EPA'S POSITION ON THE HEALTH IMPLICATIONS OF AIRBORNE LEAD

PREPARED BY

U.S. ENVIRONMENTAL PROTECTION AGENCY
401 M STREET, S.W.
WASHINGTON, D.C. 20460

NOVEMBER 28, 1973

TABLE OF CONTENTS

I. Introduction

On February 23, 1972, EPA published a notice in the Federal Register (1), setting forth proposed regulations promulgating Federal standards for the use of lead and phosphorus additives in gasoline. Pursuant to the notice, several public hearings were held. In addition, numerous written comments were received by the Agency during an extended public comment period. After consideration of the hearing testimonies and written comments, and after further consideration of the available information on health effects of airborne lead, including the adverse effects of leaded gasoline on emission control devices, the regulations were divided into two separate pieces of regulatory action: final regulations providing for the general availability of lead-free gasoline were promulgated on January 10, 1973 (2) and regulations based upon the health effects of airborne lead, providing for the reduction of lead in leaded gasoline were reproposed (3). The regulations on reduction of lead in leaded gasoline for health reasons were reproposed because the Agency's basis for this reduction was substantially revised and had not been available for review by the scientific community.

At the time of the reproposal, the Agency was aware that there had not been unanimity either within or outside the

government with respect to the need to reduce lead in leaded gasoline. In light of this controversy, the Agency requested that informed and concerned members of the public provide EPA with their opinions on the matter. This commentary was encouraged by announcement in the Federal Register (3) and through approximately 150 individual letters specifically requesting comments from members of the scientific and industrial communities and from public interest groups.

As a result of these requests, numerous comments were received by the Agency. Although a substantive response to each comment was not possible, all comments were considered and are available for public inspection at the Freedom of Information Center of the Environmental Protection Agency in Washington, D.C.

The present paper is not intended as an encyclopedic treatment of all that is known about lead. Rather, it is intended tc be a presentation of the pertinent evidence upon which a decision could be made as to whether or not there is a health justification to regulate lead in gasoline. While the emphasis in the paper is on lead as a gasoline additive, it has been necessary to consider other sources of lead in the environment in order to understand the role of leaded fuel in relation to human lead exposure. The Agency has also considered whether replacements for lead additives will result in deleterious health effects to as great or greater degree than the

lead additives themselves. This issue is discussed in a separate paper entitled, "Lead in Gasoline, Impact of Removal on Current and Future Automotive Emissions" (4).

The present paper was prepared by a committee of EPA scientists.

Members of the Committee Include:

John Buckley, Ph.D., Chairman

Kenneth Bridbord, MD	Magnus Piscator, MD
Douglas I. Hammer, MD	Lawrence Plumlee, MD
Robert Horton, MD	Steven Reznek, Ph.D.
Marty Kanarek, MPH	Richard Rhoden, Ph.D.
Wellington Moore, DVM	Jerry Stara, DVM

At least two committee members read and considered each of the approximately 130 comments received on the reproposed regulation. The results of this review process are reflected in the present paper as is additional information which has become available since reproposal of the regulation.

On October 1-2, 1973, EPA co-sponsored with the National Institute of Environmental Health Sciences a Conference on Low Level Lead Toxicity. A detailed evaluation of all presentations at the Conference is beyond the scope of the present document.

The proceedings from this Conference are due to be published in the near future.

The present document is an extension of the earlier paper entitled, "EPA's Position of the Health Effects of Airborne Lead," dated November 29, 1972. At the time the earlier paper was prepared, it seemed likely that catalytic converters would be the principal means of reducing pollutants from automobiles for a decade or more, and that the associated requirement of lead-free gasoline to protect the catalysts would result in near-elimination of automotive-related lead in the environment. It now appears that the impact of catalytic converters may be more transitory and that, unless regulated otherwise, lead additives will continue to be used in substantial amounts. Thus the health implications of leaded fuels become central to the regulatory decision process.

REFERENCES

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

2. Federal Register, Vol. 38, No. 6; pp. 1254-1256, January 10, 1973.

3. Federal Register, Vol. 38, No. 6; pp. 1258-1261, January 10, 1973.

4. Moran, J.B., "Lead in Gasoline, Impact of Removal on Current Automotive Emissions." Environmental Protection Agency, National Environmental Research Center, Research Triangle Park, North Carolina, October 1973.

II. Occurrence and Sources of Lead in the Environment

Lead occurs naturally in the earth's crust and is also found in the atmosphere and hydrosphere due to airborne dust, and to gases diffusing from the earth's crust (1). Lead was one of the first metals to be used by man, and evidence of the adverse health effects caused by lead and its compounds is found in the earliest annals of man's history. Deleterious effects from the present use of lead can be a problem, particularly in certain segments of the population. This Section will examine the influence of man upon the distribution of lead in the environment, especially with respect to the use of lead as a gasoline additive.

Lead or its compounds may enter and contaminate the environment at any step during the mining, smelting, processing, and use of the metal and its derivatives. The annual increase in lead consumption in the U.S. during the 10-year period 1962-1971 averaged 2.9%, resulting largely from an increased demand for batteries and gasoline additives (2). During the same period, lead used in pigments and caulking and in metal products declined. In 1971 the total lead consumption was 1,431,514 short tons with 596,797 short tons (42%) coming from recycled lead (3).

Because lead and many lead salts or oxides are relatively inert or immobile in the environment, a judgment can be made as to the ease with which lead from various use categories can reach and be transported through the environment. Table II-1 summarizes lead consumption patterns in the United States for 1968 and 1971 (4,5) according to categories of environmental impact. At this time, not all information required to compile a detailed lead balance for the United States is available; thus the above inventory of lead products in terms of their potential release to the environment must be recognized as subjective.

As shown in Table II-1, 352,614 short tons or approximately 25% of the lead consumed in 1971 was in the form of metallic lead or lead alloy. Most of this lead is chemically inert under typical environmental conditions. As long as the size of the piece of metallic lead or lead alloy is large, as is usually the case within the metal products category, it is relatively environmentally immobile. Recognized exceptions to this generalization do exist and include small pellets of lead shot often eaten by waterfowl, and the incineration of lead foils and tubes, with resultant release of lead into the atmosphere.

Storage batteries accounted for 679,803 short tons of lead consumption in 1971. This represents approximately 50% of the total. Since a large fraction of the lead in storage batteries

is recycled, little lead is believed to reach the environment from disposal of storage batteries.

Lead is also commonly used as a coating material. In 1971, some 53,198 short tons were in this category. Because of the possibility of erosion or wear, the lead thus used must be considered as potentially releasable into the environment.

More difficulty exists in classifying lead pigments in terms of their potential for reaching the environment. Lead pigments accounted for 81,258 short tons of lead consumption in 1971. Much of this amount is tabulated simply as red or white lead or litharge, rather than according to the actual category of use. Based on shipping information, approximately one-fourth of the lead classified under pigments in Table II-1 is used in the ceramics industry (6) and would thus have a relatively small environmental impact. However, this form of lead could contribute directly to human exposure through, for example, improperly glazed dinnerware.

Lead pigment is used in printing inks and paints. Lead in printer's ink, especially colored ink, has definite potential to reach the environment as a result of incineration. The use of lead-based paints in residential applications has been greatly reduced in recent years, although the total used in the past is still significant in terms of eventual environmental impact. The

amount of lead released into the environment from pigments is dependent on the relative quantity of waste disposal by incineration compared to other disposal methods, as well as wear and erosion of painted surfaces. On a national basis, approximately 10-20% of all solid waste is incincerated (7). This figure is assumed to be applicable as a first approximation to estimate the release of lead to the environment from incineration of lead pigments. The estimate does not necessarily account for all lead incineration from demolished buildings, since old paint tends to have a higher lead content. Further, this does not reflect entrance of lead into the environment from wear and erosion of exterior painted surfaces which has been shown to be substantial immediately adjacent to such surfaces. (8)

In 1971, 264,240 short tons of lead consumed were in the form of lead additives in gasoline. This amount is about one fifth of the total consumption for that year. It can be calculated that about 70% of the lead additives used in gasoline are emitted to the environment as particulate matter from the tailpipe (9). Much of the balance remaining within the engine or exhaust system of the vehicle is believed to eventually be removed from engine surfaces by transfer to the oil or by flaking of deposits in the exhaust system. The combustion of leaded gasoline accounts for by far the largest portion of all lead now reaching the environment, comprising about 90% of airborne lead emissions (9).

As evidence of this contribution, a 43% reduction in lead additives consumed within New York City during 1972 resulted in a 37% decrease in annual average ambient lead concentrations in the city for 1972 compared to 1970-1971 (10). In a more detailed calculation annual averages of air lead data from 38 sampling sites across the city were as follows: July 1969-June 1970, 1.62 $\mu g/m^3$; July 1970-June 1971, 1.71 $\mu g/m^3$; and July 1972-June 1973, 1.18 $\mu g/m^3$. The latter figure reflects the decrease in air lead following limitation of lead content of gasoline sold within the city to 1.0 gram per gallon on January 1, 1972. Though meteorologic differences from year to year, as well as sampling and analytic variability could have accounted for some of this decrease, the relatively constant city wide average in the two years preceding reduction combined with the decline following reduction make it evident that the contribution of lead additives to airborne lead in New York City is substantial.

Table II-2 (9, 11, 12) summarizes the major discharges of lead into the environment. Since current knowledge regarding the mobility or activity of lead in the environment is incomplete, the information in this table can only be regarded as semi-quantitative. Of the total amount of lead found in the atmosphere, the contribution from natural sources, such as the re-entrainment of surface soil in the form of dust, is small. This contribution has been estimated to be about 0.0005 $\mu g/m^3$ of air.(13) The lead concentrations in today's urban atmospheres are

at least 100 to 1000 times greater than the amount which can be attributed to natural sources.

As noted above, over two thirds of the lead added to gasoline reaches the air environment (14). As lead leaves the exhaust pipe of an automobile, initially all of it is airborne. The heaviest particles fall to the ground within several hundred feet of roadways,(15,16), whereas others remain suspended for longer periods of time. The length of time that lead particles emitted in auto exhaust remain airborne is determined primarily by particle size and weight as well as by meteorological factors. Lead is removed from the air by gravitational settling of larger particles, aggregation and subsequent settling of smaller particles, and through scavenging by various forms of precipitation.

Fallout of automobile exhaust lead contributes to lead levels found in dust and dirt which may in turn be resuspended in air. Surveys of street and sidewalk dirt in Washington, D. C. and in Boston showed lead concentrations commonly in the range of 1000 to 2500µg/g (ppm) with some values as high as 5000-10,000µg/g (17, 18). Samples of sidewalk dirt taken in the Bedford-Stuyvesant area of Brooklyn, New York over a five month period ranged from 900 to 4900 ppm lead (19).

In a study of 77 midwestern cities, the average concentrations of lead in dust collected at residential, commercial, and industrial sites were 1,636 µg/g, 2413 µg/g, and 1512 µg/g respectively (20). Lead concentrations in surface soil from three California city parks ranged from 194 to 3,357 µg/g (21).

Lead levels in urban housedust commonly ranged from 1000-2000 µg/g in Boston (22) and averaged 500-900 ppm in Brattleboro, Vermont, being higher in homes near roadways (23). In middle class residential areas of New York City, lead in house dust averaged 600-700 µg/g (24) or 2-3 times higher than similar measurements made in a suburban community. Lead emissions from automobiles, and industrial sources, as well as weathering of leaded paint all contribute to this contamination.

A very small part of the atmospheric lead resulting from lead fuel additives is in an organic form (gaseous lead alkyls). Alkyl lead concentrations are rarely as high as 10% of ambient air lead values (25, 26). Higher but transient peaks of alkyl lead concentration have been demonstrated near specific sources such as spills of gasoline containing lead alkyl compounds.

Air lead concentrations have been shown to be closely related to the density of vehicular traffic, being highest in large cities and progressively lower in the suburbs, smaller towns and

rural areas (27). Data from non-urban sampling sites in the National Air Surveillance Network (NASN) showed mean air lead concentrations of approximately 0.1-0.5$\mu g/m^3$ during 1953-1966. The mean concentrations at urban sites for those years were 1-3$\mu g/m^3$.

In Pasadena and Los Angeles, air lead concentrations sampled for two to seven hours have averaged from 9.4 to 23.6 $\mu g/m^3$ during weekday traffic and have ranged up to 29.3 $\mu g/m^3$ during the morning rush hour (28). Studies at different locations of the Queens Midtown Tunnel and Triborough Bridge in New York City showed that ambient lead levels averaged from 2.1 to 35.6 $\mu g/m^3$ per 24 hours with individual 24 hour peaks ranging up to 57.3 $\mu g/m^3$ (29). About 68% to 88% of these airborne lead particulates were in the respirable range.

One study has shown that air lead concentrations measured during 1968-69 in Los Angeles, Philadelphia, and Cincinnati increased 56%, 19%, and 17%, respectively, over concentrations measured in 1961-62 (30). The average lead concentration consistently increased at 17 of the 19 sampling sites and meteorological conditions at each sampling site were consistent for the two time periods. NASN data, which comes largely from the central city commercial areas, does not show such a marked increase during the same period, perhaps because traffic

densities were consistently high in these areas in the 1961-1969 period.

The extent to which airborne lead has affected the lead content of the earth's waters is difficult to ascertain. Lead enters the aquatic systems through precipitation, lead dust fallout and erosion and leaching of soil, as well as through municipal and industrial waste discharges. In addition, deposited lead (fallout) is washed off of streets, highways, and other surfaces into rivers, lakes and marine waters. Extrapolations from recent studies (31,32) indicate that on a national basis the contribution of lead from fallout washing off streets into water as urban runoff may be as high as 5,000 tons each year. Today, surface waters in the Mediterranean and Pacific Oceans contain up to 0.20 and 0.35 µg of lead/kg water respectively, which is about 10 times greater than the estimated pre-industrial lead content of marine water (33). The lead content of rivers and lakes also has increased in recent times.(33) A study of rainfall in various cities of the NASN network indicated an average concentration of 36 µg of lead/liter of rain which was approximately 40 times as high as that found in rainwater at non-urban sites (34).

Of concern is the possibility that continued contamination of the environment by atmospheric lead has affected the

concentrations of lead in biological systems and subsequently in man's diet. Data suggest that some biological systems are subjected to increased concentrations of lead in their environment and certain portions of the food chain (earthworms) have shown increases in lead content. (35). Shellfish that are eaten by man are known to take up and store lead. Vertebrate animals tend to store absorbed lead in bone rather than in flesh, and therefore do not so readily transmit lead up the food chain.

The quantitative contribution of atmospheric lead to the food chain of man is not known. Lead is a natural, although minor, constituent of plants. Plants can absorb lead to varying degrees (36). The solubility of lead is important when considering lead uptake by plants. Experimental studies have shown that plants can absorb and translocate only the soluble forms of lead (37). There have been no reported observations that would indicate that plants in the field are adversely affected by atmospheric lead. The most important aspect of lead fallout on plants is the potential hazard to humans or animals from the consumption of contaminated and unwashed foliage, fruit or roots. In areas with heavy lead dustfalls, such as along busy highways, substantial amounts of lead are found on exposed surfaces of plants. Since much of the atmospheric lead particulate is insoluble, a large portion of the lead contamination remains on the surfaces of plants. There is evidence of this source being important in lead poisoning of grazing animals (38). To the extent that lead

remains when plants are consumed by humans, it can add to man's total body burden of lead.

Marine species, with the exception of shellfish, usually do not concentrate lead from seawater and, hence, would not contribute much to human lead exposure. In one study, eastern oysters, soft-shelled clams, and northern quahogs were shown to concentrate lead from seawater (39). Although concentrations of lead reported in shellfish (range 0.17-2.5 ppm) are not high, they do indicate the ability of certain edible aquatic species to concentrate lead from the surrounding medium (40). Lead levels in soft clams, hard clams, and surf clams have at times been found that exceeded the maximum acceptable level proposed by federal experts (41).

The World Health Organization reports that according to the results of total diet studies in industrialized countries, the total intake of lead from food generally ranges from 200-300 µg per person per day. WHO further states that based upon available data, these levels are similar to those found in the past 30-40 years and that no upward trend in lead levels in food is evident (42).

Though lead in food would be the most consistent contributor to total lead exposure for the general population, lead in food is not the source that is most reduceable in the event that total

exposure to lead is found to be undesirable. For the general population, "the lead in air is probably the contribution that is most accessible to action for reducing the total body burden of lead especially where this fraction is large compared with that absorbed from food." (43) Lead levels in food, of course, still require careful monitoring to assure that excess exposure does not occur from this source.

In conclusion, man made sources of lead contribute most to the lead which enters the environment. Of these sources, the combustion of leaded gasoline is the single most significant contributor. Potentially, human exposure to environmental lead may result from a number of sources, among them being the inhalation of airborne lead and the ingestion of lead-contaminated dusts. Reducing the amount of airborne lead in the environment constitutes an accessible means for reducing potential human exposure to environmental lead.

