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UNITED STATES ENVIRONMENTAL
PROTECTION AGENCY

14

INTERNATIONAL SYMPOSIUM

Environmental health aspects of lead

Die gesundheitlichen Aspekte der Umweltverschmutzung durch Blei

Les problèmes sanitaires posés par le plomb présent dans l'environnement

ILZRO'S RESEARCH TO DEFINE LEAD'S IMPACT ON MAN

COLE JEROME F. and LYNAM DONALD R.

International Lead Zinc Research Organization Inc.
NEW YORK, USA

Amsterdam, October 2 - 6, 1972

Summary

The International Lead Zinc Research Organization (ILZRO) has sponsored a comprehensive program of research on the environmental health aspects of lead. These projects have included studies of the character and concentrations of atmospheric lead and also of the impact of environmental lead on public health.

Specific emphasis has been placed on determing the dynamics of absorption of lead by man and animals from the environment, and also an assessing the biological impact of exposure to lead. It was concluded, that, although man's activities have caused a generalized ecologic translocation of lead and that detrimental effects to health are caused only by exposure to lead far in excess of the concentrations to which the public is continuously exposed.

Introduction

The International Lead Zinc Research Organization, Inc. (ILZRO) is the research arm of the world-wide lead and zinc industry. ILZRO's member companies account for more than three-fourths of the world's lead and zinc production outside the Communist bloc of nations.

Since its inception ILZRO, alone and in cooperation with other industrial and governmental groups, has sponsored a broad program of research in order to further our understanding of the effect of lead on health. This research has been responsive to the potential problems associated with the use of lead and its products and indicates the industry's interest in protecting its employees, the general public and the environment.

Lead in the Environment

Sources

Lead is a natural constituent of soils and thus has always found its way into the food man eats, the beverages he drinks, and the air he breathes. Superimposed upon the naturally-occurring lead, however, is a quantity caused by man's industrial and social activities. Of course, among the best known sources of additional lead are emissions from the smelting of non-ferrous metals, the manufacture of lead products, and the usage of lead alkyl antiknock additives in gasoline.

Lead in Air

One of the earliest and the most extensive study of the size distribution of atmospheric lead aerosols was conducted by Stanford Research Institute under ILZRO sponsorship (1),(2). This study utilized a Goetz aerosol spectrometer to determine the size distribution of lead aerosols at sites in Los Angeles, Chicago, Philadelphia, Cincinnati, San Francisco Bay area along with remote sites in Oklahoma and Arizona. The results indicated that the size distributions of lead at the different locations were fairly constant (mass median equivalent diameter of 0.12 micrometer to 0.31 micrometer) even when remote locations were compared with urban areas. Recently, however, Lawther, et. al. (3) have commented that sampling devices dependent upon inertial impaction, such as the Goetz aerosol spectrometer, may underestimate the size of normally aglomerated atmospheric lead particulates. Also, because of the irregularity of shape of the particles and the greater influence of diffusion rather than inertial forces on the movement of particles of this size, models relating mass median equivalent diameter to lung deposition may not apply to atmospheric lead particles.

The recently completed "A Survey of Air and Population Lead Levels in Selected American Communities", better known as the "Seven-City Study" provides much information of the concentrations of lead in the atmosphere of urban and non-urban areas in the United States (4). This study was carried out by the Kettering Laboratory of the University of Cincinnati and was jointly sponsored by the U. S. Environmental Protection Agency, the American Petroleum Institute and ILZRO.

Air lead concentrations were measured continuously for one year at 59 sampling sites located in and near Cincinnati, Philadelphia, Los Angeles, Washington, New York, Chicago, Houston and Los Alamos. The yearly means and confidence intervals of lead concentrations are summarized for each station in Table 1. The lowest mean annual atmospheric concentration occurred at a site in the desert community of Los Alamos, New Mexico (0.14 $\mu g/m^3$) and the highest mean annual concentration was found at an urban site in Los Angeles (4.55 $\mu g/m^3$). The data for the non-urban area in Okeana and Los Alamos represent an almost "background" situation while the data from Los Angeles represent levels found in an urban situation with a very heavy concentration of automobiles along with atmospheric and topographical factors conducive to the achievement of high levels of air pollution.

One of the purposes of the Seven-City Study was to determine if a change in concentration of airborne lead had occurred at a series of reference locations designated in 1961-62 (5).

The data showed higher lead concentrations at most of the sampling sites in the later study. However, data obtained from the U. S. Environmental Protection Agency National Air Surveillance Network (NASN) cast doubt on our ability to conclude that there has been an upward trend in airborne lead concentration over the 8-year period. Figure 1 shows the NASN data for the years 1960-69 at the same site in Los Angeles as one of the "Three City Survey" and "Seven-City Study" sites. Obviously the year-to-year variability in air lead concentrations, as demonstrated by the NASN data, preclude the possibility of defining a trend, either upward or downward, with only two points.

Lead in Crops

The impact of airborne lead concentrations on foods was investigated in another major program sponsored by ILZRO and conducted by the University of California at Riverside (6),(7) and (8). The lead content of 27 varieties of consumer crops and plants was determined in samples collected at various distances from highways with heavy traffic. The results of the study demonstrated that lead accumulations on plants were caused principally by aerial deposition and not, to any great extent, by absorption of lead from soil. Increases of lead in surface soil were restricted to distances from the highway of less than 50 meters and occurred only in the surface (0 to 7.5 cm. depth). Lead in soil was not related to distance from the highway at depths in the soil profile of 50 cm. It was concluded that lead from automobile exhaust exists as simple surface deposits on vegetation, that the highest lead concentrations in vegetation were those plant portions which have the highest surface to volume ratio, and that the concentration of lead on vegetation is a function of distance from the highway.

Despite the influence of proximity to the highway on the lead content on crops, the lead content of the portions of crops normally eaten were generally below 1.0 $\mu g/Gm$ on a fresh weight basis. The few exceptions were grown quite close to the roadways and in nearly all instances washing reduced the lead content to below 1 $\mu g/Gm$.

