newborn.

4. Considerable difficulty exists in defining a single safe blood lead level protective of everyone in the population. Variable responsiveness to lead probably exists among different age groups and even within age categories. In this context, available scientific evidence supports the following guidelines defining excessive lead exposures. Blood lead levels above these guidelines in individuals do not necessarily indicate that clinical disease is actually present. These guidelines reflect a judgmental decision with regard to which levels of lead exposure may be associated with a greater possible occurrence of adverse clinical and/or subclinical effects.

a. Blood lead levels of 40ug/100g or above in adults are considered evidence of excessive lead exposure.

b. For expectant mothers the upper acceptable blood lead level should probably be no more than 30ug/100g. Low calcium diets have been shown in experimental situations to increase gastrointestinal lead absorption as well as lead storage in the soft tissues. Since there is a requirement for more calcium than usual during pregnancy, this factor may be important with respect to determining acceptable lead exposures for expectant mothers.

c. A safe blood lead level protective of all children is no more than 40ug/100g.

d. Blood lead levels of 30ug/100g or above in newborn babies obtained from umbilical cord blood should be considered evidence that excessive lead exposure has probably occurred to the fetus in utero.

5. Though food and water usually account for more lead exposure than airborne lead among the general population, airborne lead levels around 2ug/m^3 have been demonstrated to contribute to blood leads in adults. These same air levels are associated with blood lead elevations in children perhaps reflecting the dustfall lead exposure mechanism.

6. Though lead paint is considered to be the prime causal factor in childhood lead poisoning, other environmental sources such as air lead and lead which settles out from the air to contaminate dirt and dust are also capable of contributing to this problem. Large percentages of children are known to ingest non-food objects including dirt and dust. For these children, possible ingestion of lead contaminated dirt and dust should be viewed as potentially harmful.

7. Levels of lead in street dirt and house dust in urban areas have been found to be far greater than those considered safe in paint by the Food and Drug Administration. Evidence exists to indicate that the presence of lead in gasoline contributes to high levels of lead in dust and dirt found in areas and homes which are located near busy roadways.

8. Individuals within groups may often be excessively exposed to lead even though average lead exposures for the group are well within normal limits. On this basis, although average blood lead levels among urban populations are well within normal limits, considerable numbers of

individual urban residents are found to have blood lead levels exceeding 40ug/100g.

a. Small increases in average blood lead levels found among adult residents in urban compared to suburban areas may well account for the relatively large number of individual urban adults found to be excessively exposed to lead. Recent surveys of adult populations indicate that approximately 1-2% of urban females and 3-5% of urban males probably have blood lead levels of 40ug/100g and above. Residence in areas where air lead levels are greatest is consistently associated with this finding. Approximately 5-10% of women residing in urban areas have blood lead levels of 30ug/100g and above, a level which in expectant mothers should be considered a potential hazard to newborn babies.

b. Excessive lead exposures among children have approached what many consider an "epidemic" proportion. Extensive surveys involving over one quarter of a million children, document that approximately 25% of children tested have abnormally elevated blood leads of 40ug/100g and above. Although these adversely affected children are often residents of homes coated with lead based paints, lead in the air and consequently in the dust and dirt present additional sources of exposure which may contribute to and aggravate this problem.

c. Recent preliminary data suggest that excessive lead exposure may already be occurring before birth among babies born to mothers living in urban environments. Significant numbers of babies born in the central city may have umbilical cord blood lead levels well above 30ug/100g, and even approaching 40ug/100g, a level close to those at which clinical symptoms of lead poisoning in children have been observed. Exposure of

expectant mothers to airborne lead in urban environments could be an important factor contributing to these blood lead elevations.

9. Over 90% of airborne lead emissions are a result of combustion of gasoline containing lead additives.

Recommendations

These results cast doubt on the adequacy of the previous position taken by EPA that achievement of a $2ug/m^3$ air lead goal would assure a reasonably complete degree of public health protection. This is especially true in view of the possibility (a) that blood lead levels at or above 30ug/100g in mothers might contribute to similar blood lead levels among their newborn babies and (b) that air lead levels around $2ug/m^3$ may be associated with potentially harmful levels of lead in dirt and dust. On this basis, further air lead reductions below $2ug/m^3$ would seem indicated.

Though none of the above findings viewed individually and in the context of possible experimental error can be taken as conclusive evidence that airborne lead by itself is a current public health problem, considered together, they do suggest that airborne lead is contributing to excessive total lead exposures among the general urban population. Every effort should, therefore, be made to reduce all preventable lead exposures, including airborne lead, to the fullest extent possible.

APPENDIX A - OVERVIEW OF EPA'S CONCLUSIONS REGARDING RESPONSES RECEIVED TO QUESTIONS WHICH APPEARED IN THE FEDERAL REGISTER (Vol. 37, No. 115, pp. 11786-11787, June 14, 1972)

Question 1: In the light of any criticisms you may have of the Goldsmith-Hexter approach and the Environmental Protection Agency's use of a regression equation based upon it (see Figure 3-3 of "Airborne Lead in Perspective," National Academy of Sciences, 1972; and Table 7 of "Health Hazards of Lead," Environmental Protection Agency, revised April 11, 1972, which was corrected in "Corrections and Additions to Health Hazards of Lead," April 27, 1972), what are the permissible uses and limitations in its application for obtaining reasonable estimates of blood lead levels as a function of air lead exposures?

