DUPONT

Petroleum Chemicals

DATE 10/26/72

TO: Alex Azar

Would appreciate your comments on this EPA "position paper."

Welcome back!

Lee

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N40688

Air Pollution - Regulations 1848

Preliminary Draft "EPA's Position on the Health Effects of Lead" 9-22-73

United States Senate

COMMITTEE ON COMMERCE
WASHINGTON, D.C. 20510

October 20, 1972

The President
The White House
Washington, D.C.

Dear Mr. President:

On June 19th of this year, 49 members of Congress wrote to Administrator Ruckelshaus of the Environmental Protection Agency urging the removal of all lead from gasoline by 1977 under the authority of the Clean Air Act. The letter was in response to the Administrator's proposed regulations of February 23 which called for only a two-thirds reduction of the lead content by 1977.

Our judgment was based on two primary considerations: (1) that the dangers posed by lead to human health had not been adequately considered in the proposed regulations; and (2) that a more severe schedule of lead reduction was economically feasible. At that time, extremely high levels of lead had been found in the dirt of a number of cities in this country. Levels ranged from 600 up to more than 5,000 parts of lead per million parts of dirt. Our conclusion was that even if lead in dirt were reduced by two-thirds (which testimony suggested would be a likely consequence of a two-thirds reduction of lead in gasoline) substantial quantities of lead -- enough to pose grave dangers to children -- would remain. Dr. Jane Lin-Fu, a leading pediatrician, testified that it would not be unusual for ordinary children to consume enough dirt (about 1/8 teaspoon) at 500 parts per million lead to exceed the "maximum daily permissible dose", the dose above which actual poisoning could occur. Children with pica could be expected to consume considerably more. The danger is compounded by the fact that these same children might also be eating leaded paint.

Further evidence indicated that even at an ambient level of 1.5 micrograms per cubic meter of air -- which is a level lower than that expected to be achieved in many areas under the proposed regulations -- lead levels could still reach nearly 5,000 parts per million. At this level, only about 1/40 of a teaspoon of dirt would need to be consumed for a child to exceed the maximum daily dose.

The record of the hearings of the Subcommittee on the Environment indicated that less than a 2¢ per gallon increase in the cost of gasoline would be

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necessary to achieve total elimination of lead by 1977. Those costs seemed completely reasonable when balanced against the dangers posed, In addition, the letter suggested several means of preserving and enhancing the viability of independent competition in the event that a stiff schedule of lead removal were adopted.

Since the June 19th letter, additional information has been gathered which argues even more forcibly for a stiff schedule of lead reduction. In a September 22 draft document distributed by the Environmental Protection Agency to other Federal agencies, scientists within the Agency summarized the latest information with respect to the health hazards of lead. Some of the major points in the draft are as follows:

1. Recent data on excessive lead exposure of children residing near a lead smelter in El Paso, Texas, further emphasized the extent to which lead in dirt can contribute to elevated blood lead levels in children. Approximately 90% of the 1-5 year old children sampled who live near the smelter had blood lead levels of over 40 micrograms per 100 grams -- a level above which actual lead poisoning could occur. The information available to EPA indicates that lead paint was not a significant causal factor of the high blood levels and most of the airborne lead was judged to be nonrespirable. Soil lead levels in the vicinity of the smelter averaged between 4,000 and 5,000 parts of lead per million parts of soil with a range of 1500 to just over 10,000. The levels of lead found in the soil were not significantly different from those now found in many urban streets and parks. A larger percentage of the 1-5 year old children living near the smelter had abnormally elevated blood lead levels compared to a 6-17 year old group residing in the same area. This suggests that ingestion of lead-contaminated dust contributes significantly to excessive exposures in the group more likely to ingest non food items, the 1-5 year olds. The EPA draft concludes, "... levels of lead in street dirt of this magnitude found near the smelter must be viewed as a definite hazard for children with pica."

2. Other recent studies suggest that blood lead levels of 30 micrograms per 100 grams of blood in the newborn and the fetus should be considered abnormally elevated and unsafe. Further, studies in Boston and New York have demonstrated that umbilical cord blood levels of 30 micrograms per 100 grams or above were present only in babies born to urban mothers. Mothers of these children are not suspected of eating paint, dirt, or dust. Increased exposures to airborne lead in these urban environments must therefore be considered the primary factor contributing to this problem. While the number of babies tested was small, the studies are highly suggestive that airborne lead is indeed causing high blood lead levels in newborn infants. To quote the EPA draft, "... these studies indicate the probable existence of abnormally elevated umbilical cord blood lead levels among babies born in urban environments." The paper continued, "If this trend is 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."

Other data submitted to EPA by the Center for Science in the Public Interest has revealed that 20 of about 200 samples of dirt taken in Washington, D.C. averaged more than 6,000 parts of lead per million parts of dirt. 12,820 parts per million were found at the intersection of T and 13th Streets, N.W. and 9,300 parts per million at the intersection of Connecticut Avenue and Ordway Street, N.W. Even if a two-thirds reduction in the lead content of soil in Washington takes place under the EPA regulations, these 20 sites could still average better than 2,000 parts of lead per million parts of dirt. At this level, the consumption by a child of only 1/32 of a teaspoon of dirt per day would be enough to exceed the maximum daily permissible dose.

The evidence against lead in gasoline thus continues to build. As the authors of the EPA draft urged, "Every effort must be made to reduce all preventable excessive lead exposures to the fullest extent possible, especially in light of the particular susceptibility to lead of children and the newborn." In light of the evidence cited the authors conclude: "EPA's previous lead in gasoline regulation calling for a 60-65% reduction to achieve a 2 microgram per cubic meter air lead goal must be considered inadequate to protect the public health." We strongly concur in this assessment.

As the original proposed regulations were issued on February 23, it was reasonable to expect that final regulations would now be in effect. Yet, despite our expectations the final regulations have yet to be issued. It is our understanding that EPA's recommendations are now before the Office of Management and Budget. While constructive debate on the content of the regulations must be provided, further delay in the issuance can only work to the detriment of the urban poor and others who may now be poisoned by lead in gasoline. We urge you to use your influence to speed the regulations through the review process, and to advise that the regulations provide for the complete removal of lead in gasoline by 1977.

