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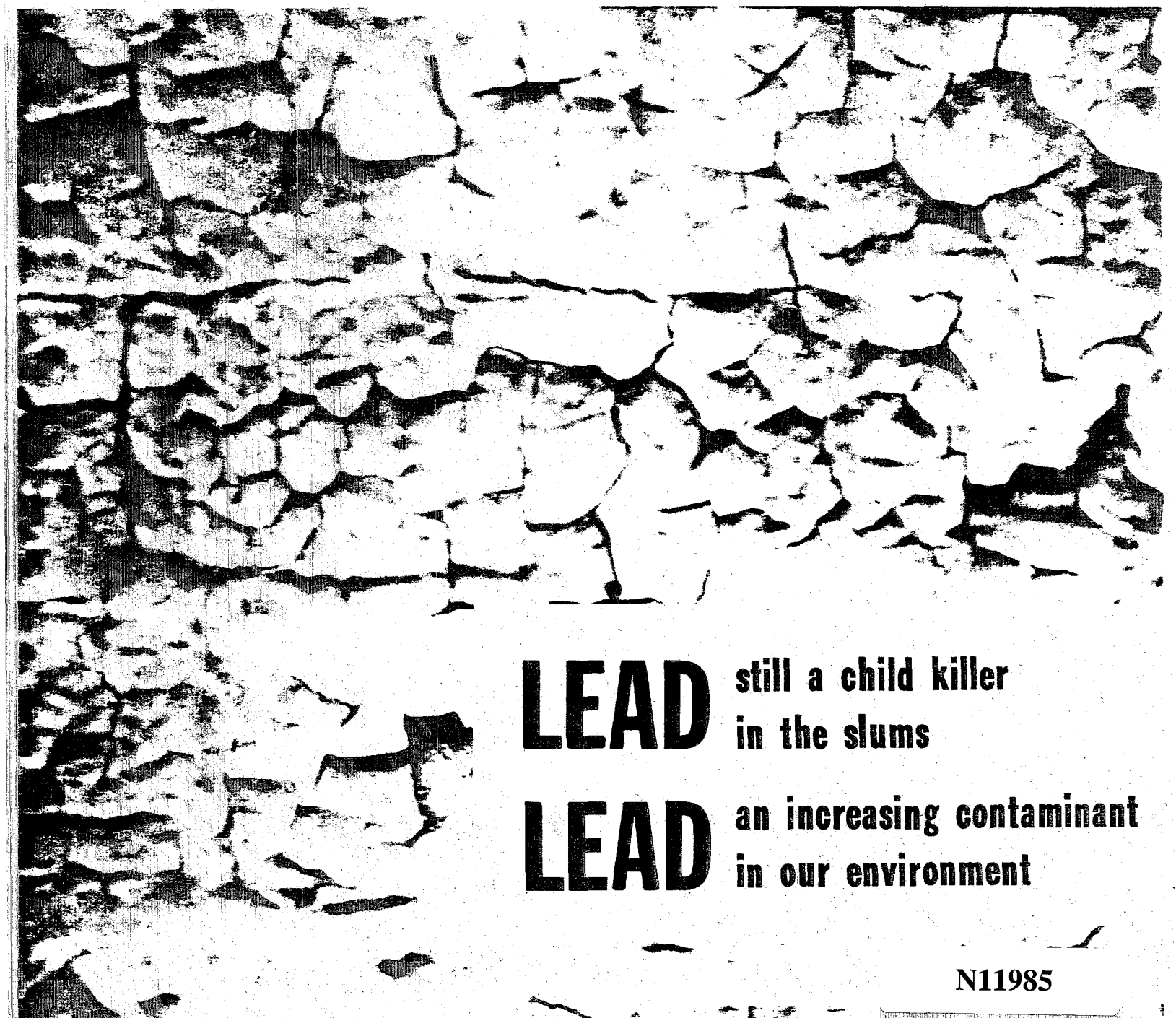

LEAD still a child killer in the slums

LEAD an increasing contaminant in our environment

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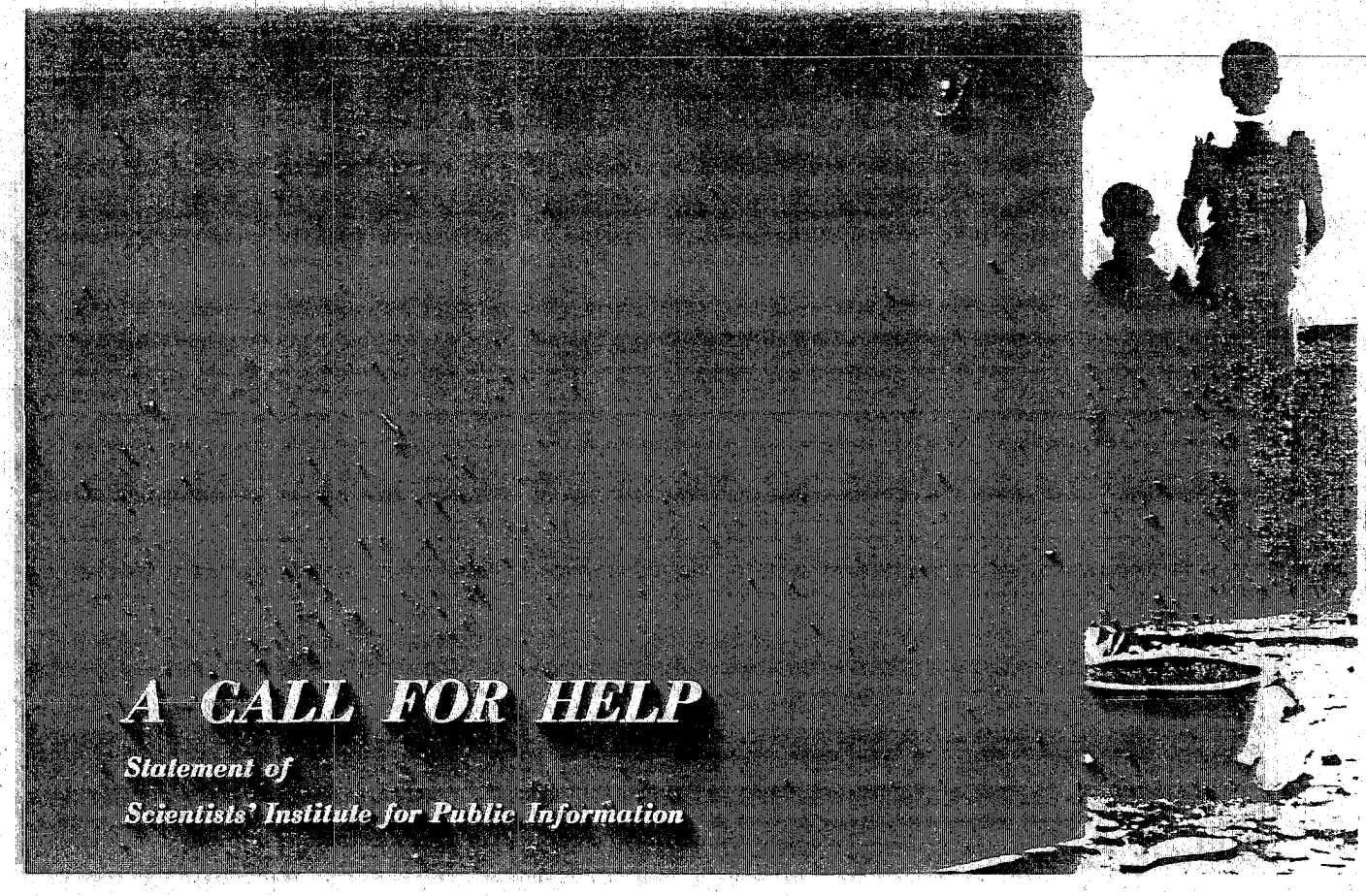

A CALL FOR HELP

Statement of
Scientists' Institute for Public Information

Lead poisoning among preschool slum children is an environmental problem resulting from the living conditions of its victims. Its epidemiology is clear, its victims can be predicted, and its consequences and treatment are known to the medical profession. They are not known, however, to the people who live in the slums, nor are the scope and intensity of the hazard clearly recognized by health workers in many cities.

Lead poisoning occurs in five to ten per cent of slum children, aged one to six, who contract the ailment by eating lead-containing paint from cracked and peeling walls. Of the victims with frank clinical symptoms, about one in four dies. Of those who survive, about one in three develops some kind of neurological disorder.

Statistics on reported cases uniformly underestimate the problem. But it is clear from studies in Chicago, Rochester, Cleveland, New York and Baltimore, that lead poisoning in children exists wherever dilapidated housing exists. It is also clear that many subclinical cases are not being detected. Physicians who are not alert to the problem are not diagnosing it in its early stages when symptoms include such vague complaints as loss of appetite, drowsiness and crankiness. For example, in New York City, where hundreds of confirmed lead poisoning cases are reported every year, one large health center did not report a single case in three years. The penalty for failure to identify lead poisoning early is heavy. The child will continue to ingest the paint and his central nervous system may be damaged.

Lead poisoning has a high recurrence rate and a high rate among siblings, and it will continue to be a problem as long as houses with flaking paint and broken plaster remain.

Since it is a problem of the environment in which the children are living, the only sure cure is a change in that environment.

Scientists' Institute for Public Information urges scientists to bring information on lead poisoning in understandable terms to their communities, particularly to people living in substandard housing, and to help alert the health community. Community programs may then include such projects as health education, case finding and treatment, and preventive action through housing improvement.

Science-information committees in Rochester and Chicago (pp. 58 and 60) have undertaken information programs in cooperation with community groups. Similar programs are also planned in two other cities. Scientists and laymen who want additional information or wish to develop programs in their communities should write S.I.P.I., 30 East 68th Street, New York, N.Y. 10021.

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A RECORD of man-made contamination has come back from hundreds of layers of snow in the Arctic (see page 66).

Introduction to a Controversy

"SOME MAINTAIN that a large segment of the population is already perilously close to the threshold of lead toxicity as a result of environmental exposure; others take an almost diametrically opposed position," says Dr. Leo J. Gehrig of the Public Health Service.[1] Is the public forever to be at the mercy of the inability of the experts to agree, or is there an alternative to hopefully awaiting the rarely-seen unanimity?

When scientists disagree, *Scientist and Citizen's* approach is to present as clearly as possible the best available evidence and explain the differences which exist among the experts. We believe that an informed public is best equipped to decide the social issues that so often arise from decisions which *must* sometimes be made, despite scientific uncertainty.

In the case of lead contamination, the question is clear: Should steps be taken to limit the amount of lead in our environment? It has been proposed that new and stricter standards be set for lead concentration in the air, in food and in water. It has been suggested that lead additives in gasoline be prohibited. (In Sweden, a scientific task force on environmental problems has already recommended this step.)

Much of the disagreement about lead is centered on the work of Dr. Clair C. Patterson. Patterson, in a series of studies and articles, has argued that the average lead concentration in the bodies of Americans is many times the natural lead concentration in human tissues. He has presented geochemical studies which estimate the natural lead concentrations in air, water and food to be far below current levels. He places the blame for the drastic increase in environmental lead on recent industrial contamination, especially from airborne lead emitted from auto exhausts.

Patterson says that industrial lead has so contaminated the oceans, surface waters, air and foods, that man today bears a high body burden of lead from this artificially accelerated intake. He warns of the likelihood of chronic, low-level damage from lead as contamination of the air from industrial sources—especially leaded gasoline—increases.

Lead poisoning has been detected by clinical signs and symptoms such as those outlined in Dr. David Elwyn's article in this issue, or by chemical tests of blood and urine. In general, blood tests are the physicians' chief tool for detecting lead poisoning. Blood lead levels from 0.05 to 0.4 parts per million have been considered within the range of normal exposure; those of 0.4 to 0.6 indicative of occupational exposure to lead; those of 0.6 to 0.8 abnormally high and indicative of danger from lead poisoning. A blood level above 0.8 parts per million is considered strong evidence of lead poisoning. According to most studies, the average blood lead level of the population is about 0.25 parts per million, usually thought of as "normal," and well within safe limits of exposure.

The crux of the problem is that the classical and generally accepted concept of acute lead poisoning lacks terms to answer Patterson's arguments. There is no recognition, for example, of "low-level" lead damage: lead poisoning to the clinical practitioner is defined by the presence of recognizable signs and symptoms; to the experimental researcher, lead poisoning is conceived of in terms of labelled ranges of blood lead concentration: normal, occupational exposure, dangerously high, intoxicated. With few exceptions, there was a general dismissal by occupational toxicologists of Patterson's most significant points: that the increasing concentrations of lead in the air are bound to show up as increasing amounts of lead in the body, and that this process has in fact already resulted in dangerously high levels of lead contamination, as represented in the "normal," average blood lead level of 0.25 parts per million.