A number of fresh and frozen foods and one canned food - tomatoes, were purchased from markets in the study area. The results of this survey are shown in Table 2. It was concluded that these foods have estimated normal values and consequently are not considered to represent a significant source of lead contamination.

Lead in Water

In the field of water pollution ILZRO's only major project has been to define the current practices for the control of lead effluent from battery manufacturing. A survey of lead-acid battery plants, varying in size of production from 250 to greater than 12,000 batteries per day was conducted. The survey indicated that chemical precipitation with hydrated lime or sodium hydroxide was the most common treatment practice. It was concluded that lead concentrations in waste water from battery manufacturing plants can be reduced to 1.0 ppm with only nominal expense and care; the concentration can be reduced to 0.5 ppm with good design and operation; and concentrations of 0.3 to 0.2 ppm can be obtained only with very good design and extraordinary careful operations.

Biological Effects of Exposure to Lead

Several projects have been sponsored to determine the effects of lead on biological organisms. The scope of these various projects has spanned a range from the subcellular effects of lead to effects on the organism of our major concern, man.

Subcellular and Cellular Effects

The purpose of these projects have been twofold: (1) to provide a better understanding of the mechanisms of lead's toxicity and (2) to determine the concentrations of lead required to produce biological effects. The effects of lead on certain enzymes, nerve cells and immature red blood cells (reticulocytes) have been investigated.

One study has been aimed at the determination of the effect of lead on certain enzymes (10), (11). Though lead is known as a sulfhydryl enzyme inhibitor, it's affinity toward monovalent sulfur ligands in proteins and enzymes is actually considerably smaller than that of many other elements, e.g., silver, mercury, or cadmium. Concentrations of 10^{-3} to 10^{-4} M lead far in excess of concentrations present in tissues and body fluids in states of lead intoxication *in vivo*, are required to inhibit monothiol sulfhydryl enzymes.

Figure 2 shows that lipoamide dehydrogenase, an oxidative, FAD-dependant, di-thiol enzyme, was found to be inhibited by lead at lower concentrations *in vitro*. Fifty percent inhibition was caused by 6.5×10^{-6} M lead, but no inhibition was noted at a concentration less than 1.0×10^{-6} M. This inhibition was reversed by EDTA and partial protection was afforded by adding substrate and co-enzymes to the medium after lead was added, suggesting that lead binds to the active dithiol center of lipoamide dehydrogenase.

In another study the effect of lead on mitochondrial oxidative phosphorylation was investigated.(12). It was found that the threshold of beef heart mitochonodrial respiration is 24 x 10^{-6} M lead and 119 x 10^{-6} M lead is required to completely shut off mitochrondrial oxidative activity. It was found that the concentration of lead required to inhibit oxidative phosphorylation was much reduced by the absence of inorganic phosphate in the incubation medium. Since intracelluar inorganic phosphate is present in tissue, inhibition of mitochondrial respiration requires relatively high concentrations of lead. Through the use of a recording Fragilograph, it was found that lead-incubated human red cells were divided into osmotically sensitive and osmotically resistant populations. This differentiation of red cells, however, occurred only after incubation with very high lead concentrations (10^{-4} and 10^{-5} M of lead) which are well above normal physiological levels.

In another study at the cellular level it was shown that, unlike mercuric and thallium salts, lead acetate produced no morphological damage to tissue cultures of newborn rat and mouse cerebellum, spinal cord, and spinal ganglia (13). The highest concentration of lead acetate tested was 10^{-1} M. There was, however, a constant and marked degenerative reaction of macrophages followed by hypertrophy of glial cells. It was further found that there was no evidence of absorption of lead by the nerve cell in vitro from a lead-containing medium. Heavy uptake was, however, observed in macrophages, menigeal cells, and to a less extent by the glial cells. This different absorption of lead may explain the differences in the morphological damage effects noted.

Further studies examined the effect of the addition of sera from lead poisoned humans and laboratory animals to mature myelinated cultures of the nervous system. The exposure produced partial or complete degeneration and breakdown of the myelin sheath, which in most instances was accompanied by complete preservation of the nerve cell bodies and axons, but not of the glial cells. From the in vitro work it appears that lead has no direct effect on myelin and nerve cells, but rather than the excessive absorption of lead by mesenchymal tissue and the moderate absorption by glial cells is responsible for the breakdown of myelin sheath. This work, however, produced two unexplained but intriguing results:

1) The effects of sera from lead poisoned animals and patients on myelinated nervous system cultures was not correlated to the actual concentration of lead in these sera. Therefore, it is possible that the observed myelin disturbances was not due to the actual presence of lead but rather to other substances secondarily present in these sera.

2) Lead appeared to facilitate the development and differentiation of nervous tissue in young cultures, although the mechanism and meaning of this observation is not clear.

Though the above investigations of the celluar and sub-celluar effects of lead were undertaken to provide a better understanding of the toxic mechanisms of lead, one study had a, perhaps, more direct purpose. Microorganisms from an activiated sludge plant were used to determine inhibitory effects of lead on

secondary sewage treatment plants (9). The results of the study which included turbidity, BOD, and bacteria count experiments, indicated that lead in concentrations ranging from 0.1 to 20 ppm has little, if any effect on the growth of the microorganisms during the usual sludge digestion period. A lead concentration of 100 ppm definitely inhibits the activated sludge microorganisms and could completely disrupt an activated sludge process. Lead in concentrations of 50 ppm appears to inhibit the growth rate but probably would not upset an activated sludge system.

Effects of Lead on Animals

Because of the practical difficulties in conducting some types of experimental work on humans, several projects have been aimed at defining the effect of lead on laboratory animals. In one such study, rabbits and guinea pigs were exposed for their entire lives (periods up to 4 years) to urban air. This air contained a mean concentration of 2.46 ug/m^3 (14), (15). Another group of animals was exposed for an equivalent length of time to air which has been passed through prefilters and absolute filters. The lead content of the liver, lung, kidney, heart, gastrointestinal tract, pelt, spleen, muscle and bone was determined. Of these, only bone showed a statistically significant difference in lead content with that of both the rabbits and guinea pigs being higher than their respective controls. Mortality and longevity were identical in the exposed and non-exposed colonies and no effect on rate of gaining weight was attributable to exposure. Exposure of rats to 10 $ugPb/m^3$ continuously and intermittently in an inhalation chamber for a year produced similar results (16), (17). Animals were sacrificed periodically and lead content of the total body, bone, kidney and blood was determined. It was determined that lead in blood and solft tissue reaches equilibrium much more rapidly on continuous exposure than does bone and, consequently, total body lead content.