Respectfully yours,

PHILIP A. HART

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THIS DOCUMENT IS A PRELIMINARY DRAFT. It has not been formally released by EPA and should not at this stage be construed to represent EPA policy. It is being circulated for comment on its technical accuracy and policy implications.

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I. INTRODUCTION

This document is a preliminary draft. It has not been formally released by EPA and should not at this stage be construed to represent Agency policy. It is being circulated for comment on its technical accuracy and policy implications.

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 for controlling 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 re-evaluation of existing data. This paper will be used to help form the basis for an environmental policy relative to lead in gasoline that is fully protective of public health.

REFERENCES FOR SECTION I - INTRODUCTION

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

[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.

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 often 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, they are often difficult to recognize as being specifically due to lead. Hence, lead may be a significant, but unrecognized, contributing factor in many clinical situations.

Children with abnormally elevated blood leads are labeled as "asymptomatic" lead poisoning cases if no symptoms or signs of lead intoxication are evident. Moreover, subtle indications of lead poisoning are difficult to detect. Perceptiveness of both parents and physicians is therefore an important factor influencing whether symptomatic lead poisoning cases are identified. For example, children with no initial symptoms of adverse lead effects (asymptomatic cases) have been found, on follow follow-up medical examinations, to be mentally retarded.

One large survey involving 425 children with lead poisoning indicated that 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 brain 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 unclear from the article. Further, of 58 children treated for asymptomatic lead poisoning, 5 or nearly 10% were found during follow-up studies to be mentally retarded. One, however, can never be totally positive that mental retardation was not present before these children were poisoned by lead.

In another study[4] eleven children who had been treated for mild lead poisoning but apparently cured and hence considered asymptomatic were reexamined 5-10 years later. Mental deterioration was not obvious and physical and laboratory tests in general did not reveal striking abnormalities. However, specialized tests of visual motor performance indicated subtle brain damage in the majority of cases.

Children originally considered to be cases of asymptomatic lead poisoning with blood leads of 50ug/100g and above have demonstrated improvements in behavior and language ability following treatment with drugs that remove lead from their bodies.[5] Although these are subjective findings they do suggest that central nervous system damage was present but previously undetected. Findings such as these cause speculation,

in the opinion of EPA, that many children considered to be asymptomatic with blood leads in the 40-50ug/100g range may, in fact, 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, 1956.

2 Chisolm, J. Julian: "Chronic Lead Intoxication in Children," Develop. Med Child Neurol 7:529-536, 1965.

3 Perlston, M. A. and Attala, R.: "Neurologic Secuelae of Plumbism in Children," Clin Ped 5:292-298, 1966.

4 Thurston, D. L.; Middlekamp, J. N., and Mason, E.: "The Late Effects of Lead Poisoning," J Ped 47:413-423, 1955.

5 Sachs, H. K.; Blanksma, L. A.; Murray, E. F., and O'Connell, J. G.: "Ambulatory Treatment of Lead Poisoning: Report of 1,155 Cases," Ped 46: 386-396, 1970.

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.[1,4] 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] The National Academy of Sciences had concluded that, at blood lead levels of 40ug/100g and above, inhibition of this enzyme is physiologically significant.[3] ALAD inhibition has been demonstrated to occur in man at blood leads in the low 20ug/100g range.[4] However, measurable increases in urinary ALA resulting from ALAD inhibition are generally not found until blood lead levels have reached 30-40ug/100g. Thus some degree of ALAD enzyme reserve probably exists, possibly offering protection against adverse effects at blood leads below 40ug/100g in most individuals.

Inhibition of ALAD by lead may not necessarily be confined only to red blood cells. In laboratory animals this effect has been demonstrated to occur in brain, kidney, liver and bone marrow.[5] Whether such widespread inhibition of ALAD by lead also exists in man is at present unknown.

Lead is also an inhibitor of other biochemical pathways besides those involved in making hemoglobin. Mitochondria isolated from rat renal tubules demonstrate impaired oxidative phosphorylation and defective membrane structures when exposed to lead.[6] Lead is also known to inhibit the dithiol enzyme lipoamide dephydrogenase which is involved in intermediary metabolism. This inhibition may be representative of an adverse effect of lead upon other similar dithiol enzymes in the body. The National Academy of Sciences had concluded that studies such as these "lend support to the concept that very small concentrations of lead can inhibit critical enzyme systems."[7]

Inhibition 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 would 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. In addition, lead has been demonstrated to interact with ribonucleic acid (RNA) in both test tubes and living organisms thereby inhibiting incorporation of amino acids into transfer RNA.[8] Since RNA is involved in the delivery of information from the gene to the rest of the cell, this indicates that lead may interfere with biochemical processes at the gene level.

This hypothesis is consistent with chromosomal abnormalities in men that have recently been associated with excessive lead exposures. For

example Muro and Goyer first reported evidence of experimental chromosomal damage caused by lead in 1969.[9] In this study chromosomes derived from leukocyte cultures of mice fed 1% lead acetate in their diets demonstrated increased gap-break aberrations. The authors 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[10] and in the lymphocytes of workers occupationally exposed to lead.[11] In a community located near a lead smelter, chromosomal abnormalities were found in 13 of 15 randomly selected exposed individuals.[12]

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

1 Hernberg, S.; Mikkanen, J.; Mellen, G., et al: "Delta Aminolevulinic Acid Dehydrase As a Measure of Lead Exposure," Arch Environ Health 21:140-145, 1970.

2 Ibid.

3 "Airborne Lead in Perspective", A report prepared by the Committee on Biological Effects of Atmospheric Pollutants of the Division of Medical Sciences, National Research Council, National Academy of Sciences, Washington, D. C., 1972.