TABLE II-1 (4,5)

LEAD CONSUMPTION (SHORT TONS)
(UNITED STATES)

Product	1968	1971
Metal Products		
Ammunition	82,193	87,567
Bearing metals	18,441	16,285
Brass & bronze	21,021	20,044
Cable covering	53,456	52,920
Casting metals	8,693	7,281
Collapsible tubes	9,310	10,041
Foil	6,114	4,417
Pipes, traps & bends	21,098	18,174
Sheet lead	28,271	27,607
Type metal	27,981	20,812
Weights & ballast	16,768	17,453
Solder	74,074	70,013
TOTAL	367,420	352,614
Storage Batteries		
Grids & posts	250,129	322,236
Oxides	263,574	357,567
TOTAL	513,703	679,803
Coatings & Miscellaneous		
Caulking lead	49,718	29,993
Annealing	4,194	4,068
Galvanizing	1,755	1,395
Plating	389	582
Terne metal	1,427	1,409
Other	17,924	15,751
TOTAL	75,407	53,198
Pigments		
White lead	5,857	4,731
Red lead & litharge	86,480	61,838
Pigment colors	14,163	13,916
ZnO	3,234	773
TOTAL	109,734	81,258
Chemicals		
Gasoline additives	261,895	264,240
Misc. chemicals	629	401
TOTAL	263,155	264,461
GRAND TOTAL	1,329,419	1,431,514

TABLE II-2 (9, 11, 12)

LEAD EMISSIONS (TONS) TO THE ENVIRONMENT FOR 1968
(UNITED STATES)

Atmospheric		
Gasoline combustion		181,000
Coal combustion		920
Lead alkyl manufacturing		810
Secondary smelting		811
Incineration of solid wastes		11,000
Other		755
	TOTAL	195,316 1/
Water		
Printing inks		10
Petroleum storage		24
Paint production		320
Battery production		600
	TOTAL	954
Insecticides		2,081
	GRAND TOTAL	198,351

1/ It is estimated that 5,400 tons of lead due largely to automotive lead emissions deposited on city streets reach the aquatic environment. Additional, but unknown, amounts are washed out of the atmosphere by precipitation and land directly on rivers, lakes and marine water.

REFERENCES

1. Airborne Lead in Perspective, A Report Prepared by the Committee on Biological Effects of Atmospheric Pollutants, National Research Council, National Academy of Sciences, Washington, D. C., 1972, p.5.

2. Ryan, J.P., Minerals Yearbook 1971. U.S. Department of the Interior. U.S. Government Printing Office, Washington, D.C., p. 671.

3. Ibid., p. 684.

4. Ibid., p. 684.

5. Airborne Lead in Perspective, op.cit.; p. 11.

6. Ryan, J.P., op.cit., p. 686.

7. Klee, A.J., et.al., "Preliminary Data Analysis, 1968, National Survey of Community Solid Waste Practices." USPHS Publication #1867, Solid Waste Program, Cincinnati, Ohio.

8. Comments submitted by the Ethyl Corporation to the Environmental Protection Agency, March 9, 1973, pp. V-16 - V-19.

9. Airborne Lead in Perspective, op.cit., pp. 11-13.

10. Personal communication from Fred C. Hart, Commissioner of Air Resources, New York City, to William D. Ruckelshaus, Administrator of EPA, March 20, 1973 and personal communication from Edward F. Ferrand, NYC Department of Air Resources to Kenneth Bridbord, EPA, Oct. 19, 1973.

11. "Recommended Methods of Reduction, Neutralization Recovery or Disposal of Hazardous Waste," Prepared by TRW Systems Group, EPA, Report #21485-6013-RU-00, February 1, 1973, Vol. I, p. 92-94.

12. The Pesticide Review, 1972, U.S. Department of Agriculture, Agriculture Stabilization and Conservation Service, Washington, D. C.

13. Airborne Lead in Perspective, op.cit., p. 5.

14. Ibid., pp. 11-13.

15. Motto, H.L., R.H. Daines, D.M. Child, and C.K. Motto, Lead in Soils and Plants. Its Relationship to Traffic Volume and Proximity to Highways. Environ. Sci. Tech. 4:231-238, 1970.

16. Airborne Lead in Perspective, op.cit., p. 29.

17. Krueger, H.W., Krueger Enterprises, Cambridge, Mass. Testimony submitted to EPA, July 10, 1972.

18. Fritsch, A., and M. Prival, Center for Science in the Public Interest, Washington, D. C., Testimony submitted to EPA, 1972.

19. Lombardo, L.V., The Public Interest Campaign, Washington, D. C., Personal Communication to Mr. William D. Ruckelshaus, Administrator, U.S. Environmental Protection Agency, March 9, 1973.

20. Airborne Lead in Perspective, op.cit., p. 139.

21. Ibid, p. 30.

22. Krueger, H.W., op.cit.

23. Darrow, D.K. and Schroeder, H.A., "Childhood Exposure to Environmental Lead," paper presented before the American Chemical Society, Chicago, Ill., August 29, 1973.

24. Pinkerton, C., J.P. Creason, D.I. Hammer and A.V. Colucci, "Multi-media Indices of Environmental Trace Metal Exposure in Humans," presented at 2nd Int. Symp. on Trace Element Metabolism in Animals, Madison, Wisconsin, June 1973.

25. Airborne Lead in Perspective, op.cit., p. 193.

26. U.S. Department of Health, Education and Welfare, Public Health Service, Division of Air Pollution. Survey of Lead in the Atmopshere of Three Urban Communities. PHS Pub. No. 999-AP-12. Cincinnati: Public Health Service, 1965. pp. 57-58.

27. Airborne Lead in Perspective, op.cit., pp. 18-21.

28. Ibid, p. 19.

29. Ferrand, E.F., Assistant Commissioner for Scientific Affairs, New York City Department of Air Resources, Testimony submitted to EPA April 11, 1972.

30. Tepper, L.B. and Levin, L.S. A Survey of Air and Population Lead Levels in Selected American Communities. Department of Environmental Health, College of Medicine, University of Cincinnati, Cincinnati, Ohio. Final Report (Contract PH-22-68-28), December 1972. 73 pp.

31. Water Pollution Aspects of Street Surface Contaminants, Environmental Protection Technology Series, EPA-R2-72-081, November 1972, p. 3.

32. Environmental Pollution by Lead and Other Metals, NSF Rann Grant 6I-31605, University of Illinois at Urbana-Champaign Progress Report May 1 - October 31, 1972, Chapter 6.

33. Airborne Lead in Perspective, op.cit., p. 6.

34. McCabe, L.J., J.M. Symons, R.D. Lee, and G.R. Robeck, Survey of Community Water Supply Systems. J. Am. Water Works Assoc. 62(11): 670-687, 1970.

35. Gish, C.D. and R.B. Christensen, "Cadmium, Nickel, Lead and Zinc in Earthworms from Roadside Soil", Environmental Science and Technology, 7:1060-1062, November 1973.

36. Airborne Lead in Perspective, op.cit., p. 33-37.

37. Ibid., pp. 33-37.

38. Ibid., pp. 178-182.

39. Pringle, B.H., D.E. Hissong, E.L. Katz, and S. Mulawka, Trace Metal Accumulation by Estuarine Mollusks. J. Sanit. Eng. Div.; Proc. Amer. Soc. Civil Eng. 94:455-475, 1968.

40. Airborne Lead in Perspective, op.cit., p. 189.

41. Metals in Shellfish with Particular Reference to Lead. Prepared by the Northeast Water Supply Research Laboratory of the U.S. Environmental Protection Agency, May 19, 1972, p. 17.

42. Joint FAO/WHO Expert Committee on Food Additives, Evaluation of Certain Food Additives and the Contaminants Mercury, Lead, and Cadmium; WHO Technical Report Series, No. 505, 1972, p. 19.

43. Ibid., p. 20.

III HEALTH ASPECTS OF LEAD EXPOSURE

It is apparent from our knowledge of the distribution of lead in the geosphere and biosphere that lead in small amounts is a natural part of living things. Lead enters animals from plants which pick it up from the soil. In spite of its ubiquity, no functional requirement for lead has been clearly demonstrated in living organisms.

Because lead is easy to extract and purify, it has been used for centuries. Lead, if improperly employed, is capable of producing serious poisoning and even death. All of the situations in which it is harmful are of human origin. For example, no known cases of lead poisoning have originated from natural amplification in the food chain. Much of our knowledge of lead poisoning comes from occupational exposures to the metal or its compounds and from ingestion of lead by children.

The commonest symptoms of lead poisoning are anemia, severe intestinal cramps, paralysis of nerves, particularly of the arms and legs, and fatigue. The symptoms usually develop rather slowly. High levels of exposure produce severe neurologic damage, often manifested by encephalopathy and convulsions. Such cases are frequently fatal. Today, severe cases of lead poisoning in occupational situations are rarely seen due to improved working conditions. Lead encephalopathy is now predominantly found in young children who have consumed flakes of

peeling lead paint, and also in some people who consume illegal whiskey, which often contains lead.

Exposure to lead is usually assessed by means of lead levels in blood and urine. Ordinarily, blood lead levels in the general population are between 10 and 30 µg/ 100g of whole blood. The expressions "µg/100g of blood" and µg/ 100ml of blood" are for all practical purposes equivalent and will be used interchangeably in this paper, depending upon which is used in the original references. Clinical symptoms of lead poisoning usually do not appear until blood lead levels reach 80-100 ug/100g or higher. Symptomatic lead poisoning may, however, be associated with blood lead levels in the range of 50-60 µg/100g in instances where the patient's response to lead in the tissues is modified as for example in the presence of anemia (1).

When the most severe effects of lead poisoning, e.g.encephalopathy, have occurred and not been fatal, recovery is frequently incomplete (2). Permanent, irreversible damage to the nervous system often results, even though no further high level exposure cccurs. With less severe effects such as anemia and intestinal symptoms, residual, irreversible injury is not observed if the exposure is discontinued. Recovery may be slow, however.

Most injurious substances produce less conspicuous effects at exposure levels somewhat below those resulting in overt symptoms.

These less conspicuous effects must therefore be specifically searched for by examinations or tests; otherwise, they may not be observed. Lead is strongly suspected to produce such subtle effects, but they have been much less well documented than have the more conspicuous ones.

The suggestion that subtle lead effects occur comes in part from studies of overt lead toxicity in which residual neurologic effects were found during followup examinations among children who initially were not deserved to have such symptoms. It should be noted, however, that the problem of subclinical lead effects is distinct from that of residual neurologic damage following lead poisoning. Whether subclinical lead effects themselves can result in residual neurologic damage is another question which has not been adequately resolved.

In this regard, a study involving 425 children with lead poisoning attributed primarily to ingestion of lead-based paint found that 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 many instances neurologic symptoms were not found at initial examinations. This study, however, dates back to 1955, when diagnosis of lead poisoning often depended upon tests for urinary coproporphyrins, which are not usually positive below 80-100 µg of lead/100 g. whole blood. It is therefore probable that large amounts of lead had been ingested. Extrapolation of these

results tc children with blood lead levels below 80 µg/100g is not appropriate.

Smith, et al (4) examined the neurologic sequelae in children diagnosed with lead poisoning at least five years prior to study. Permanent neurologic damage was found only in children who had lead poisoning accompanied by encephalopathy. Subtle damage due to lead exposure, however, could not have been recognized in this study since sophisticated psychological and fine motor function tests were not employed.

More recent studies have employed a greater degree of sophistication in testing behavioral changes associated with exposure to lead. Kotok et al (5) studied children with similar socio-economic backgrounds to determine whether increased lead body burden interferes with normal neurologic and motor development. A group having a mean blood lead level of 81 µg/100 g was not found to differ significantly from a control group having a mean blood lead of 38 µg/100 g. On this basis, it was concluded that blood lead levels below those usually associated with encephalopathy do not interfere with childhood development. The mean blood lead level of the control group selected, however indicated significant exposure to lead. Thus, firm conclusions cannot be drawn from these data regarding whether or not lead adversely affects the nervous system at blood lead levels of 80µg/100g or below.

Albert, et al. (33) employed psychological tests, medical examinations, nerve conduction studies and school records to evaluate whether deleterious effects could be associated with childhood lead exposure below overt toxicity. Excess lead exposure was measured in terms of lead levels in teeth. In the absence of a diagnosis of lead poisoning or a blood lead level in excess of 60 µg/100g, high levels of lead in teeth were not associated with deleterious health effects. In constrast, adverse effects were seen in children with blood lead levels in excess of 60 µg/100g who received no chelation therapy as well as among these who were treated for severe lead poisoning.

A study by Pueschel et.al. (6) suggests that in children lead body burdens below those usually associated with clinical poisoning contribute to renal as well as neurological damage. Fifty-eight children between the ages of 6 months and 6 years were selected for study from a larger sample of children living in a high-risk lead poisoning area. The majority of these children were known to ingest paint and dirt. The children examined had blood leads over 50 µg/100ml or had blood leads over 40 µg/100ml with urine leads exceeding 500 µg/24 hours following EDTA provocation.

While most of the children did not exhibit symptoms uniquely attributable to lead poisoning, over one-third were found to have a history of mild CNS symptoms. The symptoms were observed significantly more frequently in these children than in a matched control group. Subsequent follow-up study revealed that many of

the children examined still had behavioral and school difficulties, even though sources of lead exposure had been removed in most cases. Since complete data on the control groups were not presented, it is difficult to fully evaluate the significance of these findings.

Of particular interest in Pueschel's work is the observation of anemia, aminoaciduria, proteinauria, and casts in a high percentage of the children. It may be noted that studies by Betts, et. al. suggest anemia can occur in children with blood lead levels between 37-60 µg/100ml and studies by Tola, et al. found mild anemia occurring in lead workers with blood leads between 40-60 µg/100g (7-8). Although factors such as nutritional status and deprived environments may have been responsible for the anemia and renal and neurological damage observed by Pueschel, these effects are consistent with damage that has been related to lead exposure.

It has been suggested by David, et al. (9) that childhood behavioral disturbances such as hyperactivity may be associated with blood lead levels between 25-55 µg/100ml. About half of the hyperactive group studied (28 of 54) had blood leads between 25-55 µg/100ml compared to less than one third (10 of 37) in the control group. Both the hyperactive and the control groups were similar with respect to age and sex, but socio-economic, racial and dietary differences between the groups may have confounded the results. Further, the children studied for hyperactivity may have had much higher lead absorption at an earlier age with a

subsequent decline in blood lead level by the time of study. Because the hyperactive child is more likely to have pica (abnormal ingestion of non-food substances), it is possible that the hyperactivity caused the increased lead intake rather than having been the consequence of it. The David study suggests, although it does not prove, that exposure to lead in quantities not generally considered excessive or toxic may contribute to hyperactivity in children. Additional studies are necessary to define the relationship between hyperactivity and causal factors, including lead. Studies by Silbergeld, et al. (34) may be important in this regard. Mice exposed to high levels of lead from birth developed hyperactivity which, as in children with this disorder, responded atypically to CNS stimulants and depressants.

A study by de la Burde, et al. (10) concluded that children ingesting paint or plaster containing lead are more likely to have abnormal or suspect behavior and fine motor disabilities than children not so exposed. Subjects in this investigation were followed prospectively and were selected to eliminate pre-existing neurologic abnormalities. The lead-exposed group had a mean blood lead level of 58 µg/100 g (range 40 to 100 µg/100g); blood leads were not measured in the controls. Lead intake differences between the groups were established on the basis of measures cf urinary coproporphyrins.

Results indicate that the control group performed better than the exposed group in the areas tested. Most notable differences

were with respect to fine motor ability and behavior. These results, however, may have been confounded by the failure to measure blood leads in the controls and by the failure to exclude, in the exposed group relative to the controls, an increased incidence of pica. Increased behavioral deficiencies observed in the exposed group could have been related to a greater frequency of pica in this group rather than the ingestion of lead per se.

Some studies have recently been undertaken to determine the lowest exposure levels at which lead-induced injury to the peripheral nerves can be detected (11-13). One study shows that in adults disturbances in conduction velocity can be found at blood lead levels of 80 µg/100g and above. Preliminary results also noted in this study indicate the presence of mild neuropathy in lead workers at blood lead levels under 50 µg/100 g (13).

The potential problem of subclinical neurological damage caused by lead is an important one which deserves further attention. Whether subtle adverse effects on CNS function such as small decreases in IQ, interference with fine motor function and altered behavior can be attributed to lead exposure is still uncertain. If such effects do occur they may be related to a degree of expcsure less than that recognized as producing overt lead poisoning.

In recent years evidence has been accumulated that significant exposure to lead will produce injury involving the

kidneys and other organ systems. This knowledge of complex and varied effects has recently been reviewed by the National Academy of Sciences (NAS). Table III-1 which is reproduced here from Table 4-11 of the NAS review of lead displays a detailed outline of effects at various exposure levels (14). It should be noted that some of these effects are more typically associated with lead exposure in children than in adults.

