292 Madison Avenue · New York, N. Y. 10017 · Telephone: (212) 679-6020

A G E N D A

Meeting on

Atmospheric Lead Contribution to Pediatric Lead Poisoning

on

Tuesday, May 18, 1971

at the offices of

LEAD INDUSTRIES ASSOCIATION, INC.
292 Madison Ave.
New York, New York
14th Floor

9:30 a.m.

I. Introduction and Statement of the Problem . . . . . J. F. Cole

II. Hill & Knowlton Report of Available Information on Childhood Lead Poisoning (copy enclosed). . C. Thompson, R. Rogers

III. Possible Future Action Including Recommendation to ILZRO--General Discussion

a. Evaluation of Proposal From Environmental Sciences Associates (copy enclosed)
b. Extension of "Seven Cities Study"
c. Cooperative Study with APCO and the Bureau of Community Environmental Management, EPA
d. Preparation of a Position Paper
e. Other Suggestions.

It is hoped that the following persons will be in attendance:

Mr. H. Hesselberg
Dr. G. Roush, Jr.
Mr. R. Butler (representing Dr. Diggs)
Dr. J. C. Calandra
Mr. K. W. Nelson
Mr. C. Thompson
Mr. R. Rogers
Dr. S. F. Radtke
Mr. D. Borcina
Mr. J. L. Kimberley
Dr. J. F. Cole

cg:

LIA-76194

LEAD PAINT AND CHILDHOOD LEAD POISONING

"In the history of modern medicine, few childhood diseases occupy a position as unique as lead poisoning. The cause, epidemiology and symptoms have all been well defined. Methods for prevention, screening, diagnosis and treatment have long been available....

"Lead poisoning, or plumbism, is largely an occupational disease in adults, but in children it is almost invariably caused by repeated ingestion of chips and flakes of lead-containing paint and plaster from the walls, window-sills and woodwork of old and poorly maintained pre-World War II houses. Children between one and six years old are the main victims, and those between one and three years of age comprise about 85-90 percent of the cases reported."(1)

The speaker is Dr. John J. Hanlon, Deputy Administrator for Environmental Health of the U.S. Public Health Service. He was addressing a committee of the U.S. Senate on November 23, 1970.

The unanimity of opinion among pediatricians, toxicologists, epidemiologists and public health specialists is impressive. Childhood lead poisoning is almost invariably caused by lead paint pica -- the eating of chips of old paint peeled from the walls of substandard slum housing. The medical literature contains a few aberrant histories of cases originating from other causes, such as that of a child who liked to chew on a toy lead soldier. Otherwise, lead paint pica is the first thought of every physician when child lead poisoning is mentioned. To compile a complete bibliography on this well-established etiology would be both laborious and needless. A few sample quotations from recognized authorities summarize what is known.

The City of Baltimore has had a model control program for childhood lead poisoning for decades. Closely associated with that program for the past twenty years has been Dr. Julian Chisolm, one of the most eminent

specialists in the field. In 1970, Chisolm described childhood plumbism as "a specific insidious disease which has on the one hand definable consequences which are totally preventable and unnecessary and on the other hand, is due to a single factor. This single factor is quite clearly the presence of old paint applied 20 to 50 years ago when lead pigment paints were widely used, and this paint of course has never been removed. It is now chipping and flaking from dilapidated substandard housing." (2)

On another occasion last year Chisolm wrote: "The disease is almost entirely limited to children between one and five years of age who reside in old deteriorated urban housing built and painted prior to 1940."(3)

And again in 1970: "A few small chips of such paint may contain 100 mg. or more of lead.... Elimination of this environmental hazard would almost completely eliminate the problem of plumbism in young children as seen in the United States today." (4)

Chisolm confirmed this view in 1971: "Childhood lead poisoning in the U.S. is seen almost exclusively in children of pre-school age who live in deteriorated housing built before 1940.... The causative factors are commonly a triad: a dilapidated old house, a toddler with pica and parents with inadequate resources.... The three factors interact to increase the likelihood that the child will eat chips of lead paint." (5)

