An Assessment of the Scientific Justification for Establishing 2 µg/m³ as the Maximum Safe Level for Airborne Lead

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A comprehensive report on the subject of safe levels of environmental lead in relation to health and a sincere effort on the part of the author to present a balanced picture in establishing maximum safe levels.

INTRODUCTION

During recent months a number of important reports dealing with environmental lead have appeared. Special significance attaches to two of these: (1) *Airborne Lead in Perspective* prepared by the Committee on Biological Effects of Atmospheric Pollutants of the Division of Medical Sciences, National Research Council-National Academy of Sciences (NRC-NAS); and (2) *Background Information-Airborne Lead* prepared by the Office of Criteria Development in the Office of the Director, National Environmental Research Center, Environmental Protection Agency. The latter document became available on or about January 7, 1972. (Some copies bear the date January 7, 1971, apparently a typographical error.) Its introduction states that "The major single source of its information was the comprehensive review and summation prepared under contract for the Environmental Protection Agency by the National Research Council."

In the following discussion, frequent reference will be made to the two documents mentioned above which will be designated NAS and EPA, respectively. Other material will be introduced where appropriate, in an effort to present a balanced picture.

LEAD IN THE ENVIRONMENT

The first chapter of the NAS report, *Airborne Lead in Perspective,* includes a general account of the geochemistry and ecological relationships of lead. Points of particular relevance to the present discussion are (1) the ubiquity of lead in all parts of man's environment, (2) the effects of man-made translocation of lead, especially since the time of the industrial revolution, and (3) the great increase in the production and consumption of the metal during the past three or four decades.

Refinements in analytical techniques have made it possible to measure minute quantities of metals in various parts of the environment where but a few years ago they could not have been detected. This has resulted in what may be properly termed a revolution in the entire field of trace metals. Lead has not escaped. A number of metals, e.g., selenium, chromium, cadmium and arsenic, formerly held to be poisons, are now recognized as essential micronutrients. Lead can induce as well as inhibit enzyme activity and in low concentrations can stimulate the synthesis of heme. The NAS report states (p. 231) that "There is no evidence that lead is an essential trace element, although the hypothesis that minute amounts may serve some essential function in metabolism has not been examined." Every element is toxic in high concentrations, but every element potentially has a biologic function which can be assessed properly only against the background of a deficiency state. The presence of lead everywhere in man's environment makes it virtually impossible to create a lead-free diet and adds credence to the possibility of an essential role. In the course of man's evolution he has been able to develop a tolerance to his

environmental lead. Tolerance frequently is followed by dependence. Drastic increases in exposure to any element can overwhelm man's adaptive mechanisms. Whether or not recent increases in environmental lead, and specifically the amount attributable to the use of leaded gasoline, has had or is likely to have any deleterious effect on human health is a matter of universal concern. The principal reason for the NAS study and report was this very problem. A significant passage in the NAS report states that "... the high degree of dispersal associated with the venting of burned lead alkyls into the air has minimized the effect of the rapid rise in the consumption of lead, with the result that the average lead content of the air over most major cities apparently has not changed greatly over the last 15 years. The net result is that correspondingly little change in the character and magnitude of the effects of atmospheric lead on biologic systems will occur for some years to come" (p. 312). Many students of the subject believe that the decrease of older uses of lead, such as in paints, pesticides and plumbing, is an important factor in maintaining fairly constant levels of lead in the total environment.

In another highly relevant paragraph the NAS report says that: "From the point of view of contamination of the total environment with lead, the lead-using industries have contributed more than has the single source of combusted lead fuel. Two to three times as much lead is added to the environment in the form of paint pigments and metallic products as in the form of lead alkyls. Some undefined fraction of this nonrecycled lead finds its way into the ecosystem by surface weathering and leaching, dumping, and burning. The environmental fate of this lead is ill-defined, but it is probably chiefly returned to the soil without emission to the air. Any proposal for the removal of lead from automotive fuels to rid the environment of lead pollution must take into consideration the fact that the fate of other lead products, such as paints and manufactured items, is largely unknown" (p. 313).