There are, of course, areas of general agreement. Because lead is so commonplace and so useful and because it has long been recognized as a poison, its occurrence in nature and its physiological effects have been studied for centuries.

There is no significant disagreement on the amount of lead in the environment. This is largely a matter of direct measurement of lead concentrations in air, water, foods and organisms. The studies which have been made to date offer clear and relatively unambiguous data about the prevalence of lead in the environment.

There is no significant disagreement about the source of lead in the environment. The natural distribution of lead is well known, and industrial lead contamination can be determined by direct measurement.

There is no disagreement about the harmful effects of large doses of lead on human health. Classical lead poisoning has long been familiar to doctors, and it has been the subject of detailed and comprehensive monographs.[2,3] Although much uncertainty remains as to the mechanisms by which lead causes harm, there is no doubt that continuing large doses of lead—or a single massive dose—can cause serious, often fatal illness. There is general agreement that blood lead levels above 0.5 parts per million are indicative of abnormal exposure, and that blood lead levels in the neighborhood of 0.8 parts per million are consistent with the appearance of clinical symptoms of acute lead poisoning.

Finally, there is no evidence that any amount of lead has beneficial effects on health.

The areas of disagreement about lead are also easily identified, and they are many. But four key questions are at the heart of the disagreement about lead contamination:

- Has the rate of environmental lead contamination increased in recent years?
- Does a rise in environmental lead contamination necessarily result in an increase in the average body burden?
- Has the body burden of lead in human beings increased in recent years?
- Does long-term, low-level exposure to lead damage health?

The articles by Schroeder, Hardy, and Patterson in this issue seek to cast light on some of these questions. Patterson's studies of polar lead, for example, show a sharp increase in *atmospheric* lead contamination. Goldsmith and Hexter (see page 71) have found a clear relationship between increasing atmospheric contamination and increasing blood lead levels. However, the overall effects of increasing lead contamination on human health remain uncertain, and the most important question — whether low-level exposure to lead is harmful — cannot be answered clearly and unequivocally.

What must be weighed, then, are the unknown health costs of continued exposure versus the known economic cost of reducing the risk. Only the public can judge whether the threat of illness from current levels of lead exposure is worth the cost of curtailing lead contamination.

Lead is a more immediate and dangerous threat to a small segment of our population—children in the slums. Here the danger is not in doubt: the facts are clear. The social decision requires weighing the lives of children against the cost of new or rehabilitated housing in the slums. The articles by Elwyn and Simon and the report from the Rochester Committee for Scientific Information deal with this aspect of the problem.

Should standards be set for the control of lead contamination? As always, the answer depends on the public's ability to weigh the extent of the risk against the cost of protection. Only in this way can we avoid decisions based on groundless fears or blind complacency.—J. S.

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CHILDHOOD LEAD POISONING

By DAVID ELWYN

IN THE LAST DECADE, 138 children have died of lead poisoning in Chicago; from 1954 through 1964 128 New York children were victims of this disease (see Table I). These are relatively small numbers when compared with those who died in the same period from bacterial or viral diseases, but they assume added significance when it is realized that lead poisoning, unlike the others, is virtually unknown in a natural environment. Lead poisoning is as much a product of modern society as auto accidents, but it can be prevented much more easily. Yet it continues, even today, to threaten thousands of children in cities across the nation.

Lead poisoning is a hangover from the recent past, and a deadly one. Lead is one of the oldest known minerals; it has been used in industry for hundreds of years, and its effects on the human body are not news to the medical profession or to the general public. The sources of lead contamination in the environment are well known, and the means to control them are readily available. In view of this widespread awareness, and in the absence of any pressing social necessity for risking the effects of lead poisoning, how can such a hazard continue to exist? Why do children die from a disease which can be eradicated now with the means at hand? The answers are disturbingly simple.

Lead poisoning is a man-made disease. Lead in assimilable form may be almost entirely absent from a "natural" environment. The use of lead and its derivatives was introduced early by civilized man, and with lead came lead poisoning. Today there is lead in our food, in our drinking water, and in the air we breathe. The body of an average adult in the U.S. contains about 200 milligrams of lead, as compared to a "natural" body content of about two milligrams.[1] The extent of biological damage from this "normal" lead exposure will be, for some time to come, difficult to assess. But beyond this "normal" exposure is the problem of high level chronic or acute exposure to lead: lead poisoning, a disease which has been widely studied and reasonably well defined. The two groups mainly affected by lead poisoning are workers in lead-using industries and small children; it is the latter who are the concern of this article.[2-8,15]

Childhood lead poisoning is almost entirely restricted to slum neighborhoods, where lead poisoning afflicts about one of every fifteen children between the ages of one and five. Most display no symptoms, but about three per cent of those afflicted develop the physical symptoms that indicate acute lead poisoning.

Besides being a major cause of death among young children, lead poisoning causes permanent injury to many survivors. A recent study[8] of 425 Chicago children suffering from lead poisoning reports that thirty-nine per cent had neurological injury, mental retardation, and epileptic seizures. Cerebral palsy and optic atrophy were less frequent results.

The main cause of the disease is the eating of lead-bearing non-food objects by children. The main source of ingested lead is old paint peeling from walls and ceilings in slum dwellings. Until twenty years ago, most interior paints contained lead pigments, but these have now been replaced by titanium pigments. Walls and ceilings in good repair, even though containing lead, are not an important source of ingested lead.

For hundreds of years, men put lead into paint and paint into buildings, with no inkling of the hazard they created for themselves and for future generations. We know now of the effects of lead poisoning—retardation, cerebral palsy, epilepsy, death—and we know now the cause: lead in paint flaking and peeling from deteriorated surfaces.

The victims of lead poisoning are the children of slums where paint, plaster and putty falling from walls, ceilings and window sills are available to those children who like to eat them. (Pica, as the eating of non-food objects is called, occurs in about twenty per cent of all children between the ages of one and five; it is prevalent in all economic classes. Pica is possibly related to emotional disorders, but is not related to physical hunger.)

Once inside the body, the lead borne by paint and plaster begins a deadly cycle that culminates, in severe cases, in organic and nervous tissue damage such as that demonstrated in the Chicago study cited above.

Effects of Lead on the Human Body

When lead is absorbed by the body, it is distributed in soluble form throughout the soft tissues. It is subsequently precipitated in an insoluble form in the

bones. It may be several months after he stops taking in lead before the precipitation is completed in a child's body.

The toxic effects of lead occur while it is in the soluble form, and the extent of damage is a function of the concentration of lead in the soft tissues and the length of time high concentrations remain. The relative concentration of lead in the soft tissues can be determined approximately by measuring the concentration of lead in the blood.

Concentrations of six parts per ten million or higher in the blood are considered abnormal and suggestive of lead poisoning.[7,9] In its insoluble form in the bones, lead is presumed to be inert. However, under certain conditions, such as increased acidity, or during treatment for lead poisoning, the body can reverse the precipitation process, and release deposited bone lead into the blood stream. Bone lead is therefore a potential source of increased soluble lead. In children, the mobilization of bone lead is unpredictable and may cause severe lead poisoning even when lead is no longer being ingested.

While it is probable that lead has a damaging effect on all tissues, the effects on blood, kidneys, gastrointestinal tract and nervous system are most apparent.

The effects on blood are mainly related to interference with production of the hemoglobin of red blood cells, which is responsible for transport of oxygen to the tissue cells. Disorders include anemia, a stippled appearance of the red blood cells, and the excess production and excretion in the urine of coproporphyrin, a side product of hemoglobin production. One of the most sensitive and widely used tests for lead poisoning is the determination of the amount of coproporphyrin in the urine.[10]

Kidney damage may take the form of increased excretion of sugar or amino acids, or decreased urine output. An Australian report[11] indicates that childhood lead poisoning may result in severe chronic nephritis, a disease which can cause hypertension, kidney failure and death.

Vomiting, constipation, and stomach cramps are usual early symptoms of lead poisoning.

Nervous system disorders include behavioral problems, convulsions, and coma due to massive swelling of the brain. These may develop slowly or rapidly, and if not caught in time may lead to permanent mental retardation, seizures, cerebral palsy, and death.

Diagnosis and Treatment

Any or all of these symptoms may be absent in lead poisoning. Furthermore, all can be simulated by other diseases or conditions. Diagnosis of lead poisoning, therefore, is made only when the physician is looking

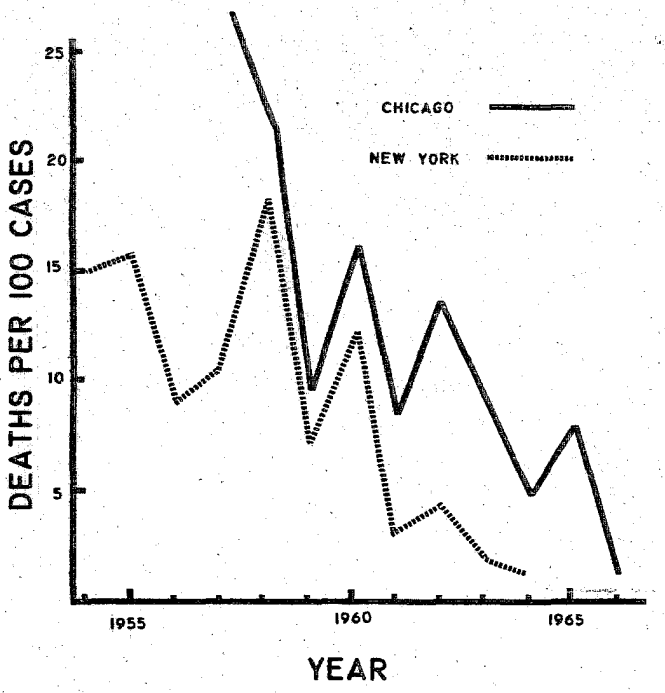

FIGURE 1. Lead poisoning case fatality rates in New York and Chicago, 1954-1966.

for it. As a result, the reported incidence of lead poisoning parallels public interest and education on the subject, and official figures on lead poisoning almost certainly underestimate the true incidence of the disease.

Lead poisoning may be divided into two overlapping categories, symptomatic and asymptomatic.

Symptomatic lead poisoning is normally diagnosed when the child is brought to the physician evidently ill with gastrointestinal or nervous system disorders. Lead poisoning may be suspected and can be confirmed by laboratory tests. These include microscopic examination of red blood cells, determination of coproporphyrin in the urine, blood and urinary lead determinations, X-ray of the abdomen for radio-opaque objects (paint and plaster), and X-ray of the long bones for evidence of lead deposition in the bones.

Asymptomatic lead poisoning is diagnosed from laboratory findings in the absence of other observed symptoms of lead poisoning. Usually two or more positive tests are considered necessary to confirm a diagnosis.

The most important treatment of lead poisoning is to administer a drug, edathamil, which forms a complex with lead and greatly increases its rate of excretion. One of the most senstitive measurements of the extent of lead poisoning is the urinary lead output on the first day after treatment with edathamil.

Extreme care is necessary with this treatment, however, for oral administration of edathamil also increases the rate of absorption of lead from the gastrointestinal tract; if lead-containing objects are present, it may kill the patient. All lead-containing objects must therefore be removed from the gastrointestinal tract before treatment, and the child must be completely removed from the lead-contaminated environment thereafter. Edathamil may also mobilize lead from the bones, thus increasing the concentration in soft tissues; this is a particular hazard in acute, severe cases.

Combatting Lead Poisoning in the Community

The reported incidence of symptomatic lead poisoning in New York and Chicago is shown in Table I. The figures show a steady rise in reported lead poisonings and a decrease in the case fatality rate. There is no reason to believe that the actual incidence of lead poisoning has increased since the 1950's and therefore the rise in reported cases reflects a growing awareness on the part of the medical community. The decrease in case fatality rates indicates that more cases are being discovered in the early stages. There is as yet no way of estimating how many cases are not properly diagnosed. It is possible that, despite growing awareness, the true incidence of lead poisoning is two or three times that reported.

At least two studies have been made of the occurrence of asymptomatic lead poisoning, one in Chicago[12,13] and one in Cleveland.[14] The conduct and results of both studies show many similarities. Total sample groups ranged in number from 900 to 1500, and included persons from poor areas with dilapidated housing as well as from control areas where housing was new or in good condition. Urine samples were collected from children aged one through five and tested for abnormal coproporphyrin or lead levels, or for both. Children with abnormal urines were given further laboratory tests to definitely establish the presence or absence of lead poisoning.

Incidence of asymptomatic lead poisoning (about seven per cent of those tested) is about twenty-five times that of symptomatic lead poisoning (about 0.3 per cent of those tested). On the basis of these studies, then, approximately 2,500 children in Chicago and 50,000 children throughout the country are victims of asymptomatic lead poisoning. It is clear that these projections, based as they are on a very small sample, are quite inaccurate. They do, however, give a valid indication of the magnitude of the problem.