The concensus of organized medicine is similar. For example, here is a 1970 statement of the American Academy of Pediatrics: "Lead poisoning in childhood is a preventable disease. Virtually all cases occur in children who live in old, deteriorated houses which were built and painted years ago when the use of lead-based paints on housing surfaces was widespread." (6)

The Medical Committee on Human Rights, 1970:

"Dr. Rene Dubos, well-known scientist and environmentalist, has said, 'The problem is so well-defined, so neatly packaged, with both causes and cures known, that if we don't eliminate this social crime, our society deserves all the disasters that have been forecast for it.'

"Any plan for the eradication of this tragedy should include four points:

"(1) Testing of all deteriorating and dilapidated housing for the presence of greater than 1% lead paint.

"(2) Where such a lead hazard is found, it should be removed or the walls covered with a suitable material. Local governmental provisions for enforcement and penalties for non-compliance are absolutely necessary. People from the afflicted communities should be employed by those engaged in removing lead paint.

"(3) Testing of all exposed children between the ages of one and six years for the presence of an abnormal amount of lead in their blood.

"(4) Prompt treatment of all detected cases of lead poisoning and comprehensive follow-up on these cases as well as those children with high blood lead levels." (7)

American Public Health Association, 1970:

"Prompt and adequate removal of lead pigment paints from housing surfaces accessible to young children offers, currently, the only practical and effective means of preventing recurrent severe acute lead poisoning in young children." (8)

Finally, the U.S. Public Health Service revised and reissued in 1970 its basic pamphlet, "Lead Poisoning in Children," by Dr. Jane S. Lin-Fu. In a section entitled "Factors Contributing to Lead Poisoning," the publication says: "Although lead can be absorbed into the body via various routes

such as inhalation and skin absorption, in children lead poisoning results almost exclusively from ingestion of flaking and peeling lead-containing paints." A nine-point discussion, "Epidemiology of Lead Poisoning," devotes only one sentence to a cause other than lead paint: "Some cases occur in winter when leaded battery casings are burned for fuel and the fumes are inhaled or there is prolonged contact with the ashes." (9)

Established Cases of Child Lead Poisoning Not Associated with Paint Pica

As noted above, the practice of burning leaded battery cases for fuel occurs often enough among the extremely impoverished to merit mentioning as the cause of an occasional case of childhood plumbism. Obviously, old battery cases are not readily available even to all the poor families who might wish to burn them for winter fuel. Nevertheless, this rare situation appears to be the second most common cause, according to a 1966 study in Chicago by Perlstein and Attala. (10) These investigators reported the sources of lead for 425 cases:

Pica from paint or plaster	405
Burning of battery cases	17
Lead toys	2
Contaminated water supply	1

The 1970 review by Chisolm (4) notes that "unusual sources include":

1. Lead toys and baubles.
2. Plastic beads, necklaces and jewelry coated with lead to simulate pearl.
3. Lead nipple shields.
4. Toys and child furniture repainted with lead pigment paint by benevolent but unaware relatives.
5. Ashes of lead of painted wood or old battery casings used for fuel in stoves and fireplaces.
6. Improperly lead-glazed ceramic ware used for acidic foods such as tomatoes, tomato juice, vinegar and fruit juices.

As of 1971 there has never been a case of childhood lead poisoning traced to lead particles in the ambient urban air -- either inhaled or ingested in the form of dust.

Childhood Plumbism in Rural Areas

Chisolm has observed that "childhood lead poisoning is a real problem in many of the older urban areas of the U.S. and perhaps in rural communities as well," [5] indicating that even this expert knows little about rural prevalence of the disease in this country. However, reports from other countries indicate that when childhood plumbism occurs in rural and semi-rural areas, it can be traced to pica.

