Memorandum Compiled for Hearing

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Subcommittee on Air Pollution

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SUMMARY

Lead is a normal constituent of the earth's crust and therefore, along with other elements, becomes incorporated in man's food chain. Thus a certain lead content can be considered a normal background amount for plants and animals, including man.

With the coming of industry there was an increase in the amount of lead introduced into the food chain; but with improvements in food technology, this has been reduced in the last century and for the last 30 years has been stable. Since the introduction of lead antiknock agents into gasoline some 45 years ago, there has been no evidence of a general rise in the human body burden of lead and no evidence of any effect of lead on the health of even those groups in the population most exposed to automobile exhaust.

The position of the Du Pont Company is that the present lead levels in the population from all sources (including lead antiknock agents) are at a level well below the point at which effects on health occur. The Du Pont Company recommends that the body burden of lead and the atmospheric lead level be monitored on a regular basis and is itself engaged in this process on a large scale. Should an upward trend in the body lead levels of the general population due to lead antiknock agents be detected, reduction in the amount of antiknock added to gasoline to bring about an appropriate reduction in the population's body burden of lead would be in order.

Lead is a Normal Component of the Earth's Crust

Lead is a normal and ubiquitous constituent of the earth's crust being present in an average concentration of about 16 parts per million.

Lead is a Normal Component of Plants and Animals

Because of its ubiquitous nature, lead is absorbed along with other trace elements by plants and animals which in turn form the diet of man.

Lead is a Normal Component of Man

Since lead has been a constituent part of the diet of man for many millions of years, it seems reasonable to conclude that man has always had a level of lead within his body which was derived largely from the diet and was not the result of man's industrial activities. This level of lead constitutes a "background" body burden which is always present, although varying in amount from place to place on the earth's surface, depending upon the local concentration of lead in the soil and water.

Lead Levels in Primitive Man

We at Du Pont have been able to analyze the blood of groups of people in remote portions of the world, far removed from cities and man-made sources of lead. In South America, we were fortunate to obtain specimens from the Caraja tribe in the Xingu National Park area of central Brazil. Eleven male Indians had blood samples drawn and analyzed and the average blood lead concentration of these 11 subjects was found to be 23 μg/100 g of blood. In southeastern Peru, on the eastern slopes of the Andes, live tribes of Indians who have seldom seen a white man. They are completely self-sufficient in regard to their food supply and are hundreds of miles away from any man-made sources of air pollution other than their wood fires used for cooking. Samples obtained from 39 Indians had an average blood lead range from 12 to 23 μg/100 g.

Lead Levels in Modern Man

In North America, using the same laboratory for the analyses and the same analytical technique, over 6000 working men and women are being sampled on a yearly basis at 25 different locations ranging from Michigan to Florida and New York to Los Angeles. The average blood lead value for the men in 1968 was 20 μg/100 g of whole blood; and for the women in the same year, the value was 17 μg/100 g of whole blood. These data are presented because they suggest that there are no gross differences between the blood lead levels of persons living in remote

areas of the world away from automobiles and those of an industrial population, most of whom live in cities and commute to work by car. These figures also suggest, to the somewhat limited extent that remote primitive tribes resemble our own ancestors, that such blood lead levels have been present in man for long periods of time.

Are there Trends in the Lead Levels of Modern Man?

Because lead is not destroyed in the body and has a relatively slow rate of excretion, the concentrations of lead in the blood and urine of individuals and of groups of individuals reflect their environmental exposure to lead. Since it has been possible to measure lead with a satisfactory degree of accuracy for many years, it is possible to follow the trends in the lead levels of man over a period of time. In a study of the lead levels in the blood and urine as reported in the world literature, it has been shown that contrary to what might have been expected there is no evidence of an upward trend over the past 30 to 40 years. Figure 1 shows these data for the levels of lead in the blood, while Figure 2 shows the lead concentration reported in the urine.

While it would be most useful if there were earlier data available, the span of time covered in the figures is one of the most important with respect to lead emissions since this is the period during which the volume of lead antiknock agents sold has risen steeply and, therefore, the absolute amount of lead introduced into the urban air has greatly increased. It has been suggested that one reason why the lead levels in the blood and urine have not risen is because absorption of lead from some sources has been falling and has counterbalanced the possible increase from automobile exhaust. The evidence available would suggest that the lead levels from sources other than the air did in fact drop due to improvements in food technology, replacement of lead-containing insecticides by chlorinated hydrocarbons, and the replacement of lead water supply pipes by copper. These changes in the lead levels in the diet largely took place, however, before the period covered in Figures 1 and 2; and it is believed that at least in North America the lead intake from food has not changed appreciably in the past three decades.

Lead as an Air Pollutant

The major portion of the lead in the atmosphere is in the form of very small particles of inorganic lead salts; there may also exist trace amounts of organic lead vapor.

The inorganic lead particles are derived from two sources and constitute the lead aerosol or particulate. The first source may be termed "natural" because it is not related to man's activities and is

composed of:

- silicate dust
- volcanic halogen aerosols
- volcanic silicate smoke
- forest fire smoke
- aerosolic sea salts
- meteoric smoke.