LEAD IN THE ATMOSPHERE

Figures on lead consumption compiled by the Bureau of Mines are quoted in the NAS and EPA reports. The apparent omission of some 30,000 tons used annually in the glass and ceramics industries probably does not materially affect the picture. Data on lead emission in both reports are taken from the National Inventory of Air Pollutant Emissions and Controls compiled by the Bureau of Air Pollution Sciences of EPA. Consumption is listed in 26 categories while emissions are given for nine, suggesting possible incompleteness in the latter. Most striking is the fact that storage batteries, which are responsible for about 40% of consumption, apparently are not responsible for any emissions. Other major users, including some involving high temperatures, also are not charged with any emissions nor do incinerators appear on the list of emitters. These comments may have some relevance to the statement in the EPA report that "... almost 99 percent of the estimated lead emissions to the atmosphere were from gasoline combustion, gasoline transfer, and gasoline antiknock additive manufacturing with over 98 percent of the emissions coming from gasoline combustion alone" (p. 3). Without implying that gasoline additives do not contribute appreciably to atmospheric lead, the proportion of total emissions from this source might be somewhat less than 99% if all emissions were to be counted. The EPA report notes (p. 3) that smelters and incinerators are potential sources of lead emissions but it does not include these in calculating the national figure since "... their significance has not yet been evaluated." This seems like poor reasoning, since the omission of these sources obviously results in a distorted picture, as pointed out in the NAS report in the passage cited above.

LEAD IN URBAN AIR

Systematic monitoring of atmospheric lead in American cities has been conducted for more than thirty years, starting in Cincinnati in 1938 (NAS, p. 21). Sampling in additional cities was initiated at later dates resulting in what have become known as the "Three Cities Study" involving Cincinnati, Los Angeles, and Philadelphia, and the "Seven Cities Study" embracing Chicago, Houston, New York and Washington, in addition to the original three. (Los Alamos, N.M., a relatively isolated community of about 17,000 persons, was added for purposes of comparison.)

The findings of various studies on atmospheric lead are discussed in some detail in the EPA and NAS reports. Commenting on yearly trends, the latter (p. 21) states: "The consumption of lead alkyl fuel additives is increasing substantially every year. It cannot be assumed, however, that the concentration of lead in urban air is increasing in parallel. Indeed, most of the available data suggest that the concentration of lead in air, even over the largest cities, is increasing only very slowly if at all." A summary of annual average particulate lead concentrations covering 18 cities for the period 1957-1969 has recently been prepared by EPA (Feb. 3, 1972). As might be expected, there are differences between the cities and fluctuations from year to year but, with the exception of Los Angeles, there is strikingly little difference between the earliest and the latest observations. Several cities showed slightly higher lead levels in 1969 than in 1959 or 1960 but almost as many showed lower levels. During the period in question (1959-1969), the consumption of lead antiknock gasoline additives rose about 70%, from 160,000 tons in 1959 to 271,000 tons in 1969. If lead additives are responsible for some 95% to 98% of lead emissions, as maintained by EPA, a concomitant increase in urban atmospheric lead would have been expected. Commenting on this point, the NAS report (p. 23) notes: "The NAS data cited earlier for 1953 through 1966 are from samples taken at many different sites and exact information is not available when sites were changed. It is difficult, therefore, to tell whether the atmospheric concentration of lead increased or decreased during that period, but the *data seem to indicate that the concentration remained fairly constant*" (emphasis added). The EPA report states (p. 11): "Unequivocal evidence with regard to long-term trends in the concentration of lead in the atmosphere is

not available." Only in Los Angeles does there appear to have been a definite increase in atmospheric lead between 1962 and 1969.