In the sparsely settled state of Queensland, Australia, climatic conditions apparently encourage pica, for as early as 1904 Gibson [11] traced the high prevalence of childhood plumbism to dried and crumbling lead paint on the porches of dwellings. Legislation restricting the lead content of paint has practically eliminated childhood plumbism in Queensland. However, as recently as 1970 Freeman [12] reported several cases associated with pica in the country areas of New South Wales and the suburbs of Sydney.

Oliver and O'Gorman [13] determined blood lead levels in hospitalized children in rural Oxfordshire, England, finding significant and direct correlation between blood lead ("normal, intermediate, high") and intensity of pica ("no pica, occasional, frequent"). Their 1966 report classified blood lead below 36 micrograms/100 milliliters of blood as "normal."

Control Studies of Childhood Plumbism

The empirical evidence indicting lead paint pica as the only significant cause of childhood plumbism has been so overwhelming that relatively few researchers have been inspired to investigate the etiology of the disease through control studies. Indeed most of the data comparing pica-plumbism cases with pica-free, plumbism-free groups of children have come not through studies of the disease itself, but rather through attempts to establish standards for diagnostic laboratory tests such as blood, urine and fecal lead levels.

A series of 906 children in a low-income neighborhood in Cleveland were studied by Griggs et al. in 1966. Of 801 children living in old houses, 216 (27 percent) had abnormal urine lead levels, and 38 (4.7 percent) were already afflicted with plumbism. Of 105 children in a new housing project, only three (2.8 percent) exhibited abnormal urine lead levels, and there were no cases of plumbism. (14)

Another index for potentially dangerous body lead burden is urinary coproporphyrin. Christian and associates (15) in 1964 examined urine samples for coproporphyrin from 500 children in a run-down Chicago neighborhood, from a second group of 500 who lived in a newly built area, and from a third cohort of 500, also in an area where "the buildings were relatively new and in a good state of repair, no lead containing paints were used inside the homes and no cases of lead poisoning were reported during the past three years." However, this third area was in proximity to a blast furnace emitting "a dense plume of smoke which enveloped the area during the greater part of the day and night. This air was found on repeated sampling to have a concentration of lead which was approximately ten times as great as that found in other areas of the city." The results of this control study are most significant. Of the children from the old housing area, 91 (18 percent) had elevated urinary coproporphyrin; 14 (2.8 percent) of the children in the second area showed elevated coproporphyrin; 16 (3.2 percent) of the children in area three, exposed to a ten-fold burden of atmospheric lead, had positive coproporphyrin. Of the 16 children from area three, 12 were subjected to further tests. Only one patient was found to have "a borderline blood lead level in addition to lead deposition in bone and a history of pica."

The same year Moncrieff and co-workers [16] compared blood lead levels in 20 mentally retarded children with lead poisoning with those in 80 normal, healthy children in London. Of the plumbism victims, all but two had a history of pica or had otherwise been exposed to lead paint; the source of lead in one case was never traced, in the other case it was presumed to be "home-grown vegetables from soil containing old battery casings." The lead poisoned children had blood lead levels ranging from 44 (in the case of the child who had eaten the lead-contaminated vegetables) to 380 mcg/100 ml. Of the 80 healthy children only two had blood lead higher than 36 mcg., the highest being 42 mcg. in a child who had been treated for eczema with an ointment of strong lead content.

Kopito et al., Boston, 1967, undertook to explore the quantitative analysis of lead in hair as an aid in the diagnosis of plumbism in children. The mean hair lead level of 16 pica-plumbism cases was 282 mcg/gm. of hair; 41 healthy pica-free controls showed a mean of 24 mcg. [17]

Fecal lead levels were measured by Barltrop and Killala,[18] London, 1967, in four groups of children aged 24-35 months: Group I - Consecutive hospital patients with diseases other than lead poisoning; Group II - Healthy children from a local child care clinic with no known history of pica; Group III - Similar to Group 2 except for positive history of pica; Group IV - Three cases of plumbism with histories of intensive pica. Mean fecal lead levels for groups 1, 2 and 3 was 123 mcg. per stool. However, one symptom-free child with pica had a level of 468 mcg. The three lead-poisoned children had levels so high as to make "statistical comparison unnecessary."