Because anemia is an evidence of lead toxicity, blood and blood cell formation have been studied particularly for early changes; shortened red cell life span (15) and lowered hemoglobin (16) have been observed. Injury to enzyme systems involved with the formation of hemoglobin has also been observed, but, because some of these systems are disturbed by substances and conditions other than lead, the observations are difficult to interpret. One biochemical change, indicated by an increased urinary excretion of delta aminolevulinic acid (ALA-U), is of particular interest because it is rarely produced by anything other than lead (17). This increase in ALA-U is one of the most thoroughly studied of the metabolic alterations produced by lead. It is directly related to inhibition of the enzyme delta-aminolevulinic acid dehydrase (ALAD) which converts ALA to porphobilinogen. Elevated ALA-U has been shown to be closely related to elevated lead levels in the soft tissues and may reflect biochemical changes in these tissues (18-19). Soft tissue lead is considered to be the metabolically active portion of the body lead burden.

(20) For these reasons, increased ALA-U is considered to be an undesirable change indicative of probable health risk (21).

The appearance of excess ALA in the urine is first seen at blood lead levels of 40-60 µg/100g (22-25). If increased urinary ALA excretion is undesirable, it then follows that blood lead levels above 40 µg/100g are to be avoided, if possible. In this context, the National Academy of Sciences Lead Panel concluded (26), "...the exponential increase in ALA excretion associated with blood lead content above approximately 40 µg/100g of blood signifies inhibition of ALAD that is physiologically significant in vivo."

Recent animal experiments, using graded levels of lead exposure, show beginning injury to kidneys at about the same exposure level as that causing increased ALA-U excretion (27). While these kidney changes are probably reversible when mild, it is known that permanent damage to the kidneys results from higher doses of lead (28). The degree and duration of exposure needed to produce irreversible changes are not known, although it is unlikely that they could be caused solely by lead in the ambient air.

Animal studies indicate that lead at much higher dosages than man would generally be exposed to is capable of disturbing the biological defense mechanisms that provide protection from bacterial and viral infection (29-31). The mechanism by which lead aggravates viral disease is by reduced interferon synthesis

rather than by inhibition of interferon action (32). Because it has become apparent that lead may produce this type of injury, further studies, with expected exposure routes and realistic exposure levels, should be of considerable interest.

The studies reviewed in this Section permit no unequivocal conclusions to be drawn. On balance, the studies suggest that subclinical changes may be associated with blood lead levels in the range of 40-60 µg/100g. As blood lead levels increase above 40 µg/100g, the likelihood that significant changes will occur increases markedly. Based upon evidence from these studies, it would seem prudent to regard blood lead levels over 40 µg/100g as indicators of lead intake that should be prevented. The 40 µg/100g figure, however, does not represent a sharp demarcation between health and disease.

TABLE 4-11 Level and Types of Effects of Inorganic Lead Salts as Related to Estimates of Various Levels of Absorption—Recent and Remote

Type of Effects	Level I: No Demonstrable *in vivo* Effect	Level II: Minimal Subclinical Metabolic Effect	Level III: Compensatory Biologic Mechanisms Invoked	Level IV: Acute Lead Poisoning		Level V: Late Effects of Chronic or Recurrent Acute Lead Poisoning
				Mild	Severe	
Metabolic (accumulation and excretion of heme precursors)	Changing ALAD[a]	Slight increase in urinary ALA may be present	ALA, UCP, FEP progressively increased	ALA, UCP, FEP increased 5- to 100 fold		Increased if excessive exposure recent, but may not be increased if excessive exposure remote
Functional injury: Hematopoiesis	None	None known	Shortened red-cell life-span, reticulocytosis (±) (reversible)	Shortened red-cell life-span and reticulocytosis with or without anemia (reversible)		Anemia (±) (reversible)
Kidney (renal tubular function)	None	None known	?	Amino-aciduria, glycosuria (±) (reversible)	Fanconi syndrome (reversible)	Chronic nephropathy[b] (permanent)
Central nervous system	None	None known	?	Mild injury (??? reversible)	Severe injury (permanent)	Severe injury[b] (permanent)
Peripheral nerves	None	None known	?	Rare	Rare	Impaired conduction (wrist, foot drop usually improve slowly, but may be permanent)
Clinical effects	None	None known	Nonspecific mild symptoms (may be due in part to coexisting diseases)	Colic, irritability, vomiting	Ataxia, stupor, coma, convulsions	Mental deficiency (may be profound), seizure disorder, renal insufficiency (gout) (permanent)
Index of level of recent or current absorption:						
Blood lead, μg/100 g of whole blood	<40	40–60	50–100+	>80 With anemia, intercurrent disease: 50–100+	>80	May be normal
Urine lead (adults only), μg/liter	–	<80	<130	>130 (May be less in severe illness)	>130	Spontaneous excretion may be normal

[a]See p. 106 for discussion of changing levels of ALAD.
[b]CaEDTA mobilization test in chronic nephropathy is positive; may or may not be positive in permanent central nervous system injury.

REFERENCES

1. "Airborne Lead in Perspective," Report prepared by the Committee on Biologic Effects of Atmospheric Pollutants, National Research Council, National Academy of Sciences, Washington, D.C., 1972, p. 130

2. "Airborne Lead in Perspective," op. cit., p. 95

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

4. Smith, H.D., Baehner, R.A., Carney, T. and Majors, W.J., "The Sequelae cf Pica With and Without Lead Poisoning." Amer. Jour. Dis Child. 105: 609, 1963

5. Kotok, D., Development of Children with elevated blood lead levels, Prediat. 80: pp. 57-61, Jan 1972

6. Pueschel, S.M., Kopito, L, and Sohwachman, H., Children with an increased lead burden, Jour. Amer. Med. Assoc. 222:4:462-466, Oct. 23, 1972

7. Betts, P.R., Ashley R. and Raine, D.N., "Lead Intoxication in Children in Birmingham,"Brit. Med. Jour., 1:pp. 402-406, 1973

8. Tola, S., Hernberg, S., Asp, S., and Nikkanen, J., "Parameters Indicative of Absorption and Biological Effect in New Lead Exposure: A Prospective Study," Brt. J. of Ind. Med., 30: pp. 134-141

9. David, O., Clark, J. and Vohller, K., "Lead and hyperactivity" Lancet, 2: 900-903, October 28, 1972

10. de la Burde, B., and M.S. Choate, Does asymptomatic lead exposure in children have latent sequelae? Pediat. 81:6:1088-1091, December 1972.

11. Catton, M.J., G. Harrison, P.M. Fullerton and G. Hazantzis, Subclinical neuropathy in lead workers, Brit. Med. Journ. 2: 80-82, 1970.

12. Seppalainen, A.M. and S. Hernberg, Sensitive technique for detecting subclinical lead neuropathy, Brit. Journ. Indust. Med. 29: 443-449, 1972.

13. Hernberg, S., Biological effects of low lead doses, Proc. Int. Symp. Envir. Hlth. Aspects Lead, CED, CID, pp. 617-629, Luxembourg, May 1973.

IV. Can an Acceptable Lead Body Burden be Defined?

In Section III the spectrum of effects due to lead was reviewed with special emphasis upon subclinical changes believed to be of health significance. This Section will examine how these findings may be of assistance in helping to define an acceptable lead body burden for the general population.

Exposure to toxic substances is difficult to quantify with external measurements, especially when exposure may occur via food, water and air. It is preferable, if possible, to measure an integrated index of these exposures within the body. The blood level of lead which itself is not an adverse health effect serves as such an index, and, under most circumstances is a reasonably accurate measure widely employed in public health surveillance. This measurement serves not only as an index of continuous lead exposure, but also as a diagnostic tool in identifying cases of lead poisoning and undue lead absorption.

Due to the ubiquitous nature of lead, the general population carries blood lead levels of 10-30µg/100g. As exposure increases, blood lead levels rise, although slowly and in a nonlinear fashion, because excretion of lead also rises with increasing exposure. Thus blood lead levels are a satisfactory index of soft tissue lead burden during constant exposures, but

are less accurate during rapid changes in lead exposure or in total body burden.

Blood lead concentration is the net result of several independent equilibria. Accordingly a single blood lead determination will not necessarily indicate a high nor exclude a low body burden of lead. Serial determinations are therefore recommended to assure the validity of the initial value as well as to ascertain rates of change (1). The variability of laboratory methodology and accuracy is also often considerable, and the confidence limits of the analytical procedure must be taken into account especially when comparing results among different laboratories.

Although physicians are generally well aware of limitations with the use of blood lead levels, the even greater variability of other indices and the ease of obtaining blood specimens make determination of blood lead the single most useful parameter. Other indices are helpful, including: concentration of lead in urine, feces, and hair; ALA in the urine; urinary coproporphyrin; erythrocyte delta aminolevulinic acid dehydrase, protoporphyrin, basophilic stippling, and red cell survival time; hemoglobin; and hematocrit. A detailed discussion of the relative merits of these techniques is beyond the scope of the present paper.

As noted in Section III, blood lead levels of 40µg/100g and above have been associated with subclinical changes as well as increased lead body burdens, and as such are considered to be evidence that significant lead intake has occurred. This 40 µg/100g level has been accepted as evidence of undue exposure to lead in children and adults (2,3,4), although one must recognize that this level does not represent a sharp demarcation between health and disease.

The U.S. Public Health Service has recommended (3) that, for older children and adults, until proven otherwise by additional research, "... a blood lead concentration of 40µg or more per 100ml of whole blood, ... determined on two separate occasions, be considered evidence suggestive of undue absorption of lead, either past or present." This recommendation also established 80µg/100ml whole blood as unequivocal lead poisoning and 50-79µg/100ml as requiring immediate evaluation as a potential poisoning case.

The FEP test (free erythrocyte protoporphyrins test) developed by Piomelli et al (11), shows promise of identifying the presence of undue metabolic effects resulting from lead exposure. The FEP test is capable of detecting an interference in heme synthesis caused by lead in the bone marrow. This interference, if present to a significant degree, could reflect a degree of lead exposure associated with toxic metabolic effects.

The FEP test is especially valuable since it is well recognized that blood lead levels do not, in themselves, constitute an absolute criterion of lead effect.

There exists debate within the scientific community with respect to what concentration of FEP in the blood constitutes an abnormal elevation. As originally proposed by Piomelli, an FEP level of 250 µg/100ml of RBC appeared a reasonable upper limit above which significant adverse effects due to lead were likely occurring. This 250 µg/100ml cutoff is approximately three times the FEP level found in 1-6 year old children with blood leads under 20 µg/100ml. There are, however, scientists who advocate a lower FEP cutoff to provide a greater margin of safety in any screening program.

Piomelli found that all children studied with blood leads of 60 ug/100ml or above had FEP levels in excess of 250 µg/100 ml of RBC. Among children with blood lead levels between 40-59 µg/100ml, 55% were observed to have FEP levels above 250 µg/100ml. In contract, only 5% of children with blood leads below 40 µg/100ml had elevated FEP levels. In this regard, one must consider the possibility that iron deficiency anemia potentiates lead toxicity even at blood lead levels below 40 ug/100g (12). The widespread prevalence of iron deficiency anemia among the population at greatest risk from lead exposure should alsc be noted. A positive FEP test accompanied by a blood

lead below 40 µg/100ml urually reflects the presence of iron deficiency anemia although this could also be an early indicator of undue lead absorption. The finding that 55% of children with blood leads between 40-59 µg/100ml have elevated FEP levels, combined with the exponential increase in FEP observed at blood leads above 40 µg/100ml, suggests the more frequent occurrence of adverse metabolic effects among children whose blood leads have risen to 40 µg/100ml and above. Combined with blood lead determinations, the FEP test would provide a more objective evaluation of lead toxicity than possible by either test alone. It may well be that subclinical neurological effects due to lead are more closely related to FEP levels in the hematopoietic system than to isolated measures of blood lead (13).

Environmental insults may put certain population subgroups such as children, the aged or infirm, and pregnant women at greatest risk. Although there is no clear evidence of increased susceptibility to lead in children, they may be considered a potentially high risk group because they have greater opportunity to ingest lead from environmental sources such as paint, dust or dirt. High lead exposure gives rise to renal tubular damage in children, which is an unusual consequence in adults. In children, excessive exposure is frequently associated with sudden onset of encephalopathy which is often followed by permanent brain damage (5). The onset of encephalopathy without previous symptoms is uncommon in adults.

Angle and McIntire (6) concluded that there is a definite fetal risk, maximal in the first trimester, from intrauterine exposure to high concentrations of lead in maternal blood. Studies by Kochen and Haas et al (7,8) confirm that umbilical blood lead concentrations are similar to those in maternal blood. In the newborn the blood-brain barrier is relatively immature (9,10) and the mass of central nervous system tissue is greater, while the mass of skeletal tissue is less, per unit of body weight. With the well documented neurotoxicity of lead, it is clear that additional data are necessary to evaluate the possibility of increased susceptibility to lead exposure in the fetus and neonate.

Two recent experiments are suggestive of a special susceptibility to lead among animals exposed either prenatally or during infancy. In one instance (14), lambs exposed to maternal blood lead levels of 34 µg/100ml during gestation demonstrated slowed learning on a visual discrimination task at 10-15 months of age. This deficit is consistent with visual-perceptual problems that have been noted in children with lead poisoning. Extrapolation of these findings directly to man is, however, somewhat uncertain since a blood lead of 34 µg/100ml in sheep may be equivalent to a higher blood lead in man. In another instance (15), infant rhesus monkeys fed 0.5 mg lead acetate per kg of body weight developed hyperactivity and insomnia. Blood leads in these infant monkeys ranged from 60-100 µg/100ml. In contrast

the physical status and behavioral status was unchanged in juvenile monkeys fed lead acetate at 20 mg/kg sufficient to produce average blood leads of 135 µg/100ml. Though lead dosages administered to the monkeys were well in excess of those found in human lead poisoning, the dependence of response upon age is noteworthy.

It is not possible at this time to firmly establish a single acceptable blood lead level protective of all high risk population subgroups. It would appear prudent, however, to recommend that the current U.S. Public Health Service guideline for older children and adults, i.e., 40µg/100ml whole blood, be regarded as a strict upper limit for younger children. Whether the acceptable upper limit should be lower than 40µg/100ml for the fetus, neonate, and the woman of child bearing age will require further investigation.

Important questions that necessitate additional study to help define with greater confidence an acceptable lead body burden include the following:

(1) At what level of exposure or body burden do effects of lead become irreversible?

(2) Are some of the permanent effects not immediately recognizable, especially in children?

(3) When the less severe effects subside is there no residual, or are there some permanent changes which are not noticeable?

(4) What combinations of level and duration of exposure prcduce irreversible lead poisoning?

(5) Does injury to other organs or systems such as the liver or biological defense mechanisms occur at levels of exposure and lead body burden common among the general population?

(6) At what level of exposure and lead body burden are changes due to lead found which produce no symptoms and will such changes if long continued produce irreversible damage?

(7) Which groups among the general population are most sensitive to lead and which factors can significantly affect lead toxicity?

Considerably more research is thus necessary to clarify these as well as other incompletely answered questions related to lead.

REFERENCES

1. Lin Fu, J.S., "Undue Absorption of Lead among Children - A New Look at an Old Problem," N. Eng. J. Med., 286 (13), 702-710, 1972.

2. "Airborne Lead in Perspective," report prepared by the Committee on Biological Effects of Atmospheric Pollutants, Division of Medical Sciences, National Research Council, National Academy of Sciences, Washington, D.C. 1972, pp. 100-110

3. "Medical Aspects of Childhood Lead Poisoning," HSMHA Health Reports, 86(2), 140-143, 1971

4. Chisolm, J.J., Baltimore City Hospitals, in testimony submitted to EPA, July 26, 1972.

5. Lin Fu, J.S., op cit.

6. Angle, C.R., and M.S. McIntire, "Lead Poisoning During Pregnancy," Amer. J. Dis. Child., 103, 436-439, 1964.

7. Kochen, J.A., Mentifiore Hospital and Medical Center,

personal communication to K. Bridbord, EPA, April 18, 1973.

8. Haas, T.; K. Mache; K. Schaller; A. Wieck; W. Mache; and H. Valentin; "Untersuchungen Uber Die Okologishe Bleibelastung Im Kindesalter;" Proceedings of the International Symposium on the Environmental Health Aspects Lead, CEC, CID, Luxembourg, May, 1973, pp. 741-748.

9. Dobbing, J.: The development of the blood brain barrier Prog. Br. Res. 29: 417-27, 1968

10. Evans, C.A. et al.: The development of blood brain barrier and choroid plexus function in immature foetal sheep, J. Physiol 224:15-16, July 1972

11. Piomelli, S., B. Davidow, V. F. Guinee, P. Young and G. Gay, The FEP (Free Erythrocyte Protoporphyrins) Test: A screening Micromethod for Lead Poisoning, Pediatrics, 51:254-259. February 1973, and Letters to the Editor, Pediatrics 51:303-306. March 1973.