The Goldsmith-Hexter regression equation relates changes in the average blood lead level of various groups to corresponding changes in their exposure to atmospheric lead. EPA believes that the physiologic basis behind the Goldsmith-Hexter approach is correct; that is, at higher atmospheric lead exposures blood leads will increase. The major problem with this approach has been the difficulty correlating blood lead levels with air lead at low air lead exposures (below 2ug/m^3). At these low air lead levels, the normal lead intake from food and water is greater than that from air, but not so great that air lead exposures do not affect blood lead levels. Hence even small variations in dietary lead intake which ranges from 100-500 ug per day[1] will tend to mask any changes in blood lead due to variations in air lead exposure. Unless dietary lead exposure can be kept reasonably constant, the likelihood of observing a correlation between blood lead and air lead at low air lead concentrations is very slim.

In judging the validity of the Goldsmith-Hexter regression equation[2] for relating blood lead levels to air lead exposures several important factors must be kept in mind. All data used in this regression analysis were not always ideally suited for this purpose. Air lead exposures were at times estimated rather than measured and blood leads were not always determined at appropriate points in time in relation to air lead measurements which were made. Further, differences in dietary lead intake may have confounded blood lead differences among groups used in the regression analysis. Finally, average blood lead levels for the groups were compared under circumstances which were not justified since the groups were often of different size with unequal variances. Use of only average blood lead levels also tends to obscure a considerable quantity of useful information present in the original data, such as the occurrence of abnormally elevated blood leads.

Primarily for the above reasons, EPA does not believe that use of the specific regression curve developed by Goldsmith and Hexter[2] is the optimal approach for predicting general population responses to air lead exposures. Reluctance to use this particular approach in no way implies that the Goldsmith-Hexter equation was not valuable. If anything, it highlights the importance of considering the role played by airborne lead as a determinant of blood lead level.

EPA's reluctance to employ this equation in a quantitative way is a result of uncertainty as to its preciseness for describing responses of blood lead to air lead especially at low air lead exposures. Our decision not to employ this equation does not mean that EPA does not consider air

lead to be an important exposure mechanism in the general population. Additional methods of statistical analyses focusing upon individual rather than average blood leads demonstrate that airborne lead is a significant factor contributing to blood lead. One noted biostatistician in commenting upon the Goldsmith-Hexter regression equation concludes:[3]

> "It is interesting to note that all of the variations in fitted trend lines that have been suggested would indicate that there is some increase of average blood lead as air lead increases at any level of air lead. The various curves differ with regard to the rate of this increase, but the data certainly do not encourage the notion of a threshold below which changes in air lead are unrelated to blood lead... it would seem to be highly imprudent, with our current information, to assume that there is any safe threshold below which air lead does not affect blood lead."

Comments received by EPA in response to this question have generally supported our reconsidered position that use of the Goldsmith-Hexter equation in a quantitative way to predict population responses to air lead exposures is not the ideal approach.

For example, a recently completed study by DuPont[4] which measured blood leads in various occupational groups using personal air lead sampling devices capable of measuring individual air lead exposure comes to a similar conclusion:

> "Any attempt to predict blood lead levels solely on the use of the average relationship line developed in this study (which is similar

to the Goldsmith-Hexter approach as well as the "7 City Study" approach) could be misleading because the effect of lead intake from other sources such as food and drink is significant."

Several prominent biostatisticians also concur with EPA's preference to consider individual blood lead values as well as average blood leads when comparing responses of groups exposed to various air lead levels. For example, according to Dr. Enterline:

> "...A test of statistical significance of these data (data used in Goldsmith-Hexter regression equation) is difficult to interpret, however, since what must be of interest is the relationship between air lead levels and individual blood lead levels...not means or groups of people."[5]

Dr. Robert Reed, Chairman of the Department of Biostatistics at the Harvard School of Public Health is in agreement with this approach:

> "A trend line is essentially an average relationship between air and blood lead. From the public health point of view, we must be concerned with individual variation in blood levels. It is almost inevitable that at ambient air levels which produce borderline 'acceptable' blood levels there will be an important fraction of the population with higher 'unacceptable' blood levels. This variation may be due to a number of factors. An important aspect of this issue is the possibility of a serious additive effect of air-lead and dust-lead from air to the lead paint exposure of children in certain central city areas."[6]

REFERENCES FOR APPENDIX A - Question 1

1
"Airborne Lead in Perspective" National Academy of Sciences, Washington, D.C. 1972, p 50.

2
Goldsmith, J. R. and Hexter, A. C., "Respiratory Exposure to Lead: Epidemiological and Experimental Dose - Response Relationships," Science, 158:132-134, 1967.

3
Reed, Robert, Professor of Biostatistics, Harvard School of Public Health, Testimony submitted to EPA, August 4, 1972.

4
Supplemental Statement by E. I. DuPont De Nemours & Company, Inc. Relative to EPA's Request for Additional Information on the Health Effects of Airborne Lead, July 12, 1972, Section 6, "Relationship of Airborne Lead and Indices of Lead Absorption" presented at the International Symposium on Environmental Health Aspects of Lead, Amsterdam, October 2-6, 1972.

5
Cole, Jerome, Testimony presented at Dallas Public Hearings, p. 459 of Hearing Record, April 28, 1972.

6
Reed, Robert, Testimony Submitted to EPA, August 4, 1972.

Question 2: How accurate a reflection is blood lead of lead body burden? What is the effect of elevated blood leads upon lead body burden? Can small increments in blood lead be expected to result in a significant lead body burden elevation? From a public health point of view, is it permissible to allow slight increases in lead body burdens among the general population when this increment can be prevented? Can the pool of body lead stored in the bone be viewed as totally "physiologically inert"? It is known that chelation therapy of children with elevated blood leads can result in acute clinical symptoms of lead poisoning as a result of mobilizing lead from bone. Is there any evidence that subtle metabolic changes could also mobilize this lead pool under other conditions?