To these natural sources of lead may be added the radioactive isotope Lead-210 which is present in trace amounts as a result of the decay of Radon and which has been used as a tracer for airborne artefactual lead. The chemical and physical identity of the naturally occurring lead in the atmosphere has been little studied and is consequently largely unknown.

The second source of the lead aerosol derives from man's activities. Primary and secondary lead smelters produce mainly oxides of lead while the burning of gasoline containing lead antiknock agents produces complex lead halogen salts. As might be expected, such activities as storage battery manufacture, pigment productions, insecticide production, and the burning of waste materials lead to a wide variety of inorganic lead salts with a considerable range of particle size.

Main Sources of the Airborne Urban Lead

Emissions of lead may occur from a variety of industrial processes, from the combustion of coal, and the incineration of waste materials. A certain amount of lead may also become airborne from the reentrainment of lead-bearing soils and street dust, although these two sources are considered to be minor contributors to the atmospheric lead level. The main source of lead in the urban environment is the exhaust from automobiles burning gasoline containing lead antiknock agents. Apart from emission inventory studies that suggest this to be true, the fact that lead concentrations in the air vary with the traffic density, the distance from the highway, and the ambient carbon monoxide concentration support this belief.

Levels of Lead in the Atmosphere

As would be expected in considering the huge volume of air overlying the United States, the amount of air sampling that has been done appears small and inadequate and yet enough is known to establish certain principles. Air lead concentrations measured at sampling locations that represent the general environment give average values ranging from 0.3 to 2.5 $\mu g\ Pb/m^3$. These values may be regarded as the

general background levels of lead in the air upon which are superimposed peak concentrations. These peak concentrations may be related to a particular geographic location such as the area downwind from the stack of a lead smelter or may vary with time at a given location as occurs during the rush hour on city streets. Air samples taken for relatively short periods in or close to traffic show average lead concentrations ranging from 9 $\mu g/m^3$ to 38 $\mu g/m^3$ in weekday samples from downtown and freeway routes in Los Angeles and Cincinnati, and from 2.8 $\mu g/m^3$ to 5.8 $\mu g/m^3$ for rural roads.

While isolated measurements of air lead values have some value, the trends of these measurements with time are perhaps even more important. In considering the trend of air lead levels, the amount of valid data is quite meager since even small changes in sampling site may produce significant changes in the measured values and very few sampling sites have remained unchanged over a number of years. In two cities for which data are available, Los Angeles and Cincinnati, a downtrend of mean lead levels is evident. In Los Angeles the mean for the period 1954-1955 was 6.6 μg Pb/m^3 and for the period 1961-1962 the mean was 4.3 $\mu g/m^3$. In Cincinnati, making use only of data obtained from fixed, continuous sampling sites, there is a fall in both the central residential and commercial areas over the period from 1946 to 1962, while the peripheral residential levels rose from 1946 to 1951 and then showed a slight drop until 1962. This information, scanty as it is, runs contrary to what might have been expected from merely considering the increased number of cars and gallons of gasoline sold in the cities in question. A possible explanation of this apparent paradox may lie in the distribution of the lead aerosol with relatively constant lead levels now being established in the core areas of large cities due to traffic saturation, while there is a tendency for a rise toward these levels to occur in peripheral areas. In addition to these factors which would tend to prevent an increase in lead concentration in the center of the city, changes in traffic patterns caused by new freeway construction, methods of home heating, slum clearance projects, etc., could lead to a decrease in air lead levels. Some support for the hypothesis that there has been no general upward trend in lead concentrations in the cities is provided by the data available from the National Air Sampling Network shown in Table I, although it must be noted that the same cities are not necessarily represented each year.

Factors Affecting Biological Effects of Lead

In considering the possible biological effects of a compound such as lead, it is necessary to know the dose of the compound administered, the route of administration, the duration of the exposure, the chemical and physical nature of the compound and the state of the organism exposed. Changes in any or all of these factors may have an important bearing on the biological effect or lack of effect of the compound on the organism.

Relationship Between Lead Levels in Man and the Atmosphere

Lead taken by mouth is largely excreted in the feces and in a topological sense has not entered the body; and in a similar fashion, lead which has entered the lung and has not yet been absorbed into the blood through the alveolar wall, can also be regarded as outside the body. The amount of lead that will be absorbed into the blood from that deposited or retained in the lung is a function of the particle size, density, solubility, and concentration. Because of the difficulties of experimentation, very little is known about the amount of lead aerosol retained in the lung under realistic conditions; and even less about the amount of lead absorbed from the lead so retained since lead, like any other particle coming to rest in the lung, can be removed from the lung by phagocytosis or by ciliary action, or by a combination of the two. From a review of the literature, it appears that the quantity of lead deposited in the lung from the type of aerosol produced by the automobile is probably less than had been previously thought.