Current practice in treatment of lead poisoning usually requires hospitalization of the child for five days or more. The problems this raises in terms of the cost of adequate treatment are obvious. Hospitalization of an additional 2500 children for five days each summer in Chicago would require 200 additional hospital beds, and an estimated additional $500,000 to $1,000,000 per year for patient care alone.

Outpatient therapy for asymptomatic lead poisoning is currently being tried by a number of institutions. Intra-muscular injection of edathamil has proved safer than oral administration. Other drugs, such as penicillamine are being tried out. Although still at an experimental stage, carefully controlled outpatient therapy, combined with elimination of lead from the environment, probably holds the most promise for wide scale treatment.

Uncertainty as to the dangers associated with asymptomatic lead poisoning add to the difficulties of treatment. There have been no adequate studies made of the extent of associated organic damage. Such studies would take several years to perform, and action based on their results would be too late for many. One can presume that in many cases there has already been severe damage, since the subjects may have had symptoms in the past, including mental retardation, without a diagnosis of symptomatic lead poisoning. Almost certainly, future cases of diagnosed symptomatic lead poisoning will come largely from this group, since both the districts of high incidence and the underlying causes (pica associated with peeling paint) are the same for both categories of the disease. Until it is proven to the contrary, the only safe assumption is that all cases of asymptomatic lead poisoning are associated with continuing organic damage.

Educational and Case-Finding Programs

Educational programs have been aimed both at the medical community and at the general population. With the former they have had a good deal of success. In many areas, pediatricians have a considerable awareness of lead poisoning, with the result that symptomatic lead poisoning is now diagnosed earlier and more frequently, and treated sooner, than was the case ten years ago. Educational programs directed to the public also have undoubtedly contributed to a greater awareness of lead poisoning, and, since they have emphasized the role of pica and peeling paint, serve to some extent as a preventive measure with regard to both symptomatic and asymptomatic lead poisoning.

A second type of approach consists of case-finding, or locating asymptomatic subjects before they present themselves to the physician. The two epidemiological studies cited above are such studies, but on a very limited scale.

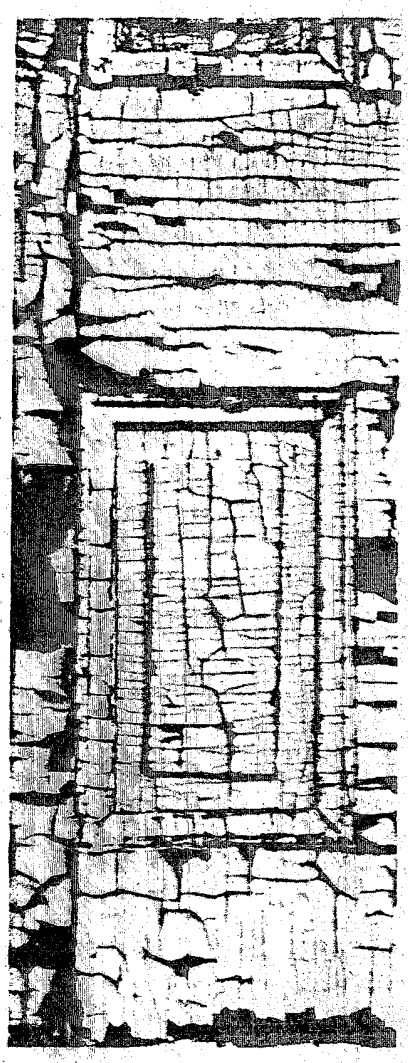

TABLE I

INCIDENCE OF LEAD POISONING IN CHILDREN IN NEW YORK AND CHICAGO

	CHICAGO[1]						NEW YORK[2]		
	All Accidental Poisonings			Lead Poisonings			Lead Poisonings		
Year	Total Cases	Deaths	Case Fatality Rate[3]	Total Cases	Deaths	Case Fatality Rate[3]	Total Cases	Deaths	Case Fatality Rate[3]
1954							80	12	15.0
1955							115	18	15.7
1956							99	9	9.1
1957	975	10	1.0	24	7	29.2	85	9	10.6
1958	2397	8	0.3	13	3	23.0	116	21	18.1
1959	3156	20	0.6	193	19	9.8	171	12	7.0
1960	3254	32	1.0	172	28	16.3	146	18	12.3
1961	3443	25	.7	178	15	8.4	181	6	3.3
1962	3827	21	.5	154	21	13.6	198	9	4.5
1963	4552	30	.7	203	19	9.4	338	7	2.1
1964	4635	13	.3	156	8	5.1	509	7	1.4
1965	5359	29	.5	218	18	8.2			
1966[4]				304[4]	5[4]	1.6[4]			
Total Through 1965	31,598	188	0.6	1,311	138	10.5	2,038	128	6.3

[1]Figures for Chicago were obtained through the kind courtesy of Dr. William Fishbein and Dr. Herbert Slutsky of the Chicago Board of Health.
[2]Figures from New York taken from Jacobziner.[6]
[3]Deaths per 100 cases.
[4]Estimated figures through November 1966.

The New York City Department of Public Health has carried on an active case-finding program since 1955.[6] Its child health stations follow over 200,000 children from birth to school entrance. Their most useful tools for locating asymptomatic lead poisoning have been blood lead determinations and histories of pica. In addition to case-finding, according to Jacobziner,[6] they have followed up all lead poisoning cases with examination of the buildings where victims lived and have enforced repainting and replastering of apartments.

A direct result of this program, as shown in Table I and Figure 1, is a marked increase in the number of reported cases and a marked decrease, particularly since 1960, in the case fatality rate.

By contrast, Chicago had not until 1966 conducted a case-finding program. Reported cases of lead poisoning had risen more slowly and the case fatality rate had decreased more slowly than in New York. In 1966, however, the Chicago Board of Health launched a major case-finding program. Over 40,000 children were tested for urinary coproporphyrin or blood lead, according to Drs. William Fishbein and Herbert Slutsky of the Board of Health. The figures for lead poisoning in 1966, though still preliminary, indicate a marked rise in cases reported and a sharp decrease in case fatality rate, consistent with the New York experience. There has also been a decline in the severity of symptoms associated with lead poisoning, according to Dr. Joseph Greengard, Chief of Pediatrics at Cook County Hospital.

The results of these programs show that active case-finding can significantly reduce death, mental retardation, and neurological disorders arising from lead poisoning. A well-informed public can take an active role in programs to reduce or eliminate lead poisoning. The effectiveness of such action by private citizens was illustrated by recent events in Chicago.

In the summer of 1965 as a result of several cases of lead poisoning, the Citizens' Committee to End

Lead Poisoning was organized in the East Garfield Park district of Chicago. With the assistance of Project House of the American Friends Service Committee, they organized an educational and case-finding campaign described in the account by Ann Simon in this issue. The Board of Health and later the Medical Committee for Human Rights assisted with urinary coproporphyrin and lead testing. High school students were trained to collect urine samples and to perform lead tests on these samples. Several hundred urine samples were collected and more than twenty cases of lead poisoning were identified. Considerable publicity attended these efforts, and other community groups in the city instituted similar programs. Most important, these events led to the decision of the Chicago Board of Health to undertake the massive case-finding program described above, which has been accompanied by a reduction of seventy per cent in deaths from lead poisoning.

Whether lead poisoning will flourish much longer is a question that has yet to be answered, and the efforts of the medical profession alone have not been enough; they can deal only with the effects. Educational and case-finding programs are also inadequate to cope with the basic cause of childhood lead poisoning, which will continue as long as we permit peeling paint and plaster in slum dwellings. For the most part, city building codes are adequate, if enforced, to eliminate peeling paint and plaster. However, such enforcement transcends the authority of Boards of Health and requires the active cooperation of other departments of city government.

The cost of lead poisoning is borne by the whole community in terms of wasted human resources, institutionalization of victims, and the resulting burdens on municipal health facilities and finances. The benefits accrue only to those owners of slum property who find it unprofitable to keep their properties in good repair.

David Elwyn, Ph.D., is Associate Director of Surgical Research at the Hektoen Institute for Medical Research, Cook County Hospital, Chicago. He is a biochemist whose research interests are the movement of amino acids between organs in the intact mammal and the metabolism of shock and trauma. Dr. Elwyn was a founder of the Chicago Science Information Speaker's Bureau in 1962.

Despite increasing attention, childhood lead poisoning remains a major public health problem. Although there are large gaps in our knowledge of the disease, enough is known to eliminate it. All that remains to be accomplished is the effective social utilization of this knowledge.

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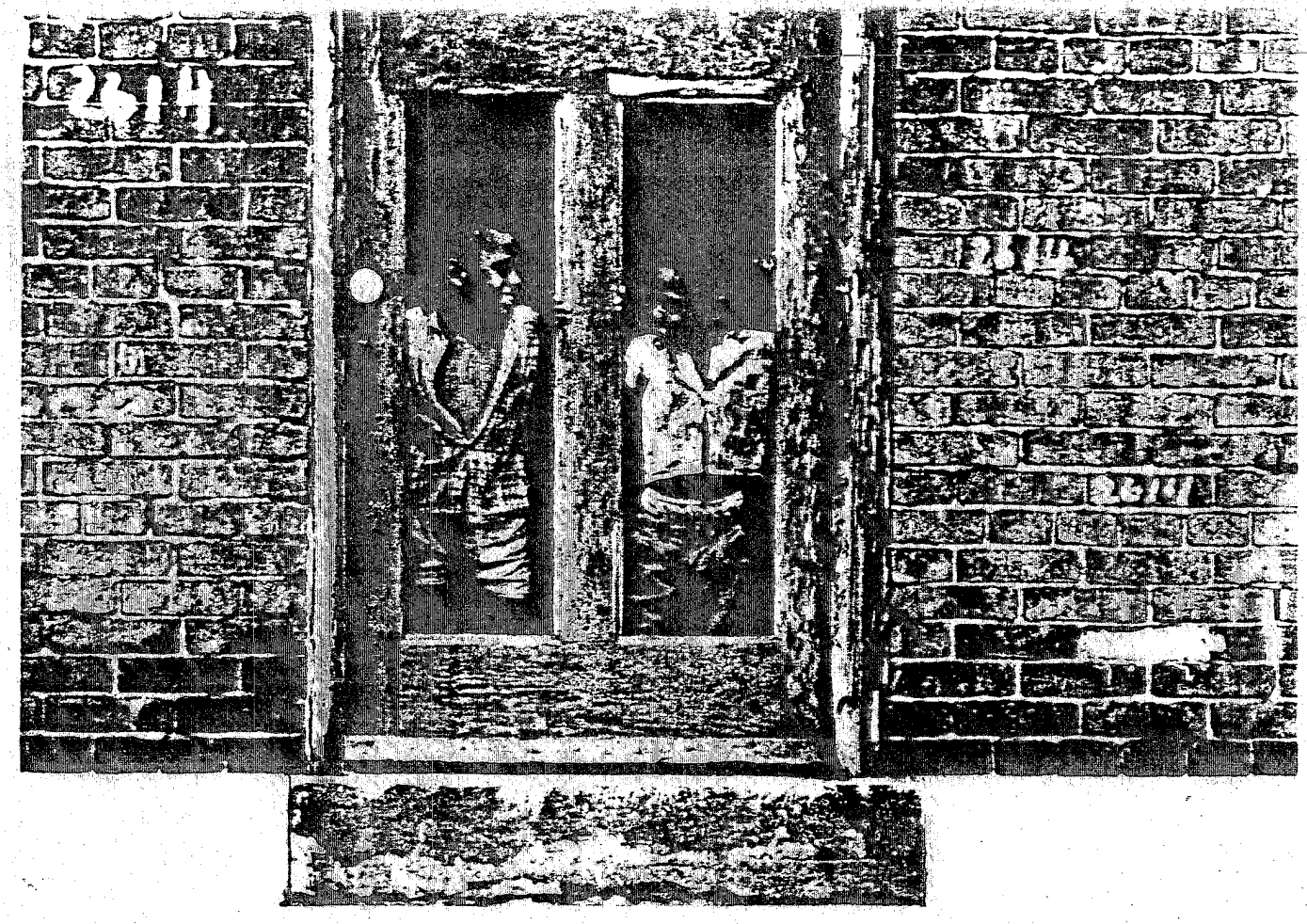


CITIZENS vs. LEAD IN THREE COMMUNITIES

1. Chicago

By ANN KOPPELMAN SIMON

FROM THE LATE SUMMER of 1965 to the spring of 1966, a small community organization on Chicago's West Side, under the auspices of the American Friends Service Committee, carried out a campaign against lead poisoning which was to influence dramatically the preventive lead poisoning program of the Chicago Board of Health. The Citizens Committee to End Lead Poisoning, basically concerned with severely inadequate housing conditions in the Negro slum community of East Garfield Park, focused on lead poisoning as one tragic result of the housing problem.