That atmospheric lead concentrations are higher in congested parts of cities than in sparsely settled rural areas should surprise no one. No doubt motor vehicle emissions contribute substantially to the lead content of urban air but it may not be assumed that this is the only factor leading to higher levels. Greater fuel consumption, industrial activity and the location of power generating plants and incinerators may play important roles. The relative contribution of these latter activities has not been adequately evaluated. One study (Cholak[2]) showed a downward trend in lead in the atmosphere of Cincinnati from 1941 to 1962 in spite of a great increase in the consumption of leaded gasoline during that period. The decrease in lead was attributed to a decrease in the amount of coal used for heating purposes (NAS p. 22).

Gasoline additives may not be the only significant source of lead found in the air and soil along highways although they probably account for a major fraction. Possible additions from tires, brake materials, bearings, paint stripes and highway surfacing materials seem not to have been evaluated.

HUMAN EXPOSURE TO ENVIRONMENTAL LEAD

Because of its ubiquity, lead must inevitably enter living beings not only through respired air but also as a component of all food and all water (Kehoe[9]). Health effects, if any, will be related to the total intake. According to the NAS report (p. 67) "Food, water, and other beverages are the major source of lead input in man and probably in most animals." More important than "exposure" or "input" is *absorption*, which means passage into the blood with subsequent distribution throughout the body. All lead which enters the respiratory or gastrointestinal tracts is not necessarily absorbed. Kehoe has shown that in humans, not more than about 10% of ingested lead is absorbed and "... that half or more of lead inhaled in finely dispersed form from the ambient atmosphere is discharged from the body in expired air." It has been estimated that not more than 15% of inhaled lead aerosol reaches the blood (Task Group[12]). As a result of his classic experiments on humans, Kehoe concluded "... that an equilibrium is established at an early age between the human organism and its usual or normal environment in the United States, whereby the stream of lead absorbed by the body from the environment is balanced by a counterstream of lead issuing forth from the tissues and from the body via excretory routes. No doubt this equilibrium is disturbed from time to time by the variability of the environmental conditions, and for a time the absorption of lead exceeds the excretion or vice versa. There is good reason to believe, however, that *the end result of the operation of these variables is the maintenance of an essential balance between absorption and excretion over the span of life of an individual*" (emphasis added). This belief is espoused by Dr. John Goldsmith[5] who states: "Although the concentration of lead in the environment, particularly from automobile exhausts, has been rising, and the equilibrium concentration in tissues of individuals living in areas of heavy automobile traffic also may be increasing, there is no evidence that this produces significant health hazards."

ACCUMULATION OF LEAD IN HUMANS

The preponderance of available evidence supports Kehoe's concepts of lead equilibrium. Until such time as large-scale controlled studies are made of total body lead, one must rely on levels of lead in blood as the most reliable indicator of body burden that can be applied in a practical manner.

A review of all available data on "normal" blood and urine lead levels from 1925 to 1965 (Stopps[11]) showed that while there was a certain amount of variation among the studies, there had been no significant change in blood or urinary lead during the period encompassed. An international study of blood and urinary lead embracing fifteen countries likewise showed some degree of variation among countries but the similarities were more striking than the differences (Goldwater and Hoover[6]). New Guinea aborigines, living in an area remote from any industrial activity, and using wooden utensils, had blood lead values similar to those of residents of congested cities. Similar findings have been noted in several other primitive peoples. A group of distinguished experts had this to say on the subject:

"In view of the very great range of lead intake between individuals and the variability in lead absorption, the consistency of blood lead levels is impressive, enough so to suggest both regulation by a biological control mechanism and possible biological need" (Tepper and Pfitzer[13]).

If factual data and actual observations are used, rather than speculation and poorly founded assumptions, the evidence clearly points to an absence of any demonstrable build-up of lead in human populations over the past forty years, the very time during which the use of leaded gasolines showed a marked increase.

DELTA-AMINOLEVULINIC ACID DEHYDRASE (ALAD)

Recent publications have called attention to the possible significance of decreased ALAD activity, with resulting impairment of hemoglobin synthesis, as an indicator of adverse effect due to lead (Hernberg et al.[8]; Haeger-Aronsen et al.[7]). The subject is discussed in both the EPA and NAS reports and both agree that an interference with ALAD activity may represent an undesirable and possibly deleterious interference with a normal body function. Further examination of this concept is in order.