4 Kennedy, Gerald, Testimony presented at Dallas Public Hearing, April 28, 1972, Hearing Record, p.340.

5 Millar, J. A.; Battistini, V.; Cumming, R. L. C., et al: "Lead and Delta Aminolevulinic Acid Dehydratase Levels in Mentally Retarded Children and Lead-Poisoned Sucking Rats," Lancet 2:695-698, 1970.

6 "Airborne Lead in Perspective", op.cit., p.165.

7 "Airborne Lead in Perspective", op.cit., p.165.

8 "Airborne Lead in Perspective", op.cit., p.166.

9 Muro, L. A. and Goyer, R. A.: "Chromosome Damage in Experimental Lead Poisoning," Arch of Pathology 87:660-663, 1969.

10 "Airborne Lead in Perspective", op.cit., p.166.

11 Forni, A. and Secchi, G.: "Chromosomal Changes in Pre-Clinical and Clinical Lead Poisoning and Correlation with Biochemical Findings," to be presented at the International Symposium on Environmental Health Aspects of Lead, Amsterdam, October 2-6, 1972.

12 Graovac-Leposavic, L.: Djarie, D.; Valjarevic, V.; Senicar, H.; Senicar, L.; Milic, S.; and Delic, V.: "Environmental Lead Contamination of Meza Valley - Study on Lead Exposure of Population," to be presented at the International Symposium on Environmental Health Aspects of Lead, Amsterdam, October 2-6, 1972.

IV. WHAT IS A SAFE BLOOD LEAD LEVEL?

The risk of developing lead poisoning increases with increments in blood lead. However, a single safe blood lead level protective of all high risk groups in the general population is, at present, difficult to define. A wide range of individual responsiveness to a given blood lead level exists in both children and adults.[1] Further, blood lead levels considered safe for adults are probably not safe for children. Available data suggest that children are more susceptible to lead than are adults. For example, clinical symptoms of lead intoxication generally occur at lower blood lead levels in children compared to adults.

Younger children may be even more susceptible to lead.[2] The majority of lead poisoning cases among children are known to occur in the 1-3 year old age category. This in part may reflect a greater susceptibility to lead among this group in addition to the high prevalence of pica at this age.

Hence, on the basis that young children are probably more susceptible to lead than older cildren and adults, the newborn and the fetus must be considered especially vulnerable to lead. Possible exposure of the developing central nervous system in utero to lead, a known neurotoxic agent, argues for a reasonable safety factor between what is considered an acceptable lead exposure among the fetus and newborn compared to older children and adults.

Symptoms of mild lead intoxication (abdominal pain) among adults employed in the lead industries with blood leads in the 40-50ug/100g range have been reported.[3] Significant physiologic disturbances in ALAD enzyme activity also occur at these blood lead levels. These factors support the establishment of 40ug/100g as a level indicative of excessive lead exposure for adults. Two leading manufacturers of lead additives, the Ethyl Corporation and DuPont[4,5] are in general agreement with acceptance of 40ug/100g as the upper normal blood lead level in adults.

NOT THE SAME

Cases of lead poisoning have been documented in children with blood lead levels in the 40-50ug/100g range.[6,7,8,9] Since symptoms of mild lead poisoning in children are often undiagnosed, the possibility that clinical disease is present but undetected at blood lead levels even under 40ug/100g cannot be ruled out. EPA's position is that a reasonable safety factor should be established between blood leads at which poisoning is reported and those that are considered normal. These above observations support establishment of the upper normal blood lead level in children at below 40ug/100g. Available data do not support a more precise definition of an upper normal blood lead level in children.

The lead poisoning problem in children has recently been reevaluated by medical authorities including the Surgeon General. This has resulted in revision of the blood lead level generally considered to be evidence of excessive lead exposure in children downward to 40ug/100g.[10,11,12]

This position is not inconsistent with that of EPA. While blood leads of 40ug/100g are certainly indicative of excessive lead exposure in children, the upper acceptable normal blood lead for all children should be established below 40ug/100g.

In the newborn and the fetus umbilical cord blood lead levels of even 30ug/100g should be considered abnormally elevated. This reflects probable vulnerability of the developing central nervous system to lead, an established neurotoxic agent. Umbilical cord blood leads of 30ug/100g or above are too close to levels known to produce clinical symptoms in children to be considered safe.

In summary, best available information indicates that to provide adequate margins of safety, upper normal blood lead guidelines for the general population could be best defined as shown in Table IV-1.

For the fetus and the newborn the upper normal blood lead limit should be under 30ug/100g; for children it should be below 40ug/100g. In adults a blood lead level of 40ug/100g or above must be considered abnormal and evidence of excessive lead exposure. Available scientific information does not presently support more precise refinements of these guidelines.

TABLE IV-1

Blood Lead Guidelines in the General Population

Group	Upper Normal Blood Lead Level (ug/100g)
Fetus and Newborn	less than 30
Children	less than 40
Adults	40

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

3
Beritic, T.: "Lead Concentration Found in Human Blood in Association with Lead Colic," Arch Env Health 23:289-291, 1971.

4
Supplement, comment of Ethyl Corporation on EPA's Proposed Lead Regulation, Ethyl Corporation, July 13, 1972, p.12.

5
Diggs, D. E., letter to the Deputy Assistant Administrator for Air Programs, EPA, in "Supplemental Statement by E. I. DuPont De Nemours and Company, Incorporated, Relative to EPA Request for Additional Information on the Health Effects of Airborne Lead," July 12, 1972.

6
Jacobziner, H.: "Lead Poisoning in Childhood: Epidemiology, Manifestation, and Prevention," Clin Ped 5:277-286, 1966.

7
Moncrieff, A.; Koumides, O.; Clayton, B., et al: "Lead Poisoning in Children," Arch Dis Child, 39:1-13, 1964.

8
Freeman, R.: "Chronic Lead Poisoning in Children: A Review of 90-Children Diagnosed in Sydney, 1948-1967, Australian Pediat. J., 5:27-35, 1969.