Sparked by the experience of one family, CCELP was born overnight through the joining of forces of a number of local organizations and teenagers. One August evening a young mother of two arrived at her block club meeting distraught with the news that both her children in recent weeks had become feverish and convulsive with what doctors reported as lead poisoning. What was this disease, she wanted to know, how did they get it, and what could be done about it? The block club president relayed the question to the local Project House of the AFSC's Urban Affairs Program, and an emergency information meeting was called for the following week.

Representatives from a number of block clubs, parents groups, social agencies, churches and other organizations heard a report from the mother and an explanation of lead poisoning and its causes—mainly peeling paint—from a pediatrician with the Medical Committee for Human Rights. It became clear to the group present that this disease was indeed caused by the living conditions of their slum community when the doctor mentioned that in his thirty years of subur-

How common a problem is lead poisoning? Is it much of an issue in the United States? The answer seems to be, it depends on how hard you look. I must say very frankly that if you live in an American city with a slum population—and there are kids living there—and you don't have many cases of lead poisoning—then your health department isn't doing its job.

I make this statement because the communities that have looked at this problem — Chicago, Cleveland, Baltimore, and my own community of Rochester—have all found the same thing. If you look carefully into the slums of your own community—look where the paint is peeling—and if you test the kids between one and five for lead, you will find that something like five per cent of them are poisoned by lead.

Dr. Evan Charney, remarks at the SIPI Workshop, St. Louis, 1968

ban practice he had treated only one case of lead poisoning, while during the summer it is a daily occurrence at the clinic of Cook County Hospital. So the group decided to become the permanent Citizens Committee to End Lead Poisoning with the goal of launching a large scale effort to alert the community to the dangers of lead poisoning and to try to prevent it.

Through representatives of the Mayor's Committee on Youth Welfare, CCELP learned that the Board of Health had termed the twelve lead poisoning deaths reported so far for 1965 "of epidemic proportions," and was also discussing plans for a preventive campaign. With its offer of volunteer manpower and extensive community contacts, CCELP was able to provide the Board of Health with a missing link in its existing program: systematic community canvassing for children's urine samples. An experimental project was established with CCELP collecting urine samples and the Board of Health testing them for urinary coproporphyrin (see "Childhood Lead Poisoning," p. 54). The Board of Health also conducted further testing and treatment when necessary, and reported to the city Building Commission the addresses of apartments in which lead poisoning cases were found.

A dedicated group of teenagers spent their weekends canvassing from September through November, and during that period close to 600 samples were tested and four children with definite signs of lead poisoning were identified and treated.

This arrangement enabled the Board of Health to experiment with the use of non-trained personnel doing field work. The availability of door-to-door canvassers also made it possible to conduct a systematic screening program for lead poisoning victims who had not yet displayed acute symptoms. In December the Board of Health announced plans to launch a similar city-wide testing program with canvassing to be carried out by local War on Poverty neighborhood workers. CCELP decided to continue its campaign in East Garfield Park (with the same group of teenagers performing a dithizone test for urinary lead) in order to continue to serve as a reminder to the city of the seriousness of community concern about lead poisoning and the need for a high quality preventive testing program.

Laboratory facilities were provided by a local small private hospital where testing was carried out several evenings a week from February through May. Seventy-three samples were tested and nine children were identified and taken by CCELP volunteers to hospitals for further testing.

CCELP ended its canvassing and testing program in April when the city program was operating full scale in East Garfield Park and doing a more extensive job than CCELP could attempt. Through September 1966, the Chicago Board of Health tested 30,000 urine specimens. In October they switched to a blood lead test, using an atomic absorption spectrometer. An additional 8,000 blood lead tests were performed. More than 700 children were treated for asymptomatic lead poisoning as a direct result of this screening program. While the greatest number were treated at Cook County Hospital, many private hospitals participated.

The city is continuing to screen by blood lead tests. In addition, a special center for diagnosis and treatment of childhood lead poisoning has been established by the Board of Health.

Despite this great advance in finding and treating lead poisoning, the work that CCELP set out to accomplish is not complete. A great deal of effort is needed to alert parents to the dangers of lead poisoning and to proper preventive measures.

Finally, it is clear that there will be no solution to childhood lead poisoning as long as there are thousands of slum apartments where peeling paint and plaster are constantly available to young children.

Ann Simon has worked in civil rights and community organizations for several years, and is now a graduate student in the Master of Arts in Teaching program at the University of Chicago. At the time the Citizen's Committee to End Lead Poisoning was formed, she was assistant director of the Urban Affairs Program of the American Friends Service Committee's Chicago Regional Office.

SAMPLES of peeling paint for lead analysis are collected by Christine Johnson, a Rochester teenager in Project Uplift, working through the Urban League. (Robert Myricks, Project Uplift)

2. Rochester

By DAVID J. WILSON

ABOUT A YEAR AGO three members of the Rochester Committee for Scientific Information — George Berg, Tom Fink, and I—attended a workshop of the Scientists' Institute for Public Information in New York City. We had gone to discuss our Rochester water pollution studies (see *Scientist and Citizen*, March, 1967). As is so often the case, we returned to Rochester with the feeling that we had gotten more from the meeting than we had brought to it. Since it had become apparent that in the Rochester Committee for Scientific Information we had an extremely effective instrument for attacking certain types of community problems, we had sought an entrance into the arena of the really big community problems. This arena is our urban slums. We were not interested in trying to compete with the Community Chest, the County Welfare Agency, or various other groups — we were looking for something on which we could bring to bear the experience and knowledge our Scientific Information group had developed during the water pollution operation.

At last year's symposium Dr. David Elwyn presented a summary of a Chicago group's work on lead poisoning in young slum children, and we realized instantly that this was our entrance. Rochester's slums, while not so extensive as those of Chicago, are older and at least as badly run-down. It seemed unlikely to us that a lead poisoning problem did not exist in Rochester.

George Berg, President of the RCSI, then did what is probably the single most important thing in carrying out a scientific information project. He appointed a chairman of our new lead poisoning subcommittee. His choice was Dr. J. D. Hare, Associate Professor of Microbiology at the University of Rochester Medical School. Hare was professionally qualified to quarterback the project, and he showed the patience, tenacity, and ability to deal with people which the job required.

We soon found that we had been beaten to the punch on lead poisoning. When Hare began to do his homework for the project, he learned that in the fall of 1964 Drs. Evan Charney and Arthur Kopelman had carried out a study of lead poisoning in Rochester. The RCSI's first report on lead poisoning was therefore a presentation to the general public of Charney's and Kopelman's findings. Their report, which they had submitted to the Monroe County Health Department, had not, to our knowledge, ever been made public, nor had the health department done anything to try to solve this serious health problem. The essence of the Charney-Kopelman report is as follows:

In September, 1964, five cases of lead poisoning were found in a group of twelve children living in an apartment house in Rochester. This stimulated Kopelman and Charney to attempt an estimate of the true prevalence of lead poisoning among children under five years of age in a slum area in Rochester's Third Ward, a part of the city's "Black Belt."

A screening test for lead poisoning, the urinary coproporphyrin determination, was carried out on urine samples from sixty out of sixty-five pre-school

children in an area of about two city blocks. Eleven out of sixty showed a positive test and eight of the eleven were still positive on a repeat exam. This test is sometimes positive in certain other illnesses; therefore, to confirm the presence of lead poisoning, they took blood samples from the eight children with positive repeat tests and analyzed the blood for lead. Three of the eight children were found to have elevated blood lead levels. A fourth child, who was not included in the original screening but was a sister of one of the children found to have lead poisoning, was tested for blood lead level and also found to be positive.

On this basis then, it was shown that four out of sixty-one children (6.6%) from a small slum area in Rochester were poisoned by lead. This figure coincides reasonably well with the incidence of lead poisoning among pre-school slum children in Chicago (8.8%), Baltimore (7.1%) and Cleveland (6.4%). The seriousness of the situation is accentuated since lead poisoning produces a variety of grim effects, including permanent brain damage and death.

The report noted that lead poisoning is a significant health menace among young children who swallow peeling, crumbling lead-containing paint and putty from the walls and woodwork of rundown, dilapidated dwellings in Rochester. Properly painted and repaired dwellings present less of a hazard, for lead-containing debris is removed or covered thoroughly. (Modern paints sold for indoor use do not contain lead.) It is clear, then, that this serious problem is directly related to the existence of inadequate and substandard housing.

The report made five recommendations:

(1) Screening of children for lead poisoning by the County Health Department.

(2) Establishment of a blood lead testing service by the Health Department.

(3) Treatment of recognized cases and adequate testing of children exposed in the same manner or who lived in the same house.

(4) Education of parents, doctors, and visiting nurses to the hazard of preschool children eating paint in slum buildings.

(5) Slum clearance.

Despite the fact that the report had been submitted in the winter of 1964-65, we found that little had been done to implement these recommendations. In view of our previous experiences with the County Health Department on water pollution, this came as no surprise to us.

Our resurrection of the Charney-Kopelman report last fall produced a spate of publicity in the local press, with whom we enjoy a very cordial relationship, but did not seem to produce any activity on the part of the County Health Department or the City Building Bureau. Dr. Hare therefore prepared a report on recent cases of lead poisoning, and I began a study of the incidence of lead paint in slum dwellings. Hare contacted the Pediatric Department at the teaching hospital associated with the medical school. Through the help of house officers and the out-patient clinic staff, he was able to locate nine children ranging in age between two and six years who were being treated last fall at that hospital for clinically significant lead poisoning. He noted that there was no way of knowing how many more cases remained to be discovered.

The report continues, "It is significant that three children of one family and two children of another were poisoned. There were no deaths in the group and in general the indications are that all children will respond favorably to treatment. One child, for instance, came to the hospital in convulsions and delirium, and is now alert. However, at present, it is too early to predict whether there will be residual impairment of health. Residual effects do occur in many cases of lead poisoning . . ." The children lived in the Third and Seventh Wards, which are Rochester's slum wards. The report concluded that a serious menace to the health of pre-school children continues to exist in Rochester, and that this will only be eliminated by aggressive control over the conditions of the paint on the inner walls of dilapidated slum housing.

We were displeased but not particularly surprised that the fairly wide publicity given this second report in the local press failed to stir our city and county governments from their torpor. The County Health Department kept saying that the whole problem of poisoning in children was gargantuan in size, that they certainly couldn't go at it in a limited way, and that a program they were studying would cost hundreds of thousands of dollars to implement.

David J. Wilson is professor of chemistry at the University of Rochester and vice president of the Rochester Committee for Scientific Information. This article was presented at the Scientists' Committee for Public Information Workshop on the Urban Environment, St. Louis, March 29-30, 1968.

Some weeks before the publication of our second report I had begun a study of the occurrence of lead paint in slum dwellings. At the advice of a Negro friend and colleague, Dr. Walter Cooper, I contacted Mr. David Anderson, deputy director of the Urban League of Rochester. We needed people to collect paint samples for us, a group that could reach people in the slums and warn them of this hazard to their children. The Urban League of Rochester, with a well-earned reputation for hard-nosed, rational militancy and for carrying out boot-strap self-help projects in the slums, seemed a good representative of the people most in need of the information we had to offer.

I gave Anderson the information we had on the lead poisoning problem, explained what we wanted to do next and why, and asked for help. We got it. The Urban League people decided to interest a group of "Project Uplift" teen-agers in the problem of collecting paint samples. We had a briefing and organization meeting with these young people at the Urban League, where I gave them a rundown on the problem, demonstrated the methods of testing for lead in paint, and turned over to them envelopes and information sheets for their use in collecting samples. Anderson and his staff workers took complete charge of this phase of the project. My next visit to the Urban League office was to pick up 112 catalogued paint samples which the Project Uplift people had collected.

Let me quote now from the report we published jointly with the Urban League this January.

> Previous RCSI reports of August and December, 1967, have documented the occurrence of chronic lead poisoning in young children in the inner city. We considered it likely that these children were getting poisoned by eating peeling paint which contained lead pigments, as was found to be the case in Chicago. Other possible causes of lead poisoning (battery burning at automobile junkyards, contamination of foodstuffs with lead-containing insecticides, etc.) we thought would produce different patterns of poisoning (poisoning of adults as well as children, poisoning in a very localized area, etc.). Actually only young children seem to be poisoned; and, aside from the fact that they come nearly without exception from older homes in the inner city, there does not appear to be any geographical factor.
>
> If our suspicion that the children are poisoned by lead-based paint is correct, we should find such paint on the inside walls of homes in the city. The paint would probably be old, since modern indoor paints are not prepared with lead pigments. Further, the poisoning would be most likely to come from deteriorated walls, with paint peeling, flaking, and dropping within the reach of children.