One of the most important factors determining the effect of the lead aerosol lies in its physical characteristics. Being made up of fine particles these will deposit in different portions of the respiratory system depending upon their size and will then be subject to various body processes which tend to remove them. In the most complete theoretical treatment of this dynamic process of deposition and clearance of aerosols so far attempted, an international committee has formulated a model which, based on present knowledge, enables the dose of lead actually absorbed from the lungs to be roughly calculated. Any such model must be used with caution since it rests on a large number of assumptions and approximations, but it may be useful in providing an idea of the amount of lead actually absorbed from the particles of lead in the air since direct experimental measurement of the fraction absorbed is very difficult. Using as input to the model the available information about the physical and chemical nature of the lead aerosol, it can be calculated that about 15 per cent of the inhaled lead aerosol would actually reach the blood compared to an absorption of about 10 per cent from lead which is swallowed.

A different approach to the problem of relating air lead levels to blood lead levels has been taken by Goldsmith and Hexter who made use of published data to estimate the daily air lead exposure level for groups of persons for whom the average blood lead levels were known. While such a relationship based on rough estimates of average airborne lead exposures in a 24-hour period must necessarily be tentative, it does suggest a tendency for blood lead levels to rise along with air lead levels, although this tendency becomes considerably less marked as the air lead measurements rise above the present range of average values found in the ambient air. The data show considerable variance and some anomalies are apparent when they are compared with some other studies.

In one such study, lead levels in the skulls and ribs of persons dying in Los Angeles were examined to see whether they correlated with the length of residence in the Los Angeles area with its generally higher lead levels; it might be expected that those who had recently moved to the Los Angeles area would have shown lower lead levels than those who had been residents of that area. No such difference in levels was found, suggesting that other factors such as occupation may quite overshadow an effect due to length of residence.

The Goldsmith-Hexter view of the relationship between the levels of lead in the air and those in the blood may be a true representation of the relationship between average air lead concentration and blood lead level; but in the absence of evidence that the individual groups used in constructing the regression line were properly investigated to see whether group averages could, in fact, be used, judgment on the value of the Goldsmith-Hexter line must be deferred. To allow such group averages to be used to calculate a linear regression line, individual regression lines should have been fitted to each of the groups of data points comprising the population representing each sub-population. These individual regression lines should then be subjected to the following three tests before a single regression line can be fitted: (1) the variance about each of the regression lines must be similar; (2) the slopes of the regression lines must be the same, that is to say, they can be considered parallel; (3) the separate parallel lines must be shown to be segments of one straight line. To perform these three tests, the individual data points are required.

It is a much more powerful statistical technique to use all of the individual data points at one time rather than to take group averages. This statistical argument is not to deny that there is a relationship between high levels of lead in the air and blood lead levels. Such a relationship has been demonstrated and used for years in safeguarding the health of industrial populations. What seems more open to question is the form of the relationship in persons exposed only to the relatively low levels of lead encountered in the community air. To further illustrate the difficulty of making general statements about the relationship between the quantity of lead inhaled and the amount absorbed, it has been generally assumed for some years that smoking cigarettes caused an elevation of blood lead levels.

In 1967, however, G. Lehnert (Intern. Arch. Gewerbepathol. Gewerbehyg. 23: 358-363) published a paper showing no difference in the blood lead levels of 116 men between 20 and 22 years of age living under identical environmental conditions and eating similar foods. Seventy-one of the subjects were smokers and 45 were nonsmokers. The average blood lead levels for the nonsmokers was 16.3 μg Pb/100 g of blood; and for the smokers, 16.4 μg Pb/100 g of blood. Since the average number of cigarettes smoked was 17, this would give an average value of 13.61 μg of lead inhaled in the smoke; therefore an appreciable amount of lead was presented to the lungs in a very

finely divided form, but this did not result in an increase in the blood lead level. Being intrigued by these results, we investigated the smoking habits of our industrial population referred to earlier and attempted to correlate the smoking habits with the blood lead levels. The lead level of smokers was 19.71 μg Pb/100 g of blood and the lead level of nonsmokers was 19.78 μg Pb/100 g of blood. Thus we have confirmed that in our series there is no difference between smokers and nonsmokers in blood lead level and that this holds whether a person smokes four packs of cigarettes per day or none at all. In a similar fashion, pipe and cigar smoking was found to be without effect on the blood lead level. These results run counter to those found in the Three Cities Survey and no immediate explanation is apparent; but if confirmed, it suggests that amounts of inhaled lead originating from cigarettes, up to about 64 μg per day for the four-pack-per-day smoker, have no effect on the blood lead level. This digression into the effects of smoking is intended to underline the apparent importance of the form and composition of the lead aerosol in determining absorption. Dr. Goldsmith has demonstrated that an aqueous lead acetate aerosol is easily absorbed, as one would predict, but we still know far less than we need to know about the retention and absorption of finely divided solids in the lungs.