EPA's position after having reviewed the responses received to this question is that there is no simple answer regarding any of these issues. Whether blood lead is in all instances an accurate reflection of lead body burden is difficult to say. Blood lead appears to be a reasonable indicator of recent lead exposure. Certainly the majority of available evidence regarding adverse clinical and/or subclinical effects of lead is related to blood lead measurements as an index of either body burden or recent exposure. Two lead additive manufacturers support the use of blood lead as a reasonable indicator of lead body burden.[1,2] Traditional use of blood lead as an exposure index in occupational situations and the correlation of biological effects with blood lead support the continued utility of blood lead determinations as indices of both recent exposure and body burden.[3]

Whether small increments in blood lead can be expected to result in significant lead body burden elevations is a complex problem. One manufacturer of gasoline lead additives feels that any significant sustained increase in lead exposure will produce increments in lead body burden.[4] The prime difficulty centers around how much of an increase in lead exposure is required before definite increases in lead body burden occur. Most of the body's lead content (90-95%) is stored in bone. Hence slight increases in blood lead level may not raise total body burden per se, but may still pose a health hazard in terms of additional lead available for storage in soft tissues including the central nervous system. Further, mobilization of even a small portion of lead from bone into the soft tissues could pose a definite threat to health.

After reviewing the evidence EPA concludes that while most lead stored in bone is probably not generally available for mobilization,[5,6] under certain instances, expecially rapid physiologic alterations, lead from bone may well be mobilized into the soft tissues. Conditions such as pregnancy, and/or any intercurrent illness which cause demineralization of bone could result in mobilization of lead from bone, which in some instances could be hazardous.[7]

Although several investigators have tried unsuccessfully to mobilize lead from bone under experimental conditions, this does not constitute proof that under all conditions lead stored in bone is in fact physiologically inert.[8] Recent evidence suggests that lead stored in bone may, in fact, inhibit hemoglobin synthesis in the intact animal.[9]

Studies involving the possible effect of dietary deficiency on lead metabolism indicate that low calcium diets may significantly change the partition between the amount of lead stored in bone and that stored in the soft tissues with more lead being found in soft tissues compared to bone under these conditions.[10] On this basis, calcium deficient diets may possibly result in the mobilization of lead from bone. Since relatively more calcium is required during pregnancy, this physiologic state might predispose to a similar mobilization of lead from bone.

Dr. Laurence Finberg, a member of the American Academy of Pediatrics, Committee on Environmental Hazards, is in general agreement with EPA's position and notes:[11]

> "I am quite sure that the pool of lead in the skeleton is not physiologically inert under all circumstances. A number of metabolic events which affect hydrogen ion or divalent ion metabolism will affect the lead pool. Since lead does not appear to have any necessary role in life processes, its presence may be looked upon as the biologic equivalent of a loose monkey wrench in the machinery."

REFERENCES FOR APPENDIX A - QUESTION 2

[1] "Comments of Ethyl Corporation on EPA's Proposed Lead Regulations," July 13, 1972.

[2] "Supplemental Statement by E. I. DuPont De Nemours & Company, Inc. Relative to EPA's Request for Additional Information on the Health Effects of Airborne Lead," July 12, 1972.

[3] Hammond, Paul B., Testimony submitted to EPA, June 8, 1972; and Goldsmith, John, Testimony submitted to EPA, July 11, 1972.

[4] "Comments of Ethyl Corporation on EPA's Proposed Lead Regulations," July 13, 1972, p. 11.

[5] Calandra, J. C., Testimony submitted to EPA, July 14, 1972.

[6] Barry, P. S. I. and Mossman, D. B., "Lead Concentrations in Human Tissues," Brit J Indust Med 27:339-351, 1970.

[7] Goldsmith, John, Testimony submitted to EPA, July 11, 1972.

[8] Hammond, Paul, Testimony submitted to EPA, June 8, 1972.

[9] Kahn, Ephraim, Testimony presented at L.A. Public Hearing, May 2-4, 1972, p. 163 of Hearing Record.

[10] Goyer, R. A., and Mahaffey, K.R., "Susceptibility to Lead Toxicity," Env. Health Pers;. Exp. Issue No. 2, Oct. 1972.

[11] Finberg, Laurence, Testimony submitted to EPA, June 20, 1972.

Question 3: The Environmental Protection Agency has relied upon the National Academy of Sciences' Report (Appendix C. p. 249, footnote "A") for estimates of daily respired air by an average adult in its own calculations in Table 7 of the "Health Hazards of Lead" paper. How accurate are these estimates of pulmonary physiology (a) that an adult male breathes 23 cubic meters of air per day, (b) that 30% of respired lead particles will be retained, and (c) that nearly 100% of retained lead particles will be absorbed? Is there additional evidence available in this area besides that which is cited in the NAS Report?

The wide variance of opinion received in reply to this question emphasizes the importance of considering the entire spectrum of biological response to lead that exists in the general population. The real world is simply not adequately described in terms of only the average response.