In yet another experiment rats and monkeys were exposed to an average of 21.5 $ugPb/m^3$ in a chamber for a twelve month period (18). Figure 3 shows the blood lead concentration of the animals exposed to lead versus unexposed animals both during the exposure and post-exposure periods. It is clear that the exposed animals clearly showed an elevated blood lead concentration in both monkeys and rats though the magnitude of the difference is apparently species dependent. In both species, however, the blood lead concentration appeared to stabilize after 4 months of exposure indicating an approach to an equilibrium condition.

An increase in the lead content of tissues other than blood also occurred in animals as a result of exposure to airborne lead. Measurements were made in the rats after six and twelve months of exposure and also in some rats six months after exposure was terminated. In bone, kidney, and liver tissues of exposed rats the concentration of lead was ten or more times as great as that of control animals. There was less increase in lung tissues. There were no striking differences between the levels found at six and twelve months as shown in Table 3. This indicates that a stable condition had been attained by 6 months in the rats, but is in some disagreement with earlier data which appeared to show a continuing increase in bone lead and total body lead content in rats during 12 months exposure to $10ug/m^3$ (16).

Delta-aminolevulinic acid dehydrase (ALA-D) activity in red cells was determined but the natural level of activity in the rhesus monkey is apparently too low to permit meaningful results. Natural levels of activity in rats, however, were higher and it was shown that after 12 months exposure, the activity of this enzyme in red cells was only about one-third that of the unexposed controls. The effect was reversible, however, and within one month after termination of exposure, the activity of ALA-D in red cells had returned to normal. Despite the depression of the ALA-D activity in red cells of exposed rats, there was no difference between exposed and control animals in ALA-D activity in liver and brain tissues obtained at the twelve month autopsy.

Examination of the animals also included routine blood studies selected to disclose the general well-being and clinical status of the animals. Routine clinical chemistry examinations of the rats and monkeys did not reveal any deviation of either the control or exposed groups with regard to hematology and histopathology. Both groups, then, appeared to be equally healthy with the exposed animals seemingly unaffected in any detrimental way by exposure to 21.5 $\mu g/m^3$ for a twelve month period.

Another study was directed at determining the effect of lead exposure on neurological behavior in rhesus monkeys (19). Animals were divided into the following dose groups receiving the following daily dosages of lead: 0,0.05, 0.50, and 5.00 mgPb per kilogram of body weight. During thirty-three months of testing, total blood lead, blood ALA-D activity and urinary delta-aminolevulinic acid (ALA) were determined to monitor the level of systemic intoxication. In additon, two standarized behavorial tests, a conditioned behavior performance test and a delayed response performance test were conducted weekly. No performance decrement was shown in any of the exposure groups in either test. The only significant indicators of lead intoxication were elevated blood lead and urinary ALA, as expected. Red cell ALA-D activity was measured, but as indicated previously, is naturally too low to serve as a useful indicator in this species. All other physiological parameters yielded no evidence of toxic effects due to chronic lead ingestion even at a level of 5 mgPb/kg body weight. It is of interest that this dosage produced a perhaps, unexpectedly, low stable blood lead concentration of 40-60 $\mu g/100G$ after reaching in excess of 80 $\mu g/100G$ early in the experiment.

Effects of Lead on Man

Several ILZRO projects have been aimed at defining the impact of lead directly on man. One experiment utilized human volunteers exposed to airborne lead in a respiratory chamber. Subjects were exposed to $10\mu g/m^3$ and $20\mu g/m^3$ of lead in air for increasing lengths of time during successive 16 week periods. The subjects' intake of lead in food and drink and output in urine and feces were carefully measured. A slight elevation of blood lead and urinary lead was noted in the subject exposed to 10 $\mu g/m^3$ but this was attributed to an unexplained increase in his intake of lead in food during the latter part of the exposure period. Two subjects exposed to 20 $\mu g/m^3$ for as long as 84 hours per week for 16 weeks produced equivocal results. It was apparent that the practical limitations as to the number of subjects available and the fact that exposure, while lenghty, was required to be intermittent prevented a true assessment of the effect of the effects of continuous exposure to these concentrations of lead in air.

We were fortunate, however, in that, with the co-sponsorship of the American Petroleum Institute and the U.S. Environmental Protection Agency, we were able to initiate a new project in which it was possible to expose 14 human volunteers simultaneously and almost continuously (23 hours/day) for up to 4 months (18), (20). The mean concentration of airborne lead in the chamber was 10.9 ± 3.1 $\mu g/m^3$, as determined by daily sampling and analysis. Inaddition there was some indication that an additional 10-15% lead passed the particulate filter. While this lead was not characterized, it may have been organic lead which was not changed to lead oxide particulate in the particulate generating flame and which, because of the recirculation of air in the chamber, may have built up over the course of the exposure period. Studies of particles collected on electron microscope grids indicated that the mean geometric diameter of the particles produced in the flame was about 0.05 micrometers.

The mean concentration of lead in the blood of eight men who remained in the study throughout the four month exposure period and 16 week post exposure period is shown in Figure 4. It is clear that exposure to this concentration of lead in air, under these conditions, produced a significant elevation of lead in the blood of the exposed subjects. The increase in blood lead concentration appeared to be limited to the first 10 weeks of exposure suggesting that a stable situation with regard to blood lead had been attained within the limits of the experiment.

The activity of red cell ALA-D was also estimated in both exposed and unexposed men. A significant reduction in the activity of ALA-D in the red cells of these men was noted shortly after exposure commenced and was maintained throughout the exposure period. Urinary excretion of lead was also elevated in exposed men.