A summary of the rest of the report is as follows: Of 112 samples of cracked and peeling paint collected

CRUMBLING PLASTER samples are analyzed for lead by the Rochester Committee for Scientific Information. Clara Jones of the Urban League's Project Uplift, collects a sample for analysis. (Robert Myricks, Project Uplift)

in the north end of our Third Ward, twenty-seven were found to give positive tests for lead, demonstrating that ample opportunity exists for inner-city children to get lead poisoning by eating paint indoors. Of the fifty-nine households from which samples were taken, twenty-two yielded samples containing lead.

The teen-age collectors removed samples of peeling paint from the walls of rooms and sealed them in envelopes. Cataloging data were immediately recorded on each envelope at the time the sample was taken—collector, name, address and phone number of occupant, room and color of paint, and whether or not young children live in the household.

The samples were tested qualitatively for lead by two means; by treatment with sodium sulfide solution, which turns lead-containing samples black by forming lead sulfide, and by ashing a sample and doing a benzidine spot test on the ash for lead. Only samples giving positive tests by both procedures were counted as positives — one sample giving a weak positive with benzidine and a negative with sulfide was counted as negative. Ten of the twenty-seven positive samples were then analyzed spectrographically by Dr. Luville Steadman of the Medical School and

Mr. John Temmerman of the County Public Safety Laboratory. All ten samples were found to contain lead as a major constituent (of the order of ten per cent by weight), giving ironclad verification of our qualitative tests. Anderson and the Urban League were appalled at the results, as they were fully aware of their significance in terms of illness, brain damage, suffering, and death of children in the inner-city. They got mad. What followed was one of the more constructive applications of "Black Power" to our local governments. One of the results was that about three weeks ago Dr. Hare, our lead subcommittee chairman, was asked to supply the city building inspectors with instruction sheets for testing paint samples for the presence of lead. I wrote up a detailed description of our sodium sulfide procedure, and such testing will now be routinely carried out during the inspection of slum housing by the City Building Bureau. The Urban League has also launched a large-scale public education campaign to warn parents living in the slums of the hazard to their children of lead poisoning. The visiting Nurses' Association has alerted its people to the problem. We are also collecting more paint samples. And Dr. Hare, a couple of medical students, and the Urban League are now doing urine tests on the kids living in the houses where we found lead paint, so that any of them that have been poisoned can be given treatment.

The Price of Missing Early Indications of Lead Poisoning

"By far the most important damage from lead poisoning is to the child's central nervous system. The exact mechanism isn't entirely clear yet, but certainly the increased lead burden is reflected in increasing cerebro-spinal fluid pressure. The pressure around the brain increases, and as this continues, the child becomes irritable and lethargic—later come coma, convulsions, and death.

The symptoms of lead poisoning are very insidious. All too often we are not sensitive enough to detect them. Initially, when the child is presented at the clinic, he may be irritable or sleepy and a little bit cranky. He may have a little bit of diarrhea or he may be a little constipated. He may be a little pale due to anemia, and possibly due to some lead deposited in the skin.

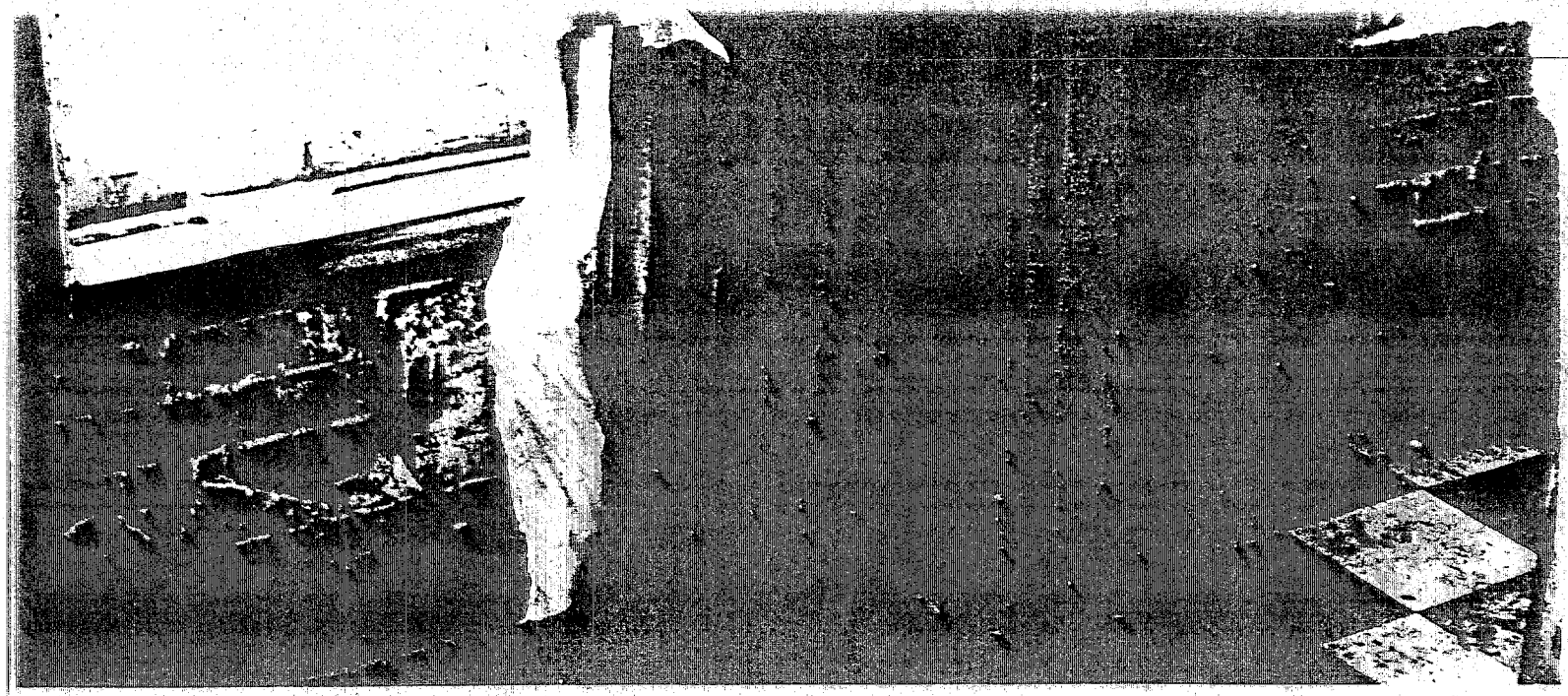
The clustering of symptoms I just mentioned describes half of Rochester's kids in the summertime—so you can imagine why it is hard to identify lead poisoning unless you are really looking for it.

Unfortunately, the penalty for not identifying lead poisoning at that level is a harsh one. If the doctor doesn't diagnose the case early, and the child's lead-eating continues, the central nervous system symptoms will become prominent and ataxia will develop, and stupor, and then coma and convulsions.

Unfortunately, it's only then that the kids are brought to medical attention. When they have a convulsion, they are brought to the emergency room of one hospital or another, but at that point it's often too late. The case fatality rate, once the child has a seizure, is about twenty-five per cent.

So one out of four will die. Of those who recover, about half will suffer long-term residual damage. They will have continuing seizures, they will show evidence of mental retardation, of brain disorder, of behavior disorder. Half of them will have these problems for life. So the price of missing the early case is a tremendous one—for the child. . . .

Dr. Evan Charney, remarks at the SIPI Workshop, St. Louis, 1968



I. Perry

3. New York

The New York Scientists' Committee for Public Information has announced that it will assist a community action group in a case-finding study of lead poisoning in New York slums. *Scientist and Citizen* received this letter from SCPI:

The New York Scientists' Committee for Public Information has recently started to concern itself with the problem of lead poisoning in slum children. We have profited greatly in our early stages from the experiences of the Chicago and Rochester groups, which we learned about at the SIPI workshop in St. Louis earlier this spring, and also from later personal communications.

Now that we have the scientific information, we plan to use it in two ways. First, we will attempt to carry the information to the affected populations in ghetto areas in New York City. We started this process at a meeting held April 22. Over thirty people were present, including representatives of ten community action groups from Central and East Harlem, the South Bronx, the Lower East Side, and the East New York section of Brooklyn. At this meeting we described the problem of lead poisoning, and we in turn learned several interesting things about other ghetto phenomena. We foresee having more such meetings, as well as sending speakers to community groups in the areas.

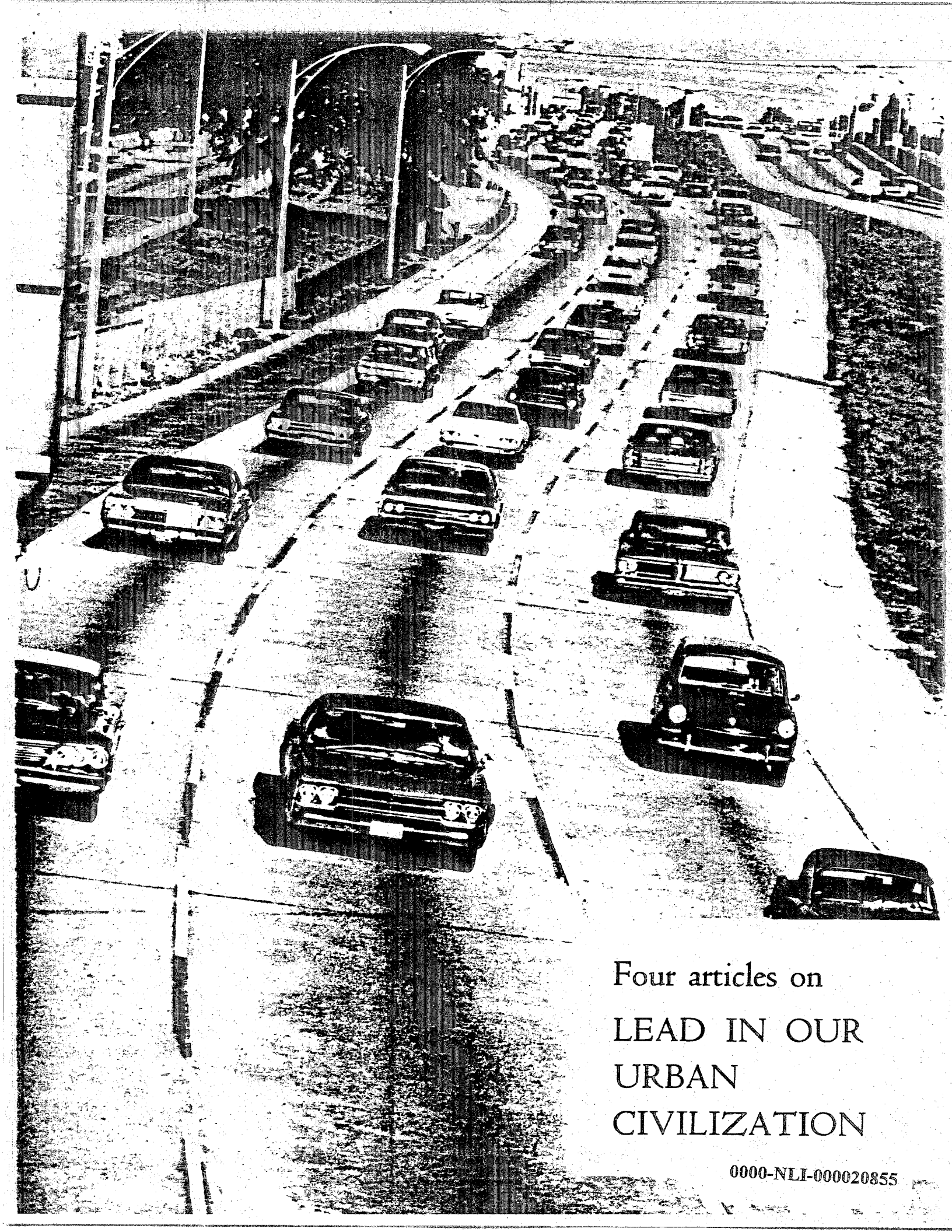
Second, we hope to find a community group that will join with us to carry out a study on the actual incidence of lead poisoning in a slum area of New York. We plan to screen a large number of children during the summer months, identify the children needing treatment, and publicize the results of our study in all possible ways. So far we have not found a definite group to work with, but we have received some encouragement in our search.

"This program is a new departure for New York SCPI, involving as it does the necessity of forging a close link between the scientist and the citizen to approach a problem unique to the urban ghetto. Those of us involved are finding it an exceedingly challenging and stimulating task: we may, in fact, end by learning more than we teach.

Glenn L. Paulson
Edmund O. Rothschild
Joel Buxbaum
Scientists' Committee for Public Information
30 E. 68th St.
New York, N.Y. 10021

CORRECTION

Apologies to the Missouri Water Pollution Board for the statement in our January, 1967 issue that "Witnesses at the air hearings were sworn in and all testimony was recorded verbatim; neither of these procedures was followed at the water hearings." Jack K. Smith, Executive Secretary of the Missouri Water Pollution Board informs us that verbatim transcripts of this and all other MWPB hearings were made and are available for inspection in Jefferson City.