For example, estimates of daily ventilatory volume can be developed by extrapolating from metabolic oxygen requirements. On this basis a figure of 23 cubic meters per day as an average daily respiratory volume is too high. Even considering the wide spectrum of metabolic requirements within the population, a more reasonable estimate would be in the range of 13-20 cubic meters per day.[1] Dr. Goldsmith from the California State Department of Health also feels that the $23m^3$ figure is too high and that $15\text{-}20m^3$ is probably a better estimate.[2]

On the other hand, Dr. Paul Hammond, Chairman of the National Academy of Sciences Lead Panel which wrote "Airborne Lead in Persepective" considers the $23m^3$ figure to be acceptable. An International Council on Radiation

Protection report (still in draft stage) recommends 23 cubic meters as an appropriate estimate for average daily respiratory volume.[3] Dr. J. C. Calandra of Northwestern University and Medical Director of the Houston and NALCO Chemical Companies, however, critizes the basis upon which the ICRP arrived at this figure.[4]

A similar difference of opinion exists with regard to how much inhaled lead is ultimately retained in the lung. The National Academy of Sciences' Report on lead concluded that 30-37% was a reasonable figure for pulmonary lead deposition.[5] Dr. Paul Hammond believes this figure to be based upon sound scientific evidence;[6] Dr. Calandra, however, considers that available evidence supports a much lower figure.[7]

Disagreement also exists with respect to how extensively particles which have been retained in the lungs will actually be absorbed into the blood stream. A report of the National Academy of Sciences concluded that virtually all lead deposited in the lung is retained.[8] All this lead is probably ultimately absorbed into the blood stream.

A task group on lung dynamics of the ICRP considers a figure of 17-18% to more closely describe total blood absorption related to respiratory lead inhalation including factors for both particle retention and ultimate absorption of retained particles.[9] Other medical opinions consider this overall absorption figure to be even lower.[10] Dr. P. Lawther sums the situation up this way:

> "It would appear that little of the speculation on the uptake of lead inhaled in the form of aerosols in the exhaust from petrol

engines is based on solid and established fact... In the absence of such data, the only evidence relating to the effect of these exhaust gases is from epidemiological studies on man."[11]

Hence, EPA concludes that the entire adult population cannot be well characterized in terms of simple average physiologic parameters. A range of responses is a much more reliable reflection of the real world. On this basis, EPA feels that available evidence supports 13-23 cubic meters per day as the range for ventilatory volume in the general adult population and 17-30% as the overall range for absorption of lead particles in the lung, including factors for pulmonary deposition as well as absorption of these retained particles. Ultimately, however, epidemiologic studies provide the best evidence regarding effects of automotive lead emissions upon man.

REFERENCES FOR APPENDIX A - QUESTION 3

1 Earle, Richard, Testimony presented at EPA Public Hearing, Dallas, Texas, p. 277 of Hearing Record, April 28, 1972.

2 Goldsmith, John, Testimony submitted to EPA, July 11, 1972.

3 Hammond, Paul, Testimony submitted to EPA, June 8, 1972.

4 Calandra, J. C., Testimony submitted to EPA, July 14, 1972, pp. 8-11.

5 "Airborne Lead in Perspective" National Academy of Sciences; Washington, D. C., 1972 pp. 57 & 66.

6 Hammond, Paul, Testimony submitted to EPA, June 8, 1972.

7 Calandra, J. C., Testimony submitted to EPA, Table 1, May 19, 1972.

8 "Airborne Lead in Perspective, " National Academy of Sciences, Washington, D. C., 1972, p. 62.

9 Cole, Jerome, Testimony presented at Dallas Public Hearing, p. 455 of Hearing Record, April 28, 1972.

10 Calandra, J. C., Testimony submitted to EPA, July 14, 1972.

11 Lawther, P. et al "Airborne Lead and its Uptake by Inhalation," Lead in the Environment, Proceedings of a conference held at the Zoological Society of London, pp. 8-28, January 27, 1972.

Question 4: What is an appropriate safety factor for extrapolating industrial threshold limit values (TLV) to the general population? Should such an extrapolation to the general population even be permitted? The proposed TLV for lead is due to be revised to 150ug/m^3 for a 40 hour week. On a weekly basis this corresponds to breathing air continually at between 35-40 ug/m^3 of lead. If TLV's can be extrapolated to the general population, what would be an appropriate safety factor for this purpose so that all groups, including those most susceptible to lead, will be protected?

A review of the evidence presented does not support extrapolation of industrial threshold limit values to the general population. Such extrapolation would not assure protection of those groups within the general population who are most susceptible to lead.

When extrapolating from occupational to general population situations, the following factors must be considered: (1) the wider variation of age in the general population compared to the occupational population. Included in the general population are the very young and the very old, precisely those who are almost always most susceptible to pollution in any form; (2) the physical health of occupational workers. Those in occupations tend to be healthier and hence less susceptible to pollution than those in the general population. Occupational groups, for example, do not usually include those with chronic diseases; (3) occupational groups receive pre-employment medical examinations to exclude those highly susceptible individuals--people exposed in the general population are not so excluded; and (4) occupational groups receive periodic medical examinations while on the job to detect early disease changes. This opportunity is not always available to those exposed in the general population.

A British industrial health physician noted that in over 10 years of industrial lead health experience, he had performed some 50,000 medical examinations covering 8,000 man-years of risk.[1] This corresponds to an average of over 6 medical exams per man per year and reflects the potential gravity of the situation with respect to increased lead exposure. The general population is not afforded the opportunity for this close medical supervision to detect effects associated with excessive exposures to lead.