Biological Effects of Low Concentrations of Lead

No research workers have suggested that the present levels of lead in the air are associated with the classical signs of lead poisoning. These classical signs are anemia, muscular paralysis, and abdominal pain. Therefore if there is an effect of the urban lead aerosol on man, it must be sought in more subtle effects. Of the possible subtle effects which have been looked for, the only one that appears to have some merit as an early sign of lead absorption is the effect on the synthesis of hemaglobin. This effect has the additional merit that measurement of the degree of interference by lead is relatively easy by determining the δ-aminolevulinic acid (ALA) level in the urine. This inhibition by lead is almost specific, not being shared by other heavy metals, and the appearance of excessive amounts of ALA in disease states is confined to certain rare porphyrias in which, in contrast to lead, the urinary porphobilinogen level is also raised. The level of lead in the urine above which an increased excretion of ALA occurred was 50 μg/lit as reported by Haeger-Aronsen for lead-exposed workers. In a study carried out by Du Pont's Haskell Laboratory, the relationship in lead-exposed workers between an increasing concentration of lead in the urine and the concentration of ALA is curvilinear (Figure 3). "Non-lead-exposed" office workers showed no significant change in ALA concentrations over the range of urine lead concentrations encountered in the study (Figure 4).

A point worth making about studies of the relationship of gradually increasing levels of lead intake to an index of metabolic

derangement, such as ALA concentrations, is that such relationships tend to be curvilinear, so that there is a range of lead intake which has no biologically significant effect on the ALA level and then as the level of lead intake rises above this "no effect" region, a rise in ALA concentration takes place at a rate greater than the rate of increase of the lead intake. This concept of a level of lead intake below which no biologically significant effect occurs is open to some philosophical arguments; but as a practical guide in regulating the use of food additives, in controlling pollutants, and in setting safe levels of chemical exposures in industry, it has served us well.

Figure 3 shows the type of curve derived from measurements made on lead-exposed workers, with the virtually horizontal portion of the curve covering the range of lead values found in the normal population.

In dealing with the health effects of lead, Dr. Goldsmith has stated that metabolic effects of lead exposure, such as a raised ALA level, may be reflected in the blood and urine at lead levels that are considered to be within normal limits. This statement is naturally disturbing because if it is true, it means that we cannot necessarily place reliance on the blood and urine lead levels to guide us in assessing the health risk from exposure to lead. The relationship between the level of lead in the blood and the level of ALA in the urine has been well studied in man and animals and is fairly well understood. If the level of inorganic lead intake by the body is substantially raised above the normal level, first the urine and later the blood will reflect this increased intake by showing an increased concentration of lead. After this rise in blood lead has occurred, and if it is sufficiently great, the level of delta-aminolevulinic dehydrase will be depressed and this in turn is followed by a rise in the ALA levels in the blood and urine. If now the lead intake is reduced to normal, the blood and urine values will return to their normal values at a rate which is dependent upon the degree and duration of the previous elevation. The enzymatic depression will take longer to return to normal and will lag behind the fall in the blood and urine lead values. If the lead levels have been elevated for a period of months or years, the ALA levels may remain elevated for several months to a year after the lead levels have fallen to normal. With this background, if we reexamine Dr. Goldsmith's statement, we can see that high ALA levels could be found in the presence of normal blood and urine lead values if the person had previously had a fairly severe lead exposure. The confusion on this point has probably arisen because most of the studies of ALA levels have been carried out in populations with an abnormal exposure to lead. This point is illustrated in Figure 5 which superimposes two sets of data, one from lead-exposed workers, and one from office workers with no occupational lead exposure. It can be seen that for the same range of urine lead values, the lead-exposed workers tend to have higher ALA values.

Some persons concerned about the possible effect of lead on health have suggested that an increased amount of lead in the blood should be classed as an "effect" of lead and used as an air quality criterion in setting air quality standards. This seems illogical since body lead levels appear in general to follow the environmental lead levels in a passive manner and since lead has presumably always been present in the diet. A range of blood lead values must therefore be considered normal. If one accepts this premise, then an increase of storage may be perfectly acceptable if it occurs within the normal range; but at some point above this range an effect on health will occur. It is the level of this transition zone between safe and unsafe levels which causes so much debate. The debate drags on because we lack vital data on the long-term effects of various tissue levels of lead or disturbances of porphyrin metabolism. Here, the epidemiologists must help by studying populations with moderately elevated ALA levels and comparing the health of such groups with those without raised ALA levels. If there is no difference in the health of the two groups, there would seem to be no reason to use a slightly elevated ALA level as a criterion for setting an air quality standard. Of course, the problem posed is the measurement of health, and this is not really such a semantic slough of despond as some would believe, and in fact, is the daily bread-and-butter of the epidemiologist. What is important is that we keep our eyes firmly fixed on the functioning of the whole man rather than on individual enzyme systems.

Are There Groups in the General Population Who are More Susceptible to the Effects of Lead?