That ALAD activity can be depressed by lead has been established beyond any reasonable doubt, but the circumstances associated with this effect and the interpretation of the phenomenon have not been fully elucidated. Nevertheless, decreased ALAD activity in human blood has been equated with lead intoxication, either clinical or subclinical, in spite of the fact that it is most unusual for any change of this type to have diagnostic specificity. Furthermore, alterations in

activity of red blood cell enzymes seldom result from a single disease or even a single diseased organ. Information has not been published whether or not these changes are found in a spectrum of conditions which are not necessarily causally related. Inhibition of ALAD activity as a result of alcohol consumption has been reported (Ball and Sorensen[1]).

ALAD is one of several important "copper enzymes" (Underwood[14]) and, at least under some circumstances, it depends for its activity on copper. The functioning of copper enzymes can be disturbed by a number of things including zinc, iron, molybdenum and inorganic sulfates. It remains to be examined and established whether or not ALAD activity can be markedly depressed in a variety of common diseases such as diabetes, pneumonia, alcoholism, cancer, heart failure, and kidney disease, and notably by alcohol.

The lack of specificity in decreased activity of ALAD is of great significance in the interpretation of studies which are frequently cited to support the causal relationship between lead absorption and depression of ALAD. The occupational groups studied by Hernberg[8] and by Haeger-Aronsen et al.[7] included printshop workers, traffic policemen, automobile repairmen, storage battery makers and ship scrappers. All of these occupations involve significant exposures to chemicals in addition to lead. No data are given as to alcohol consumption (notoriously high in Scandinavia) nor to the presence or absence of any acute or chronic diseases. Failure to control significant variables in any epidemiological investigation can result in misleading conclusions or, at best, to the finding of associated phenomena with unproven causal relationships. No amount of statistical manipulation can correct this weakness in the basic data.

The significance of depressed ALAD activity has not been clearly established although it is known to be associated with the synthesis of heme, one of the building blocks of hemoglobin. Experimentally it has been shown that dogs with ALAD activity reduced virtually to zero by lead feeding showed no demonstrable adverse effects, and when one-half of their blood was removed, regeneration was unimpaired (Tepper and Pfitzer[13]).

The question of ALA and ALAD in relation to lead absorption is discussed in some length in the NAS report. Following a review of the literature on ALA excretion the report states that "None of these studies is entirely satisfactory by itself from the point of view of methodology, completeness, or groups studied. Nevertheless, all are consistent with the hypothesis that the exponential increase in ALA excretion associated with blood lead content above approximately 40 μg/100 g of whole blood signifies inhibition of ALAD that is physiologically significant *in vivo*." The report goes on to say that "At blood lead concentrations lower than about 35 μg/100 g of whole blood, neither stimulatory nor inhibitory effects of lead have been observed *in vivo* in relation to ALAD activity. At these 'normal' levels of lead in blood, hemoglobin synthesis and red blood cell formation do not increase [sic] and ALA excretion is apparently unaffected" (p. 147). Elsewhere (p. 323) the NAS report states: "At concentrations of blood lead below about 40 μg/100 g of whole blood . . . inhibition of ALAD in circulating red blood cells in proportion to the concentration of lead found in the cells can be demonstrated *in vitro*. This relation seems to apply even at the lowest concentrations of lead detectable *in vivo* in the peripheral blood of man, but its biological significance is dubious, because it is unaccompanied by any detectable biologic effects in intact man." In another part of the discussion of ALAD (p. 142), the NAS report emphasizes the necessity for caution ". . . in the transfer of *in vitro* observations to *in vivo* situations." A phenomenon which can be demonstrated in *all* individuals, regardless of the amount of lead in their blood, can hardly have much significance as an indicator of a toxic effect.