Numerous authorities on lead support EPA's position that extrapolation of industrial lead standards to the general population cannot be justified. For example, Drs. T.J. Chow, of the Scripps Institute of Oceanography and a consultant to the National Academy of Sciences' lead panel, and Claire Patterson, of the California Institute of Technology, feel that industrial threshold limit values are not based upon valid scientific data and eventually will be shown to be harmful.[2] Dr. John Goldsmith, an authority on general population as well as industrial lead exposure, believes that extrapolation of threshold limit values to the general population is inappropriate, especially for children.[3] Dr. Finberg, a member of the American Academy of Pediatrics Committee on Environmental Hazards has written:

> "I would think, emphatically, that it is not safe to extrapolate industrial threshold limit values to the general population. For example, the general population has in it pregnant women with their developing

fetuses. It also has young children and many sick people, including those with cerebral vascular disease."[4]

Two leading manufacturers of lead additives are also in general agreement that TLV's should not be extrapolated to the general population:

"There exists no factor that permits the simple extrapolation of TLV values (established for the industrial population) to the general population."[5]

"Because of the wide differences between industrial groups and the general population in exposure time, the types of populations involved, and the opportunity to monitor both health and exposure, it does not seem appropriate to use TLV's as a basis for developing air quality criteria."[6]

Finally, the Department of Health, Education and Welfare is in agreement with this position and concludes:

"We do not believe that the industrial threshold limit values (TLV) should be extrapolated to the general population."[7]

REFERENCES FOR APPENDIX A - QUESTION 4

[1]Williams, M. K., Testimony presented at EPA, Washington, D. C., Public Hearing, pp.342-355 of Hearing Record, April 12, 1972.

[2]Patterson, C. C., and Chow, T. J., Testimony submitted to EPA, July 6, 1972.

[3]Goldsmith, John, Testimony submitted to EPA, June 11, 1972.

[4]Finberg, L., Testimony submitted to EPA, June 20, 1972.

[5]Comments of Ethyl Corporation on EPA's Proposed Lead Regulations, p. 24, July 13, 1972.

[6]DuPont, comments submitted to EPA, p.4, July 12, 1972.

[7]Richardson, Elliot, Secretary, DHEW, letter to EPA Administrator William D. Ruckelshaus, August 11, 1972.

Questions 5 and 6:

5. In regard to the dustfall lead theory (p 139 of the NAS report): How much of a hazard is dustfall lead to children prone to pica? The Environmental Protection Agency's calculations indicate that continued ingestion of even small amounts of lead contaminated dust and dirt containing as much as 0.25-0.35 percent lead could theoretically result in dangerously elevated blood leads among children, or could contribute significantly to additional unnecessary lead burdens in children with other known lead exposures (such as lead paint). Will the Environmental Protection Agency's proposed 60-65 percent reduction of leaded automobile emissions significantly reduce the risk of this potential contamination?

6. Although lead paint has traditionally been considered the prime causal factor in childhood lead poisoning, how effective would reductions in other known environmental sources of lead exposure (such as dustfall) be in helping to reduce the risk of undue lead exposure among children also exposed to peeling lead paint? How clear is it that all lead poisoning in children is, in fact, caused only by lead paint? Since many years are required to solve the lead paint problem, would the risk of undue lead absorption and possible lead poisoning not be reduced by also decreasing airborne lead and consequently lead in dust?

EPA agrees that peeling lead based paint from dilapidated housing is a problem. This Agency has supported HEW in its effort to reduce the hazard associated with lead paint among future generations.[1] The main question is whether reductions in the use of gasoline lead additives will also help to decrease the risk of not only lead poisoning but also excessive lead exposure among children. Previous investigations of the lead paint poisoning problem have not always considered the magnitude of paint exposure with respect to other environmental lead sources which may also be contributing to abnormally elevated blood leads. Clearly blood lead is a function of all sources of lead exposure.

For example, a recent publication by Dr. Vincent Guinee, Chief of the Lead Poisoning Control Bureau of New York City[2] indicates that only 76.3% of children with lead poisoning lived in homes containing lead paint (defined as paint containing 1% or more lead). Although on reinspection of these homes additional peeling paint surfaces of 1% or more lead will probably be found, this by itself does not completely put this problem in true perspective. A considerable number of lead poisoning cases (for this purpose defined as blood leads of 60ug/100g or greater in children) are associated with lead paint environments containing lead paint predominantly at lead concentrations of 1% or below.

In testimony before the Senate Health Subcommittee,[3] Dr. Guinee presented the fact that of 418 samples of paint removed from broken surfaces in 25 apartments where a lead poisoning case resided, nearly two-thirds of these samples were found to contain lead paint at

concentrations of 1% or less. Over half were found to contain lead paint Of 0.5% or less. Although the breakdown was not available with respect to whether most of the samples containing markedly elevated paint concentrations were predominantly found in a selected number of homes, this is a reasonable possiblity. Accordingly, a considerable number of lead poisoning cases are probably associated with home paint environments containing predominantly paint of 1% lead or less. As evidence of its concern for the potential harm caused by lead paint at this concentration, the FDA has recently established 0.06% as what it believes to be a safe level of lead in paint.[4]

When this observation that excessive lead exposure is associated with paint of 1% lead or below is put into the context of other environmental lead exposures (including food, water, air, dust and dirt), the potential contribution of these additional sources to the problem cannot be ignored. Of these sources cited, lead content of food and water are not at the moment always easily controlled. However, exposures through lead in air and consequently lead falling out from the air to contaminate dust and dirt can be readily reduced. Further, exposures to airborne lead as well as lead in dust and dirt must be considered additional burdens to children already exposed to peeling lead based paint. These additional factors may in part explain why such large numbers of urban children have abnormally elevated blood leads.

One recent study designed to test the possible effect of these additional factors upon blood lead supports this point of view.[5] In

this investigation, 230 rural children and 272 children from an urban poverty area were examined for excessive lead exposure in the summer of 1971. Nearly all of the rural children with excessive lead body burdens lived in homes containing at least one surface with 1% lead paint or greater. However, this paint hazard could be found on accessible indoor and exterior surfaces in homes of only 60% of the urban children found to have excessive lead exposure. These findings are consistent with the position that excessive lead exposure of young children in urban areas is caused not only by lead in paint, but also by lead in air, in dust, and in dirt.