It is noteworthy that the volunteers showed a significant weight gain during the exposure period. Whether this increase in weight was due to inactivity while in the chamber, to greater food intake, or to both is not clear. Fecal excretion of lead from the exposed men was higher than from controls, however, suggesting that food intake may have been a factor. Whether or not greater food intake by the exposed group may have, in part, accounted for the increase in blood lead concentration is also not clear.

Other studies of the human volunteers, which included comprehensive studies of serum chemistry, hematology, urinalysis, and excretion of heme precursers, including ALA, in the urine were normal and were unchanged during exposure. At present a similar experiment with a lower exposure level has been completed. However, the data have not yet been fully analyzed.

In interpreting the results of these experiments one must be mindful of the comments of Lawther, et al., (3) regarding experiments of this type. It has been pointed out that electron micrographs of atmospheric lead particles show them to be quite different from particles produced by burning tetraethyl lead in a propane flame, which was the means of generating lead particles in all the inhalation chamber projects. It may be that exposure to the more or less regularly shaped particles in these experiments, essentially unaccompanied by extraneous "smoky" material may create an exaggerated situation with regard to lung deposition and absorption.

The final data which will be discussed are the biological data arising from the "Seven-City Study." Blood samples were obtained from groups of volunteers in each of the eight cities in which air-lead measurements were made. Total fecal output was also obtained from some of these women in each city. In addition, smoking history was obtained. With the exception of some volunteers in Los Alamos, and in the case of special studies in Cincinnati, Philadelphia and Alpine County, California, volunteers were exclusively women. In order to be included in the study the women must have resided for at least 5 years within about a 1 mile radius of one or more of the air sampling stations. Women were selected because it was believed that they would be less likely to be occupationally exposed to lead and would be more likely to spend a greater portion of each day in and near their homes than men, i.e., within a one mile radius of one or more of the air sampling stations. The mean blood lead, fecal lead data, and the annual mean air lead concentration at the station(s) nearest their homes are shown in Table 4.

It was found that smokers exhibit a statistically significant greater blood lead concentration than non-smokers and previous smokers. However, there was no significant regression of age on blood lead concentration (age 20 to 79) of fecal lead content on blood lead concentration or of air lead concentration and blood lead concentration from 0.17 to 3.39 ug/m^3. Figure 5 shows the lack of a relationship between air lead concentration and blood lead concentration from 0.17 to 3.39 ug/m^3.

In addition an attempt was made to determine if the blood lead concentration of selected populations had changed over the period from the Three Cities Survey to the Seven City Study. In Cincinnati, 45 policemen, showed a mean blood lead in May, 1962 of 23.5 µg/100G and in March, 1971 they showed a mean of 17.9 $\mu g/m^3$, a statistically significant decrease, (P=0.00). In Pennypack, Pennsylvania, the mean blood lead concentration of 23 persons in April 1961 was 12.4 µg/100G and in April was 13.8 µg/100G, not a statistically significant change (P=0.09).

Another special survey conducted in Alpine County, California--a rural mountain area--showed a two month air lead concentration of 0.03 µg/m . The average blood lead concentration of 37 males and females, in 1961 was reported as 11µg/100G, while the mean blood lead concentration of 39 different persons in 1971 was 17.7µg/100G. While this was an apparent statistically significant difference (P=0.002), it was concluded that the 1961 Alpine County blood lead concentration, which constituted the lowest point of the controversial Goldsmith-Hexter regression was in error. This conclusion was based on the belief that neither a higher nor lower air lead concentration prevailed in Alpine County in 1961 than in 1971 and that the 1961 Alpine County blood leads were determined by a laboratory which exhibited highly erratic results (21).

Summary

It is apparent that man's use of lead has caused a generalized ecological translocation of lead. Further it has been shown that lead, at higher concentrations than to which the population is continuously exposed, does cause an increase in blood and bone lead concentration in man and animals. There is some evidence that lower airborne concentrations may cause some elevation in the bone lead concentration of laboratory animals. On the other hand, there is no evidence that harmful effects of lead occur at other than concentrations well in excess of those to which the public is continuously exposed.

ILZRO's research into the environmental impact of lead is continuing and hopefully it will continue to provide useful data of benefit to both the industry and the general public.

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TABLE 1

YEARLY MEANS AND CONFIDENCE INTERVALS OF AIR PB CONCENTRATIONS FOR EACH STATION

Area	Station	Geometric Mean ($\mu g/m^3$)	95% Confidence limits ($\mu g/m^3$)
Cincinnati	30	1.92	(1.63, 2.27)
Dec 1968	31	1.45	(1.16, 1.83)
Nov 1969	32	2.13	(1.75, 2.60)
	33	0.85	(0.71, 1.01)
	34*	0.31	(0.24, 0.41)
	35*	0.33	(0.20, 0.40)
Philadelphia Suburban	19*	1.28	(1.06, 1.55)
Dec 1968	20*	1.01	(0.86, 1.19)
Nov 1969			
Philadelphia Urban			
Dec 1968	11	1.89	(1.63, 2.20)
Nov 1969	12	1.74	(1.52, 2.00)
	13	3.75	(3.30, 4.26)
	14	2.18	(1.90, 2.52)
	15	1.39	(1.19, 1.65)
	16	1.09	(0.90, 1.33)
	17	1.08	(0.92, 1.27)
	18*	1.67	(1.42, 1.93)
Los Angeles			
Dec 1968	1	4.03	(3.40, 4.76)
Nov 1969	2	4.34	(3.58, 5.26)
	3	3.46	(2.96, 4.05)
	4*	3.72	(3.20, 4.32)
	5*	3.06	(2.62, 3.57)
	6	2.36	(1.92, 2.90)
	7	3.39	(2.62, 4.39)
	8	4.55	(3.71, 5.60)
Los Alamos			
Dec 1968	41*	0.14	(0.11, 0.18)
Nov 1969	42*	0.20	(0.16, 0.25)
New York Suburban			
Apr 1970	88*	1.14	(1.03, 1.27)
Mar 1971	89*	1.10	(0.98, 1.24)
New York Urban	81	1.76	(1.44, 2.15)
Apr 1970	82	2.04	(1.74, 2.38)
Mar 1971	83	1.73	(1.57, 1.90)
	84	1.71	(1.54, 1.89)
	85*	2.08	(1.79, 2.43)
	86	1.34	(1.13, 1.58)
May 1970	87	1.22	(1.06, 1.39)
Apr 1971			
Chicago Suburban	60	1.30	(1.17, 1.45)
Mar 1970	61	1.35	(1.23, 1.49)
Feb 1971	62	1.53	(1.33, 1.76)
	64	1.83	(1.67, 2.02)
	65	1.57	(1.39, 1.78)
	66*	1.18 (7 mo.)	(0.83, 1.68)
	67	1.55	(1.37, 1.76)
	68	1.87	(1.51, 2.31)
Chicago Urban	63*	1.76	(1.60, 1.93)
Mar 1970			
Feb 1971			
Houston	50	1.15	(0.92, 1.43)
Mar 1970	51	1.02	(0.86, 1.21)
Feb 1971	52	2.13	(1.83, 2.47)
	53	2.26	(1.87, 2.73)
	54	1.32	(1.16, 1.49)
	55	0.87	(0.73, 1.03)
	56*	0.85	(0.75, 0.96)
Washington, D.C.	70	1.10	(0.95, 1.27)
Oct 1970	71	1.64 (7 mo.)	(1.36, 2.00)
Sept 1971	72	2.21	(1.91, 2.55)
	73	1.78	(1.53, 2.06)
	74*	1.19	(1.07, 1.32)
	75	1.13	(0.99, 1.28)
	76	2.34	(2.05, 2.67)
	77	1.08	(0.94, 1.24)