Uncertainties in the application of ALAD measurements to the prediction of lead effects include:

1. The questionable significance of reported correlations between ALAD activity and blood lead in normal persons who have had no unusual exposure to lead and in whom blood lead levels are within the normal range.
2. The dubious propriety of extrapolating data from normal humans to foretell incipient disease of unknown type or to denote that lead causes toxicity, albeit not discernible clinically and at blood concentrations of lead long accepted to be within the normal range.
3. The unsuitability of enzymatic kinetic standards in the assay systems commonly employed.
4. Variability of results in analyses of blood for lead.
5. Absence of suitable controls to eliminate the possibility of decreased ALAD activity being due to causes other than lead (see above).
6. Possible failure to realize that ALAD activity decreases quite rapidly in blood samples even when they are stored at 4°C.

Somewhat related to the use of ALAD activity as an index of lead effect is the proposal, principally by Goldsmith and Hexter,[4] that a regression line which they constructed would indicate at what levels of lead in air and blood effects on ALAD activity will occur. The subject is covered in both the NAS report (p. 89) and that of EPA (p. 31). Much of the discussion centers around the so-called Goldsmith-Hexter Regression Line, using, according to its designers, *estimated* average respiratory exposures and mean blood lead levels from the "Three City Study," supplemented by data obtained from four experimental human subjects. As pointed out in the NAS report, the air data and the blood lead determinations were not taken at the same time and place, a serious defect. It would be possible to design a regression line of any type that one might desire by taking combinations of unrelated air and blood lead values. The concepts that the atmosphere can be a source of absorbed lead and that there may be a relationship between atmospheric and blood lead are not in question.

The "Seven City Study" has made available a large body of data which make it possible to construct a

regression line, using the same method as that employed by Goldsmith and Hexter, but using air and blood analyses performed at the same times and places. When this is done the slope of the line is much closer to horizontal than that of Goldsmith and Hexter and indicates a correlation coefficient of 0.22. This means rather weak positive correlation. Not specifically mentioned by Goldsmith and Hexter, nor in either the NAS or EPA report, is the matter of confidence limits when this type of statistical analysis is employed. Lines representing confidence limits are hyperbolic and diverge markedly when applied to extrapolations at either end of the regression line; in other words, there is little validity to extrapolations beyond the ranges encompassed by actual observations. This is recognized in the NAS report in the statement that ". . . the regression line cannot be applied with confidence to exposure conditions affecting the general population; this is so even for the general population of most large urban centers, inasmuch as average ambient air concentrations even in these centers do not generally exceed 2$\mu g/m^3$." It thus appears that the Goldsmith-Hexter calculations have little or no bearing on the question of lead emissions due to gasoline additives.

No doubt there are various possible interpretations of these findings. The EPA press release states: "It has not yet been determined whether there is a true relationship between the ambient air lead levels and the blood lead levels measured in the . . . communities. EPA officials stressed that further statistical analysis of the data is needed before firm conclusions can be drawn." Such caution is commendable but it seems fairly obvious that at least, as far as these findings are concerned, and if the data truly portray the situation, there is no correlation whatsoever between lead in the ambient air and in the blood. It would appear that at air levels such as those found in Pasadena (3.39 $\mu g/m^3$) excretory mechanisms are capable of maintaining blood lead within normal limits. In any event, the findings show that use of mathematical formulas to predict air/blood ratios is fraught with danger. They also show that levels of atmospheric lead at least as high as 3.39 $\mu g/m^3$ are not likely to result in increases in blood lead above the normal. In fact, data for Los Angeles in the "Seven City Study" show that atmospheric concentrations of lead of 3.82 $\mu g/m^3$ are associated with blood lead levels averaging 17.5 $\mu g/100$ g, well within the normal range. Perhaps the "further statistical analysis" referred to above will yield other conclusions, but the information at hand cannot support a requirement of ambient air levels lower than 3.82 $\mu g/m^3$.

Some anomalous findings in data which accompanied an EPA press release of June 4, 1971, raise questions which find no ready answers. One of the tables gives air and blood lead levels for five locations, including Los Alamos and Pasadena. The air lead for the latter is 20 times greater than that of the former (3.39 against 0.17 $\mu g/m^3$) while the blood lead is only slightly greater (17.6 against 15.4 $\mu g/100$ g). Another comparison of locations shows that the highest blood lead value (20.6 $\mu g/100$ g) was found in an area with air lead one-half of that in Pasadena. Because of the possible significance of these findings the table is reproduced in its entirety.