Evidence accumulated by the Environmental Protection Agency,[6] indicates that dustfall lead and concentrations of lead in dustfall generally decrease with increased distance from roadways. Levels of lead in dustfall of 0.3% were commonly found and levels of 0.5% or more were observed.[7] These findings suggest that vehicular lead emissions may be contributing significantly to high concentrations of lead in dustfall found in urban areas. Street dirt in urban areas has been documented to contain as much as 1% lead.[8,9]

Elevated lead concentrations have also been found in dust collected from indoor urban dwellings. Concentrations of lead in indoor dust in central city areas averaging 0.2% are reported.[10] Although lead from peeling paint may have contributed in part to lead contaminated dusts found in older homes collected as vacuum cleaner samples, this factor was not a reasonable explanation for the often high dust lead values found by this study in homes built in the 1950's and after. Airborne

lead was felt to be a significant source of this lead contamination.

Lead in dustfall and consequently lead in street dirt are probably related to the total quantity of automotive lead emissions, although no simple relationship has been demonstrated between the quantity of lead in the air and that in the dust.[11] This in part may be explained by settling of large lead particles deposited close to emission sources compared to the movement of smaller respirable lead particles much farther distances. Thus, it is difficult to relate specific levels of airborne lead directly to levels of lead in dust. However, the role of automotive lead emissions in contributing to urban lead fallout from the air has been demonstrated. For example, average soil lead levels collected in front yards of homes in urban areas are two to three times greater than soil lead concentrations in back yards which are located further away from roadways.[12] Automotive lead emissions are felt to contribute significantly to this difference.

Precise information with respect to the gastrointestinal absorption of lead contaminated dirt and dust relative to lead containing paint are not presently available. However, cases of clinical lead poisoning or excessive lead exposure among children known or suspected tp eat dirt, but without known excessive lead exposure directly from paint, have been reported.[13,14]

Demonstration of excessive lead exposures among children residing near a lead smelter in El Paso, Texas, further emphasizes the importance of the dustfall lead exposure mechanism. Information available to EPA[15] indicates that lead in paint could not have been a major factor in the

etiology of these abnormally elevated blood lead levels found among children residing near the lead smelter. Soil lead levels in the vicinity of this smelter averaged 0.4%-0.5% lead with a range of 0.15% to just over 1%. These average levels are not significantly different from levels of lead in dirt and soils reported in many urban streets and parks.

Though air lead levels were also significantly elevated near the smelter, most of the airborne lead (approximately 75%) was judged to be in the non-respirable range. The fact that a larger percentage of 1-5 year old children (89.2%) had abnormally elevated blood leads compared to 6-17 year olds (64.7%) suggests that combined exposure to airborne lead as well as ingestion of lead contaminated dusts was contributing significantly to this problem in the group most likely to ingest non-food items, the 1-5 year olds. Hence, levels of lead in street dirt of the magnitude found near the smelter must be viewed as a potential hazard for children with pica.

Many medical opinions submitted in testimony to EPA expressed concern for this potential hazard. Dr. Finberg of the American Academy of Pediatrics writes:

> "The dustfall lead theory seems quite reasonable and I believe that dustfall lead will represent a hazard to some children. In our own clinical experience, we have seen children who were dirt eaters with elevated blood leads and signs of toxicity where we could not incriminate painted surfaces in the household or other parts of their environment."[16]

Dr. Paul Hammond, Chairman of the NAS Lead Panel notes:

> "The lead panel of the NAS expressed no firm conviction as to the actual contribution of dustfall to the total lead input of young children. It definitely was concerned that street dust might in some cases be a major contributor to the total lead assimilation of some children who have been found to have blood lead concentrations of 40ug/100g. I do not think it is at all clear that all childhood lead poisoning can be attributed to paint. The relatively large number of city children with blood lead levels in excess of 40ug/100g may or may not be attributed to eating paint...street dust may well be a significant source."[17]

Dr. Anthony Mustalish of the New York City Department of Health generally agrees:

> "Although to my knowledge no cases of lead poisoning have been attributed to atmospheric lead alone, there is growing evidence that atmospheric lead contributes to this body burden and in inner city children this contribution may aggrevate an already compromised system."[18]

Finally, Elliot Richardson, Secretary of the Department of Health, Education and Welfare, has written in a recent letter to EPA Administrator William Ruckelshaus:

> "For those children with pica who eat dirt, the danger from exposure to lead containing dust and dirt is great."[19]

Although preliminary data failed to show any difference in blood lead levels among groups of children residing in an area of high soil lead content compared to those residing in a low soil lead area,[20] EPA does not believe that this study contradicts the potential importance of the dustfall lead exposure mechanism. Levels of lead in soil were not reported in a way to relate concentrations around the homes of individual children to their specific blood lead levels. Further, levels of lead were not measured in the housedust from homes in which the children actually lived and the data available to EPA were not broken down according to age so that blood leads in the youngest children could be compared. Finally, the lead exposure gradients between areas reported in this study were rather low with soil lead concentrations averaging approximately 0.1% in the high exposure area compared to 0.05% in the low exposure area. The high exposure area was thus characterized by soil lead levels considerably less than those reported to occur in street dirt, in soil and in dust inside homes from several American cities where these data have been collected.