12. Angle, C. R., and M. S. McIntire, "Red Cell Lead, Whole Blood Lead and Red Cell Enzymes," Paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, North Carolina, October 1-2, 1973.

13. Chisolm, M M., D. Mellits, J. E. Keil and M. B. Barrett, Variations in Hematologic Responses to Increased Lead Absorption in Young Children, paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, North Carolina, October 1-2, 1973.

14. Carson, T. L., G. A. Van Gelder, G. G. Karas and W. B. Buck, Development of Behavioral Tests for the Assessment of Neurologic Effects of Lead in Sheep," paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, North Carolina, October 1-2, 1973.

15. Allen, J. R., P. J. McWey and S. J. Suomi, "Pathobiological

and Behavioral Effects of Lead Intoxication in the Infant Rhesus Monkey," paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, North Carolina, October 1-2, 1973.

V. Relationship of Environmental Lead Exposure to Human Lead Intake

In Section II it was demonstrated that lead emitted from motor vehicles reaches the environment in substantial quanities by several routes including lead suspended in air and fallout of lead into dust and onto soils. This widespread dispersal of lead into the environment resulting from the presence of lead in gasoline, may contribute to man's intake of lead. The routes of intake of lead into man from the environment are through ingested food and non-food items, water, dust, and air inhalation. This present Section examines the extent to which man's lead intake could be affected by lead emissions from motor vehicles and what effect, if any, this may have upon the people so exposed.

Lead is added to gasoline as tetraethyl lead, an organic compound. Exposure to organic lead is dangerous and an important occupational health problem. As indicated in Section II, a very small part of the atmospheric lead resulting from fuel additives is in an organic form (gaseous lead alkyls) and there is little opportunity for general population exposure. General population exposure is much more likely to come from inorganic lead contained in the aerosol particulate emitted from automobiles.

To more fully understand the factors affecting man's lead intake and any resulting biomedical effects, it is useful to discuss the routes of lead absorption, the quantity of lead available through each route, and the differential absorption among the various routes. It is in this context that the sources of lead intake can be examined with respect to the highest amount of total daily lead intake that is generally considered safe or acceptable over a long period of time. For the general population, absorption of lead through the skin is nil. The lead that is swallowed into the gastrointestinal tract is available for absorption regardless of source, but only a small portion is actually absorbed. Similarly, lead inhaled into the respiratory tract is available for absorption through the lungs or from the gastrointestinal tract after it has been moved to the throat by ciliary action and subsequently swallowed. Only a portion of the inhaled lead is absorbed.

Commonly accepted figures for daily dietary lead intake in adults are on the order of 200-300 µg/day with ranges of 100-500 µg/day (1, 2). Gastrointestinal absorption of lead is primarily a function of the chemical composition of the diet and is usually estimated to be 10%, although it has been reported to be lower in adults (3, 4). Young children, however, may absorb considerably more lead, up to 50% of their oral lead intake (5). Recent experiments in animals indicate that diets low in calcium greatly increase lead absorption from the gastrointestinal tract

and affect lead distribution in the body with more lead being stored in the soft tissues (6). Diets low in calcium and low in iron have both been shown to increase lead toxicity in rats fed subtoxic quantities of lead in drinking water (7).

Respiratory absorption of lead is dependent upon the size, shape, and density of the inhaled particles as well as the rate and volume of respiration. For respiratory absorption, 30% is a commonly accepted figure, assuming essentially complete pulmonary retention of deposited lead particles of the size found in ambient air. (8) Other estimates of respiratory lead absorption have ranged from 17% to 37% (9, 10). Thus, for equal amounts of available lead, an average of about three times as much of a given amount of lead would be absorbed from the respiratory tract as from the gastrointestinal tract. Table V-1 summarizes the results of EPA's calculations to estimate the contribution of airborne lead to total daily lead absorption among adults in the general population. A lead isotope tracer technique developed by Rabinowitz et al. has been helpful in further clarifying the relative contribution made by airborne lead to total lead absorption in man (11). Preliminary results from these isotope studies are consistent with the calculations in Table V-1. Since, as noted in Section II, lead in gasoline is by far the largest contributor to airborne lead, the calculations in Table V-1 indicate that lead in gasoline can make a substantial contributicn to lead absorption in man.

In the classic experiments which he summarized in the 1960 Harben lectures, Kehoe fed known amounts of lead to four subjects and monitored their daily intake and output of lead as well as the lead concentrations in their blood and urine. (12) Each subject had a daily diet containing about 300 µg of lead to which additional lead was added in the following quantities: approximately 300 µg (Subject S. W.), 1000 µg (Subject M. R.), 2000 µg (Subject E. B.), and 3000 µg (Subject I. F.). The average daily total intake for each study subject was 600 µg, 1270 µg, 2350 µg, and 3270 µg respectively. Gastrointestinal absorption was determined to be about 10% of the dietary lead dose. In his Harben lectures, Kehoe plotted the average blood lead level for each subject for consecutive 28-day periods during the course of the study. Although Kehoe did not give the regression lines of blood lead versus time for each subject, they can be calculated from the data in his original figure, reproduced as Figure V-1 (12). The source data from Subject S. W. for the calculation of this line are retabulated in Table V-2.

The slope of the regression line for Subject S. W. while ingesting a total of 600 µg of lead each day was significantly different from zero ($t=2.82$, $p < .01$), whereas the slope was not significantly different from zero during his control period. The actual regression line as calculated from the data in Table V-2 and shown in Figure V-2 is as follows:

$$y = 36.5 + 0.70 (x - 7.5)$$

where y = predicted whole blood lead level at time x

and

x = time in 28-day periods

The best fit for these data may be a curvilinear rather than a linear function, in which case, blood leads would level off about 40 µg/100g rather than continuing to increase indefinitely.

This equation predicts that a blood lead concentration of 40 µg/100g would be reached in 12.5 28-day time periods with a 95% confidence interval ranging from 8.9 to 16.1 time periods. In other words, a subject ingesting only a 300ug lead supplement to the 300 ug in his diet would theoretically reach a blood lead level of 40 µg/100g in just less than one year. This prediction may, of course, be dependent upon the relatively high blood lead baseline (30-32 µg/100g) in subject S.W. when supplemental lead feedings were initiated and may not accurately reflect any additional lead intake in subject S.W. due to airborne lead. It should also be noted that this 600ug figure is consistent with the World Health Organization's maximum recommended intake of lead for adults of 50µg of lead/kg/week, the equivalent of 500µg of lead intake daily for a 70kg man (13).

As described in Section III, urinary delta-aminolevulinic acid excretion begins to increase as blood lead content rises above a level of about 40µg/100g. Such an increase in urinary ALA excretion is generally considered indicative of biochemical changes in the tissues that are undesirable and physiologically

significant (14). Based upon Kehoe's data, a daily lead ingestion of 600µg (with an absorption of 60µg) could cause blood lead to increase to a level associated with increased urinary ALA excretion (i.e., 40µg of lead/100g). In this context, one can estimate the additional daily lead intake in the diet or in the air necessary to increase lead in blood to this level.

Calculations of this nature are presented in Table V-3. If average dietary intake is estimated to be 200-300 µg of lead, or an absorbed amount of 20-30 µg daily, an ordinary man would have to regularly absorb only an additional 30-40 µg of lead daily to reach a biologically undesirable blood lead level. Assuming 10% absorption, he could do this by an additional dietary ingestion of 300-400 µg daily, or by combinations of dietary ingestion and inhalation. The main concern here, however, is what air lead exposure level could also result in an increased absorption of 30-40 µg daily?

The daily amount of lead absorbed from inhaled air is the product of: the airborne lead concentration; the volume of air inhaled daily; and the percent of lead reaching the respiratory tract that is absorbed. The airborne lead concentration varies from place to place. The volume of air inhaled daily has been estimated at about 23 cubic meters (m^3) for a "standard man" (weighing 70kg, 20-30 years of age, 175 cm tall, and having a surface area of 1.8 m^2) engaged in light work; about 85% of this

air being inhaled during waking hours (15). Other estimates have been lower, in the range 15-20m^3 of inhaled air daily. As an example, the amount of lead absorbed from breathing air containing 1$\mu g/m^3$ of lead assuming 30% absorption and inhalation of 20m^3 of air each day, is 6.0μg. For a constant dietary lead intake, exposure to increasing airborne lead levels will increase both the absolute amount of lead absorbed by this route and the relative proportion of total lead absorption into the blood which comes from air.

Air levels necessary to increase a man's total daily lead absorption by 30 and by 40μg are estimated in Table V-4. A range of values was calculated by using different parameters for daily air intake and daily lead absorption from the respiratory tract. These calculations indicate that a daily air lead exposure of 5.0$\mu g/m^3$ for a standard man could increase his daily lead absorption by 30μg. Such Ambient air lead levels attributable largely to lead in gasoline have been noted in portions of several U.S. cities at the present time (16).

Experimental data on humans exposed to particulate lead in a chamber support these theoretical calculations (17, 18). The chemical composition and size of the lead oxide particles in these experiments were not identical to those emitted from automobiles; nevertheless, these differences were sufficiently

small so that inferences can be made regarding effects of airborne lead among the general population.

Volunteer subjects in New York State living in a chamber were exposed to airborne lead levels of 10.9µg/m^3 and 3.2µg/m^3 for 23 hours a day in two separate experiments. Average blood lead levels among the men exposed to 10.9µg/m^3 of lead daily increased from about 20 to 37µg/100ml of lead in whole blood after 125 days (18 weeks); average blood lead levels among a second group of men exposed to 3.2µg/m^3 of air lead for 11 weeks increased from about 19 to 25µg/100 ml of lead in whole blood.

These results are compared to those of Kehoe (12) referred to earlier in Table V-5. Estimates of the daily total lead absorption for the study subjects breathing lead at 10.9µg/m^3 and Dr. Kehoe's subject S.W. are shown in this table. The results of both experiments are similar and, while the New York State subjects might have had a somewhat greater total daily lead absorption, their average blood lead levels increased from 20-37µg/100ml in only a little over four months.

The experiment in New York State at lead exposure levels of 3.2µg/m^3 also showed that the men who had been previously exposed to airborne lead at 10.9µg/m^3, responded differently to their subsequent re-exposure, compared to subjects not so previously exposed. (18) The previously exposed men had consistently higher

blood lead levels before and after exposure to 3.2μg/m^3 than the men without such exposure. This observation is consistent with Goyer's recent suggestion that repeated exposure to lead may alter pathways of lead detoxification and excretion (19).

Several epidemiologic studies have investigated the relationship between blood lead levels and airborne lead exposures. A study, commonly referred to as "The Seven City Lead Study", showed a positive but not statistically significant correlation between average blood lead levels and average annual airborne lead exposures ranging in concentration from 0.17 to 3.39 μg/m^3. (20). Exposure was estimated from measurements made at sampling stations generally located within one mile of the homes of subjects being studied. Based upon consistent differences between average blood lead levels in urban and suburban dwellers, the authors concluded, "It is probable that these observations partially reflect lead absorption from ambient atmospheres differing in lead concentration. . . but that factors other than the atmospheric lead level are of relatively greater importance in determining the blood lead levels in population groups." This conclusion was strengthened by further analysis of the study results (21) which found that air lead was a significant though not the most influential factor affecting blood lead levels.

A study by Daines et al. obtained more accurate exposure information by studying the effect of distance of residence from

highways upon blood lead levels among black females (22). Average annual air lead levels on the front porches of homes located 3.7, 38.1, and 121.9 meters away from a highway were 4.60, 2.41, and 2.24 $\mu g/m^3$ respectively. There was no significant difference between the outside air lead concentrations at 38 and 122 meters, but both were different from the air lead concentration at 3.7 meters from the highway. This rapid non-linear decrease in airborne lead with distance from the source has been observed for mobile as well as stationary lead sources.Concentrations of lead in dustfall also decrease rapidly with increasing distance from the source. (23-25) Average annual air lead levels in the front room of houses at the three sampling points also reflected this non-linear decrease being 2.30, 1.50, and 1.57 $\mu g/m^3$ as the distances increased. Average blood lead levels in the study subjects were 23.1, 17.4, and 17.6 µg Pb/100 g at 3.7, 38.1, and 121.9 meters respectively. The average blood lead levels at the two more distant sampling sites differed significantly from the average at 3.7 meters, but did nct differ significantly from each other.

It is of interest to note that among the homes closest to the highway (3.7 meters), air lead and blood lead levels were significantly lower for subjects whose homes were air-conditioned than for similar subjects in homes without air-conditioning. Another finding was that significant differences in air lead, but not in blood lead levels were observed for subjects in homes at

33.4 and 457 meters from a turnpike (22,26). The average outdoor air lead levels at 33.4 meters and 457 meters were 1.95 and 1.73 $\mu g/m^3$; blood leads at these sites averaged 15.7 and 16.1 µg/100g respectively. In this experiment the air lead levels, though statistically different, would not necessarily be reflected in higher blood lead levels, as daily respiratory lead absorption nearer the turnpike would only amount to about 1 ug of lead per day more than at the distant site.

Azar et al. studied thirty male subjects in each of five locations in the United States (27). Air lead levels were measured with personal samplers twenty-four hours a day for two to four weeks and the combined air lead exposure each day ranged from 0.81 - 1.10 $\mu g/m^3$ for office workers in Starke, Florida, and Barksdale, Wisconsin, to 3.06 for office workers and 6.10 $\mu g/m^3$ for taxi drivers in Los Angeles, California. The average daily air lead exposure for a group of Philadelphia taxicab drivers was 2.62 $\mu g/m^3$.

Biweekly blood samples were obtained during the two taxicab studies and the Los Angeles office workers study; at Starke and Barksdale, a single blood specimen for each study subject was obtained at the beginning and at the end of the study. Limitations in the study design were summarized by the authors who wrote, "The use of different occupational groups located in different cities; studying each group at a different time; the

lack of data relative to ingested lead; and the lack of detailed histories and physical examinations are obvious design deficiencies in this study." Average blood lead levels ranged from 13.8 µg/100 g in Barksdale to 24.6 µg/100 g for the Los Angeles taxi drivers. Although the relationship was not linear, average blood lead values were generally positively associated with increasing airborne lead exposure. The study also found that 79-90% of the combined total airborne particulate lead exposure was in the respirable range, which is in agreement with the results from New York City. (28)

Jones et al. studied blood lead and carboxyhemoglobin levels among London taxi drivers on both the day and the night shift (29). Significant differences were found in blood carboxyhemoglobin, but not in blood lead levels, when comparing smoking and non-smoking day-shift drivers to similar night-shift drivers. The authors concluded that the day drivers had a greater exposure to motor car exhaust than the night shift drivers because they had significantly higher blood carboxyhemoglobin levels and therefore "it would seem that little of the lead found in their blood is attributable to the lead they inhale whilst driving in London streets." Unfortunately, both differences in smoking intensity between smoking groups, as well as actual differences in air and dietary lead exposure could explain these results and neither was measured. Furthermore, information was not obtained regarding the exposure of the

drivers when they were off-duty. Thus the authors' original conclusions failed to consider at least three pertinent confounding variables.

Two studies of blood lead levels among non-occupationally exposed people living in smelter communities provide additional data which support a relationship between blood lead levels and exposure to lead in air or dustfall. Average blood lead levels among 10 year old United States school boys living in smelter and non-smelter communities reflected environmental exposure rankings, but no quantitative measurements of lead in air or lead in dustfall were given.(30) However, the study pointed out that lead fallout on home grown produce did not explain the observed differences and concluded that increased lead exposure and absorption probably occurred by the respiratory as well as the gastrointestinal route. A study of adults living around a secondary lead smelter in Finland showed that blood lead levels were negatively correlated with the log of the distance from the source in meters (31). This study did publish isopleths of monthly dustfall lead in relation to the point source, but no air lead values were given. Nevertheless, both studies indicate a qualitative relationship between blood lead levels and exposure to lead in air and in dustfall.

Observed relationships between lead in air and lead in blood did not always fit a simple straight line in the above cited

epidemiologic studies. Using the previously stated assumptions for a "standard man", a person breathing 1 µg of lead/m^3 of air would absorb a total of 6 µg daily from his respiratory tract and a person breathing 3 µg of lead/m^3 of air would absorb a total of 18 µg daily from his respiratory tract. Ultimately this difference in absorption would be reflected in different blood lead levels providing other sources of lead intake were equivalent.

Assuming 10% lead absorption from the gastrointestinal tract and a range of normal daily dietary lead intake from 100-500 µg, total lead absorption from the gastrointestinal tract would range from 10-50 µg each day. The need to measure dietary lead intake in studies of air lead effects is important since one would not be able to distinguish a person (Subject A) breathing 1 µg/m^3 of lead in air and eating 340 µg of lead daily from a person (Subject B) breathing 3 µg/m^3 of lead in air and eating 220 µg of lead daily, viz:

Lead Intake	Subject A	Subject B
Absorbed from air	6 µg	18 µg
Absorbed from diet	34 µg	22 µg
Total Daily Lead Absorption	40 µg	40 µg

Furthermore, variations among individuals with respect to gastrointestinal as well as respiratory lead absorption must be considered in evaluating such data.