Air and Blood Lead Levels

Name	Stations	Air Lead $\mu g/m^3$	N	Blood Lead $\mu g/100$ g
Los Alamos	41, 42	0.17	204	15.4
Okeana Farm	34, 35	0.32	166	15.8
Ardmore-Wynnewood	19, 20	1.15	156	18.0
Rittenhouse Square	18	1.67	137	20.6
Pasadena	4, 5	3.39	209	17.6

N – Number of subjects
From EPA press release of June 4, 1971

OCCUPATIONAL EXPOSURES

Occupational exposures to lead are of only indirect relevance to the present discussion. Their importance relates to the knowledge of chronic lead poisoning gained through many extensive studies of workers in lead industries. Most significant, perhaps, is the observation that overt evidence of toxic effects is unlikely to occur with blood lead levels under 80 $\mu g/100$ g of whole blood and unless atmospheric lead exceeds 200 $\mu g/m^3$. The relatively minute amounts of lead in ambient air would not be expected to have any significant effect when added to that to which industrial workers are exposed. The prevention of occupational lead poisoning lies within the realm of industrial hygiene rather than that of air pollution.

CHILDHOOD LEAD POISONING

The NAS report states (p. 174): "Today in the United States, lead poisoning in children is believed to be due almost entirely to the repetitive eating of leaded house paint." It is pointed out, however, that there may be substantial amounts of lead in street dust and that the continued ingestion of a gram per day of street dust could add significantly to the intake of lead. The chemical composition of street dust, including its lead content, depends on a multiplicity of factors, just as in the case with dust along highways (see above). No data are available as to the extent to which automobile exhausts contribute to the lead content of street dust.

Theoretically it is possible to conceive of a situation in which a child who eats paint could tolerate the amount of lead ingested from that source but would develop lead poisoning as a result of adding to the lead intake by eating street dust. There is no way of knowing how often this has occurred, if ever; the data are simply not available. The NAS report notes: "The extent to which airborne lead in congested urban areas contributes to increased lead absorption and lead poisoning in children is not clearly defined. . . . It may be estimated that dust-fall 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."

Elimination of old, dilapidated slum dwellings would almost certainly put an end to most of the present childhood lead poisoning. The effect of eliminating leaded gasoline is conjectural and uncertain. The former would yield important social and economic benefits, while the latter would have an opposite effect.

SUMMARY AND CONCLUSIONS

The evidence that ". . . airborne lead levels exceeding 2 micrograms per cubic meter, averaged over a period of three months or longer, are associated with a sufficient risk of adverse physiologic effects to constitute endangerment of public health" has been examined as has the role of leaded gasoline in contributing to atmospheric lead. The following points are of particular cogency:

1. The claim that leaded gasoline is responsible for almost 99% of lead emissions to the atmosphere is not justified since a number of important sources of emissions were not included in the calculations which led to this figure.

2. With the possible exception of Los Angeles, there is no clear evidence that there has been any significant increase in atmospheric lead in large cities or elsewhere.

3. There is no evidence that there has been a build-up of lead in persons living in large cities or elsewhere over the past 30 or 40 years.

4. Studies purporting to show that decreased ALAD activity points to an imminent threat from atmospheric lead are not convincing, since adequate controls were not included in these studies.

5. Mathematical calculations (pointing to a lead hazard) based on unsubstantiated assumptions are unconvincing, particularly when they are in disagreement with similar calculations based on actual observations.

6. Populations exposed to atmospheric lead concentrations in excess of 3.0 $\mu g/m^3$ and up to nearly 4.0 $\mu g/m^3$ have shown no evidence of build-up of lead in blood. There is no evidence that these were not specifically investigated.

7. An ambient air standard for lead of 4.0 $\mu g/m^3$ or higher would have fewer scientific objections than a standard of 2.0 $\mu g/m^3$.

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