In summary, EPA's position is rather straightforward. If paint containing less than 1% lead can contribute significantly to abnormally elevated blood leads and even to lead poisoning, then the potential contribution to this problem of dust and dirt containing similar quantities of lead cannot be ignored.

Although lead paint and lead in dust and dirt may not always be equally absorbed from the gastrointestinal tract, current levels of lead in street dust and dirt are considerably higher than that recommended as a safe level of lead in paint. Lead in dust and dirt would pose an additional hazard to a child aready exposed to peeling lead based paint. Reduction of airborne lead levels for purposes of decreasing the concentration of lead found in urban dust and dirt would thus be a prudent decision.

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Question 7: What is the consequence upon the environment in general of allowing large quantities of lead to be expelled into the atmosphere from motor vehicle exhausts? Does this environmental contamination pose any direct or indirect threat to man?

A concise answer addressing the problem of possible general environmental damage caused by lead is difficult to give. One area of concern that has recently become apparent involves the possible role played by leaded gasoline emissions in the contamination of shellfish. At the 1968 Shellfish Sanitation Workshop conducted by the U.S. Public Health Service, guidelines for trace metals were proposed. Maximum acceptable levels for trace metals in shellfish were established at 2 milligrams per kilogram (PPM) wet tissue weight for cadmium, lead, mercury, and chromium (combined). These proposed levels assumed an average serving of shellfish meats to be about 200 grams (7 ounces on a wet weight basis). Lead levels in shellfish from many areas have already been shown to exceed this proposed maximum acceptable level in soft clams, hard clams, surf clams, and oysters.[1] These data indicate that over 18% of oysters collected off the shores of two states exceeded the proposed maximum acceptable lead level. In the Raritan Bay, lead levels in shellfish were approximately 10 times higher than normal. This report concludes that contamination of edible shellfish by heavy metals may present a serious health hazard.

There is mounting evidence that lead from gasoline probably contributes to the lead content of shellfish. A study conducted on contract

to EPA indicates that hundreds to thousands of pounds of lead particulate matter fall out from the air to the ground, and are then regularly washed off the street during heavy rainstorms.[2] These street washings containing large amounts of lead eventually reach our waterways through the sewer systems, where they may potentially contaminate shellfish. Lead also enters these waterways via improper disposal of petroleum products containing lead additives directly into sanitary sewers.

Thus there appears to be a relatively rapid turnover of lead contaminated street dirt via periodic rainstorm washing of streets. Reductions in the use of lead as a gasoline additive can be expected to decrease the concentrations and total amounts of lead currently found in urban street dirt. Consequently, decreases in lead water pollution are also anticipated by this reduction.

REFERENCES FOR APPENDIX A - QUESTION 7

[1] "Metals in Shellfish with Particular Reference to Lead", prepared by the Northeast Water Supply Research Laboratory of the U. S. Environmental Protection Agency, p. 17.

[2] "Water Pollution Effects of Street Surface Contaminants," EPA Contract #14-12-921, prepared by URS Research Co., January 1972, Draft of Final Report.

APPENDIX B A SURVEY OF AIR AND POPULATION LEAD LEVELS IN SELECTED AMERICAN COMMUNITIES (SEVEN CITY LEAD STUDY)

In 1961, a special study to evaluate the problem of atmospheric lead in urban areas was begun.[1] Blood and urine samples from selected populations in the cities of Cincinnati, Los Angeles and Philadelphia were analyzed for their lead content and these data were compared to atmospheric lead levels to which these people were exposed.

This study (often referred to as the "Three City Study") concluded (1) that a definite difference in atmospheric lead levels existed between urban and rural areas with highest levels being recorded in the central city and (2) that increased blood lead levels were measured among people working or residing in urban areas compared to people in rural areas.

Seven years after completion of the Three City Study, a follow-up investigation was begun to determine whether atmospheric lead levels had changed significantly with time and if blood lead continued to be elevated in regions of high atmospheric lead levels. This work was carried out by the Kettering Laboratory of the University of Cincinnati and was supported by the American Petroleum Institute, the International Lead Zinc Research Organization and the Environmental Protection Agency. Sampling sites used in 1961-1962 were reestablished and additional sampling sites were set up in the original three cities as well as in five new cities including Los Alamos, Chicago, Houston, New York and Washington, D. C. A comprehensive preliminary report of these data (referred to as the "Seven City Study") was presented at the EPA Los Angeles Public Hearing on May 3, 1972.

The following analysis represents initial comments by Agency staff regarding the results of this study. Much testimony was presented during the public hearings and subsequent comment periods that the Seven City Study failed to demonstrate any significant relationship between air lead exposure and blood lead level. While a significant correlation between blood lead and air lead was not found when all geographical areas were compared, within each area blood lead levels were consistently elevated among urban residents as compared to those residing in the surburbs.

EPA does not believe that failure to demonstrate a significant correlation between blood lead and air lead in this study proves that no relationship exists between blood lead and air lead. A significant correlation would never be expected to result from this particular investigation in part because a wide enough air lead exposure range was not examined. The observed increases in blood leads among urban residents compared to suburban residents supports the probable contribution of airborne lead in establishing this difference.

When discussing these results one key factor must be kept carefully in mind. That is, although food and water contribute more to lead absorption than air at low air lead exposures, if lead intake from food and water can be kept reasonably constant, then differences in blood leads can be more easily detected. Variation of dietary lead intake tends to be greater between geographic areas than within geographic areas. Hence urban-suburban comparisons are less confounded

by differences in dietary lead intake, thus increasing the probability of detecting differences in blood lead due to variable air lead exposures. This in part accounts for the failure to obtain a significant correlation between areas while within areas consistent effects of increased air lead exposures upon blood lead were found.