Of course it is possible to bring forward for discussion any number of hypothetical groups that might be more susceptible to the effects of lead than the general population; but to make such discussions fruitful, there should be some reasonable grounds for believing that the effect claimed is relevant to the actual levels of lead now being absorbed or that might be expected to be absorbed by the population in the future. In the case of women and children, the special concern for the wellbeing of these two groups makes an examination of their susceptibility to lead important. It is necessary however to define what is meant by "more susceptible" before any worthwhile conclusion can be reached. In this paper "more susceptible" is taken to mean that under the same conditions of exposure leading to the absorption of equal concentrations of lead one population group will exhibit more severe effects, the onset of the effects will be quicker, or new, serious effects will appear.

That the child may exhibit more alarming symptoms and face a more grave prognosis when suffering from lead poisoning is true, but what is also true is that the dose of lead which the child ingested is usually manyfold greater in proportion to his size than that received by most adults suffering from lead poisoning. The mean daily fecal output of lead by the lead-poisoned children in Chisolm's series (44 mg Pb/day) exceeded by approximately sixfold that of a group of severely exposed industrial workers (7.6 mg Pb/day). The group of industrial workers

chosen for comparison were exposed to lead-containing dust much of which is swallowed in the saliva. To quote Dr. Chisolm: "The more frequent occurrence of encephalopathy in children as compared with adults may depend in part upon their more intense exposure rather than upon any inherent biologic differences between child and adult." The relatively massive lead exposure usually encountered in pediatric practice is not directly relevant to the study of the chronic low level exposure to lead in the community air.

One of the other groups in the population that has been the subject of concern with regard to the possible harmful effects of lead are pregnant women. In this case there seems to be some evidence in the older literature, published at a time when controlled studies were not usually employed, that lead does affect both the capacity to conceive and the fetus. Unfortunately, none of these studies give any quantitative measurement of the lead exposure; but from knowledge of conditions at that time, it is known that industrial and personal hygiene were often conducive to severe chronic lead exposure.

Due to the evidence that bad working conditions contributed to infertility, abortion and stillbirths, women in most industrial countries have been prevented by governmental regulations from working in occupations in high lead exposures. Therefore, it is not possible to obtain current data on the effect of lead on women in industry. In one of the few studies in which modern epidemiological methods were used, the U. S. Public Health Service, in 1939, studied the fertility of groups of men and women exposed to lead arsenate insecticide in Wenatchee, Washington. Although the lead levels in the lowest and highest exposure groups were significantly different, there was no detectable effect on their fertility.

The result of this scarcity of modern data on the effect of lead on human reproduction means that some reliance must be placed on studies using animals. In one of the few such studies so far reported, workers fed two levels of lead acetate in the diet (64 and 512 mg/kg of diet) to rats from four weeks until one year of age. Four litters of offspring were produced by most of the females during this time. No significant differences were noted in fertility and fecundity between the experimental and control groups. Equal numbers of animals were then taken from the second litters of each group and bred to provide an F_1 generation. Again, no difference was found in the fertility, fecundity, mortality or the ability of dams to rear their young between any of the groups. That the lead in the diet was absorbed is shown by the fact that the average lead content of newborn animals from dams receiving 64 mg Pb/kg was eight times that in the newborn animals from dams on the control diet.

With the admittedly scanty information available, it cannot be denied conclusively that groups of persons may exist who are unusually sensitive to lead in the concentrations now present in the ambient air, but it is also true that such groups have not yet been found.

Based on Health Effects, What is a Safe Level of Atmospheric Lead for the General Population?

While it is obvious that much of the data are conflicting and it is always tempting to retire from the field to await the results of more research, many eminent men have counseled against this saying that to wait for more research is to wait forever and the problem is here and now. If then we accept the condition of working within the confines of the existing data, those charged with promulgating the regulations must accept the condition that any standard arrived at may need to be changed in the light of new information, and they should be willing to do so.

Since standards to be meaningful must not change too frequently, their basis must be carefully examined by knowledgable persons in the field from at least two main points of view. First, is it scientifically sound?; and second, if adopted, can it lead to satisfactory maintenance of air quality? The second point is of particular concern in the case of lead since it has a long biological half-life and measures to control lead emissions must necessarily take time to be effective. While the evidence taken as a whole suggests that the body burden of lead is not increasing, this does not mean that it may not in the future. A biological monitoring program must, therefore, be instituted on a regular yearly basis not only for lead but perhaps for other pollutants to provide a series of reference points for the detection of trends. If the trend is found to be upward, then a series of steps should be taken to reduce emissions; and such steps must be known to be effective.

There is no evidence that the present body burdens of lead in the general population are causing any deleterious effect on human health, and furthermore there is no convincing evidence that the present levels of lead in the air exert a significant effect on this body burden. Nevertheless, some persons have expressed a strong sentiment for setting an air quality standard for lead, and indeed two states have already done so. To the extent that an air quality standard set at this time will be based on inadequate and fragmentary data, the sentiment should be resisted. If, however, it is felt that an air quality standard for lead is necessary, then the data upon which a tentative standard should be set would be derived from two main sources.

The first would be the extensive experiments of Kehoe using human volunteers at the Kettering Laboratory and which were summarized in his statement before the Division of Air Pollution Control of the Department of Health of the Commonwealth of Pennsylvania on January 19, 1967. These experiments which are still in progress would lead to a figure of 10 μg Pb/m^3 as being a conservative level at which to set an air quality standard, and in Kehoe's opinion further information now being obtained may well allow this figure to be raised.