9
Berman, E.: "The Biochemistry of Lead: Review of the Body Distribution and Methods of Lead Determination," Clin Ped, 5:287-291, 1966.

10
"Medical Aspects of Childhood Poisoning," HSMHA Health Report; 86:140-143, 1971.

11
Lin-Fu, Jane, S.: "Undue Absorption of Lead Among Children - A New Look at an Old Problem," New Eng J Med, 286:702-710, 1972.

12
Chisolm, J. Julian, Testimony submitted to Environmental Protection Agency, July 26, 1972.

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, (ingestion of non-food objects) are generally exposed to lead not only via air, food, and water, but also via lead contaminated paint, dirt, and dust. Lead contaminated dirt and dust are as readily available for children to ingest as is peeling lead based paint. Fall-out of lead from the air is a major 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 expecially in urban areas are related to 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]

Effects of lead exposure upon blood leads in the general population have been documented for both air lead and dustfall lead exposure routes. Blood lead levels in children as well as adults are affected by low airborne lead exposures under $2ug/m^3$. For example, urban adult female residents exposed to air leads in the range $1.5\text{-}2.0ug/m^3$ have higher blood leads than suburban residents exposed to air lead levels between 1 and $1.25ug/m^3$.[3]

Elevated blood leads have also been found among children attending school in higher air lead areas compared to those in lower air lead areas.[4] This effect similarly was observed at air lead exposures under 2ug/m^3.

Additional investigations support the observation that greater air lead exposures in urban environments are associated with elevated blood lead levels.[5,6] Further, persons living near highways generally tend to have higher blood lead levels than those living away from highways, again implicating lead in gasoline as a causal factor.[7,8]

Long term (20 weeks) carefully controlled chamber exposures to airborne lead at approximately 3ug/m^3 in human volunteers confirms that blood lead increments can be expected at these air lead levels.[9] Analysis of these results suggests that blood lead levels did not plateau after 20 weeks exposure and hence would be expected to increase even more had air lead exposures been continued. This study generally supports epidemiologic findings showing effects of air lead exposures below 2ug/m^3 upon blood lead levels in the general population.

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Called Coulston today + he stated that these statements are not true.

A careful analysis of existing data indicates that environmental exposures to lead contaminated dirt and dust can contribute significantly to excessive lead exposure in children.[10] 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%).[11,12,13] These surveys of urban environments in Boston and Washington demonstrate elevated concentrations of lead in street dirt commonly 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%.[14] 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.

As indicated above, lead contaminated dirt and dust are as readily available for children to ingest as is peeling lead based paint. The prevalence of pica (ingestion of non-food items) among children is high, perhaps exceeding 50%.[15] While large concentrations of lead in individual paint chips are especially hazardous, cases of lead poisoning in children are often associated with paint surfaces containing less than 1% lead.[16,17] Data from the City of New York Lead Poisoning Control Bureau demonstrate that among children with abnormally elevated blood leads between 35 and 44ug/100g, only half can be associated with peeling paint of 1% lead or greater.[18] Further, nearly 20% of cases in this blood lead category lived in homes in which peeling paint was not identified.

Though ingestion of paint containing less than 1% lead is still a definite hazard, continued ingestion of lead contaminated dirt and dust

of below 1% lead would also contribute to dangerously elevated blood lead levels. Rats fed samples of lead contaminated dirt collected from the Queens Midtown Tunnel in New York, for example, demonstrate significantly increased blood lead levels compared to controls not fed this material.[19] Combined ingestion of lead based paint and lead in dirt and dust is thus probably responsible for the large numbers of urban children found to have abnormally elevated blood leads.

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 40-60mg/100g."[20]

One recent study specifically designed to test the possible effect of lead contaminated dirt and dust as well as airborne lead upon blood leads in children supports this point of view. In this investigation, 230 rural children and 272 children from an urban poverty area were tested for excessive lead exposure in the summer of 1971.[21] Nearly all of the rural children (18 out of 19) 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 urban children found to have excessive lead exposure. Further, one quarter of the urban children tested had abnormally elevated blood leads compared to less than 10% of

the suburban children. Hence, young children living in urban areas are more excessively exposed to lead than those residing in rural areas. These findings are consistent with the position 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 health 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."[22]

Demonstration of excessive lead exposures among children residing near a lead smelter in El Paso, Texas, further emphasizes the potential importance of the dustfall lead exposure mechanism.[23] 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.[24] 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%, which 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, most of the airborne lead (approximately 75%) was judged to be in the nonrespirable range. A larger percentage (89.2%) of 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 significantly 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 must be viewed as a definite hazard for children with pica. A more in depth study supervised by HEW, with EPA participation, is currently underway to further clarify the etiology and extent of this problem.

In summary, blood lead must be viewed as a function of all exposure routes. Available evidence indicates that living in urban environments where air lead exposures are elevated, is associated with higher blood lead levels. Even air lead levels below $2ug/m^3$ can contribute to elevations in blood leads among both children and adults. Especially for children who are known to ingest non-food items, lead falling out from the air and in turn contaminating dirt and dust is a particular 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.

REFERENCES FOR SECTION V

SOURCES OF LEAD EXPOSURE AMONG THE GENERAL POPULATION

1 Office of Air Programs Data File of Nationwide Emissions, 1970, Environmental Protection Agency, Research Triangle Park, North Carolina, July, 1972, Table G-2.

2 Pinkerton, C., Hammer, D. I., Hinners, T. A., Kent, J. L., Hasselblad, V., Lagerwerff, J. V., and Ferrand, E. S., "Trace Metals in Urban Soils and Housedust," paper to be presented to Environmental Section, APHA Centennial Convention, Atlantic City, New Jersey, Nov. 16, 1972.

3 Tepper, Lloyd. "A Survey of Air and Population Lead Levels in Selected American Communities" (Seven City Study), presented at Los Angeles Public Hearings, May 3, 1972, plus additional data submitted to EPA.