Chisolm [5] in Scientific American, 1971, reproduced a simplified table showing results of fecal lead measurements performed by Chisolm and Harrison, 1956 [19]. The simplified 1971 table is reproduced below. All the plumbism cases resulted from pica.

PATIENTS	LEAD OUTPUT (MILLIGRAMS PER 24 HOURS) MEAN	MEDIAN	RANGE
Unexposed Controls	.132	.157	.012-.175
Household Controls	.832	.651	.087-1.93
Increased Lead Absorption, No Symptoms	2.16	1.11	.116-9.60
Lead Poisoning, With and Without Brain Damage			
During Exposure:	44.0	27.0	5.040-104.0
After Treatment:	.362	.240	.062-0.850

EXCRETION OF LEAD in feces is an index of exposure to lead. These results of a study by the author and Harold E. Harrison illustrate the massive exposures seen in lead poisoning. Unexposed controls were children with no known exposure to lead. The other groups were children with increased lead absorption (high blood lead), children with lead poisoning and members of their households with neither high blood values nor overt symptoms.

Most recently, Weissburg et al., [20] 1971, reported on a rapid, reliable method of screening for plumbism through measurement of micro-quantities of delta-aminolevulinic acid dehydratase (ALAD) in the blood. Decreased ALAD blood levels indicate higher lead levels. In the course of their study the authors compared ALAD levels in children from Philadelphia slum areas, presumably exposed to lead paint, with those in a group of children from more modern homes: "The mean level of ALAD activity among the 60 children of slum areas, from whom capillary blood was obtained by finger stick, was 18.56 $\pm$ 8.51 ($\pm$ S.D.) U. The 50 children from modern urban or suburban areas, upon whom the finger-stick method was also employed, exhibited a mean ALAD activity of 28.11 $\pm$ 8.84 U. The difference between these two means is highly significant (p less than 0.001)."

Does Atmospheric Lead Contribute Significantly to Childhood Plumbism?

Voluminous literature shows pica and old lead paint as the overwhelmingly important factors in childhood plumbism. Nevertheless, the question has arisen whether airborne lead may constitute a possibly contributory source to childhood lead poisoning. Automobile exhaust fumes contain measurable amounts of lead and the concentration of auto fumes is higher in the city than in the country. The possibility has been partially investigated, and the data so far adduced indicate that airborne lead is not a significant factor in childhood plumbism.

The possibility of airborne lead as a factor in childhood plumbism involves three hypothetical types of exposure:

1. Children may inhale harmful quantities of lead directly.
2. Their food supply may be contaminated with lead particulates washed out of the atmosphere.
3. Lead particles may accumulate in hazardous concentrations in the dust of the city streets. Children play in the dust, suck their dirty hands, and so may intake lead.

Each of these three possible sources and the relevant evidence from published scientific studies is considered.

1. Hypothesis: Children may inhale harmful quantities of lead from auto exhausts directly.

Evidence in Support: Nil.

Evidence Against: One study specifically concerned with children is that of Christian[15], mentioned above. In a Chicago area where the concentration of lead in the air was approximately ten times as great as that found in other areas of the city, there were no cases of lead poisoning, and among 500 children, only one elevated "borderline blood lead level" which was subsequently found to be due to pica.

In a 1969 review Barltrop [21] has noted that as regards atmospheric lead "few studies concerning children have been made." Barltrop cites some examples of heavy air lead concentrations from lead smelters in India and Italy (none in the U.S.), but has no data on the effects on children.

However, there have been many studies of adult exposure to varying levels of airborne lead, together with measurements of blood lead levels. These studies indicate generally slightly higher blood lead among men occupationally (excluding smelters and factories) exposed to higher air lead. But there is no clear correlation between the two factors, and even the highest blood levels found are well within safe limits. Since little children do not work in underground garages and vehicular tunnels, they are obviously not exposed to these highest urban concentrations of air lead.