Since none of the epidemiologic studies of air lead and blood lead considered both lead in diet and variability of gastrointestinal and respiratory absorption among individuals when evaluating air lead-blood lead relationships, it is understandable why simple straight line relationships between the two variables are not consistently found. As exposure to airborne lead increases, dietary variations would become less likely to confound true differences in blood lead due to air lead exposure.

The above calculations were made for a "standard man;" comparable calculations for women are difficult since respiration and absorption data for women are less complete than those for

men. While the total dietary intake and total daily respiration of women are generally less than those of men, women generally are smaller with proportionately lower caloric and oxygen requirements. Thus the relative relationships with respect to lead absorption from the gastrointestinal and respiratory tracts should be rather similar to those for men. It is even more difficult to extrapolate such calculations to children because they are in a phase of rapid growth and the parameters of interest change in a non-linear fashion. For example, a one year old child has only about 1/7 the body weight of an adult, yet has about 1/3 to 1/4 the total daily air intake ($6m^3$) and about 40-60% of an adult's total dietary lead intake (130 µg)(32). Hence, a child takes in less lead on a total basis, but proportionately more lead on a body weight basis than an adult. At present, the ultimate effect of these differences upon lead intake and absorption in children when compared to adults is not fully known.

Lead from automotive exhaust which is deposited in city dust and dirt also is a potential source of exposure for human beings. A gram of dust or dirt would be only about 1/4 teaspoon, although this may vary somewhat depending on its characteristics. Since these dusts often contain 1000-2000 µg/g of lead or more, the safe intake level of lead for children would be greatly exceeded if only a small fraction of a teaspoon of dust were swallowed daily. (33)

In summary, lead from automotive exhaust contributes to human exposure both from the air and from fallout. Blood lead levels exceeding 40 µg/100 g are considered medically undesirable and may ultimately be harmful. Blood lead levels depend on daily dietary intake and absorption of respired lead. For a "standard man" with average dietary lead intake, exposure to airborne lead concentrations of 5.0 to 6.7 $\mu g/m^3$ could cause his blood lead concentrations to reach 40 µg/100 g within a year. Airborne lead levels near to or in excess of 5 $\mu g/m^3$ have been observed in several U. S. cities.

Lead in dust and dirt poses another important source of exposure, especially for small children. Daily ingestion of relatively small quantities of dirt or dust (less than 1 gram or about 1/4 of a teaspoonful) containing 1,000-2,000ppm of lead would be medically undesirable. Although dustfall exposure to lead is still often considered an hypothesis, much of the available evidence is consistent with this hypothesis. Thus, present day lead exposure to air or dusts in some sections of large cities leaves little or no margin of safety compared to those concentrations associated with biomedical harm.

FIGURE V-1

The average concentration of lead in the blood of subjects S.W., M.R., E.B., and I.F. (from reference 12)

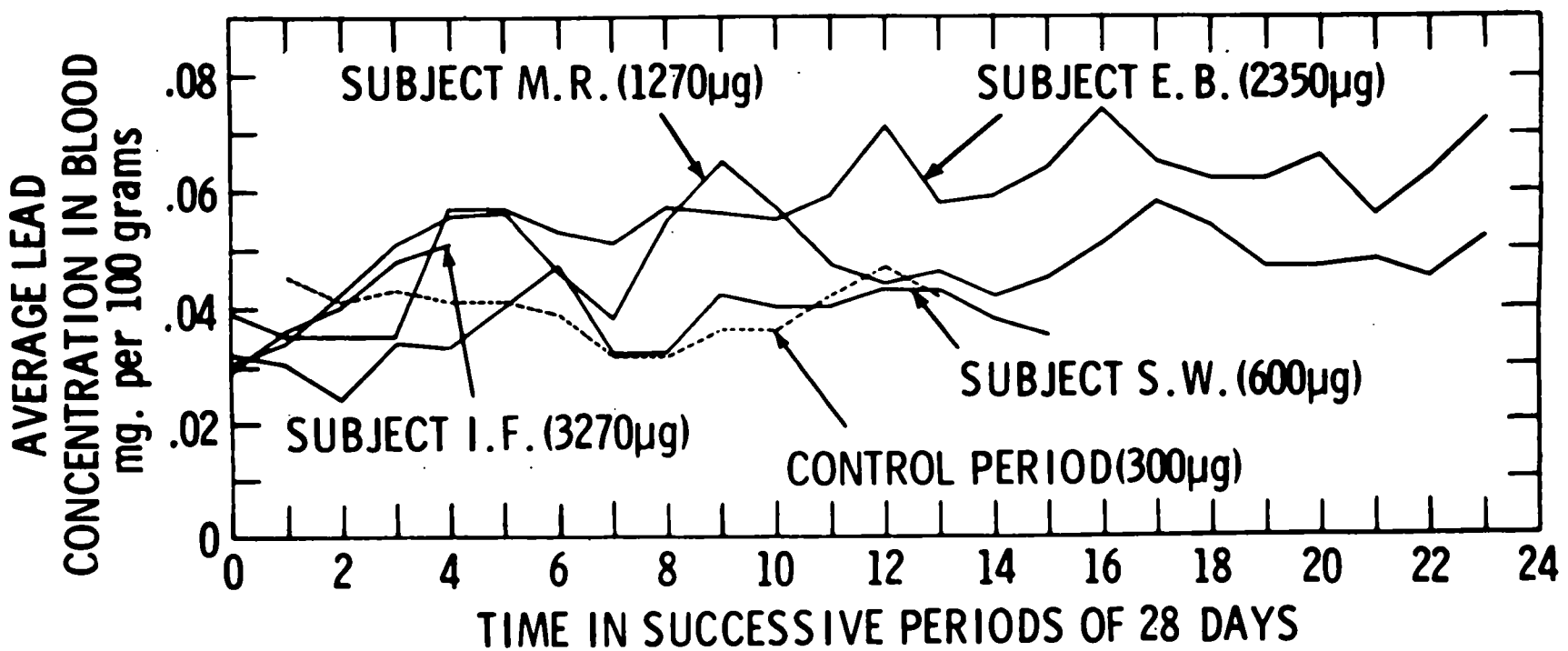

FIGURE V-2

Regression of average blood lead versus time in a subject ingesting a total of 600ug of dietary lead daily (adapted from reference 12)

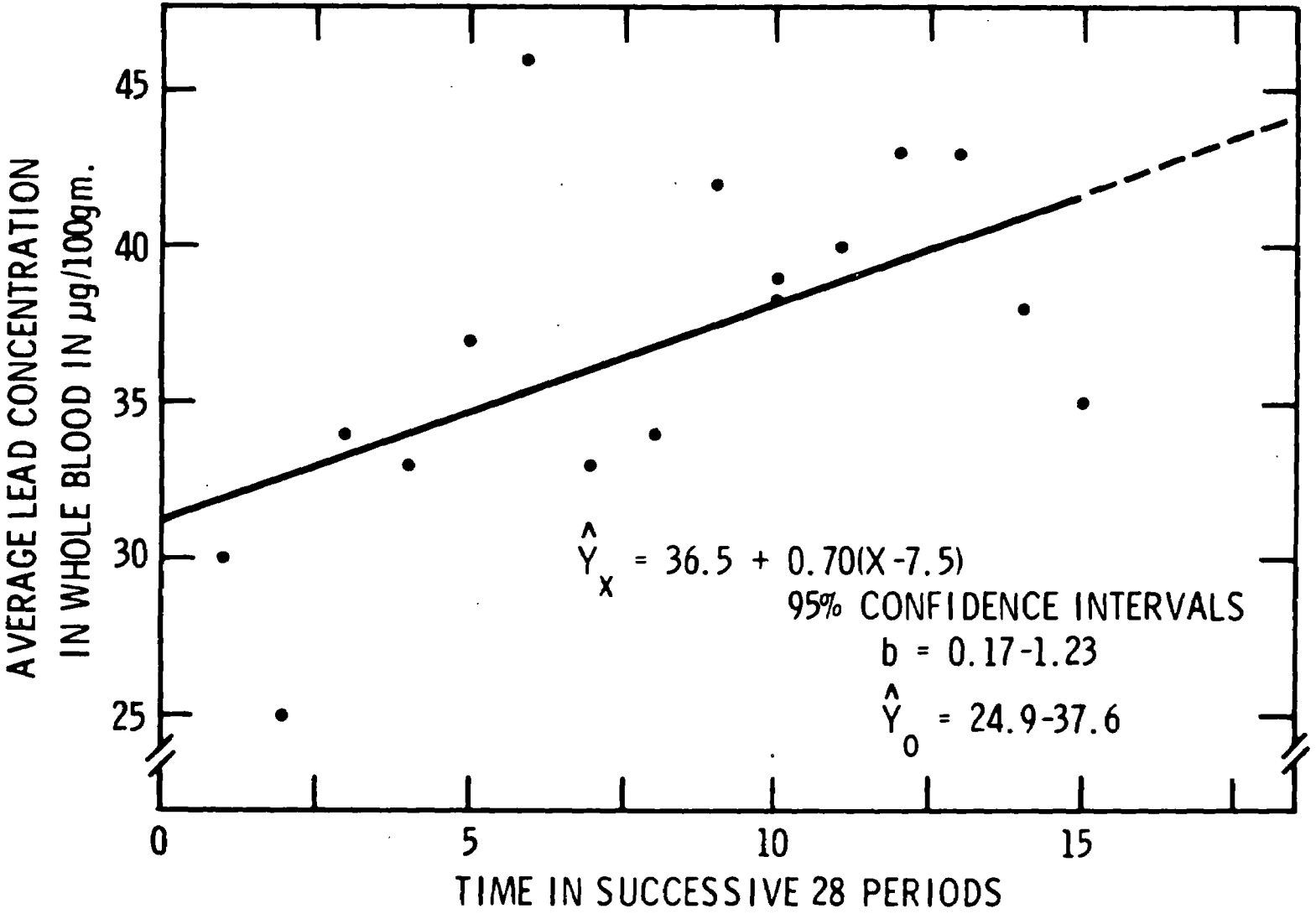

TABLE V-1

Amount of Lead Absorbed from Air
in Relation to
Airborne Lead Concentrations
Among Adults

Lead in Air ($\mu g/m^3$)	Daily Lead Absorbtion		Percent From Air
	From Air+	Total*	
0	0	30	0
1	6	36	17
2	12	42	29
3	18	48	38
4	24	54	44
5	30	60	50

+Assumes 30% absorption and 20m^3 daily respiration.

*Assumes 10% absorption of a daily dietary intake of 300 µg of lead.

TABLE V-2

Regression of Average Blood Lead Level
Versus Time in a Subject* Ingesting a Total of
600 µg of Dietary Lead Daily

x	y
0	32
1	30
2	25
3	34
4	33
5	37
6	46
7	33
8	34
9	42
10	39
11	40
12	43
13	43
14	38
15	35

where:

x = time in 28-day periods

y = average whole blood lead level for a 28-day period in µg/100 g of blood.

$y = 36.5 + 0.70 (x - 7.5)$

$S_x = 4.76$
$S_y = 5.53$
$S_{y.x} = 4.56$

*Reference 12. Subject S. W., average blood lead values taken from Figure 10., Lecture II.

TABLE V-3

Calculated Difference in Daily Lead Intake
Between a Biologically Undesirable Amount and the
Amount in the Normal Adult Diet

	Daily Lead Intake	
	Ingested	Absorbed
Biologically Undesirable Amount+	600 µg	60 µg
Normal Adult Diet	200-300 µg	20-30 µg
Difference	300-400 µg	30-40 µg

+Continued daily intake of this amount could cause blood lead levels to rise to about 40 µg/100 g of whole blood with the consequent increased excretion of delta-aminolevulinic acid in the urine.

TABLE V-4

Estimated Airborne Lead Exposures Which Could Cause
Adult Male Blood Lead Levels to Reach
40 µg/100 g of Whole Blood

Common Daily Dietary Lead Absorption	Increase Required to Reach 60 µg Daily+	Airborne Lead Exposures Which Would Cause the Designated Increase		
		Standard(a) Man	Least(b) Case	Highest(c) Case
20 µg	40 µg	6.7 µg/m^3	15.7 µg/m^3	5.4 µg/m^3
30 µg	30 µg	5.0 µg/m^3	11.8 µg/m^3	4.0 µg/m^3

+Experimentally in a human, absorption of 60 µg of lead daily caused his blood lead levels to reach 40 µg/100 g of whole blood. (12)

(a) Standard Man = 20m^3 x 30% absorption
(b) Least Case = 15m^3 x 17% absorption
(c) Highest Case = 20m^3 x 37% absorption

TABLE V-5

Estimated Daily Total Lead Absorption
in Two Experimental Studies

Exposure Source	Daily Lead Absorption	
	New York (17,18) Chamber Exposure	Kehoe's Subject (12) S.W. Exposure
Diet	10 µg*	30 µg*
Experimentally Added	49-60 µg+ (Air)	30 µg* (Diet)
Daily Total Lead Absorbed	59-70 µg	60 µg

*Assumes 15m^3 daily respiration (since the men in both experiments were sedentary) and 30-37% absorption of 10.9 ug/m^3 of lead in air.

*Assumes 10% absorption of dietary intake.

REFERENCES

1. Joint FAO/WHO Expert Committee on Food Additives; "Evaluation of Certain Food Additives and the Contaminants Mercury, Lead and Cadmium; WHO Technical Report Series, No. 505, 1972.

2. Airborne Lead in Perspective, A Report Prepared by the Committee on Biologic Effects of Atmospheric Pollutants, National Academy of Sciences, Washington, D. C., 1972 p.50.

3. Airborne Lead in Perspective, op.cit., pp. 52-53.

4. Thompason, J.A.; Balance Between Intake and Output of Lead in Normal Individuals. Brit. J. Industr. Med., 28:189-194, 1971.

5. Alexander, F.W., H.T. Delves, and B.E. Clayton, the uptake and excretion by children of lead and other contaminants, Proceedings of An International Symposium on the Environmental Health Aspects of Lead, CEC, CID, Luxembourg, May 1973, pp. 319-331.

6. Goyer, R.A., and B.C. Rhyne, "Pathological Effects of Lead," Int'l. Rev. Exp. Path., 1973, 12:pp. 1-77.

7. Ibid.

8. Airborne Lead in Perspective, op.cit., pp. 65-67.

9. Cole, J.; International Lead Zinc Research Organization, New York City; Testimony presented to EPA, Dallas, Texas, April 28, 1972.

10. Airborne Lead in Perspective, op.cit., pp. 65-67.

11. Rabinowitz, M.A., G.W. Wetherill, and J.D. Kopple, "Study of lead metabolism using stable isotope tracers," paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, N.C., Oct 1-2, 1973 and M. B. Rabinowitz, G. W. Wetherill and J. D. Kopple, "Lead Metabolism in the Normal Human: Stable Isotope Studies," Science 182:725-727, November 1973.

12. Kehoe, R.A.; The Metabolism of Lead in Man in Health and Disease. The Harben Lectures, 1960. McCorquodale and Co., Ltd., London, England, 1961. 81 pages. Reprinted from the J. Roy. Inst. Public Health Hyg.

24:81-97, 101-120, 129-143, 177-203; 1961.

13. Joint FAO/WHO Expert Committee on Food Additives, op.cit.

14. Airborne Lead in Perspective, op.cit., p. 110.

15. Airborne Lead in Perspective, op.cit., p. 51.

16. Shearer, S.D., EPA; Testimony presented at EPA Public Hearings on Regulation of Fuels and Fuel Additives, Los Angeles, California, May 2-4, 1972.

17. Knelson, J.H., R.J. Johnson, F. Coulston, L. Goldberg and T. Griffin; "Kinetics of Respiratory Lead Intake in Humans:" Proceedings of the International Symposium on Environmental Health Aspects of Lead;" CEC, CID, Luxembourg, May 1973, pp. 391-401.

18. Knelson, J.H., F. Coulston, L. Goldberg and T. Griffin; The Role of Clinical Research in Establishing Standards for Atmospheric Lead. Presented at the Conference on Heavy Metals, VDI-Kommission Reinhaltung der Luft, Dusseldorf, Germany, February 22, 1973.

19. Goyer, R.A.; "Lead and the Kidney" in Current Topics in Pathology, Springer-Verlag, Berlin, 55:147-176, 1971.

20. Tepper, L.B. and L.S. Levin; A survey of Air and Population Lead Levels in Selected American Communities. Department of Environmental Health, College of Medicine, University of Cincinnati, Cincinnati, Ohio. Final Report (Contract PH-22-68-28), December 1972. 73 pages.