TABLE 2

LEAD CONTENT OF VARIOUS FRESH AND FROZEN FOODS PURCHASED FROM LOCAL MARKETS

Type of food	Pb Content* As received	Water washed
	µg Pb/gm	
Fresh foods		
Celery	0.02	B.D.
Collards	0.50	0.25
Spinach	0.25	0.10
Romaine lettuce		
outer leaves	0.18	0.10
inner leaves	0.01	B.D.
Artichokes		
outer leaves	0.04	0.04
inner leaves	B.D.	B.D.
Broccoli		
flower	0.30	0.19
stalk	0.04	0.04
Carrots		
peels	0.06	0.05
inner portion	B.D.	B.D.
Potatoes		
peels	0.11	0.02
inner portion	0.02	0.02
Peas		
pods with peas	0.02	B.D.
pods	0.02	B.D.
Frozen foods		
Beans	B.D.	--
Peas	B.D.	--
Carrots	B.D.	--
Spinach	0.07	--
Strawberries	0.02	--
Potatoes	0.01	--

* Results are expressed as µg Pb per gram of fresh or frozen vegetables as received.

B.D. indicates below the limit of detectability, or less than 0.01 µg Pb/gm.

TABLE 3. AVERAGE TISSUE LEVELS WITH STANDARD DEVIATIONS OF LEAD IN RATS EXPOSED TO AIRBORNE PARTICULATE LEAD*

Group	Tissue			
	Bone	Kidney	Liver	Lung
6 Months Exposure-				
Control	0.36 ± 0.12	0.19 ± 0.04	0.24 ± 0.06	0.11 ± 0.04
Exposed	4.49 ± 1.14	2.30 ± 0.47	3.63 ± 0.98	0.72 ± 0.53
12 Months Exposure-				
Control	0.43 ± 0.14	0.21 ± 0.04	0.27 ± 0.06	0.12 ± 0.03
Exposed	5.13 ± 1.36	2.03 ± 0.60	2.83 ± 0.53	0.43 ± 0.17
6 Months Recovery-				
Control	0.50 ± 0.19	0.22 ± 0.03	0.27 ± 0.07	0.14 ± 0.05
Exposed	5.42 ± 1.09	0.45 ± 0.15	0.71 ± 0.20	0.14 ± 0.05

TABLE 4

MEAN AIR, BLOOD AND FECAL CONCENTRATIONS--SEVEN CITY STUDY

Community	Location	Sampling Site	Mean Air Lead ug/m^3	Number	Mean Blood Lead ug/100G
Philadelphia	Rittenhouse Square	18	1.67	136	20.5
Philadelphia	Ardmore	19,20	1.15	150	18.0
Cincinnati	Okeana	34,35	0.32	166	15.7
Los Angeles	Pasadena	4,5	3.39	193	17.5
Los Alamos	Los Alamos	41,42	0.17	191	14.9
Washington	Washington	74	1.19	219	19.2
New York	Greenich Village	85	2.08	140	16.6
New York	Port Washington	88,89	1.13	198	15.3
Chicago	Bridgeport	63	1.76	147	17.6
Chicago	Lombard	66	1.18	208	13.9
Houston	Houston	56	0.85	191	125

* Feces were collected from 20 women at each location.