2. Hypothesis: The food supply may be contaminated with lead particulates washed out of the atmosphere.

Evidence in Support: Nil. The few unusual cases of lead poisoning from vegetables grown in lead-contaminated soil are the exceptions which prove the rule. In every case the contamination resulted from buried lead wastes, not from precipitated airborne lead. It should be noted that almost all agricultural soil contains trace amounts of lead which are absorbed by food crops. Some crops have a greater affinity for lead than others. In fact, this is the major source of body lead in most people throughout the world.

Evidence Against: Ter Haar[22] reported in 1970 on the results obtained by growing various food plants in open fields at various distances from a heavily travelled highway, and in greenhouses next to the highway -- some greenhouses equipped with air filters to remove particulates. "Tomatoes,

potatoes, wheat, carrots, leaf lettuce, cabbage, snap beans, and sweet corn were grown in filtered and unfiltered air. In all of these crops, except leaf lettuce, there was no measurable effect on the lead concentration of the edible portion due to lead in air. It seems probable that the lead found in plants grown in filtered air was due to lead naturally present in the soil. Only in the case of leaf lettuce was there a measurable difference between the concentration of lead when the plants were grown in filtered and unfiltered air. This difference would add 0.5 to 1% to the daily intake of lead.

"Likewise, when these same crops were grown at increasing distances from a heavily traveled highway, there was no measurable effect on lead concentration in the edible portion of tomatoes, potatoes, wheat, carrots, leaf lettuce, cabbage, oats, rice, and sweet corn. Snap beans had a higher lead concentration near the road, which could add about 0.75 to 1.5% to the daily intake of lead."

Schuck and Locke[23] reported in 1970 their analysis of the lead content of cauliflower, tomatoes, cabbage, strawberries and oranges, growing in areas from 50 to 2,000 feet from heavily travelled highways. The amounts of lead associated with the five crops averaged less than one part per million by weight.

3. Hypothesis: Lead particles may accumulate in hazardous concentrations in the dust of the city streets. Children play in the dust, suck their dirty hands, and so may intake lead.

Evidence in Support: Nil. Studies have indicated that dirt swept from city streets contains measurable amounts of lead. This would not be unexpected. As noted above, all dirt contains some lead, and it may be that dirt from the average city, contaminated with trash and waste, contains more lead than country dirt.

Evidence Against: Reference is made again to the study of Christian[15]. In a Chicago neighborhood surrounding a blast furnace, emitting lead particles to the level of ten as great as other areas of the city, there were no cases of childhood lead poisoning. Furthermore, the fallout of heavy lead particles from this industrial source (although not measured by Christian) must have been of even greater intensity than the airborne particles. Evidence of this comes from the review by Barltrop[21], who noted: "Much industrial air pollution is of relatively large size so that a considerable fallout occurs near the source. Figures published for an Italian plant showed maximum (lead) concentrations of 490 mcg/m^3 at 80 meters from the plant, falling to 37 mcg/m^3 at 320 meters."

Barltrop's observation concerning the weight and size of lead particles from fallout raises an interesting point. If lead from auto exhaust can be inhaled in dangerous quantities, it must be because auto exhaust contains very small lead particles which remain airborne and are readily inhaled. On the other hand, if lead fallout from auto exhaust occurs in dangerous quantities, it must be because these particles are too heavy to remain airborne. It would seem that one cannot inhale lead and eat it too.

Secondly, it is well known that almost all children get dirty and that a great many suck their fingers. But a child who eats dirt in significant quantities is afflicted with pica. This certainly can cause lead poisoning if the lead content of the dirt is high enough. If it were as high as the maximum generally considered safe in lead paint, a gram of dirt would contain one centigram of lead. Chisolm[5] points out that a chip of lead paint the size of an adult's thumbnail may contain 5 to 10 centigrams of lead.