21. Hasselblad, V. and W.C. Nelson; Additional Analyses of the Seven City Lead Study. Human Studies Laboratory, National Environmental Research Center, U.S. Environmental Protection Agency, Research Triangle Park, N.C. In-house Technical Report, March 15, 1973.

22. Daines, R.H., D.W. Smith, A.F. Feliciano and J.R. Trout; Air Levels of Lead Inside and Outside Homes. Ind. Med. Journal 41:26-28, October 1972.

23. Airborne Lead in Perspective, op.cit., p. 29.

24. Creason, J.P., O. McNulty, L.T. Heiderscheit, D.H. Swanson and R.W. Buechley; Roadside Gradients in Atmospheric Concentrations of Cadmium, Lead and Zinc. Trace Substances in Environmental Health V. A Symposium, 1972. pp. 129-142.

25. Helena Valley, Montana, Area Environmental Pollution

Study, U.S. Environmental Protection Agency, Office of Air Programs, Research Triangle Park, N.C., January 1972. OAP No. AP-91, xiv + 179 pages.

26. Trout, J.R.; Statistics Center, Rutgers University, New Brunswick, N.J. 08903. Personal communication to Dr. K. Bridbord, U.S. ENvironmental Protection Agency, March 21, 1973.

27. Azar, A., K. Habibi and R. Snee; Relationship of Community Levels of Air Lead and Indices of Lead Absorption; Proceedings of an International Symposium on Environmental Health Aspects of Lead; CEC, CID, Luxembourg, May 1973, pp.581-594.

28. Ferrand, E.F., Assistant Commissioner for Scientific Affairs, New York City Department of Air Resources, New York City, New York. Testimony submitted to EPA, April 11, 1972.

29. Jones, R.D., B.T. Commins and A.A. Cernik; Blood Lead and Carboxyhemoglobin Levels in London Taxi Drivers. Lancet, 302-303, August 12, 1972.

30. Hammer, D.I., J.F. Finklea, R.H. Hendricks, T.A. Hinners and W.B. Riggan; Trace Metals in Human Hair as

a Simple Epidemiologic Monitor of Environmental Exposure. Trace Substances in Environmental Health V. A Symposium. D.D. Hemphill, Ed. University of Missouri, Columbia, 1972. pp. 25-38.

31. Nordman, C.H., S. Hernberg, J. Nikkanen and A. Ryhanen; Blood Lead Levels and Erythrocyte Delta-Aminolevulinic Acid Dehydratase Activity in People Living Around a Secondary Lead Smelter. Work - Environm. - Hlth 10:19-25, 1973.

32. King, B.G.; Maximum Daily Intake of Lead Without Excessive Body Lead Burden in Children. Amer. J. Dis. Child. 122:337-339, 1971.

33. Ibid.

VI. Lead Exposure from Dustfall

Recent surveys of urban environments reveal substantial levels of lead contamination in dust and dirt found in streets as well as in homes. This contamination represents a potential source of lead exposure, especially for children. Weathering of leaded paint from buildings is a major contributor to lead in dust and dirt (1-4), as is fallout of airborne lead from other stationary and mobile lead sources. At the moment it is not possible to give an overall figure quantifying the contribution that each of these sources makes to lead in dirt and dust. The discussion to follow summarizes what is known about these contributions as well as the implications of this contamination.

Lead content of dirt at various distances from houses and roadways in Detroit was examined by Ter Haar, et.al. (5). Lead content of dirt two feet from painted frame houses averaged 2,010 ppm lead, while, dirt two feet from brick houses with painted trim averaged 468 ppm lead. Street gutter dirt near these homes averaged 966 and 1,213 ppm lead, respectively. Lead content of dirt decreased with increased distances from both roadways and houses. Lead concentrations in dirt at corresponding distances from frame houses were similar in city and rural yards. Based upon these findings, it was concluded by Ter Haar that lead antiknocks did not contribute significantly to the lead content

of dirt in urban areas. (5) This conclusion, however, does not adequately reflect the effect of proximity to roadways upon dirt lead content that was found. Further, these observations were made in areas with relatively low traffic density and would not be applicable to all urban situations.

In this context, it has been shown that airborne lead fallout decreases with increased distances from roadways (6), implicating lead in gasoline as a contributor to this problem. The effect of proximity to roadways upon lead levels in soils has previously been demonstrated (7). Lead levels in dust samples taken inside homes located within 5 meters from heavily travelled roadways averaged nearly 1000 ppm (0.1%) lead, or approximately double the level in samples from homes located on side streets (8).

Lead concentrations in surface soils of city parks have been found to range from 194 to 3,357 ppm and lead levels in dust from residential and commercial sites in midwestern cities averaged, 1,636 and 2,413 ppm, respectively (9). Dust lead levels in street sweepings from Washington and Boston usually ranged from 0.1 to 0.25% and at times exceeded 0.5% (10,11).

Lead levels in dust inside homes of Boston have ranged from 0.1-0.2% (12) and have exceeded 0.5% in Birmingham, England (13). Samples of dust from inside elementary school classrooms in

Philadelphia and dirt from playgrounds adjacent to those schools routinely contained 0.2-0.3% lead, with levels greater than 1% found at two locations (14). From these studies it is not possible to determine the relative contribution that erosion of lead based paint compared to airborne sources made to lead in housedusts. In some of the measurements in Philadelphia, contamination by lead fallout from industrial sources was believed to have occurred.

Concentrations of lead in house dust have been observed to vary with the amount of lead fallout from the air (15). House dust lead levels taken from middle income homes in two New York City communities which were likely free of peeling lead paint averaged 608.6 ppm and 741.5 ppm, respectively. These values were 2-3 times higher than the mean values from homes in a control community where dustfall lead was about 1/10 - 1/5 as great. In this study, lead in housedust as well as lead in soil from each community followed the dustfall gradient implicating airborne sources largely due to lead in gasoline as important contributors to this contamination. Erosion of lead based paint could, of course, also have been a contributing factor. Lead concentrations in hair among subjects living in these homes (adults and children combined) were not found to consistently follow these exposure differences. However, additional analyses are necessary to determine if lead exposure was different depending on age within each area and if hair lead levels

reflected exposure differences among the young children in different areas. It should be noted that hair leads among children in this study may not strongly reflect housedust lead exposure differences since opportunities to place dust contaminated objects into their mouths during unsupervised activities may be much lower for middle income than for low income children.

To date, ingestion of lead-contaminated dirt or dust has rarely been suspected of causing clinical lead poisoning, but this source of lead generally has not been investigated in cases of excessive lead exposure. A report from Charleston, South Carolina has shown that soil from yards of homes where cases of pediatric lead poisoning occurred contained greater concentrations of lead than soil in yards of randomly selected homes (16). The cases of lead poisoning also were most frequent in areas of the city with high soil lead concentrations. A quantitative assessment of the sources responsible for high soil lead concentrations in Charleston is not possible from this study. In particular, weathering of outdoor paint as well as ashes, pesticides and automotive exhausts were all believed to have contributed. This study concluded that " . . . these facts further support the theory that children in Charleston can get lead poisoning by eating dirt."

In contrast to these findings, a study from England has shown that average blood lead levels in children residing in an area with soil containing 10,000 ppm lead were only 4 µg/100 ml higher than in children from a community with soil containing 500 ppm lead (17). Why children in Charleston, but not in England, appeared significantly affected by residence in areas with high scil lead content is not certain. Perhaps this difference was due to a greater incidence of pica among children in Charleston or the more frequent presence of additional sources of lead in Charleston such as leaded paint. Differences in biologic availability of lead bound to soil in England compared to Charleston may also be important. Lead in dirt and dust which is not tightly bound to soil likely has greater biologic availability than lead which is bound to soil. It should also be noted that lead in the form of fine dusts may be more readily absorbed from the gastrointestinal tract than comparable quantities of lead in paint chips due to the vastly increased surface area in these fine dusts.

In this context, rats fed lead-contaminated top soil gathered near a lead smelter in El Paso at a dose of lmg lead/day for 28 days, showed blood lead increases of 20 µg/100ml over controls on a low lead diet (18). Similar results were seen after feeding rats lead contaminated dust (1-2% lead) from city highways and automobile tunnels (18). Lead contaminated dirt (6600 ppm) from expressways in Chicago has also been noted to increase blood and

soft tissue lead content after being fed to rats (19). These results suggest that lead in dust, both that gathered near the lead smelter and that originating from emissions of automobiles burning lead additives, although of different chemical composition and perhaps somewhat different particle size, is comparably absorbed when fed to experimental animals.

In recognition of the possibility that ingestion of lead-contaminated dirt and dust could contribute to pediatric lead exposure, the National Academy of Sciences Lead Panel concluded (20):

> "The swallowing of as much as 1g of such dust could result in the oral intake of an amount of lead that exceeds by a factor of 10 or more the estimated mean daily intake of lead from normal food and drink in nonexposed young children . . . For a child with pica for paint, the combination of the ingestion of a few chips of paint and an increased intake of lead from contaminated dusts would provide a total lead intake sufficient to cause symptomatic illness."

There are many factors which place young children potentially at a greater risk to lead exposure from dust than adults. The behavioral patterns of children expose them to large amounts of dust. This was demonstrated in a study from El Paso, Texas (21). In a neighborhood near a lead smelter, with high concentrations

of lead in air (5µg/m^3) and in surface soil (0.1-0.5%), blood lead levels of both children and adults were found to be elevated. Air lead, blood lead and surface soil lead concentrations all decreased with increased distance from the smelter implicating this source as the probable causal factor. Under these circumstances, 80% of the children between 1 and 4 years of age had blood lead levels in excess of 40µg/100g, a frequency much higher than seen among older children and adults. This suggests a special exposure route for young children, not only in dry areas where little or no vegetation exists to hold outdoor dust in place, but also in typical urban situations (25, 38, 43).

Preliminary data suggest that airborne lead concentrations tend to be greatest at ground level, and to decrease with increased elevation (8,22). Therefore, young children, with their shorter height, may be exposed to greater amounts of airborne lead and lead-contaminated dust than older, taller individuals. The normal play activities of children can include placing materials or their hands, contaminated by leaded dusts, into their mouths (23-25). A moistened lollipop dropped to the ground can pick up 0.5g of dirt (22), which would contain 2,500µg of lead if the dirt contained 0.5% lead. This quantity of lead is an order of magnitude greater than the amount of lead routinely found in the daily diet of children.

A Rochester, New York study has demonstrated that lead contamination on hands of young children living in homes with high housedust lead levels is significantly greater than on hands of children living in homes with low house dust lead levels (25). The lead level in household dust was also significantly elevated in inner city homes where at least one child with moderately elevated blood lead resided compared to suburban homes. Since blood leads were not determined in all subjects, it is not known with certainty whether more lead on the hands was associated with increased blood leads in these children. These data are consistent with the frequent contamination of finger prick blood lead specimens by lead on hands of children indicating the widespread occurrence of such contamination. (25)

These data do not firmly establish the sources responsible for the elevated lead concentrations in urban housedust. Of note is that pre-1940 homes had greater quantities of lead in housedust than post-1940 homes (25). This observation was also made in Boston (11). Though this difference could in theory be accounted for by higher concentrations of lead in paint in older homes, it could also be explained by greater ventilation and increased lead fallout in older homes especially since newer homes are more frequently air conditioned (11). Peeling lead paint was likely not the sole contributor to lead in housedust in the Rochester homes studied since an association was not found between the presence of peeling lead based paint in homes and

increased lead in house dust. This in part could have been due to residual leaded dusts from paint which were ground into the floor prior to repainting of deteriorating surfaces or from deterioration of repainted surfaces themselves. With the available data, one cannot quantitatively assess the relative contribution of erosion of lead paint and of other lead sources to the total lead content of house dust. As noted earlier, lead in housedust is a function of airborne lead fallout and proximity of homes to roadways.

In separate Rochester surveys (25) average blood leads among 3-4 year old urban children (about 40µg/100g) were approximately double those among suburban children. Blood leads much above 60 µg/100 g were not common in the urban children, which would be expected if ingestion of paint flakes containing very high quantities of lead were the primary mechanism of exposure. Nearly 50% of these inner city children had blood lead levels between 40 and 60µg/100g implicating sources of exposure less massive than that routinely found in chips of paint. There were no known industrial sources of lead pollution in the inner city study area and high lead levels in water were also ruled out as an important factor. To the extent that airborne lead fallout, in large part due to automobiles, is greater in urban compared to suburban areas, lead in gasoline would have contributed to lead in housedust found in these Rochester homes. Traffic density in this study area was relatively low so the contribution of lead

from gasoline was no doubt less than in areas with high traffic density. These data suggest that ingestion of leaded dusts were in part responsible for increased lead exposure among Rochester children. This study does not quantify the contributors to lead in housedust whether from airborne sources, chalking or peeling of paint, or perhaps pulverization underfoot of paint chips shed from the walls.

Lead-210, an isotope usually present at very low levels in leaded paint and in significantly higher concentrations in fallout dust was employed to evaluate the relative quantities of lead containing dust and paint ingested by young children (5). The hypothesis examined was that if a child with an elevated lead body burden had high stable lead but normal level-210 in his feces, then lead elevation was probably the result of paint ingestion. If high stable lead and high lead-210 were found in the feces, then this would favor lead in dust and dirt as contributing to the lead body burden.

Eight children suspected of having elevated lead body burdens were compared to ten children living in good housing where lead poisoning was not a problem. Fecal excretion of both stable lead and lead-210 was determined in both groups of children. The children suspected of having elevated lead body burdens, with two exceptions who were normal, had stable lead concentrations in their feces that were 4 to 400 times higher than found in the

normal children. There was, however, no difference in lead-210 fecal excretion between groups. Based upon these findings, it was concluded that children suspected of elevated lead body burdens did not ingest dust.

The effect that lead-210 in the diet or in the air could have had upon these results was not adequately considered in this study. Unless lead-210 intake from the diet and the air were known to be equivalent in each group, no firm conclusions are warranted regarding non-dietary sources of lead-210. Unfortunately, no measurements of lead-210 in the diet were made, which for adults in the United States range from 1-2 picocuries/day (26-28). Airborne sources which have been estimated to contribute 8-32% (28) and in one instance nearly 50% (27) of total daily lead-210 absorption into the blood were also not measured. From the data presented (5), one can calculate that the fecal excretion of lead-210 by these children was generally less than 1 picocurie/day. Except for one child who almost certainly had recently ingested paint chips, fecal stable lead excretion in the group suspected of elevated lead body burdens averaged only about 300 µg/day or 5 times greater than in the normal children. This level of lead excretion is far below the 44,000 µg/day associated with lead poisoning from ingestion of paint chips (2). Though these findings do not rule out ingestion of paint as dust or ingestion of paint chips prior to fecal lead determinations in these children, they also do not

rule out ingestion of dirt or dust contaminated by lead sources other than paint prior to examination.

An additional important observation is evident from this study. The lead-210 which was used as a tracer is a product of radon decay in the atmosphere. Lead-210, thus produced, attaches to air suspended particulates including lead from automobile exhausts as well as from other sources. The fact that relatively high concentrations of lead-210 originating in the atmosphere were found in vacuum cleaner sweepings, in yard dirt and in street dirt means that atmospheric particulate fallout, known to routinely contain stable lead, was contaminating these samples to some degree.

Extensive screening programs in the United States concentrating on inner city children, have demonstrated that approximately 25% of these children were significantly exposed to lead (blood lead levels of 40µg/100g or above) (29-31). Lead-based paint has long been recognized as the principle source of lead overexposure among young children. However, it is not clear that lead-based paint alone accounts for this widespread degree of exposure.

For example, screening programs have detected high levels of lead exposure in children residing outside the central city, where exposure to lead based paint would be expected to be less

than in the central city environment (24, 32, 33). In a study from Oregon, 50% of children with elevated blood lead levels were found to live in housing where lead paint was not considered accessible to them (34). In New York City, only 50% of children with blood lead levels between 35 and 44µg/ 100g could be associated with living in homes containing peeling paint of 1% or greater lead. Nearly 20% of this group lived in homes in which no peeling paint was identified (35).

The source of excessive lead exposure is thus not always directly related to paint in a significant percentage of cases. This situation may reflect exposure to lead paint in former homes, or in places other than those inspected. Further, closer or repeat inspection will often reveal sources of peeling lead paint in the home which were not initially detected. Additional sources of lead in food, water, pottery, toys, pencils, solder, etc., are also possible factors in cases of unidentified routes of exposure. Lead contaminanted dust and dirt, especially prevalent in urban areas, may also be an important contributing factor. Further information is required to arrive at a definitive assessment of the significance of this source of lead contamination.