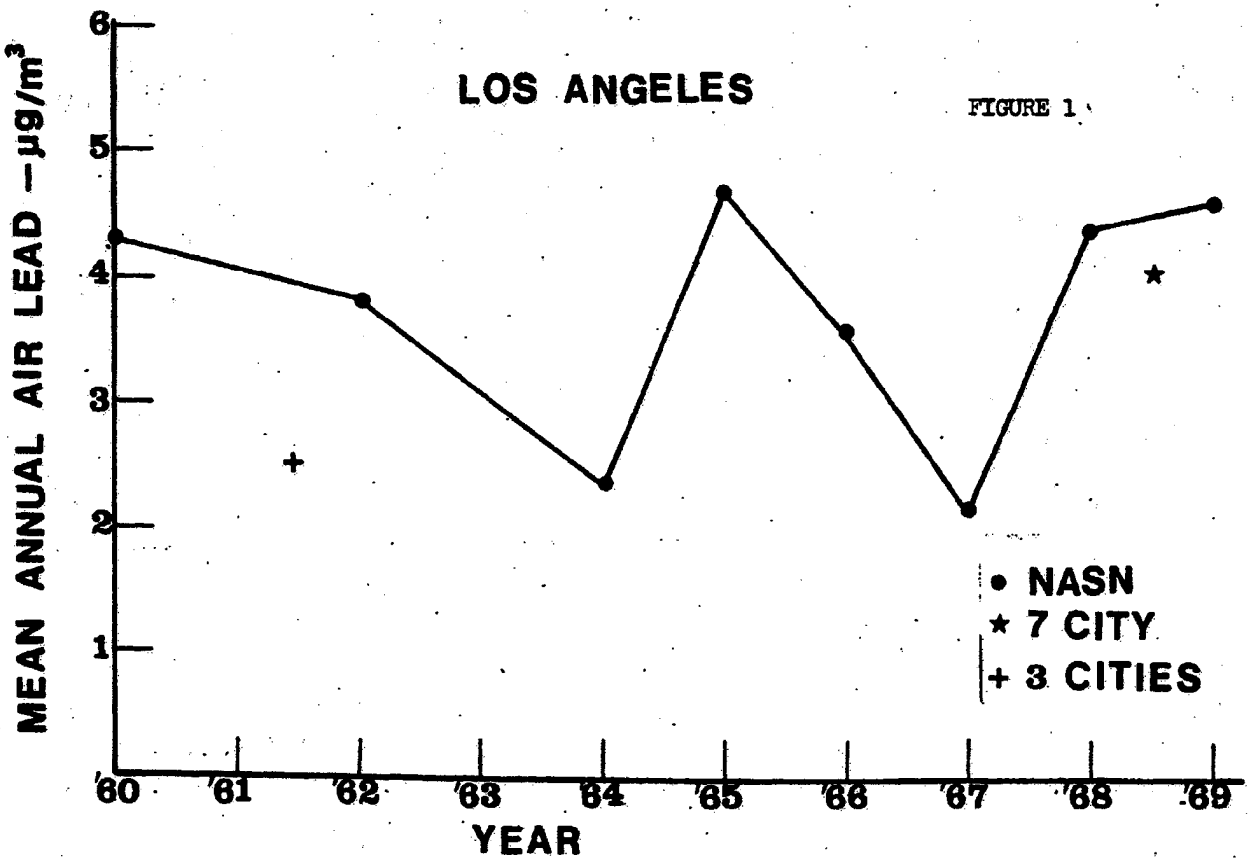


FIGURE 1

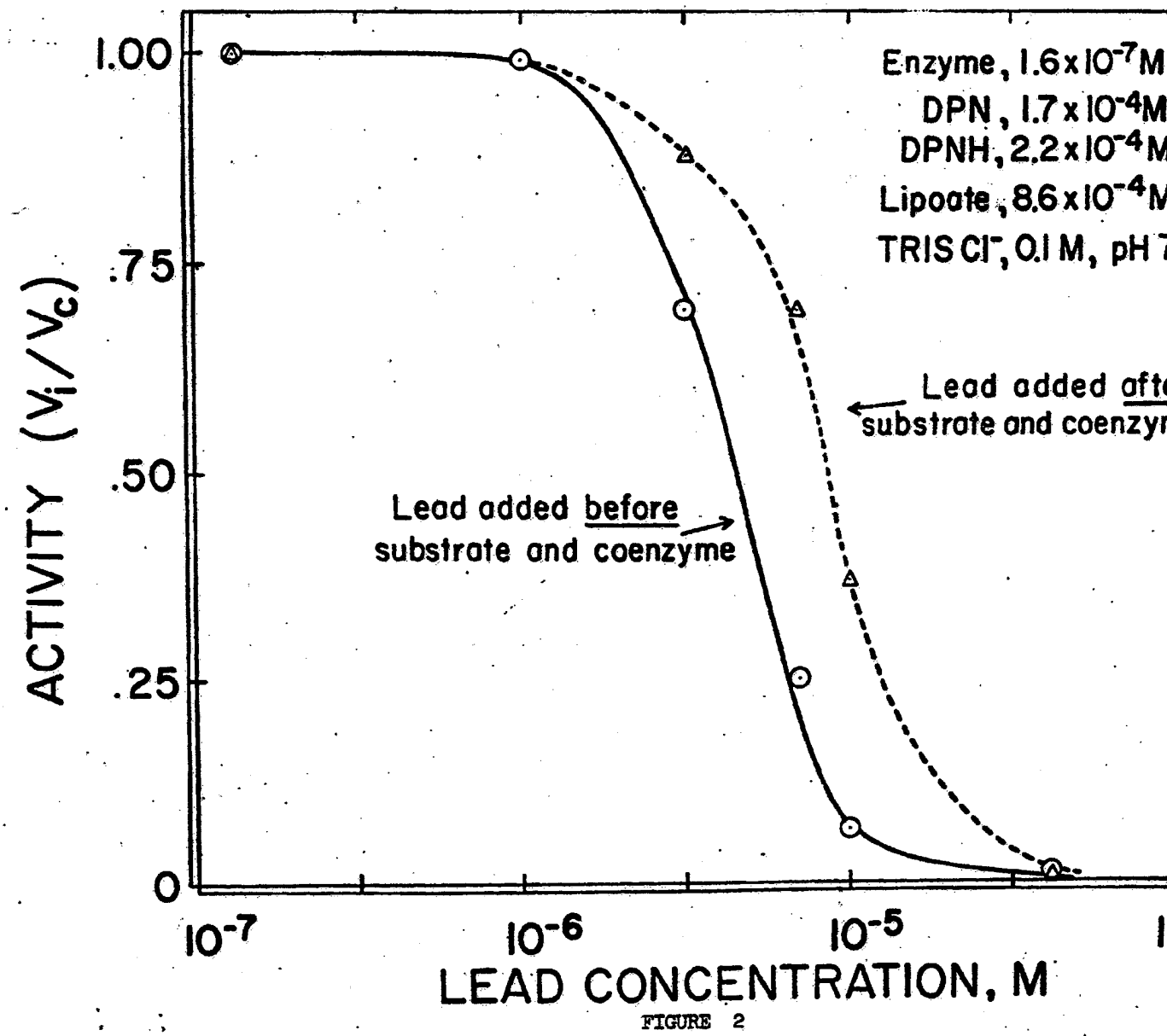

1.00
.75
.50
.25
0
ACTIVITY (Vi/Vc)
Enzyme, 1.6x10-7M
DPN, 1.7x10-4M
DPNH, 2.2x10-4M
Lipoate, 8.6x10-4M
TRIS Cl-, 0.1 M, pH
Lead added after
substrate and coenzyme
Lead added before
substrate and coenzyme
10-7
10-6
10-5
LEAD CONCENTRATION, M