In the United States, the recognized Dean among scientists interested in plumbism is Dr. Robert A. Kehoe, of the Kettering Laboratory, Cincinnati. In 1969 Dr. Kehoe addressed himself to the question of atmospheric lead[24]:

"It is almost impossible to prove that no harmful effect has been or is being induced by the presence in the human environment of a type of energy or a substance which, under certain circumstances or at some level of concentration is known to be harmful... (But) there is no probable or even rational relationship between the consequences of frank lead poisoning of either mild or severe types, as encountered in the literature and in present experience, and the consequences of exposure to lead in the common environment as it has existed during the past several decades. The apprehension expressed hitherto on this subject has disregarded completely the quantitative aspects of the matter, and has gone off into the never-never land of speculation and vivid imagination... It is not to be regarded as a reasonable hypothesis that so widespread a constituent of the general atmosphere as lead, in the concentrations in which it occurs, is responsible for shortening the lives of appreciable numbers of persons in our population... I confess to a deep concern about the many cases of frank lead poisoning which are known to occur among children in areas of poor housing in all but the newest of American cities... In the face of these existing facts of life in modern society, I find it somewhat incongruous to encounter such deep anxiety over a purely hypothetical threat. That every effort should be made to assess the present and future significance of this putative threat, is obvious. It is clearly mandatory that no obstacle, based solely on economic considerations, should be permitted to forestall effective action to eliminate any real threat which may be identified.

Moreover, a physiologically and socially acceptable standard for lead in the air, along with an applicable standard (or standards) for lead in food and beverages, should be adopted at the earliest feasible time, and an adequate surveillance of the environment and of the population should be continued."

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ENVIRONMENTAL SCIENCES ASSOCIATES, INC.
Laboratory Facility/49 Hampshire St./Cambridge, Mass. 02139 617 - 868 - 6700

22 April 1971

Dr. Jerry Cole
International Lead Zinc Research
Organization
292 Madison Ave.
New York, N. Y. 10017

Dear Jerry:

As we discussed yesterday I have enclosed a short letter-form proposal for determining the extent to which air-borne automotive lead emissions raise the blood lead levels of children and the extent to which the raised levels contribute to increased incidence of clinical lead poisoning.

Basically what I have done is to delineate the problem, and to separate it into two study areas, one dealing with the field study of children and their air environment. The second dealing with a laboratory study to determine the incremental intake level vs blood lead level function on a small group of laboratory animals. The problem you have posed is difficult, and I feel that an integrated two part approach will lead to a better documented answer to your question.

Sincerely,

Wayne

Wayne R. Matson, Ph.D.
Vice President, Technical Director

WRM/vlh

enc.

P.S. We are currently consulting in an area active in problems of lead processing and if you have any preference in operation near the sites I mention it could be arranged.

LIA-76209

Instrumentation, study, and solution of industrial and community environmental problems

N 869.02

ENVIRONMENTAL SCIENCES ASSOCIATES, INC.
Laboratory Facility
49 Hampshire St., Cambridge, Mass. 02139

22 April, 1971

THE ASSESSMENT OF THE CONTRIBUTION OF AUTOMOTIVE (AIRBORNE) LEAD TO LEAD BLOOD LEVELS, AND LEAD INTOXICATION IN CHILDREN

The Problem

The problem of assessing the effect of airborne lead on the levels of lead in children is two fold.

1) The assessment of the intake of lead from primary atmospheric sources, secondary sources such as street dirt, and tertiary sources such as food stuffs.

2) The assessment of the affect of the incremental uptake on the level of lead in the blood, and on the chemical toxicity of the increased lead levels.

Or to rephrase the problem, how much lead does a child get from airborne sources as distinct from paint, water, plaster, etc., and does this incremental lead intake cause a significant increase in lead poisoning, or is it excreted and removed faster in children at higher lead levels and hence unimportant.

The Approach to the Problem

The approach to the solution of the problem is a two-task study, frist in the field and second in the laboratory.