4 McIntire, M. and Angle, Carol R. "Air Lead: Relation to Lead in Blood of Black School Children Deficient in Glucose 6--Phosphate Dehydrogenase," Science, Vol. 15, August 1972, pp. 520-522.

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

6 Hofreuter, D. H., et al. "The Public Health Significance of Atmospheric Lead," Arch. Environmental Health, Vol. 3, November 1961, pp. 82-88.

7 Thomas, H. V., et al. "Blood lead of Persons Living Near Freeways," Arch. Environmental Health, Vol. 15 (1967), pp. 695-702.

8 Daines, R. H., et al. "Lead in Air Inside and Outside Homes and in the Blood of Women as Influenced by Proximity to a Well Traveled Roadway," data submitted to EPA, July 25, 1972. (To be published in Ind. Med. and Surg. Journal, October 1972.

9 Coulston, Frederick; Goldberg, Leon; Griffin, Travis; Johnson, Richard J., and Knelson, John H. "Kinetics of Respiratory Lead Uptake in Humans," paper to be presented at the International Symposium on Environmental Health Aspects of Lead, Amsterdam, Netherlands, October 2-6, 1972.

10 Shy, C., Hammer, D., Goldberg, H., Newill, V. and Nelson, W., "Health Hazards of Environmental Lead," DHER In-House Technical Report, EPA, Research Triangle Park, N. C., March 1971.

11 Krueger, H., Boston, Mass. Testimony submitted to EPA July 10, 1972.

12 Fritsh, Albert and Prival, Michael, Center for Science in the Public Interest. Testimony submitted to EPA.

13 Duval, Merlin, Asst. Sec. Health and Scientific Affairs, DHEW. Testimony before the Senate Subcommittee on Health, March 10,1972.

14 Krueger, op.cit.

15 Lin-Fu, Jane. "Lead Poisoning in Children," Children's Bureau Publication No. 452-1967, DHEW (1967).

16 Guinee, Vincent. "Lead Poisoning," American Journal of Medicine, Vol. 52 (1972), pp. 283-288.

17 Guinee, Vincent. Testimony before Subcommittee on Health of the Committee on Labor and Public Welfare, United States Senate, March 9, 1972. (Position of New York City on the Control of Childhood Lead Paint Poisoning).

18 NYC Bureau of Lead Poisoning Control data submitted to EPA, August 31, 1972 and September 12, 1972.

19 Stara, J. F., Moore, W. and Bridbord, K., "Blood and Tissue Levels in Rats Fed Dust Containing Environmentally Bound Lead," report of preliminary data from Environmental Toxicology Division, EPA, Cincinnati, Ohio.

20 NAS Report p. 139.

21 LePow, Martha. Testimony before Senate Committee on Commerce, Subcommittee on the Environment, Washington, D. C. May 8, 1972

22 Richardson, Elliot, Secretary, DHEW. Letter to EPA Administrator William D. Ruckelshaus, August 11, 1972.

23 Chisolm, J. Julian. Information available to EPA July 1, 1972 and information submitted to EPA July 17 and 26, 1972.

24 Ibid.

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. On this basis, although average blood lead levels among urban populations are well within normal limits, considerable numbers of individual urban residents have abnormally elevated blood lead levels exceeding 40ug/100g.

These abnormal blood lead elevations have been documented among adults and children, as well as the newborn. They consistently are associated with residence in urban areas where air lead levels are greatest. Since over 90% of airborne lead is due to lead automotive emissions,[1] these emissions must be contributing significantly to this problem. Table VI-1 briefly summarizes the extent of excessive lead exposures among urban adults.

Extrapolation from the evidence in this Table 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 from gasoline.

Within each city in Table VI-1 the percentage 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 varying amounts from dietary lead sources.[2] Table VI-2 summarizes existing data which demonstrate that abnormally elevated blood lead levels among adults are found predominantly in urban areas where exposure to airborne lead is more likely to occur.

Among children, extensive surveys (see Table VI-3) have demonstrated that excessive lead exposures have approached what must be considered an "epidemic" proportion. Approximately one quarter of the children tested showed elevated blood leads of 40ug/100g and above. Since the upper acceptable normal blood lead level for children should be below 40ug/100g, considerably more than one quarter of these children must be considered excessively exposed to lead. Although these excessively exposed children are often residents of homes coated with lead based paints, lead in the air, and consequently in the dust and dirt, is contributing to and aggravating this problem.

As in the case of adults, childhood residence in urban areas where air lead levels are greatest is associated with larger percentages of children with abnormally elevated blood lead levels. These data, which were discussed in more detail earlier, are presented again in Table VI-4. Airborne lead and consequently lead in dirt and dust were believed by Dr. Lepow to contribute significantly to the abnormally elevated blood lead levels in this urban group.

Recent preliminary data indicate that excessive lead exposure is already 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. A sizeable percentage of these babies (perhaps as high as 10%) are found to have umbilical cord blood lead levels in the 35-40ug/100g range. This borders on the level at which clinical symptoms of lead poisoning in children have been observed. Mothers of these children are not suspected to eat paint, dirt, or dust. Hence, increased exposures to airborne lead in these urban environments must be considered a primary factor contributing to this problem. For example, in a study conducted in Boston,[3] umbilical cord blood lead values of 30ug/100g or above were present only in babies born to urban mothers. In this urban group, of 13 babies tested, cord blood leads of 37 and 39ug/100g were observed. These are levels well above those considered safe for the fetus.

A second study examined umbilical cord blood lead levels among

babies born to mothers living only in New York City.[4] Of 100 urban newborns sampled, 6 were found to have umbilical cord blood lead levels in the range of 25-34.9ug/100g. Several of these babies were probably born with cord blood leads of 30ug/100g or above, an observation reasonably consistent with that reported in the Boston investigation.

A third study, however, failed to demonstrate any difference in umbilical cord blood lead levels between babies born to mothers living in urban compared to suburban environments.[5] 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.

In summary, considered as a group, these studies indicate the probable existence of abnormally elevated umbilical cord blood lead levels among babies born in urban environments. If this trend is 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.