FIGURE 2

FIGURE 3

BLOOD LEAD OF ANIMALS EXPOSED TO AIRBORNE LEAD

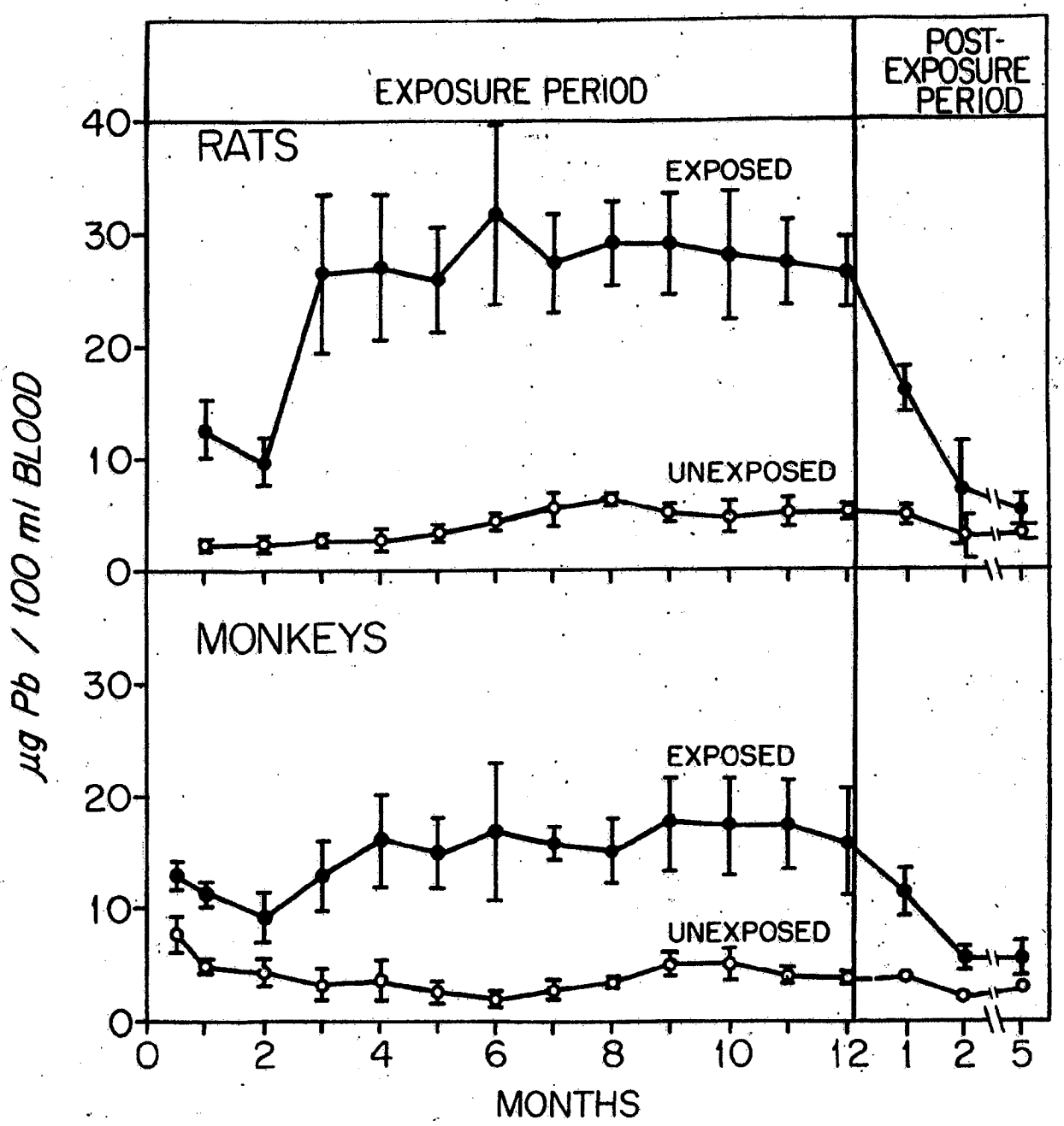
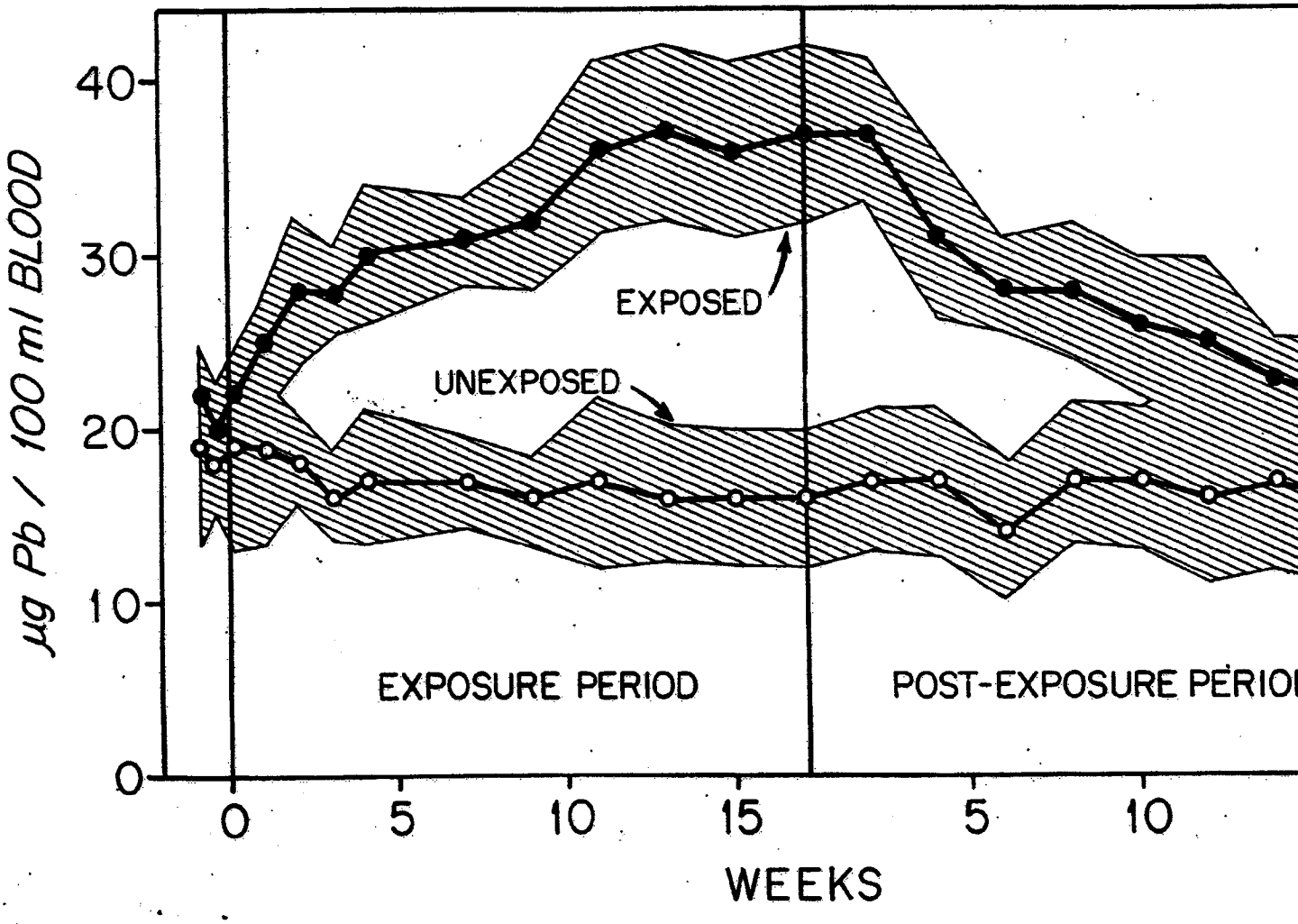