It is clear that peeling lead based paint remains an exceedingly important problem, and even exists outside of inner city areas (36). In one New York City study, 100% of apartments

in a high risk lead poisoning neighborhood showed high lead levels on paint surfaces (37). These studies, and extensive clinical experience, indicate that leaded paint is primarily responsible fcr the great majority of overt clinical lead poisoning in children, although non-paint lead sources would decrease the amount of lead from paint required to cause serious damage. Education and screening programs have been noted to decrease the reported frequency of clinical lead poisoning in children (38). Whether or not these screening programs are sufficient in themselves to adequately reduce the frequency of undue lead exposure among children is less certain.

For example, in the initial year of the Chicago lead screening program, 8.5% of children tested had blood leads of 50 µg/100 ml or more (38). In 1968 the fraction of children with blood leads in this range dropped to 3.8% and in 1969 to 1.5%. Figures for 1970 and 1971 were 2.1% and 2.3% respectively. Hence, despite an initial decline and a continued and vigorous program to decrease lead paint exposure, the percentage of children tested with blood leads of 50 µg/100 ml or above has either remained relatively constant or may have actually increased slightly in recent years. Statistics for 1972 confirm this increasing trend and show that 4.1% of children screened that year had blood lead levels of 50 µg/100 ml or above (42). Thus, though efforts to reduce lead paint exposure continued, the reported frequency of undue lead exposure but not severe lead

poisoning appears to be increasing in Chicago. These findings are unexplained by changes in method of blood lead collection or analytical techniques because these procedures have not been altered since the beginning of the Chicago screening program in 1967. This increasing trend is particularly significant since screening programs initially concentrate in highest risk areas for lead paint and then at least to some degree gradually expand to include other areas of lesser risk. These statistics indicate the continuing need for vigorous action to prevent lead paint exposure and also suggest that sources of lead other than paint are having an incremental effect upon lead paint exposure in Chicago.

There has admittedly been considerable controversy as to whether or not partial or even total removal of lead from gasoline would have any impact upon the lead exposure problem in this country (39,40). In this context, children residing in pre-World War II housing have been observed to have higher blood lead levels than children residing in newer housing projects. One such survey conducted in Washington, D.C. during 1970 indicated that the lowest blood lead levels among children tested for lead poisoning were found in areas of the city where public housing projects had been built since 1942 (43). No child living in a recently built housing project, where exposure to lead based paint would be minimum, had a blood lead level above 40 µg/100g. Blood lead determinations in this survey of 808 children showed

that 5.8% of all children tested had a blood lead of at least 40 µg/100g and that nearly 10% of children between 1-3 years of age had blood lead levels in this range. It should be noted that these results are somewhat lower than those from subsequent Washington, D.C. surveys in which 22.0 and 39.2% of children tested were found to have blood lead levels of 40 µg/100g or above (44,45).

This original Washington survey does indicate that the frequency of lead poisoning and undue lead absorption in children would be greatly reduced if lead paint were nearly eliminated. These findings however, do not rule out an important contributing role for sources of lead other than paint among children residing in home environments where lead paint is present.

For example, location of residences near urban roadways and in areas of high traffic density has been shown to significantly affect the degree of undue lead absorption among children also exposed to paint (46). In this study, the effect of residential location upon blood lead was examined retrospectively among over 5,000 children tested for lead poisoning in Newark, New Jersey during 1971. Among those children residing within 100 feet of a major roadway, 49.3% were found to have blood lead levels between 40-59 µg/100 ml and 8.1% had blood lead levels of 60 µg/100 ml and above. Children residing 100-200 feet and greater than 200 feet from major roadways were not significantly different from

each other. Approximately 25% of blood leads in both groups were between 40-59 µg/100 ml and 3-5% were at least as high as 60 µg/100 ml. When these groups, however, were compared to children residing within 100 feet of major traffic arterials a marked difference was observed with twice the frequency of undue lead absorption found in children living nearest the roadway.

In children residing within 200 feet of a major traffic artery the extent of undue lead absorption was observed to increase when average weekday traffic densities exceeded 24,000 vehicles per day. In such instances 51.3% of children had blood leads between 40-59 µg/100 ml and 10.8% of children had blood leads of 60 µg/100 ml or above. Comparable rates for children residing in areas with weekday traffic densities below 24,000 vehicles per day were 36.6% with blood leads between 40-59 µg/100 ml and 5.1% with blood leads of at least 60 µg/100 ml.

These results could conceivably be confounded by existence of poorer quality housing nearest roadways and by racial and social class differences among residents near major roadways. However, the fact that no difference was noted in blood leads among children residing within 100-200 feet of roadways compared to children residing beyond 200 feet suggests that these other factors were likely not major confounding variables.

Earlier studies in Newark found positive and statistically significant,but not overwhelming correlations,between elevated blood leads and indices of housing status as reflected by number of housing units built prior to 1950 and the number of housing units considered deteriorated and dilapidated in the census tract. (47) Correlation coefficients for these indices and rates of excessive lead absorption (blood lead of 60 µg/100g and above) and elevated blood lead (40 µg/100g and above) ranged from 0.35 to 0.49. The relatively low correlations which were observed suggests that factors other than housing status also were important. For example, in seven census tracts with low potential hazard scores based on housing status, high rates of excessive lead absorption were observed.

Findings such as these from Newark indicate that factors other than housing are important in lead poisoning and that automotive lead emissions can contribute significantly to undue lead absorption in children. Because immediate removal of old lead paint from all existing structures cannot practically be accomplished,reduction of other sources of lead exposure is important. If the results from Newark, New Jersey are in any way applicable to other urban situations, this would indicate that automotive lead emissions do have a significant incremental impact upon the lead exposure problem in this country.

In a study in Philadelphia, referred to earlier (14), 8.4% of white first grade children who lived and attended private school in proximity to a major industrial lead source were found to have dentine lead levels in excess of 300 µg/g, a level associated with frank lead poisoning. These children resided in older housing that was generally in good repair but were exposed to levels of lead in environmental dusts commonly in the range of 3,000-8,000 ppm. The community in which these children lived was also heavily exposed to automotive lead emissions. In contrast to these children, 19% of black students attending public school in the same district but who lived in deteriorating housing had dentine lead levels in excess of 300 ug/g. When both groups of children were compared to public and private school children living in a community free of industrial lead sources and residing in housing built since World War II striking differences in dentine lead levels were noted. Only 3% of the public and 6.6% of the private school students in the low exposure community had dentine lead concentrations in excess of 100 µg/g, whereas 66% of the black public school students and 43% of the white private school students in the high exposure area had dentine lead levels greater than 100 µg/g.

Interior dust samples from the public and private schools in the low exposure community contained an average of 614 ppm lead (range 293-939 ppm) compared to interior dust samples from schools in the high exposure community which contained an average

of 3,867 ppm lead (range 929-15,680 ppm). Samples of gutter dirt in the low exposure community contained an average of 1,507 ppm lead (range 270-2,626 ppm) compared to an average of 3,262 ppm lead (range 280-8,201 ppm) for gutter dirt samples in the high exposure community. These data from Philadelphia are suggestive that airborne and dust lead largely from industrial sources can contribute to undue lead absorption in typical urban areas either by themselves or when combined with paint from deteriorating housing.

Studies from Philadelphia, Chicago and Newark discussed above provide persuasive evidence to strongly suggest that sources of lead other than paint, including that resulting from the presence of lead in gasoline, play an important role in childhood lead exposure. These other sources may be especially significant at levels of exposure below overt clinical poisoning.

The National Academy of Sciences summarized available information on the ingestion of lead by children in the following paragraph from its (1972) report (41):

> "The extent to which airborne lead in congested urban areas contributes to increased lead absorption and lead poisoning in children is not clearly defined. With respect to young children, it is not a matter exclusively of inhalation and particle size, inasmuch as young children mouth and actually

eat things that are not food rather indiscriminately. Airborne lead wastes from such sources as automotive emissions and the weathering and demolition of old buildings can be expected to have a significant additive effect on the total intake. This would be sufficient to evoke compensatory metabolic responses that are now considered subclinical (such as increased urinary ALA), at the very least. It may be estimated that dustfall from airborne lead, if swallowed, can make a significant contribution to a small child's total lead intake and thereby contribute to the occurrence of lead poisoning, especially in urban areas. Even so, the direct ingestion of lead-pigment paints is clearly the principal environmental source in cases of severe acute lead poisoning in young children."

In conclusion, clinical experience indicates that leaded paint is primarily responsible for the great majority of overt clinical lead toxicity in children. There is, however, sufficient data to strongly suggest that sources of lead other than paint play an important role in childhood lead exposure. These other sources may be especially significant at levels of exposure below overt clinical poisoning.

Lead in the air, and particularly lead in dust are ubiquitous sources of lead which may well be important contributing factors to the problem. Exposure to dirt and dust sufficiently

contaminated by lead could reduce significantly the quantity of additional lead exposure required to produce clinical poisoning in a child with other sources of exposure. Though exposure to lead contaminated dirt and dust from automobile exhaust, alone, has not been shown to be responsible for cases of overt lead poisoning, automotive lead has been related to undue lead absorption in children. At this time, it would be prudent to decrease the potential air and dust lead exposure. It should be recognized that further studies are necessary to better quantitate the sources of lead contamination in dust and dirt and the magnitude of the contribution that leaded dust and dirt make to both subclinical and clinical lead overexposure.

REFERENCES

1. Hardy, H., et al., "Lead as an Environmental Poison," Clinical Pharm. and Ther., 12:982-1002, November 1971

2. Comments submitted by Ethyl Corporation to the Environmental Protection Agency, March 9, 1973. pp. V-14 - V-19

3. De Treville, T.P., "Natural Occurrence of Lead," Arch. Env. Health, 8:212-221, February 1964

4. Bertinuson, J.K. and Clark, C.S., "The Contribution of Lead Content of Soils from Urban Housing," Interface, 6: p. 1073, 1973

5. Ter Haar, G. and R. Aronow, "New Information on Lead in Dirt and Dust as Related to the Childhood Lead Problem," paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, N.C., Oct 1-2, 1973

6. Creason, J.P., O. McNutty, L.T. Heiderscheit, D.H. Swanson, and R.W. Buechley; "Roadside Gradients in

Atmospheric Concentrations of Cadmium, Lead and Zinc," Trace Substances in Environmental Health V. A Symposium, 1972 pp. 129-142

7. Airborne Lead in Perspective, A report prepared by the Committee on Biologic Effects of Atmospheric Pollutants, National Research Council, National Academy of Sciences, Washington, D.C. 1972, p.29

8. Darrow, D.K. and Schroeder, H.A., "Childhood Exposure to Enviromental Lead," paper presented before the American Chemical Society, Chicago, Ill., Aug. 29, 1973

9. Airborne Lead in Perspective, op. cit., p. 30

10. Fritsch, A. and M. Prival, Center for Science in the Public Interest, Washington, D.C., testimony submitted to EPA, 1972

11. Krueger, H. Krueger Enterprises, Cambridge, Massachusetts, testimony submitted to EPA, July 10, 1972

12. Krueger, H. Op. cit.

13. Personal communication to L. Lombardo, Public Interest Campaign, from R. Stephens and A. Townshend, University of Birmingham, Department of Chemistry, Birmingham, England, January 8, 1973

14. Personal communication from Dr. I. M. Shapiro, U. of Penn. School of Dental Medicine, Philadelphia, Penn. to Dr. K. Bridbord, EPA, July 26, 1973 and H. L. Needleman and I. M. Shapiro, "Dentine Lead Levels in Asymptomatic Philadelphia School Children: Subclinical Exposure in High and Low Risk Groups," paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, North Carolina, October 1-2, 1973.

15. Pinkerton, C., J. P. Creason, D. I. Hammer and A. V. Colucci," Multi-Media Indices of Environmental Trace Metal Exposure in Humans," presented at 2nd International Symposium on Trace Element Metabolism in Animals, June, 1973, Madison, Wisconsin

16. Fairey, F. S., and J. W. Gray, "Soil Lead and Pediatric Lead Poisoning in Charleston, S.C.", J. South Carolina Med. Assoc., 66:79-82, March 1970

17. Barltrop, D., and Strehlow, C.D., "The Significance of High Soil Lead Concentrations in Childhood Lead Burdens,"

paper presented at EPA-NIEHS conference on Low Level Lead Toxicity, Raleigh, N.C., Oct 1-2, 1973

18. "Environmental Lead: Comparison of Blood and Tissue Levels Following Oral Exposure in Rats," EPA Internal Report, ETRL, NERC-Cincinnati, February, 1973

19. Calandra, J.C., Industrial Bio-Test laboratory, Northbrook Ill., Industrial Bio-Test Research Report IBT No. E1733C, January 10, 1973

20. Airborne Lead in Perspective, op. cit. p. 139

21. Data compiled by Center for Disease Control, HEW, Atlanta, on human and environmental lead pollution in El Paso, Texas, 1972

22. Personal communication from Dan K. Darrow, Dartmouth Medical School, Trace Element Laboratory, Brattleboro, Vt. to R. K. Bridbord, EPA, Washington, D.C., February 20, 1973

23. Barltrop, D., "Children and Environmental Lead," paper presented at a conference on "Lead in the Environment," Zoological Society of London, January 27, 1972

24. Lin-Fu, J. S., "Preventing Lead Poisoning in Children," Children Today, Vol. 2, p2, January 1973

25. Sayre, J.W., Vostal, J., Pless, I.B., and E. Charney, House and Handdust as a Potential Source of Childhood Lead Exposure, Am. J. Dis Child, in press, 1973 and Vostal, J., J.W. Sayre, E. Taves and E. Charney, "Lead Containing House Dust: another Source of Increased Lead Exposure in Inner City Children," paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, North Carolina, October 1-2, 1973

26. Holtzman, R.B., "Measurement of the Natural Contents of RaD (Pb-210) and RaF (Po-210) in Human Bone-Estimates of Whole-Body Burdens", Health Physics 9:pp 385-400, 1963

27. Morse, R.S. and Welford G.A., "Dietary Intake of 210-Pb," Health Physics, 21:pp. 53-55, 1971

28. Magno, P.J., Groulx, P.R. and Apidianakis, J.C., "Lead 210 in Air and Total Diets in the United States During 1966," Health Physics, 18: pp. 383-388, 1970.

29. Lin-Fu, J.S., "Undue Absorpticn of Lead Among Children--A New Look at an Old Problem," New Eng. J.

of Med., Vol. 286, pp. 702-710, 1972

30. Fine, P.R., G.W. Thomas, R.H. Suhs, R.E. Cohnberg and B.A. Flashner, "Pediatric Blood Lead Levels, A Study in 14 Illinois Cities of Intermediate Population," JAMA: 221, p. 1475-1479, September 1972

31. Gilsinn, J.F., "Estimates of the Nature and Extent of Lead Paint Poisoning in the United States," NBS Technical Note 746, December 1972

32. Lin-Fu, J.S., "Vulnerability of Children to Lead Exposure & Toxicity," New England Journal of Medicine, In Press

33. Personal communication from W. J. Sobolesky, City of Philadelphia Department of Public Health to K. Bridbord, EPA, dated August 6, 1973

34. Personal communication from M. Deas, Department of Human Services, Multnomah County, Oregon, to K. Bridbord, EPA, dated March 6 , 1973

35. New York City Bureau of Lead Poisoning Control, data submitted to EPA, August 31, 1972 and September 12, 1972

36. Personal Communication from W. R. Smith, Baltimore City Health Department, Baltimore, Md., to M. Kanarek, EPA, Washington, D.C.

37. Lauer, G.R., R.E. Albert, T.J. Knelp, B. Pasternak, C. Strehlow, N. Nelson and F.S. Kent, "The Distribution of Lead Paint in New York City Tenement Buildings," Amer. J. Pub. Health, 63: 163-168, February 1973

38. Sachs, H.K., "Effects of a Screening Program on Changing Patterns of Lead Poisoning," Paper presented at EPA-NIEHS Conference on Low Level Lead Toxicity, Raleigh, North Carolina, October 1-2, 1973.

39. Personal communication from J. J. Chisolm, John Hopkins Univ. School of Medicine, Baltimore, Md. to Asst. Administrator for Air and Water Programs, EPA, Washington, D.C., dated March 6, 1973

40. Personal communications from H. Sachs, formerly Director, Chicago Lead Poisoning Program to Administrator, EPA, dated March 5, 1973 and June 15, 1973

41. "Airborne Lead in Perspective," op. cit., p. 140

42. Personal communication from H. L. Slutsky, Coordinator, Chicago Lead Poisoning Program to J. S. Lin-Fu, HEW, August 28, 1973.

43. Conn, R. H. and D. Anderson, "D. C. Mounts Unfunded Program of Screening for Lead Poisoning," HSMHA Health Reports, 86:409-413, May 1971.

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

45. Gilsinn, J.F., Estimates of the Nature and Extent of Lead Paint Poisoning in the United States, National Bureau of Standards Technical Note 746, December 1972.

46. Margulis, H. L., R. J. Caprio and M. K. Joselwo, Residential Location Ambient Air Lead Pollution and Childhood Lead Poisoning, Arch. Env. Health, in Press.