REFERENCES TO TABLE VI-1

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

2 Tepper, L.: "A Survey of Air and Population Lead Levels in Selected American Communities" (7 City Study), Testimony presented at EPA Public Hearing in Los Angeles May 3, 1972, and data later submitted to EPA.

3 Hofreuter, D. H., et al: "The Public Health Significance of Atmospheric Lead," Arch. Env. Health 3:82-88, Nov 1961.

4 Daines, R. H., et al: "Lead in Air Inside and Outside Homes and in the Blood of Women as Influenced by Proximity to a Well Traveled Roadway," Rutgers, U., data submitted to EPA, July 25, 1972, and to be published in Ind. Med. Surg. Journal, Oct. 1972.

5 Goldsmith, J., California Department of Public Health, Testimony submitted to EPA July 11, 1972.

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.

REFERENCES TO TABLE VI-2

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

2 Tepper, Lloyd: "A Survey of Air and Population Lead Levels in Selected American Communities," (7 City Study), Testimony presented at EPA Public Hearing in Los Angeles May 3, 1972, and data later submitted to EPA.

3 Hofreuter, D. H., et at: "The Public Health Significance of Atmospheric Lead," Arch. Env. Health 3:82-88, Nov. 1961.

Table VI-3

Percentages of Children with Abnormally Elevated Blood Leads

City	Years Tested	Numbers Tested	% Blood Leads Equal to 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

REFERENCES TO TABLE VI-3

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

2 Guince, Vincent F.: "Lead Poisoning," American Journal of Medicine, 52:283-288, 1972.

3 Challop, R. S., and McCabe, E. B.:"Childhood Lead Poisoning: A Thirty City Neighborhood Survey," BCEM, USDHEW, May 23, 1972.

4 "National Estimates of Lead Based Paint Poisoning of Children," National Bureau of Standards Report 10651, Dec. 7, 1971.

TABLE VI-4

Urban-Suburban Blood Lead Comparisons in Children[1]

Residence	Number Studied	% of Blood Leads Equal to or Greater than 40ug/100g
Urban	272	25.0
Suburban (rural)	230	10.0

REFERENCE TO TABLE VI-4

1 Lepow, Martha, MD, Associate Professor of Pediatrics, U. of Connecticut School of Medicine, Testimony presented to Senate Commerce Committee, May 8, 1972.

REFERENCES FOR SECTION VI - EXTENT OF ABNORMAL LEAD EXPOSURES IN THE GENERAL POPULATION

1 Office of Air Programs Data File of Nationwide Emissions, 1970, EPA, Research Triangle Park, N. C., July, 1972.

2 Tepper, Lloyd, Testimony presented to EPA at L. A. Public Hearing, May 3, 1972.

3 Scanlon, John: "Umbilical Cord Blood Lead Concentrations," Amer J Dis Child, 121:325-326, 1971.

4 Rajegowda, B. K., et al: "Lead Concentrations in the Newborn Infant," Journal of Pediatrics, 80:116-118, January, 1972.

5 Harris, Paul: "Lead Levels in Cord Blood," Journal of Pediatrics, pp. 606-608, April, 1972.

VII. FINDINGS AND RECOMMENDATIONS

Findings

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

2. A precise lead threshold below which no adverse health effects will ever occur is at present difficult to define. 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.

3. Susceptibility to lead appears to be increased among young children compared to adults. New born babies and especially the fetus must be considered most vulnerable to lead. Any possible exposure of the developing central nervous system in utero, to lead, an established neurotoxic agent, must be kept at a minimum. This argues for a reasonable safety factor between what is considered a safe blood lead level in children and what is considered an acceptable exposure among the fetus and the newborn.

4. Considerable difficulty exists in defining a single safe blood lead level protective of everyone in the population. Variable responsiveness to lead exists among different age groups and even within age categories. In this context, available scientific evidence supports the following

guidelines:

a. Blood lead levels of 40ug/100g or above in adults must be considered evidence of excessive lead exposures.

b. A safe blood lead level protective of all children is below 40ug/100g.

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

5. Airborne lead levels below $2ug/m^3$ have been shown to have effects upon blood leads in both children and adults.

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 also contribute significantly 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 is a definite hazard.

7. Levels of lead in street dirt and house dust 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 abnormally elevated blood lead levels exceeding 40ug/100g. These abnormally elevated blood leads have been documented among adults and children, as well as the newborn.

a. Recent surveys of adult populations indicate that approximately 1-2% of urban females and 3-5% of urban males presently have blood lead levels of 40ug/100g and above. Residence in urban areas where air lead levels are highest is consistently associated with this finding.

b. Excessive lead exposures among children have approached what must be considered an "epidemic" proportion. Extensive surveys involving well 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 are also contributing to and aggravating this problem.

c. Recent preliminary data indicate that excessive lead exposure may already be occurring before birth among babies born to mothers living in urban environments. These studies suggest that significant numbers of babies born in the central city may have umbilical cord blood lead levels well above 30ug/100g, a level close to that at which clinical symptoms of lead poisoning in children have been observed. Exposure of these mothers to airborne lead in urban environments is considered to be a primary factor contributing to these blood lead elevations.

9. Experimental evidence indicates that lead is capable of interfering with biochemical processes at the gene level. This observation is consistent with recent documentation that chromosomal abnormalities are associated with increased lead exposure in man. We do not know whether these effects occur at low level chronic lead exposures among the general population. However, they do indicate that we should be concerned about possible genetic implications resulting from general population exposures to lead.

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

Recommendations

Since this document is a preliminary draft and has not been formally released by EPA the following recommendations should not be construed to represent Agency policy.

Though none of the above findings viewed individually and in the context of possible experimental errors can be taken as conclusive evidence that airborne lead is harmful, considered in total, they are definitely indicative of widespread excessive exposures to airborne and dustfall lead which presently exist among the general urban population. Every effort must be made to reduce all preventable excessive lead exposures to the fullest extent possible, especially in light of the particular susceptibility to lead of children and the newborn. On this basis the Administrator concludes that EPA must take the most prudent position required to protect the Nation's health.