FIGURE 4

BLOOD LEAD OF MEN EXPOSED TO 10.9 µg/cu m AIRBORNE LEAD



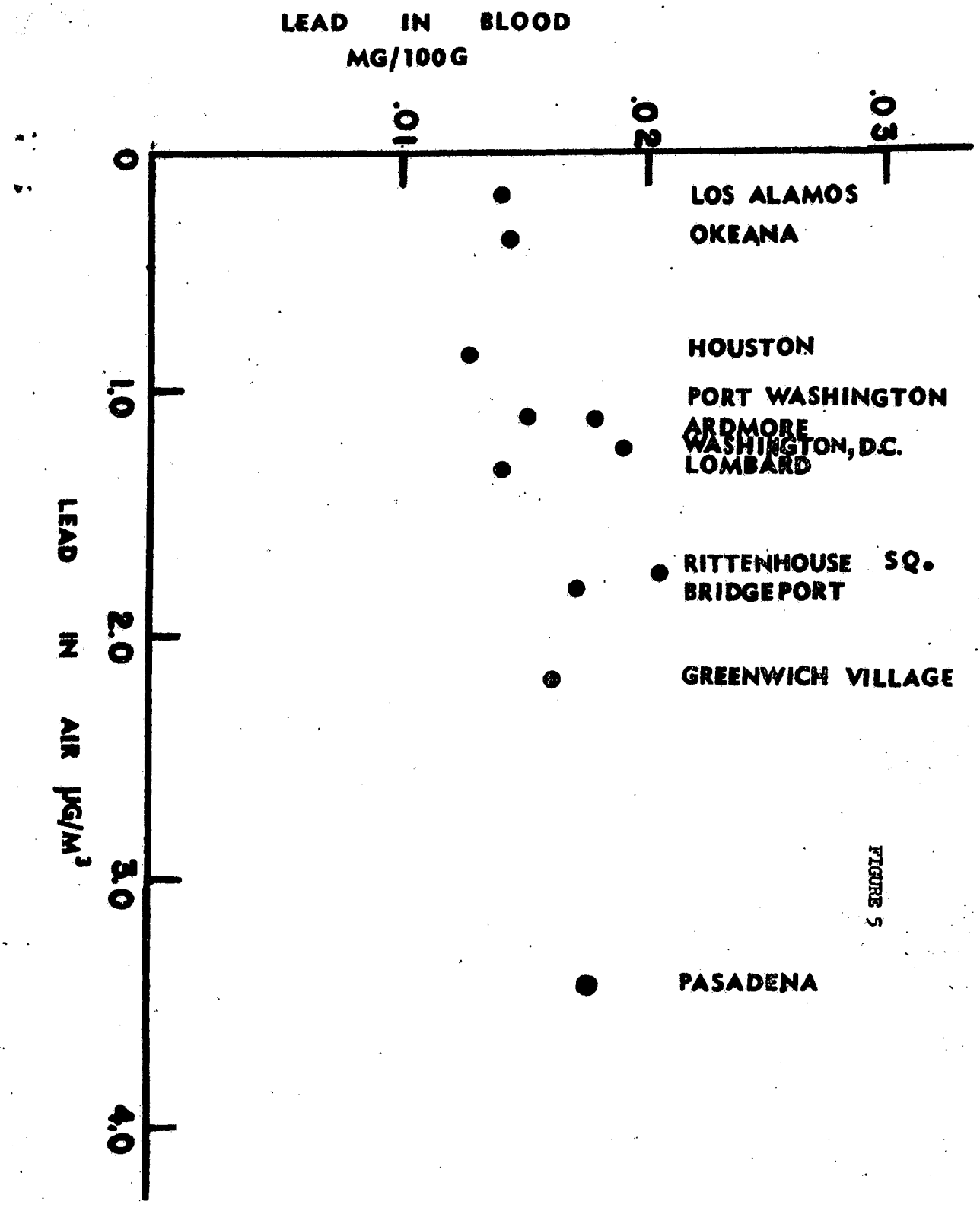


FIGURE 5