47. Margulis, H. L., R. J. Caprio, A. Stefaniwsky, and M. K. Joselow, "Surveillance of Lead Poisoning in Newark, New Jersey--An Ecological Approach,"

Rutgers University and the New Jersey College of Medicine and Dentistry, Newark, New Jersey. Prepublication draft.

VII. Extent of Lead Exposure Among the General Population

In section IV, it was stated that determination of lead in blood is at present the best index for evaluating lead body burden and exposure. However due to limitations of the studies on blood lead levels in population groups in this and other countries, it is not possible to make precise estimates of the number of persons having blood lead levels within a given range. Problems still exist in the chemical analysis of blood for lead content. The results of quality control studies from laboratories in both the United States and Europe show that sometimes there is a large variation in results among different laboratories (1). Reproducible results even within the same laboratory are not always obtained, as shown in the Seven Cities Study (2). It is difficult to accurately estimate the relative contributicns of air and food sources to such blood lead levels as are found. That air lead makes some contribution is apparent from occupational data and from experimental exposures. The limited survey data available make it clear that most of the adult U.S. population has blood lead levels in the range of 10-30 µg/100 g.

Available information on the extent of elevated blood lead levels found in this country is presented in Tables VII-1 & 2. It can be seen that a small but generally consistent proportion of the general population has elevated blood lead levels. It is also evident that a number of specific groups in the population frequently or characteristically have blood lead levels

distinctly higher than those usually found. Many of these groups are occupationally exposed to automobile exhaust. Individuals so exposed in an outdoor environment, such as traffic police, parking lot attendants, and service station attendants frequently show elevated mean blood levels with ranges of 20-50µg/100g or higher (3,4,6-9). The few data on commercial drivers(4,6,7) also show elevations compared to the general population. Likewise, the available information on groups exposed to automotive exhaust in closed environments show consistently elevated blood leads (4,8,9) (See Table VII-1). These include workers in parking structures, traffic tunnels, and service and repair garages. Repairmen and mechanics may, of course, also be exposed to lead dust and fumes originating from maintenance activities (9).

Blood lead levels found in urban areas are also somewhat higher than in suburban and rural areas(2-4) (See Table VII-2). The source of most lead for the general population is food, although air is a significant source of lead in urban areas. It is known that air lead exposures are higher in urban than in suburban and rural areas. There are no data available to determine whether or not this is also true for food. The extent of the contribution, by occupational and avocational lead exposure, to the lead burden of the general population and to urban-rural gradients is likewise not well documented.

In recent years it has been found that large numbers of central city children are overexposed to lead. Surveys have been carried out in urban areas to search for childhood lead

poisoning, which usually results from the ingestion of chips of peeling lead paint. Other evidence of overexposure was also found. In nearly all of these surveys, it has been demonstrated that approximately 25% of the young children tested have blood lead levels of 40 µg/100g or above (most below 50 µg/100g) (10-13) (Table VII-3), which is medically undesirable. The reasons for this frequency of elevated blood leads are not completely understood at present. The shapes of the blood lead frequency distributions suggest that lead sources more homogeneously distributed than peeling paint and containing less lead than routinely found in paint chips are making a major contribution. The rationale for considering lead dusts which originate from automotive exhaust as a contributing factor was discussed in the previous sections.

In summary, it is clear that levels of lead in the blood, indicating elevated exposure, exist to a small but significant extent in the general adult population, and to a very great extent among children. There are also a number of large groups of the population in which undesirable elevated blood lead levels are commonly found directly related to exposure to automobile exhausts. Thus much of the excess exposure to lead in the general population is due at least in part to lead from automotive exhaust.

TABLE VII-1

Extent of Abnormally Elevated Blood Leads
Among Urban Adults

City	Exposure Category	Number Studied	% of Blood Leads Equal to or Greater than 40 µg/100 g
Cincinnati	Post Office Employees (4)	140	2.9
	Firemen (4)	191	3.0
	Service Station Attendants (4)	130	12.3
	Police (4)	40	12.5
	Drivers of Cars(4)	59	15.0
	Parking Attendants (4)	48	44.0
	Garage Mechanics (4)	152	67.0
Los Angeles Area	L.A. Police (4)	155	0.6
	Pasadena Male City Employees (4)	88	3.3
	L.A. Female Aircraft Employees (4)	87	3.3
	L.A. Male Aircraft Employees (4)	291	5.2
Philadelphia	Male Commuters (1)	43	2.3
	Police (4)	113	3.5
	Downtown Male Residents (4)	66	4.5
Camden, New Jersey	Women Living Near Freeways (5)	55	1.8
Composite Urban Samples	Females from New York, Philadelphia, and Chicago (3)	423	0.7
	Males and Females from 6 Cities (3)	833	2.7*
Composite Urban Samples	Taxi Drivers and Office Workers from L.A.; Philadelphia; Barksdale, Wisconsin, and Starke, Florida (6)	149	0

*Only those above 40 µg/100 g blood lead.

TABLE VII-2

Urban-Suburban Blood Lead Comparisons
In Adults

Group Studied	Number Studied	% Blood Leads Equal to or Greater than 40 µg/100 g
Urban Females (2)	423	0.7
Suburban Females	556	0
Phiadelphia Males (4)		
Urban	66	4.5
Suburban	23	0
Composite (3)		
Urban	833	2.7*
Suburban	162	0

*Only those above 40 µg/100g.

TABLE VII-3

Percentages of Children with Abnormally Elevated Blood Leads

City	Years Tested	Number Tested		% Blood Leads Equal to or Greater than 40 µg/100 g
Baltimore (10)	1968	655		25.3
	1969	746		27.9
	1970	939		31.5
Chicago (10)	1967-70	120,000		20.0
New Haven (10)	1969-70	1,897		29.8
Newark (10)	1970	594		38.9
New York (10)	1969	2,648		45.5
	1970	84,368		28.7
New York (11)	1971	81,626		20.2
Philadelphia (10)	1970	3,496		34.0
Washington (10)	1970	808	(all ages)	5.8
	1970	1,152	(2 years)	22.0
Many Cities (12)	1971	2,309		9.1
Aurora, Ill. (13,14)	1971	449		24.3
Springfield, Ill. (13,14)	1971	670		30.1
Peoria, Ill. (13,14)	1971	387		31.3
E. St. Louis, Ill. (13,14)	1971	376		24.7
Decatur, Ill. (13,14)	1971	793		12.2
Joliet, Ill. (13,14)	1971	383		24.3
Rock Island, Ill. (13,14)	1971	285		21.1
E. Moline, Ill. (13,14)	1971	298		11.4
Robbins, Ill. (13,14)	1971	103		12.6
Harvey, Ill. (13,14)	1971	226		16.4
Carbondale, Ill. (13,14)	1971	264		17.0
Norfolk, Va. (13)	1971	1,225		22.7
New Haven, Conn. (13)	NA	1,339		23.7
Washington, D. C. (13)	1971	1,821		39.2
Rockford, Ill. (13,14)	NA	1,200		19.5

REFERENCES

1. Subclinical Lead Poisoning; Editorial; Lancet; 1:87, January 1973

2. Tepper, L. and L.S. Levin, "A Survey of Air and Population Lead Levels in Selected American Communities"; Department of Environmental Health College of Medicine, Univ. of Cincinnati, Cincinnati, Ohio; Final report (Contract PH-22-68-28), Dec., 1972, Cincinnati, Ohio

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

4. "Survey of Lead in the Atmosphere of Three Urban Communities", Public Health Service. Publ. No.999-AP-12, 1965

5. Daines, R.H. et. al.; "Air Levels of Lead Inside and Outside Homes"; Indust. Med. 41: 26-28, Oct. 1972.

6. Azar, A., et. al. - Relationship of Community Levels of Air Lead and Indices of Lead Absorption. - pp 581-594 In: Proceedings of International Symposium on Environmental Health Aspects of Lead, Amsterdam, October 2-6, 1972

7. Jones, R.D. et.al. - Blood Lead and Carboxyhemoglobin Levels in London Taxi Drivers - Lancet 2: 302-303, 12 Aug., 1972.

8. Bericht der Eidg. Bleibenzin-Kommission an den Bundesrat uber ihre tatigkeit im zeitraum 1947-1960. Mitt. Gebiete Lebensmitt. Hyg. (Bern). 52(3): 135-244, 1961.

9. Tola, S. et. al. - Occupational Lead Exposure in Finland. II. Service Stations and Garages. - Work Environ. Hlth. 9:102-105, 1972.

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

11. Guinee, Vicent F. - Lead Poisoning - Amer. Jour. Med.; 52:283-288,

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

13. Gilsinn, J.F., Estimates of the Nature and Extent of Lead Paint Poisoning in the United States, NBS Technical Note 746, December 1972

14. Fine, P.R., et.al. Pediatric Blood Lead Levels. "A Study in 14 Illinois Cities of Intermediate Population," JAMA 221: p. 1479, September 1972.

VIII. Summary and Conclusions

Summary

Lead occurs widely in the environment. Its present distribution is the result of natural occurrence, greatly influenced by man's activities. In 1971, in the United States, 1,431,514 tons of lead were consumed with over one million tons used as metallic lead or lead alloys and in storage batteries. About 135,000 tons were used in coatings and pigments. Of the 264,000 tons used in gasoline additives, more than two-thirds enters the environment. Combustion of gasoline contributes by far the largest fraction of lead reaching the environment. Other sources of lead in the environment are wear and erosion of lead-containing painted surfaces, and incineration of lead-containing substances.

Man takes in lead from many sources: water; food; air; and, particularly in the case of children, from ingestion of lead-containing non-food items such as paint and dust. It is probable that lead in dust and dirt is inadvertently ingested by both children and adults. It is generally agreed that food is the major source of lead for the general population. A World Health Organization expert committee reports that according to the results of total diet studies in industrialized countries, the total intake of lead from food generally ranges from 200-300 µg per person per day. WHO further states that based upon available

data, these levels are similar to those found in the past 30-40 years and that no upward trend in lead levels in food is evident. Reducing the amount of airborne lead in the environment constitutes an accessible means for reducing potential human exposure to environmental lead particularly when that fraction is large compared with that absorbed from the diet. (Section II)

Lead has not been shown to be biologically essential or beneficial to man. In sufficiently high quantities, it is clearly toxic, and, at somewhat lower levels, has been shown to cause biochemical changes. Lead is also suspected of producing subclinical neurologic damage.

The studies reviewed in Section III permit no unequivocal conclusions to be drawn. On balance, they suggest that subclinical changes are associated with blood lead levels of approximately 40 µg/100g and above. As blood lead levels increase above 40 µg/100g, the likelihood that these changes will occur increases markedly. Based upon evidence from these studies, it would seem prudent to regard blood lead levels over 40 µg/100g as indicators of lead intake that should be prevented. The 40 µg/100g figure, however, does not represent a sharp demarcation between health and disease.

Blood lead levels, under most circumstances, serve as a reasonably accurate measure of lead body burden and have been widely employed in public health surveillance. It is not

possible at this time to firmly establish a single acceptable blood lead level protective of all high risk population groups. It would appear prudent, however, to recommend that the current U.S. Public Health Service Guideline for older children and adults, i.e., 40 µg/100ml whole blood be regarded as a strict upper limit for younger children. Whether the acceptable upper limits should be lower than 40 µg/100ml for the fetus, neonate, and the woman of child bearing age will require further investigation. (Section IV)

In Section V, it is shown that lead from automotive exhaust contributes to increased exposure for humans both from the air and from fallout. Blood lead levels exceeding 40 µg/100g are considered medically undesirable and may ultimately be harmful. Blood lead levels depend on daily dietary intake and adsorption of respired lead. For a "standard man" with average dietary lead intake, exposure to average airborne lead concentrations of 5.0 to 6.7 $\mu g/m^3$ could cause his blood lead concentrations to reach 40 µg/100g within a year. Airborne lead levels near to or in excess of 5 $\mu g/m^3$ have been observed in several U.S. cities.

Lead in dust and dirt is an important potential source of exposure, especially for young children. Daily ingestion of relatively small quantities of dirt or dust (less than 1 gram or about 1/4 of a teaspoonful) containing 1000-2000ppm of lead would be medically undesirable. Although dustfall exposure to lead is still often considered an hypothesis, much of the available evidence is consistent with this hypothesis. Present day lead

exposure via air or dusts in some sections of large cities leaves little or no margin of safety in relation to those concentrations associated with biomedical harm.

In Section VI, it was shown that lead emissions from automobiles contribute to lead present in urban soils, in street dirt and in house dust. Lead content of soil has been demcnstrated to decrease with increased distances from both painted houses and roadways. Lead concentrations in fallout dust from the air also decrease away from roadways. Dust lead levels in street sweepings from large cities usually range from 0.1 to 0.25% and at times exceed 0.5%. Housedust from urban areas commonly contains 0.1% lead or higher. Concentrations of lead in housedust vary with lead fallout from the air and are reported to be higher in homes located near heavily travelled roadways than in homes on side streets. Quantities of lead in housedust do not vary directly with the presence of peeling paint in the home but they are higher in older homes, reflecting greater amounts of lead paint, as well as increased atmospheric ventilation and dust fallout in older homes. Erosion of paint is clearly a contributor to lead in housedust. Urban dust and dirt, if sufficiently contaminated with lead represent a hazard to children, if ingested. With the available data one cannot quantitatively assess the relative contribution of erosion of lead paint and of other lead sources to the total lead content of soil, street dirt and housedust. Nevertheless, it is evident that lead emissions from automobiles are major contributors to this contamination.

Clinical experience indicates that leaded paint is primarily responsible fcr the great majority of overt clinical lead poisoning in children. There is, however, sufficient data to strongly suggest that sources of lead other than paint play an important role in childhood lead exposure. These other sources may be especially significant at levels of exposure below overt clinical poiscning.

Lead in air, and particularly lead in dust are ubiquitous sources of lead, which may well be important contributing factors in the childhood lead problem. Exposure to dirt and dust, if sufficiently contaminated by lead, could significantly reduce the quantity of additional lead required to produce clinical poisoning in a child with other sources of exposure. Although exposure to lead contaminated dirt and dust from automotive exhausts has not been shown to be responsible for cases of overt lead poisoning, automotive lead has been related to undue lead absorption in children. At this time, it would be prudent to decrease the potential air and dust lead exposure. It should be recognized that further studies are necessary to better quantitate the sources of lead contamination in dust and dirt as well as the magnitude of the contribution that leaded dust and dirt make to both subclinical and clinical lead overexposure.

It is clear that undesirable levels of lead in the blood, indicating elevated exposure, and elevated lead body burdens exist to a great extent among children, and to a small, but significant extent among adults. There are also a number of

adult groups in which undesirable elevated blood lead levels are found directly related to exposure to automobile exhausts. Thus, much of the excess exposure to lead in the general population is due at least in part to lead from automotive exhaust. (Section VII)

Conclusions

A small but significant fraction of the adult population has blood lead levels of 40µg/100g or higher, and such levels occur in a much larger propcrtion of urban children. Such levels are medically undesirable and should be reduced if possible.

Sources of exposure to lead include food, water, air, and ingested non-food items such as lead based paint and dust. Food is the largest contributor of lead to the general population.

Lead based paint is the major cause of overt clinical lead poisoning in children, though sources of lead other than paint play an important role in childhood lead exposure particularly at levels below overt poisoning.

Lead in dust and dirt is believed by EPA to contribute to increased lead levels in man, both through inhalation of resuspended dusts and at least in children, through inadvertent

ingestion of dirt and dust. This source could significantly reduce the quantity of additional lead required to produce clinical poisoning in a child with other sources of exposure. Automotive lead is a major contributor to lead in dust and dirt and has been related to undue lead absorption in children.

Lead from all these sources should be reduced to the degree possible.

Actions have already been taken by the Federal and other levels of government to reduce the lead content of paint and further actions in this regard are being contemplated so that this source of lead will decline with time as older buildings with leaded paint are replaced. Action has also been taken to substantially reduce controllable sources of lead in food, and efforts to further reduce lead in food are continuing. One controllable source of lead in food is residue from use of lead compounds as insecticides. Tolerances for these residues are being reevaluated at this time. Lead in a few drinking water supplies is higher than desirable, and efforts should be continued to reduce this source of lead exposure.

In EPA's opinion, lead in gasoline is the most important remaining source of <u>controllable</u> lead entering the environment. Reduction of lead in gasoline has shown to reduce lead in the ambient air. Leaded gasoline results in direct exposure to the population through inspired air and by presence of lead in dirt

and dusts which may be inspired or inadvertently ingested. General widespread contamination of the environment by lead occurs through deposit of airborne lead directly in water, and on streets and other paved areas from which some will be washed into waters. Airborne lead also is deposited in relatively high amounts on plants, along heavily travelled roads, and in lesser concentrations but over vast areas more distant from highways. Reduction of lead in gasoline will, therefore, result in reduced exposure of man, both directly from reduction in atmospheric lead, and indirectly from reduction of lead in dirt, dust, and at least to a minor extent, on and in foods.