The second source of data upon which an air quality standard can be based is derived from animal studies. Animal studies tend to confirm the human data and suggest that only relatively great increases over presently existing community air lead levels may be expected to have a significant effect on the body burden of lead. In studies conducted by R. F. Lutmer et al., mice were exposed for 15 months to low levels of non-irradiated and irradiated auto exhaust in a cyclic 24-hour pattern. Mouse bone lead concentrations were not significantly affected when compared with control air animals until the 24-hour average atmospheric lead concentration exceeded 9.6 μg Pb/m^3. The atmospheres containing non-irradiated auto exhaust produced higher bone lead values than did the irradiated exhaust. The irradiated atmosphere more nearly represents the situation in the ambient air.

These studies using mice have a certain built-in safety factor because mice are animals which are constantly grooming themselves by licking their fur. This grooming results in the swallowing of a portion of the lead particulate that inevitably falls out on their coat, while, in addition, mice have a more primitive lung clearance mechanism than do human beings and thus a greater retention of the lead aerosol by the lungs would be expected.

Two more theoretical approaches to this problem may be made: the first making use of the Goldsmith-Hexter regression line would indicate that at an air level of 10 $\mu g/m^3$ the blood lead value might be about 34 μg/100 g, and this is a figure which is not considered hazardous to health. The second and entirely theoretical approach makes use of the ICRP lung dynamics model. Using an acceptable daily dose from pulmonary retention of 0.3 mg Pb, the highest acceptable air concentration is calculated from the model to be 18 $\mu g/m^3$.

Taking these admittedly fragmentary and approximate figures, a conservative air quality standard derived for lead would be 10 $\mu g/m^3$ based on a 30-day average. This figure is based solely on health criteria which are considered to embody a considerable safety margin, and the 10 μg figure is not to be construed as a sharp line between health and disease but a figure which would not lead to any significant, detectable, adverse effect on the population in the present or reasonably foreseeable state of our knowledge. It is recommended that

biologic monitoring of representative population groups should be continued to measure the actual level of lead absorption rather than merely relying on the levels of lead in the air. In the case of lead and other materials that behave in a similar biologic manner, biologic monitoring is a more precise indication of the exposure of human beings regardless of the route of absorption or the exact form of the compound. It is also recommended that studies be undertaken of the ALA levels of populations in areas of light and heavy lead exposure since this is one of the most sensitive tests of a lead effect in the intact human being.