Four articles on

LEAD IN OUR URBAN CIVILIZATION

LEAD IN THE MODERN ENVIRONMENT
How Much Is Natural?

By CLAIR C. PATTERSON
with JOSEPH D. SALVIA

IN JULY, 1965 a Boeing 707 took off from McGuire Air Force Base, carrying our small party from the California Institute of Technology. Within a few hours the big jet had left behind the summer heat of New England. It was bound for Thule, Greeland, where we would spend ten weeks collecting samples of snow from three types of sites: from the vertical faces of ancient glaciers at the edges of the ice caps; from deep tunnels at the polar research station Camp Century; and from surface snow layers in undisturbed virgin regions of the arctic.

Drs. T. J. Chow of the University of California, M. Murozumi of the Muroran Technical Institute in Japan, and I have collaborated for years in the study of lead contamination.

From studies of changing lead concentrations in polar snows of recent centuries and in seawater, we hoped to gather data which would determine the extent to which industrial lead has contaminated the atmospheres. The remote polar icecaps far from any immediate source of lead contamination, seemed the ideal proving ground. As rain and snow "scrub" the atmosphere, airborne dust particles and suspended aerosols are carried to earth. By careful measurement of annual samples of precipitation, changes in the relative concentrations of lead and other substances in the air can be determined. We hoped to measure long-term variations of lead contamination by gathering lead samples of snow layers from the polar icecaps.

The annual snowfall in polar regions is relatively small—about one foot per year in the Antarctic and three feet per year in the Arctic. Because of the low year-round temperature and the absence of melting or surface activity, snow is deposited in distinct annual layers which remain relatively undisturbed. Each layer is an intact record of what was in the air when the snow came down; the thickness of the polar icecaps is comprised of centuries of such records. In 1964 we had begun a series of expeditions to collect samples of ice and snow and measure the lead content. These expeditions culminated with the Greenland and Antarctica expeditions of 1965-66.[1]

I was accompanied on the Greenland expedition by three students from the California Institute of Technology. The plane carried tons of equipment, much of which had been developed for our purposes, and tested in earlier, preliminary expeditions in the United States and Greenland.

After disembarking at Thule, our party and equipment were trucked to Camp Tuto, on the edge of the ice sheet, where we gathered blocks of ice from tunnels driven deep into the vertical faces of the glacier sheets. Samples of ice, dated by carbon 14 determination at about 800 B.C.,[2] were taken from the exposed ancient edges of the ice sheet.

We then flew by helicopter 140 miles eastward to Camp Century, a center for U.S. arctic research until its abandonment in 1967. In its deep snow tunnels were year-round living quarters, shops, and laboratories, heated and powered by an atomic reactor and supplied in the summer months by a tractor from Camp Tuto. Here at Century we gathered more samples of ice and snow, this time from the walls of a thousand-foot long tunnel which slanted to a depth of more than 300 feet beneath the arctic ice. Samples of annual snow layers dating back more than 200 years were gathered by workers cutting the frozen blocks from the walls with chain saws.

Snow samples were dated by several different methods: stratigraphic techniques (annual layers are identified by variations in seasonal textures of the snow); direct measurement of accumulation on stakes; measurement compaction of successive snow layers; determination of lead 210 (a radioactive isotope of lead common in air—it is the daughter product of radon, a radioactive gas). Lead 210's twenty-two-year half life can be used to date snow as old as seventy years. An additional aid to dating recent samples was the known location of two layers of snow dated by fallout contamination from atomic and hydrogen bomb tests.

Our party, joined by a dozen soldiers, from the U.S. Army Research Support Group, embarked from Camp Century in August. We travelled hundreds of kilometers upwind in three fifty-ton caterpillar snow tractors, each pulling a train loaded with supplies, and research and communications equipment. The weather was bad and we traveled through almost continuous

storm, checking our course with a sun compass when possible. When the storms obscured the sun, we held our course by sighting back along flag-markers that we set out as we went. At a desolate, virgin site, working parties dug a trench fifty feet deep and 300 feet long to collect samples of snowfalls from the last fifteen years. (The Camp Century site, contaminated at the surface by human activity since 1954 and by slight melting of the upper layers of snow in summer, was unsuitable for the collection of recent samples.)

In November, 1965, I went to Antarctica to conduct similar experiments in the southern hemisphere, accompanied also by three Caltech students and by four students from the University of Canterbury in New Zealand. We flew from New Zealand to McMurdo Sound in Antarctica in Air Force C-130 turbo-prop planes, and from there to the mile-high Byrd Station, halfway between the South Pole and the edge of the Antarctic ice sheet, where even midsummer temperatures fall to ten degrees below zero.

Here, with electric chain saws and an electric winch, we dug an inclined tunnel more than 300 feet long and 140 feet deep. As in the arctic expedition, all personnel worked ten to twelve hours a day for many weeks, in low temperatures and at high altitudes. Because of the extreme demands, only young, strong men in top physical condition were chosen for the expeditions.

Again as in the arctic expedition, the project was concluded by gathering surface snow samples from a virgin site, uncontaminated even by the activities of the few men at tiny Byrd station. This time, we travelled many miles upwind from Byrd, on the mile-high Antarctic plateau. Our four man party travelled in a single snow-cat pulling a freight sled. At the final site, deep in this remote region, we excavated by hand a hundred-foot long inclined shaft to collect samples.

In both the Greenland and Antarctic expeditions, surface laboratories were set up at sites remote and upwind from the main camp tunnel entrances. Power lines from camp generators had to be laid to supply the electric cutting tools in the shafts and the ice-melters in the laboratories. Transformers were installed and laboratory buildings were built and their interiors lined with plastic sheeting.

The ice blocks were collected by men completely encased in plastic suits, working with acid-cleansed stainless-steel electric tools to prevent contamination of the snow. The shaft at Byrd alone required four weeks to excavate, working two ten-hour shifts per day.

Samples were taken from ten different time-levels in each deep shaft; from five different time-levels in each virgin site trench, and from several different levels at each glacier-edge site. The ice blocks gathered from each site were melted in the surface laboratories and transferred to large, chemically clean plastic drums for transport back to laboratories in the U.S., where they were analyzed for concentrations of lead and other substances.

For lead analysis, we use an extremely sensitive and accurate method called isotope dilution — a small quantity of lead isotope tracer is added to the snow water, mixed with the lead in it, withdrawn, and analyzed on a mass spectrometer to determine the ratio of tracer lead to sample lead. This method is complex, tedious and expensive, so the work is slow and costly. For determination of other elements, we use isotope dilution (potassium, calcium, titanium), neutron activation (sodium, chlorine), atomic absorption (sodium, potassium, magnesium), spectrophotometric techniques (chlorine), and emission spectrographic techniques (silicon).

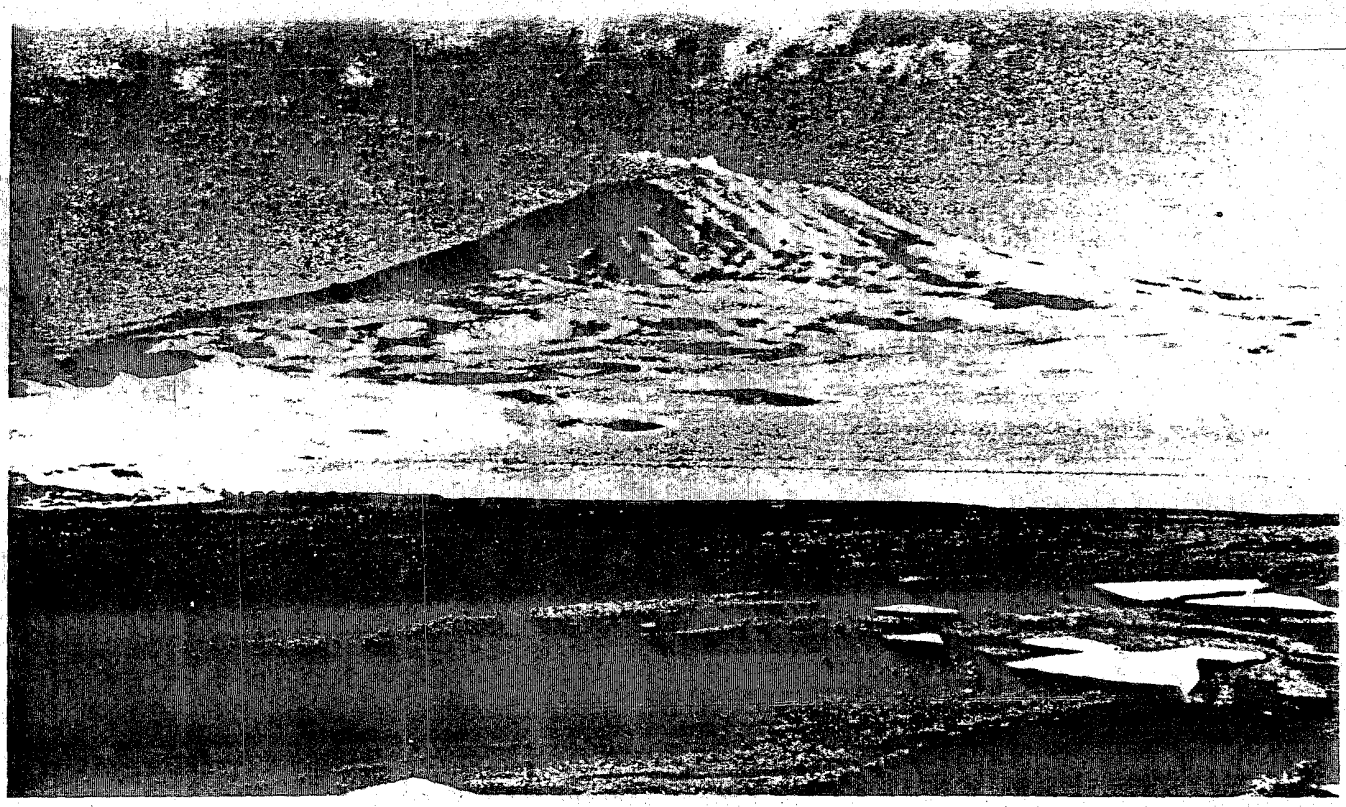
For the simple lead curve of sixteen points shown in Figure 1, about 400 lead analyses of various kinds have been performed over a period of four years. The scatter in these points is due mainly to our improper sampling techniques. At the time the ice samples were taken we were ignorant of seasonal variation effects, and we failed to mix equal proportions of rich and lean winter and summer layers in all samples, which introduced a pronounced scatter upon the yearly trend.

The analytical work was done cooperatively by Murozumi, Chow, and myself in our laboratories at the Muroran Institute of Technology, the University of California at San Diego, and the California Institute of Technology.

Three polar field expeditions were carried out between July, 1964 and February, 1966. But the polar snow studies were only one aspect of our inquiry into environmental lead contamination.

Chow is conducting a program of collecting measurements of lead concentrations in continental and oceanic atmospheres, rainfall, harbor waters, soils and grasses, and coal. Since March, 1967 he has operated a continuous air sampling program, monitoring lead in the atmosphere in the San Diego-La Jolla area. In 1967, the Scripps Oceanographic Institute's research vessel *Argo* collected samples of lead in oceanic air from thirty stations along its path from San Diego to American Samoa. Chow has found much less lead in the air over the mid-Pacific than in rural American air, indicating that country air outside cities is also polluted with lead. Chow has also analyzed lead content in coals from the major American mining areas, and in samples of soils and grasses gathered near busy U.S. highways in Maryland.

Dr. M. Tatsumoto, Dr. Chow, and I have made studies of lead and barium concentrations in sea water.[3] Most of this work has been in process for



MT. EREBUS, the only active volcano on the continent, is seen at the top of the page, as it appeared from a helicopter. Ice flows in the foreground are broken off the Ross ice shelf. The shelf reaches about a hundred feet out of the water and to a depth of about nine hundred feet below the water.

several years and it will probably be several years more before a comprehensive picture is completed.

Researchers have studied the distribution and concentration patterns of lead in sea waters all over the world. Long-term surveys have been made of the concentrations of lead in urban and rural atmospheres, and of the surface water systems which supply drinking water to most Americans. Geochemical studies and experiments have sought to determine the relationships between lead and other substances in their natural distribution.

The research of recent years has added to the already considerable knowledge of lead in the environment. The polar snow studies furnish evidence of a sharp increase in lead contamination of the atmosphere. The lead content of North polar snows has increased gradually and dramatically with time, displaying a marked parallel with the increase in lead smelting and in consumption of leaded gasoline. Near the North Pole, lead concentrations apparently have increased about four hundred per cent between 1750 and 1940; since 1940, there has been another, sharper increase, this time by about three hundred per cent. Lead content in snow dating from 1750 is about twenty micrograms per ton; from 1860 about fifty micrograms; from 1940 about eighty micrograms; from 1950, about 120 micrograms; and from 1965, about 210 micrograms (See Figure 1).