The following recommendations are thus made:

1. EPA's previous lead in gasoline regulation calling for a 60-65% reduction to achieve a $2ug/m^3$ air lead goal must be considered

inadequate to protect the public health. This decision is based largely upon a thorough reevaluation of all available health effects data, including recently completed studies. This new information was not available before publication of "Airborne Lead in Perspective" by the National Academy of Sciences or before EPA's previous health analyses examining the airborne lead problem were developed.

2. Based upon the above findings including effects of airborne lead directly and indirectly upon blood leads in adults and children as well as the newborn, air lead levels should prudently be reduced to the fullest extent possible.

p.4 DuPont Study

DRAFT:9/22/72

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 basic principle 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 considerably greater than that from air. 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 well controlled, the likelihood of observing a correlation between blood lead and air lead at low air lead concentrations is very slim.

Another limitation in the Goldsmith-Hexter approach is the reduction of many data points into simple averages for blood leads as well as air lead exposures. This averaging approach tends to lose a considerable amount of information that is present in the original data. Consequently, available information is reduced to a series of averages which do not adequately describe the situation present in the real world. This is especially true when one considers the fact that although the average blood lead in a given group may be well within normal limits, selected individuals within that group may have blood leads that are definitely elevated above normal. Any averaging that is done during the analysis will tend to obscure the presence of abnormally elevated blood leads in the original data.

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 affecting blood lead. One noted biostatistician in commenting upon the Goldsmith-Hexter regression equation concludes:[3] REED, HARVARD.

> "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 concur with EPA's preference to consider the distribution of individual blood lead values instead of merely the average blood lead among groups exposed to specific air lead levels. For example, according to Dr. Enterline:[5]

"...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."

Dr. Robert Reed, Chairman of the Department of Biostatistics at the Harvard School of Public Health is in agreement with this approach:[6]

"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' average 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."

REFERENCES FOR APPENDIX A - QUESTION 1

1 NAS Report, p. 50.

2 Science 158:132-134, 1967.

3 Reed, Robert B., 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" to be 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 B., 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 manufactureres 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 causes demineralization of bone could result in mobilization of lead from bone, which in some instances could be toxic.[7,8]

Although several investigators have tried unsuccessfully to mobilize lead from bone under experimental conditions,[9] this does not constitute proof that under all conditions lead stored in bone is in fact physiolog-

ically inert. Recent evidence suggests that lead stored in bone may, in fact, inhibit hemoglobin synthesis in the intact animal.[10] ← not true

One leading medical authority, 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, Section 6, "Relationship of Airborne Lead and Indices of Lead Absorption" to be presented at the International Symposium on "Environmental Health Aspects of Lead," Amsterdam, October 2-6, 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 Harrison's Textbook of Medicine, McGraw-Hill, p. 666, 1970.

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

10 Millar, J. A.; Battistini, V.; Cumming, R. L. C.; et al, "Lead and Delta Aminolevulinic Acid Dehydratase Levels in Mentally Retarded Children and in Lead Poisoned Suckling Rats," Lancet 2:695-698, 1970.

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 cited in reply to this question emphasizes the importance of considering the entire spectrum of biologic response to lead that exists in the general population. The real world is simply not adequately described in terms of only the average response. Hence any policy related to environmental lead must consider the widely variable responses to this substance that will occur.

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 Perspective" 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, criticizes 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, 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. The National Academy of Sciences believes 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. Pat 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 connot 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 NAS Report, 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 NAS Report, p. 69.

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 "Lead in the Environment," Proceedings of a conference held at the Zoological Society of London, p. 26, 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 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; (4) occupational groups receive periodic medical

examinations while on the job to detect early disease changes. This opportunity is not available to those exposed in the general population; and finally (5) occupational groups are being exposed more by free choice than are those 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, is convinced 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, including comments from the National Institute of Occupational Safety and Health, 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, July 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, letter to 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 elimination of lead additives from gasoline will also help to reduce 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 60 micrograms/100 grams 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 possibility. 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 readily controllable. However exposures through lead in air and consequently lead falling out from the air to contaminate dust and dirt can be 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 specifically 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 sufface 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. 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 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 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 by the author to be a significant source of the 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 lead content in urban soils 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 be primarily responsible for 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 among children known or suspected to 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 the major factor in the etiology of these abnormally elevated blood lead levels 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 nonrespirable 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 exposure to airborne lead as well as ingestion of lead contaminated dusts was contributing significantly to this problem in the 1-5 year old group. 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 received by EPA expressed concern for this 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:[17] "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."

Dr. Anthony Mustalish of the New York City Department of Health generally agrees:[18]

"Although to my knowledge no cases of lead poisoning have been attributed to atmospheric lead alone, there is grow-

ing evidence that atmospheric lead contributes to this body burden and in inner city children this contribution may aggrevate an already compromised system."

Finally, Elliot Richardson, Secretary of the Department of Health, Education, and Welfare has written in a recent letter to EPA Administrator William Ruckelshaus:[19]

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

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 already exposed to peeling lead based paint. Reduction of airborne lead levels for purposes of reducing the concentration of lead found in urban dust and dirt would thus be a most prudent decision.

REFERENCES FOR APPENDIX A - QUESTIONS 5 & 6

Bridbord, K.; Shy, C.; Hammer, D.; Goldberg, H.; Newill, V.; and Nelson, W., "A Control Strategy for Lead in Paint," Congressional Record, Extension of Remarks, pp. E1010-E1011, February 9, 1972.

2 Guinee, Vincent, "Lead Poisoning," Medicine 52:283-288, 1972.

3 Guinee, Vincent, "The Position of NYC on the Control of Childhood Lead Paint Poisoning," Submitted to the Subcommittee on Health of the Committee on Labor and Public Welfare, United States Senate, March 9, 1972.