Task I

The primary task will be a field study in two cities to obtain correlation information on environmental and blood lead levels in three different time periods. Atmospheric levels of lead in regions of expected high and low values will be obtained. Similar information will be obtained for levels of lead in street dirt from the areas, and estimates of the aerosol lead contribution to foodstuffs in the area.

Where values are not already available, blood lead levels, medical histories and environmental histories will be obtained on the children in an area.

For optimum data handling, the dirt lead, air lead and blood lead levels will be obtained over the same time period in each given target area in order to normalize seasonal differences in the environmental exposure of children.

The areas which would be considered for the study to give the widest variation in atmospheric leads, and exposure of children are the Haymarket Square area in Boston, the central city New Orleans area, and the Norfolk, Va. CNY clinic.

The distribution data obtained for blood lead levels in children is expected to be a skewed distribution to the high side of the average. All points that can be attributed by a child's medical or environmental history to housing conditions will be discarded initially from the distribution. The average and the

median of the blood lead distribution in children will then be compared to a variety of functions generated from the data on air and street-dirt lead values to ascertain if (or under what conditions) there is a correlation between aerosol lead and blood lead averages.

Teh frequency distribtuion will then be studied to see if there is any significant increase in numbers of children over 40 µg% and 60 µg% that can be attributed to aerosol leads.

The Mode of Operation

Air samples will be taken over time periods that will allow for a reasonable certainty that the values obtained are the average values that a population will be subjected to during the time period that will affect their blood lead levels. Glass fiber filters will be used in self-contained air pump cases that can strap onto telephone poles in the areas studied. Average particle size distribution will be obtained in each area utilizing an Anderson impactor.

Dirt samples will be obtained from the target areas, and the apparent interaction of the child populace with street dirt will be assessed by an observer on-the-spot during the time period when the blood lead distributions and air samples are being obtained.

Samples of foods indigenous to the areas will be obtained, the lead content determined, and the extent to which this lead content is due to aerosol lead, estimated from the area where the food arose.

Blood samples will be obtained in these areas simultaneously with the environmental monitoring from 150-250 children to obtain suitable distributions for data handling. The three sample periods will be spaced as much as possible to obtain different periods of street usage for the population.

Task II

To assess the effect of incremental added lead on blood lead which will give an added dimension to the problem we would propose to inject ten individual rats at relative levels of 10, 5, 2 and 1 of lead intake.

We propose to take five animals on a regime of 10 days at an intake level of 1, 10 days at 2, 10 days at 5 and 10 days at 10, measuring their blood lead on a daily basis. Five additional animals will be taken 10 days at 10, 10 at 5, 10 at 2, and 10 at 1, measuring their blood lead level daily. The input level vs blood lead level will be plotted for individuals, and averages, to obtain curves for the expected effect of incremental incrases on blood lead.

It is expected that the curve will be highly non-linear in the higher regions, and that significant increases in blood lead will require massive increased inputs over normal.

The data obtained would be related to the environmental data to ascertain the level to which a biological system rejects an incremental dosage of lead, and predict the probably rejection of an added aerosol lead level in children.

Statement of Work - Task I and II

The work will be performed over a six month period - one month for planning, three months for field sampling, one month for analysis of data, and one month for reporting the work. Two urban areas will be analyzed three times for the following:

- Average air lead, (5 locations within area).
- Average particle size distributions (1 location).
- Average street dirt lead (15-30 locations).
- Blood lead distribution in area children (150-250 children).
- Average lead in foodstuffs (5-15 foodstuffs).
- Assessment of street usage and dirt contact.
- Assessment of high leads from lead (paint, plaster, water).

Ten rats will be carried for ten days at four separate lead levels and curves obtained for the individual and average response of blood lead levels to incremental increases in lead.

The data will be treated to ascertain the effect of aerosol lead on the blood lead average and to ascertain the incidence of over-average children. A monthly progress report wil be submitted and a full six month report will be prepared with a complete disclosure of data, and conclusions.

The proposed budget for Task I is $40,000, fixed price.

The proposed budget for Task II is $8,000, fixed price.