This corresponds with what is known about the increase in lead smelter production following the industrial revolution, and the increase in the use in leaded gasolines in recent decades. Samples of snow taken from the North Greenland ice sheet furnish concentration profiles of other substances during the same period (See Figure 2). There has been no significant change, for example, in the concentrations of sea salts and calcium in snow during the last two centuries.

These findings become more significant in the light of the data gathered in Antarctica, where lead concentrations in snow samples taken near the South Pole were in sharp contrast with the Greenland results. The highest lead content of the Antarctic samples was about equal to the 800 B.C. sample, *lowest* value found in Northern snows. Older samples, taken from snow layers approximately two centuries old, showed no significant decrease in lead content. These findings suggest that the increase in lead content of Northern snows is due to atmospheric contamination, for most industrial contamination of the air occurs in the northern hemisphere and the prevailing air currents set up an effective barrier against the transmission of airborne contaminants from the northern hemisphere to the southern.

Measurements taken in the Atlantic, Pacific and the Mediterranean show that the recent distribution of

lead in sea waters follows a pattern of relatively high surface concentrations which decrease sharply with depth,[3,4] a pattern similar to that of strontium 90 and cesium 137, which are products of radioactive fallout from nuclear explosions,[5] and whose current concentrations in sea water are without doubt the result of recent contamination from two decades of atomic bomb testing (See Figure 3).

The lead pattern is strikingly different, almost the opposite, of the barium concentration in sea water (See Figure 4). Barium, an element closely related to lead, appears in low surface concentrations which increase with depth.[4] This reflects a history approximately in accord with the natural prevalence of barium in the environment. Since barium is of little industrial significance, this pattern is probably an indication of the distribution to be expected in the absence of industrial contamination.

Research in the field of environmental lead has not only shown an increase in lead concentration in the atmosphere and in the seas. Contamination from industrial lead is everywhere; air, water, food, and man himself carry many times the amount of lead they would bear in an uncontaminated environment. City dwellers are especially subject to high concentrations of lead, largely due to the discharge into urban air of tons of lead from the exhaust wastes of automobiles.

The *Survey of Lead in the Atmosphere of Three Urban Communities*,[6] compiled in Cincinnati, Philadelphia, and Los Angeles, by a working group from public health agencies, the Kettering Laboratory and industries producing leaded gasoline, shows *annual average* airborne lead concentrations in these cities of 1.4 micrograms per cubic meter of air in Cincinnati, 1.6 micrograms per cubic meter in Philadelphia, and 2.5 micrograms in Los Angeles. These figures range from thirty to fifty times the average existing rural concentration, and five thousand times the estimated natural concentration.

Shortly after this report was issued, I criticized the California State Department of Public Health and the U.S. Public Health Service for collaborating with the leaded fuel-producing industries in seeming to exonerate leaded fuels as a source of increased lead burdens in the bodies of city dwellers.[7] I pointed out that these agencies had ignored the obvious relationships which existed between present blood lead levels, lead concentrations in air, and material balance considerations of the sources of lead in the atmosphere.

California State Department of Public Health investigators John R. Goldsmith and Alfred C. Hexter, working with data from the above survey and from experimental data compiled by Dr. Robert A. Kehoe, now recognize a predictable dose-response relationship between increased respiratory exposure and increased blood lead levels. Goldsmith and Hexter now take issue with Kehoe, the Professor Emeritus of Occupational Medicine at the Kettering Laboratories, who says:

> So long as a larger quantity of lead is absorbed by the average citizen from food and beverages than from the air . . . the control of atmospheric lead cannot be expected to obviate a potential lead hazard to the general population, since it does not concern itself with the principal source of such hazard.[9]

On the basis of accepted measurements of intake and absorption rates for lead, and recent measurements of airborne lead concentrations, Goldsmith and Hexter now contend, as I had pointed out earlier, that total lead absorption from alimentary and respiratory tracts are of similar magnitude, and that for residents of highly contaminated areas such as Los Angeles, the amount of lead absorbed from inhalation may be greater than that from food and drink.

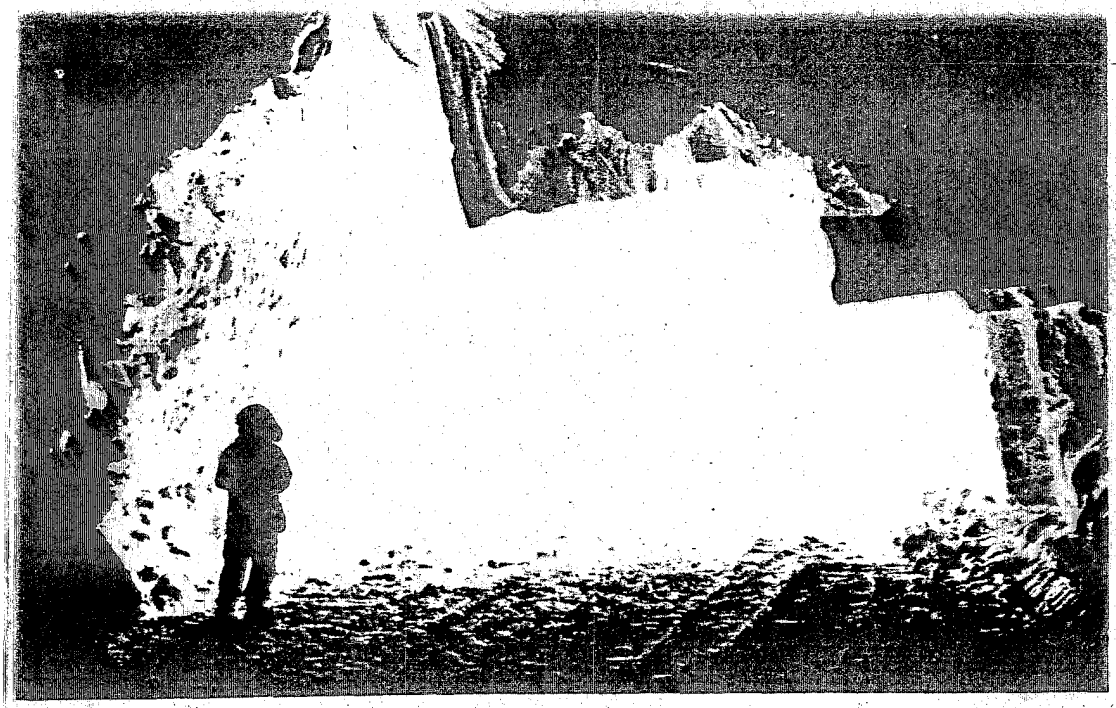
They conclude that airborne lead *is* a principal source of body lead, at least for city dwellers, and that it is a real hazard:

> . . . increased respiratory exposure *within the range observed in community air pollution* is capable of producing materially increased storage of lead in the body, as reflected in blood lead level; and that *further increases in atmospheric lead will result in higher blood lead levels in the population in a predictable relationship.* (Italics added)

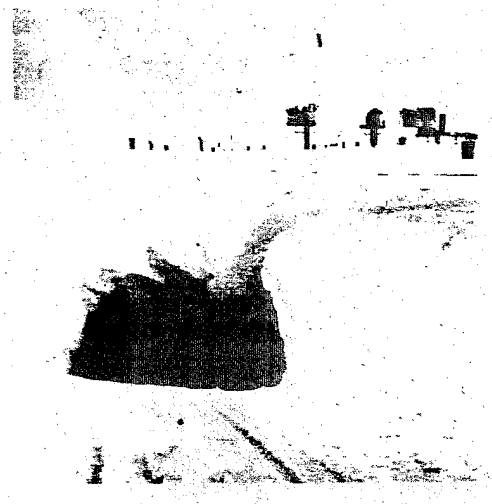
This conclusion is still not universally accepted, however. Some authorities see no immediate threat in atmospheric lead contamination. In 1960, Dr. Kehoe, had this to say of the public health implications of lead exposure:

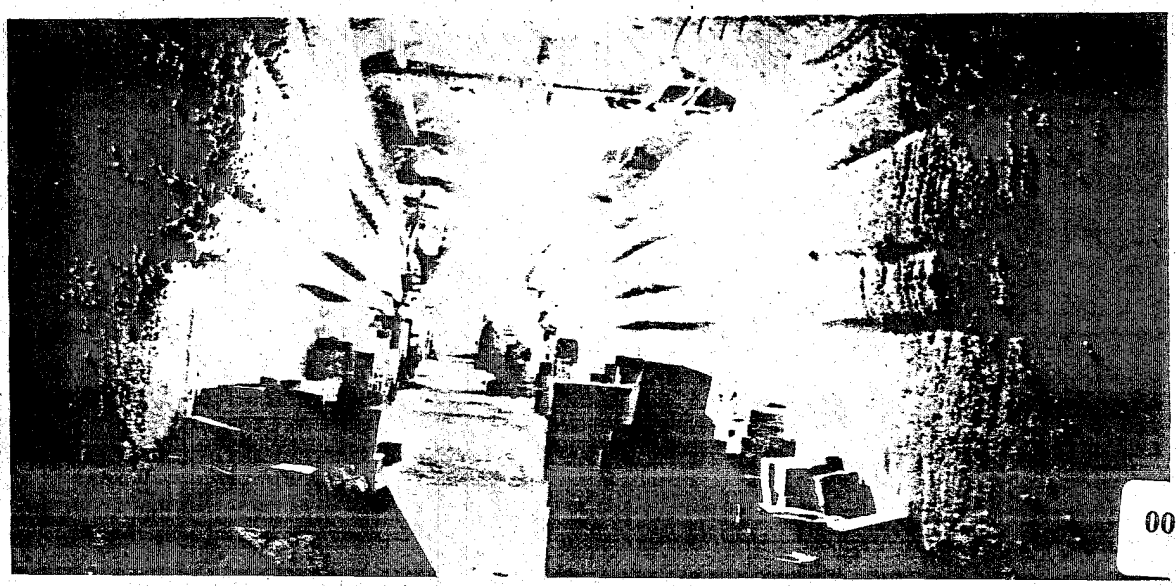
> It is fair to say that, at the present time, in the United States, the absorption of lead on the part of the public, generally, is attended by no hazard. This is not to say that there may not be situations productive of danger and of actual cases of lead poisoning within the population. . . . On the whole, however, the food and beverages of the nation contain but little more lead than that which occurs naturally in them, while the ambient air, even in the more heavily contaminated areas of our cities, is contaminated with lead only to the extent of a few micrograms per cubic meter. . . . We have found no reason to believe or to suspect that the quantities of lead involved in this normal metabolism of lead have increased within the past twenty-odd years.[10]

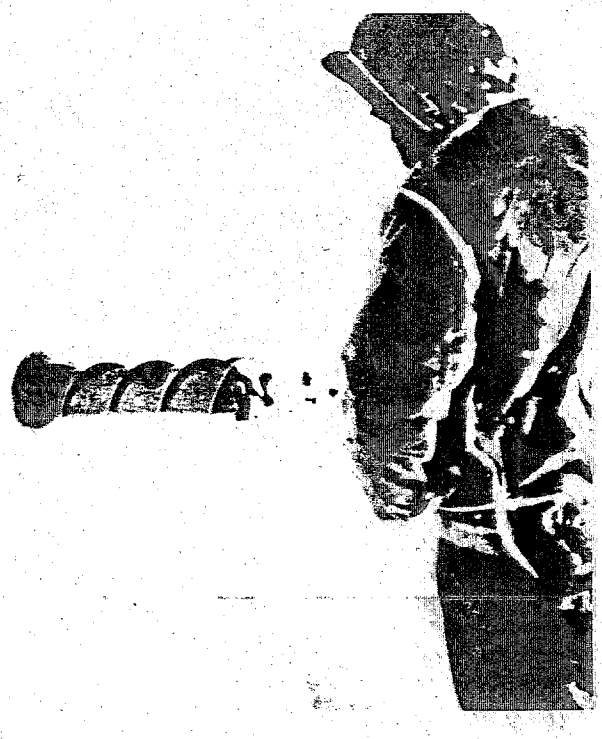
It seems to have been generally accepted by many people in the fields of public health and occupational medicine that—excluding occupational exposure to lead—the lead content in a typical man's blood and tissues is well within the margin of safety. This assumption—and it is important that we identify it as such—is based on other assumptions, now questionable in the light of recent studies of lead in the environment.