GJS/dyb

TABLE I

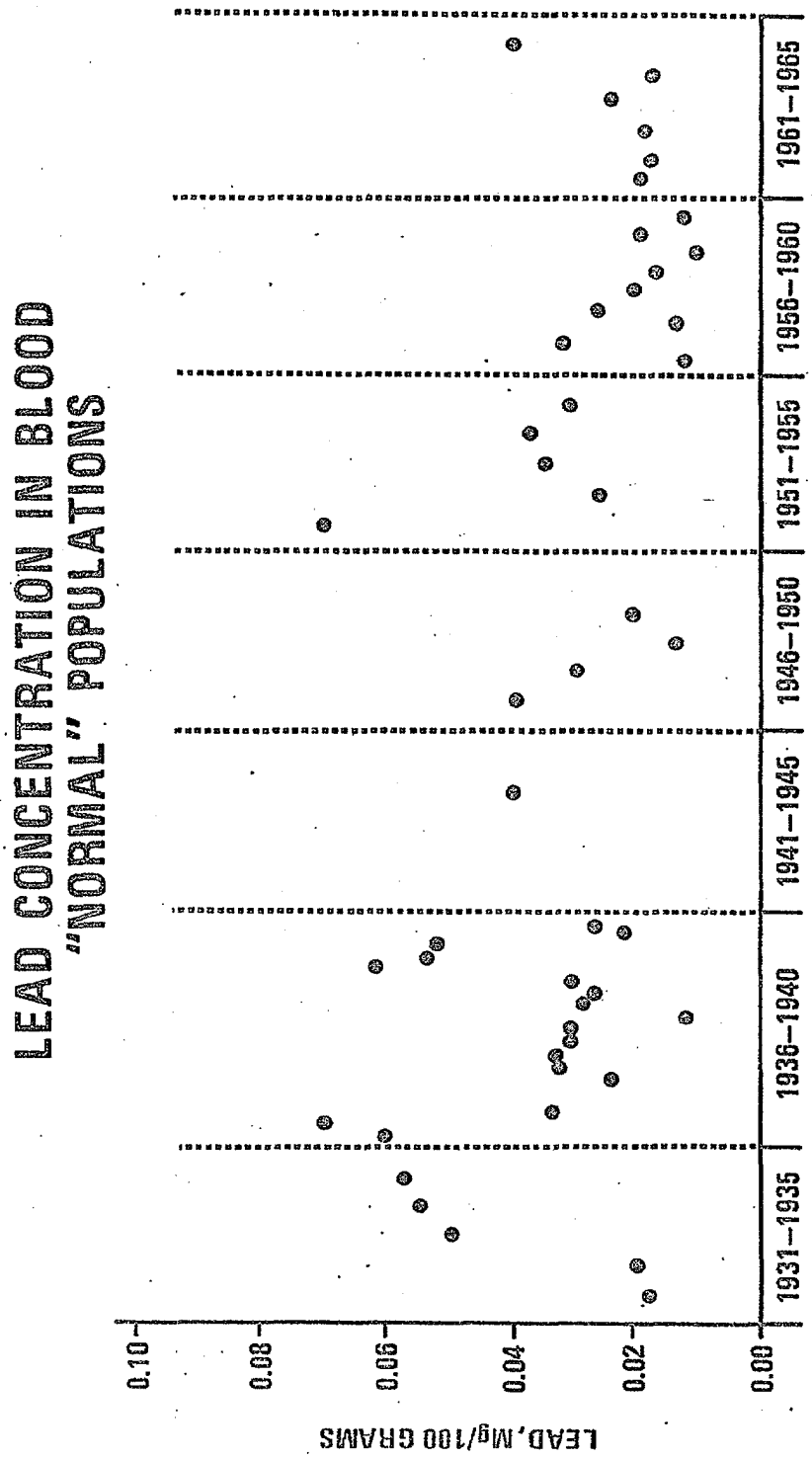
Yearly Average Air Lead Concentrations

U. S. Cities Over 100,000 Population in N.A.S.N. *

	1954	1955	1956	1957	1958	1959	1960	1961	1962	1963	1964
Average Lead in Air, $\mu g\ Pb/m^3$	2.27	1.32	0.61	0.62	0.50	1.40	0.87	1.37	0.99	0.96	0.74
S. D.	1.47	1.02	0.34	0.23	0.36	--	0.53	0.95	0.81	0.42	0.32
Number of Cities	12	19	14	5	24	1	6	11	28	17	20

* From Air Pollution Measurements of the National Air Sampling Network.