Since lead intake from food and water among areas in this study, as measured by fecal lead excretion, varied considerably, correlations between air lead and blood lead would not be expected to be very significant, especially at small air lead gradients. Although differences in fecal lead excretion were not always in a direction that could explain specific area inconsistencies, the very existence of this factor in part explains why a statistically significant correlation was not observed.

Another important consideration related to data analysis from the Seven City Study is that many thousands of individual data points were reduced into approximately one dozen simple average blood lead levels, which were then correlated with air lead exposures averaged over time. This averaging procedure resulted in the loss of a considerable amount of information present in the original data. Consequently, these results were reduced to a series of averages which did not adequately describe the real world from which they originated. This is especially important when one considers that, although average blood leads in a given group may be well within normal limits, selected individuals within that group may have blood leads that are elevated above normal. Any averaging that is done during analysis will tend to obscure the

presence of abnormally elevated blood leads in the original data. Further, had all of the original data points been plotted instead of just the averages, a statistically significant correlation between air lead and blood lead could possibly have been obtained. Comparing blood lead determinations to yearly average air lead exposures derived from monthly measurements which varied considerably is also not appropriate from a physiologic standpoint since blood lead is most likely a function of air lead exposures taken 2-3 months before blood leads are sampled.

Important conclusions regarding the study become more apparent when additional methods of data analysis are employed. For example, the hypothesis that urban and suburban exposure categories are alike with respect to observed blood lead levels can be tested by considering how many individual blood leads are above a given blood lead value by using a Chi squared analysis. This frequently used and commonly accepted statistical technique will readily demonstrate any differences in blood leads between groups as this relates to residence and consequent exposure to differing quantities of airborne lead.

Three urban-suburban comparisons can thus be established. In each instance the number of people with blood leads above 21.8 micrograms per hundred grams in urban versus suburban areas are compared. There is nothing magic about the choice of 21.8 as a cutoff in this test. This cutoff was chosen because it was well toward the middle of each distribution but slightly toward the side of higher blood lead levels. Consequently, any trend toward elevated blood leads in one group compared to the other becomes more apparent.

Table B-1 - Philadelphia

Urban - Suburban Blood Lead Comparison

Number of People

	Urban	Suburban	
Blood lead less than 21.8	76	105	181
Blood lead greater than or equal to 21.8	60	45	105
	136	150	286

$X^2=6.12$ (1df)

.01 p .02* (*Statistically significant)

	Average Air Lead (ug/m^3 -geometric mean)	Average Blood Lead (ug/100g -geometric mean)
Philadelphia Urban	1.67	20.5
Philadelphia Suburban	1.15	18.0

	% Blood leads 29ug/100g & Above	% Blood Leads 40ug/100g & Above
Philadelphia Urban	11.0	1.5
Philadelphia Suburban	4.7	0

Table B-2 - Chicago

Urban - Suburban Blood Lead Comparison

Number of People

	Urban	Suburban	
Blood lead < 21.8	118	200	318
Blood lead ≥ 21.8	29	8	37
	147	208	355

$X^2 = 23.3$ (1df)

$p < 0.01$* (*Statistically significant)

	Average Air Lead (ug/m^3 -geometric mean)	Average Blood Lead (ug/100g -geometric mean)
Chicago Urban	1.76	17.6
Chicago Suburban	1.18	13.9

	%Blood Leads 29ug/100g & Above	%Blood Leads 40ug/100g & Above
Chicago Urban	3.4	0.7
Chicago Suburban	0.5	0

Table B-3 - New York

Urban - Suburban Blood Lead Comparison

Number of People

	Urban	Suburban	
Blood lead < 21.8	119	180	299
Blood lead ≥ 21.8	21	18	39
	140	198	338

$X^2 = 2.81$

$0.05 < p < 0.10$

	Average Air Lead (ug/m^3 -geometric mean)	Average Blood Lead (ug/100g -geometric mean)
New York Urban	2.08	16.6
New York Suburban	1.13	15.3

	% Blood Leads 29ug/100g and Above	% Blood Leads 40ug/100g and Above
New York Urban	1.4	0
New York Suburban	0.5	0

In two of the three comparisons (Philadelphia and Chicago) statistically significant differences in blood lead patterns between groups are present in urban as compared to suburban residents. In the third comparison, New York, although statistical significance was not achieved at the 5% level, the results are very close to being significant.

In each of these comparisons the urban residents as a group had greater numbers (and percentages) of people with blood leads greater or equal to 21.8ug/100g than those in the suburban groups. Thus a statistically significant trend toward higher blood lead levels among urban residents exposed to higher levels of airborne lead is evident.

A second important observation is that only in urban areas are more individual blood lead levels found to be near or above the level indicative of excessive lead exposure in adults (blood lead of 40ug/100g or above).

Further, the Seven City Study concluded that men have higher blood lead levels than women at comparable air lead exposures. (Approximately 2ug/100g greater on the average.) Thus, had men been studied instead of women, a greater percentage would have been found to have abnormally elevated blood lead levels (40ug/100g and above). If these findings can be extrapolated to the general urban population, one must conclude that several million adults are probably excessively exposed to lead as a result of residence in urban environments where airborne lead exposures tend to be elevated.

As a result, the Seven City Study suggests the possibility that despite measurements showing that average blood leads in the United States are well within normal limits, there apparently are large numbers of urban adult Americans who are presently excessively exposed to lead.

REFERENCES TO APPENDIX B

[1] Survey of Lead in the Atmosphere of Three Urban Communities, Public Health Service Publication , No. 999-AP-12.