4 DuVal, Merlin K., Assistant Secretary for Health and Scientific Affairs, Department of Health, Education, and Welfare, Testimony before the Subcommittee on Health, Committee on Labor and Public Welfare, United States Senate, March 10, 1972.

5 Lepow, Martha L., Testimony before Subcommittee on the Environment, Committee on Commerce, United States Senate, May 8, 1972.

6 Creason, J. P.; McNulty, O.; Heiderscheit, L. T.; Swanson, D. H.; and Buechley, R. W., "Roadside Gradients in Atmospheric Concentrations of Cadmium, Lead and Zinc," presented at the Proceedings of the Fifth Annual Conference on Trace Substances in Environmental Health, June 29-July 1, 1971, Columbia, Missouri.

7 Ibid.

8 Krueger, Harold, Testimony presented to EPA, July 10, 1972.

9 Fritsh, Albert, and Prival, Michael, Center for Science in the Public Interest, Testimony presented to EPA.

10 Krueger, Harold, Testimony presented to EPA, July 19, 1972.

11 Creason, J. P.; McNulty, O.; Heiderscheit, L. T.; Swanson, D. H.; and Buechley, R. W., "Roadside Gradients in Atmospheric Concentrations of Cadmium, Lead and Zinc," presented at the Proceedings of the Fifth Annual Conference on Trace Substances in Environmental Health, June 29-July 1, 1971, Columbia, Missouri; Finklea, John, memorandum of June 30, 1972.

12 Pinkerton, C., Hammer, D. I., Hinners, T. A., Kent, J. L., Hasselblad, V., Lagerwerff, J. V., and Ferrand, E. S., "Trace Metals in Urban Soils and Housedust," paper to be presented to Environmental Section APHA Centennial, Atlantic City, Nov. 16, 1972.

13 Finberg, Laurence, Testimony presented to EPA, June 20, 1972.

14 Lepow, Martha, Testimony before Senate Subcommittee on Environment, May 8, 1972.

15 Chisolm, J. Julian, Testimony submitted to EPA, July 1, 1972 and July 20, 1972.

16 Finberg, L., Testimony presented to EPA, June 20, 1972.

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

18 Mustalish, Anthony, Testimony presented at Washington, D. C., Public Hearing, April 11, 1972.

19 Richardson, E., letter to EPA Administrator Ruckelshaus, dated August 11, 1972.

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. Testimony was not received advocating benefits as a result of introducing lead into the environment. 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. Elimination of lead from gasoline can be expected to decrease the concentrations and total amounts of lead currently found in urban street dirt. As a result, decreases in lead water pollution are also anticipated by discontinuation of lead as a gasoline additive.

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 [illegible] 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 that (1) a definite difference in atmospheric lead levels existed between urban and rural areas with highest levels being recorded in the central city and (2) 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 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 suburbs.

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 since 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 causal role played by 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 constant, then differences in blood leads can be more easily detected. During comparisons of blood lead levels between different geographical areas, variable levels of dietary lead intake become especially significant and tend to mask effects due to air lead differences. However, within specific urban-suburban comparisons these differences in dietary lead intake become minimized, hence, increasing the probability of detecting differences in blood lead due to varable 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.

DU PONT STUDY DOES NOT SHOW THIS

Since lead intake from food and water among areas, as measured by fecal lead excretion, varied considerably, correlations between air lead and blood lead would not be expected to show very significant results expecially at low air lead differences. Although differences in fecal lead excretion were not always in a direction that could explain specific area inconsistencies, the very existence of this variable factor in part explains why a statistically significant correlation was not observed. In this regard, any positive correlation that is found, as is the case in this study, even if not statistically significant, is suggestive of an effect of air lead upon blood lead levels.

DIFFERENCES WERE 20 FOLD

REALLY STICKING OUT THIS ONE.

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 average air lead exposures. 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 definitely 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 would probably 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.

speculative

valid point

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 cut off was chosen because it was well toward the middle of each distribution curve 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 < 21.8	76	105	181
Blood lead ≥ 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)*
Philadelphia Urban	1.67
Philadelphia Suburban	1.15

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)

Chicago Urban	1.76
Chicago Suburban	1.18

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)

New York Urban	2.08
New York Suburban	1.13

In two of the three comparisons (Philadelphia and Chicago) statistically significant differences in blood lead patterns between groups are evident 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. This suggests that if sample size had been increased, a statistically significant difference would also have been observed.

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. This implies that effects of air lead upon blood lead occur at very low air lead levels (1-2 micrograms per cubic meter).

A second important observation is that only in urban areas more individual blood lead levels found to be equal or above those indicative of excessive lead exposure in adults (blood lead of 40ug/100g or above). For example:

	Number Studied	% Blood Leads 40ug/100g and above
Urban Females	423	0.7 — 3 WOMEN.
Suburban Females	556	0

Sampling variability might in theory explain these results. However, other independent investigations confirm the observed presence of abnormally elevated blood leads in urban, but not suburban or rural populations, suggesting that this is a real finding.[2,3]

Further, the Seven City Study demonstrates that men have higher blood lead levels than women at comparable air lead exposures. Thus, had men been studied instead of women, a greater percentage than 0.7% would have been found to have abnormally elevated blood lead levels. If these findings can be extrapolated to the general urban population, one must conclude that several million adults are currently excessively exposed to lead as a result of residence in urban environments where airborne lead exposures are greatest. (FROM 3 TO SEVERAL MILLION! — PRETTY TENUOUS EXTRAPOLATION)

As a result, the Seven City Study reaffirms the fact that despite measurements showing that average blood leads in the United States are well within normal limits, there are still large numbers of urban adult Americans who are presently excessively exposed to lead. Individuals with abnormally elevated blood leads almost always reside in urban environments. Hence, exposures to elevated airborne lead levels in urban areas must be considered a significant factor contributing to these abnormally elevated blood leads.

REFERENCES TO APPENDIX B

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

2 Ibid.

3 Hofreuter, et al. "The Public Health Significance of Airborne Lead," Arch. Environ. Health, Vol. 3 (1961), pp. 82-89.

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