FIGURE 1



LEAD CONCENTRATION IN URINE "NORMAL" POPULATIONS

FIGURE 3

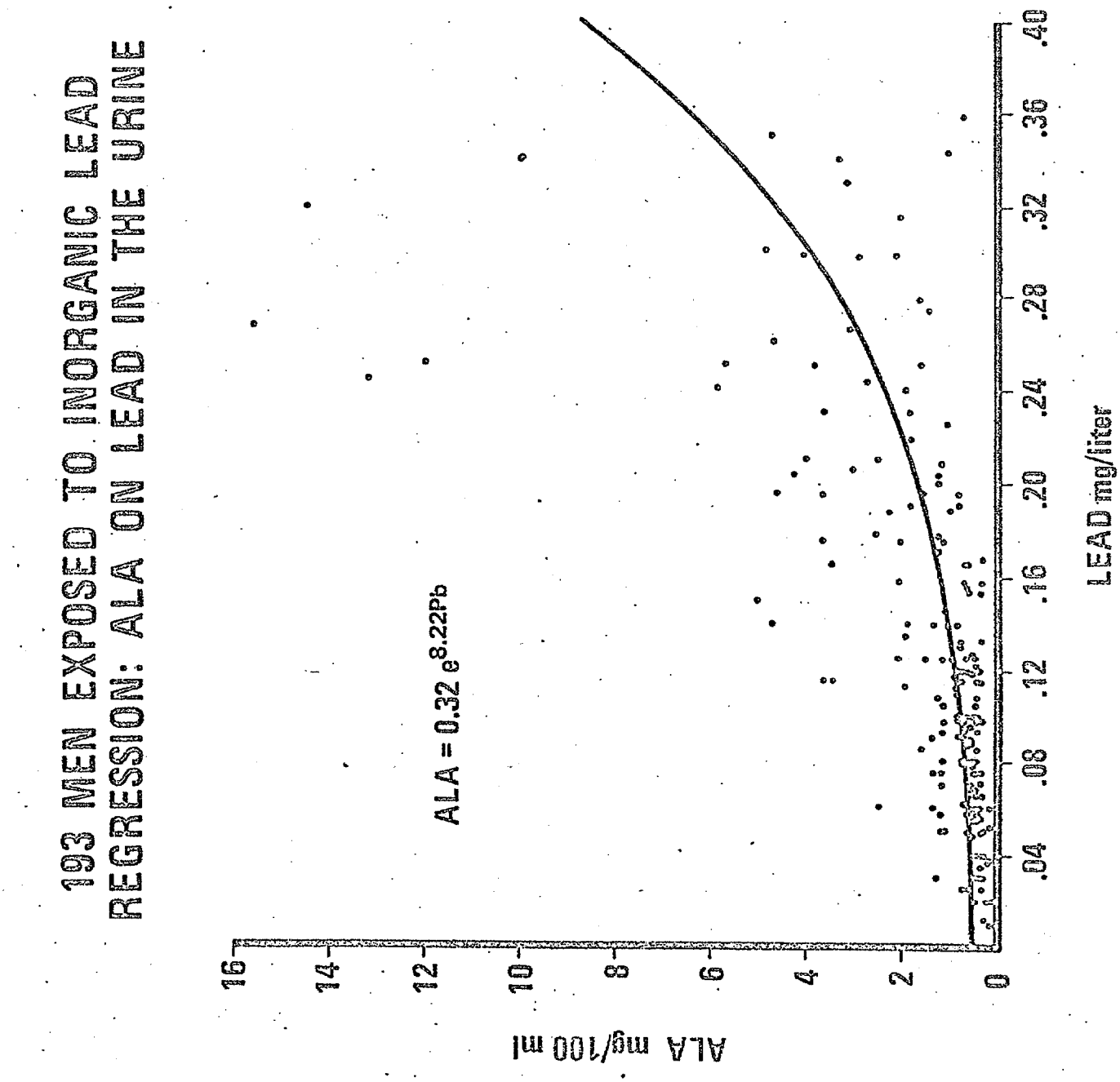


FIGURE 4

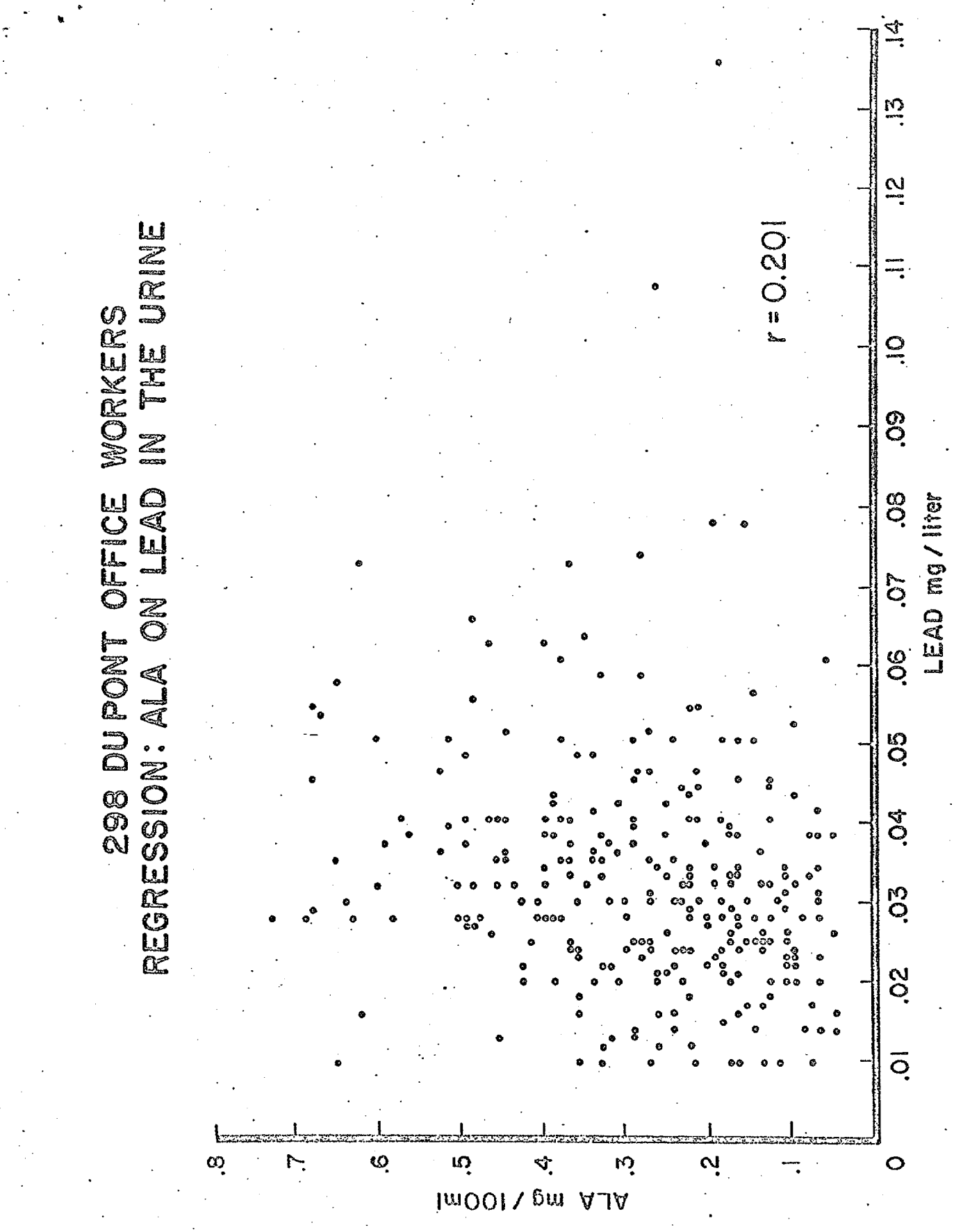


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FIGURE 5