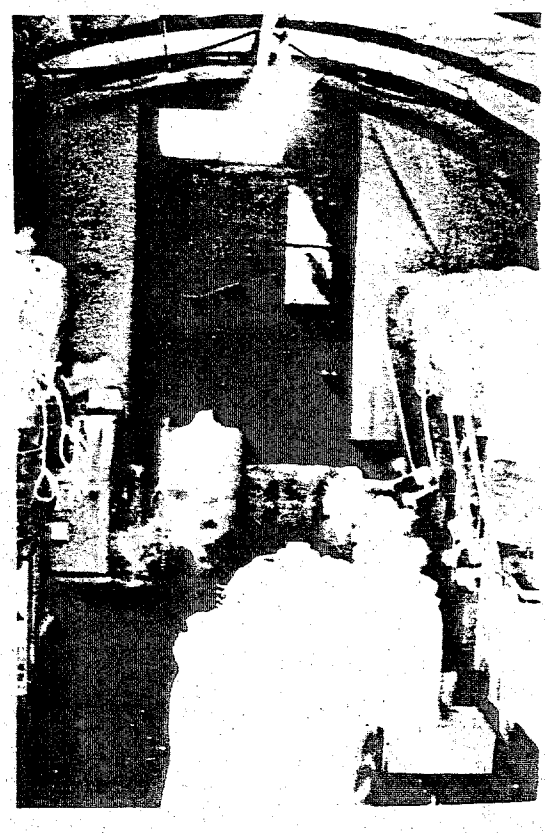


STUDIES OF POLAR SNOWS took Patterson and his party to the Arctic and Antarctic to study the content of layer after layer of snow for lead and other substances. In the Antarctic, the party's headquarters were at the mile-high Byrd Station, halfway between the South Pole and the edge of the Antarctic ice sheet where even midsummer temperatures fall to ten degrees below zero. The entrance to the tunnel used for living quarters is shown from inside (above) and outside (at right). Below is the food storage tunnel.









AN INCLINED SHAFT was dug deep into the snow. Holes were drilled into the wall of the shaft. Then blocks were hand sawn between the holes, broken off at the back and lifted out with metal tongs into plastic barrels. A laboratory for melting and bottling waters was built at Byrd Station, and lined with plastic sheeting. The water was transported back to U.S. laboratories for analysis. The ice blocks were collected by men completely encased in plastic suits, working with acid-cleansed stainless-steel electric tools to prevent contamination of the snow. The shaft at Byrd required four weeks to excavate, working two ten-hour shifts a day. Samples were taken from ten different time levels in each deep shaft.

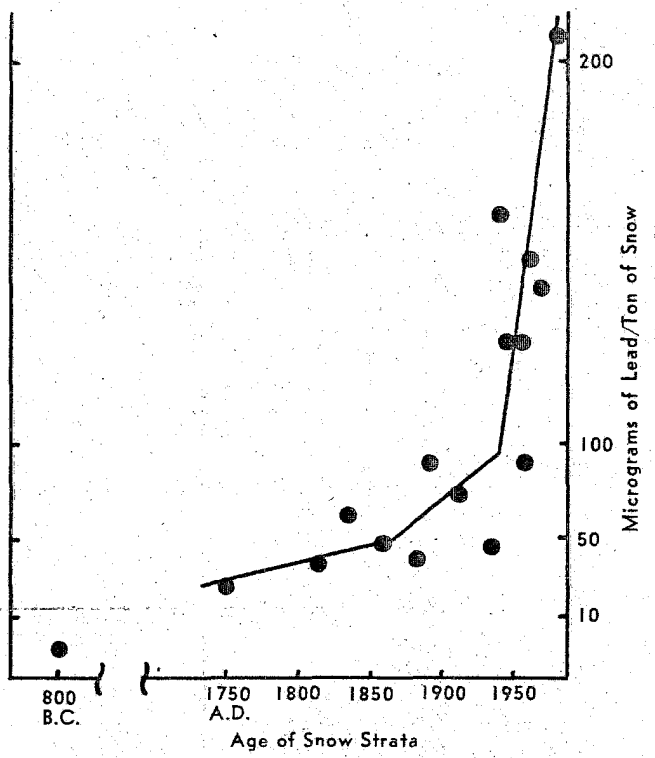


FIGURE 1. The amount of industrial lead in the snow was measured in samples taken from the Greenland Ice Sheet. The more recent the sample, the higher the concentration of lead.

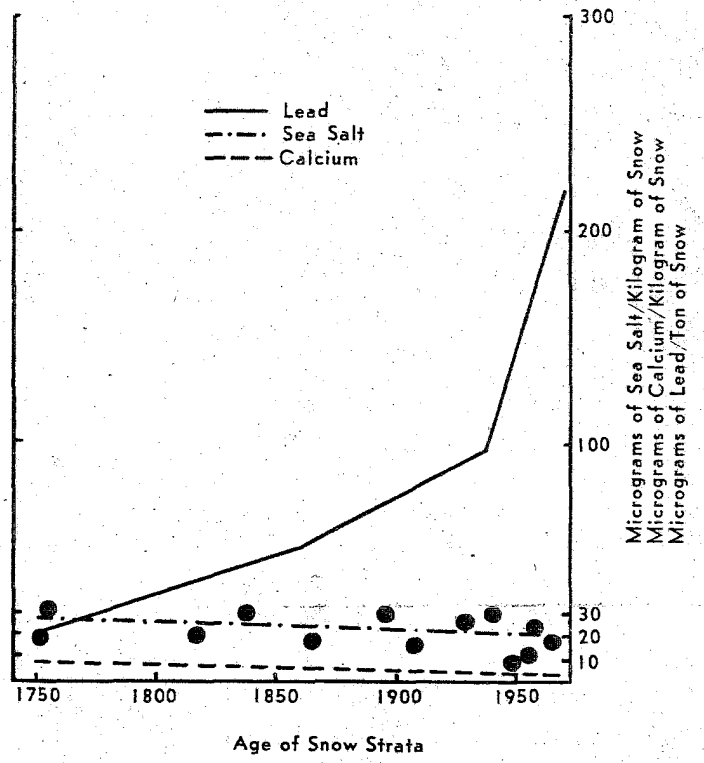


FIGURE 2. Measurements of sea salt and calcium were also taken in samples from the Greenland Ice Sheet. The presence of these substances has remained relatively stable, while the increased industrial use of lead has caused a sharp rise in the presence of that trace metal.

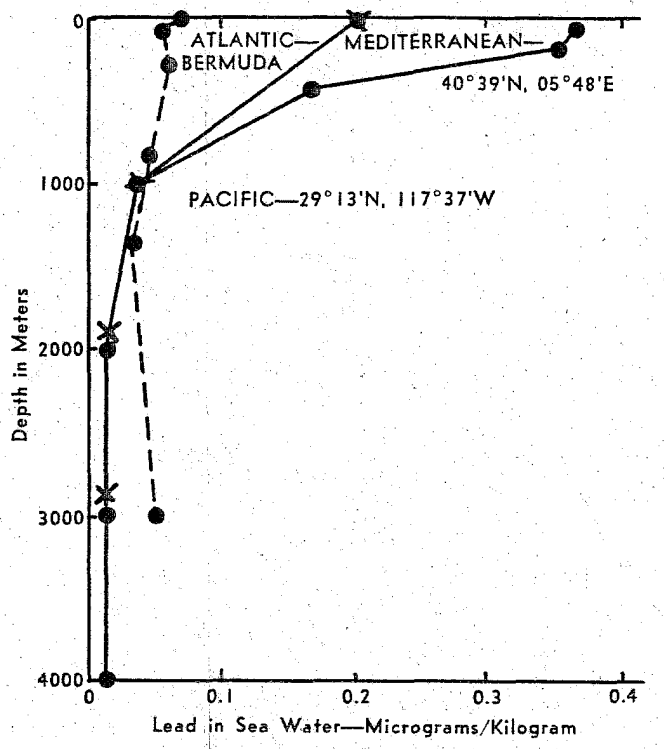


FIGURE 3. Lead in Atlantic, Pacific and Mediterranean waters. In contrast to barium, lead appears in a very low concentration in deep waters, but it increases rapidly above 1000 meters.

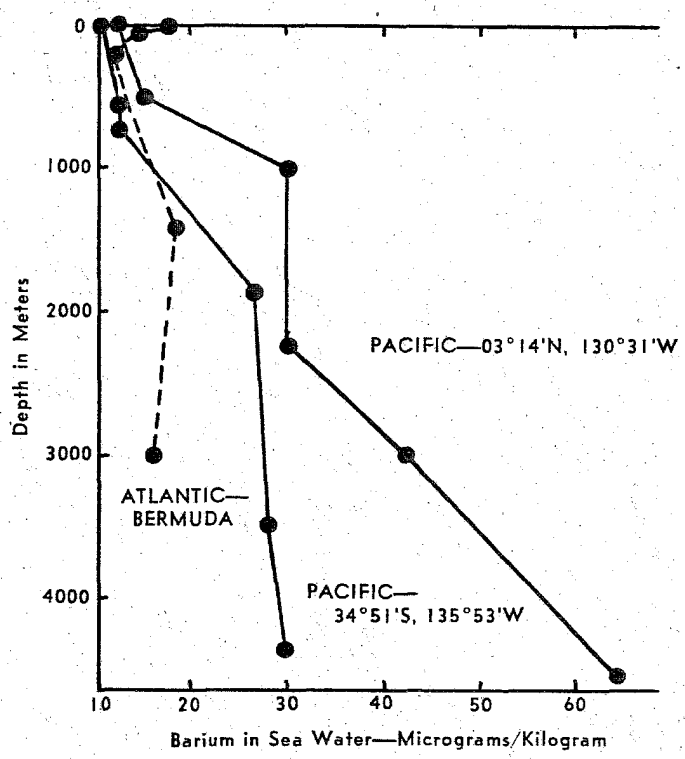


FIGURE 4. Barium in Atlantic and Pacific waters. Barium is a trace metal not used in industry. Note the greater concentration of barium in deep water than at the surface, where measurements become negligible.

Natural Lead States

It is *assumed* that lead in food and water is not much above natural levels. It is *assumed* that the typical lead burden in man today is not much above what would be expected in an entirely natural, uncontaminated environment. It is *assumed* that this lead burden is normal, or little above normal, in terms of man's body chemistry and the physical environment out of which he evolved. It is *assumed* that there has been in recent years no significant increase in lead in man's immediate atmospheric environment and therefore no significant increase in lead intake by human beings.

Drs. Leonard J. Goldwater and A. Walter Hoover of Columbia University said in 1967:

> Concern has been expressed that increased burning of gasoline by the internal combustion engine and the wider use of lead in industry are causing additional exposures to lead in our environment. At the same time the amounts of environmental lead from insecticides, lead pipe and paints have decreased. It would appear that the increase of lead from the former sources has been balanced by decrease from the latter.[11]

These assumptions are not demonstrable. They may be entirely false.

A most serious flaw in the above argument is that airborne lead pollution does not replace a segment of the lead pollution of foods with equal effect. For a unit of lead entering the lungs and a unit of lead entering the stomach, nearly an order of magnitude greater fraction is absorbed into the body by the lungs. Much of the lead entering the stomach passes through the gastrointestinal tract unabsorbed. The above-mentioned studies of lead concentration in modern and prehistoric environments casts serious doubts, furthermore, on what may be the most harmful assumption—that the typical American is in no danger from lead poisoning today and has been in no danger for decades. It may well be that *any* interpretation of the danger of lead to typical human beings today is meaningless except in the light of more accurate knowledge of a genuinely *natural* lead environment.

It is difficult to measure natural concentrations of lead in foods by studies of contemporary plant and animal tissues from relatively uncontaminated rural or primitive areas because industrial lead pollution is so widespread and of such long standing duration. I have been compiling a history of world lead production, and figures from that study show that very large quantities of lead have been smelted in southwest Asia and Europe since 2500 B.C., the time when men discovered how to obtain silver from lead ores. The huge amounts of lead that accumulated from these operations has provided the foundation for a widespread use of lead in human societies throughout the world during the last four thousand years. The Romans produced an average of more than 60,000 tons of lead each year for four hundred years, and most of this lead was shipped to Italy, where it was utilized by a population of about ten million free people and four million slaves. With an industrial per capita consumption of approximately 0.004 ton of lead per person per year, the Romans are essentially indistinguishable from modern, industrialized Americans, who have an industrial per capita consumption of lead of about 0.006 ton of lead per person per year.

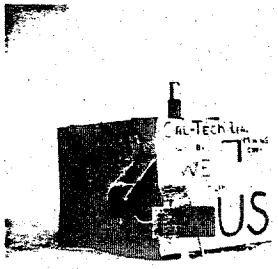
Natural lead concentrations can also be estimated by comparing the occurrence of lead with that of such commonplace elements as calcium, strontium, and barium. Because these elements, unlike lead, are not of industrial importance, their occurrence in plant and animal tissues has not changed significantly since primitive times.

Man's natural sources and rates of lead intake can be estimated from the same and similar data. These studies of processes which determine the natural lead content of air and water suggest that, in an uncontaminated setting, essentially all of man's lead intake comes from food, with insignificant amounts originating in air and water. The total lead intake of a man in a natural environment is estimated on this basis at about 20.5 micrograms per day.[7]

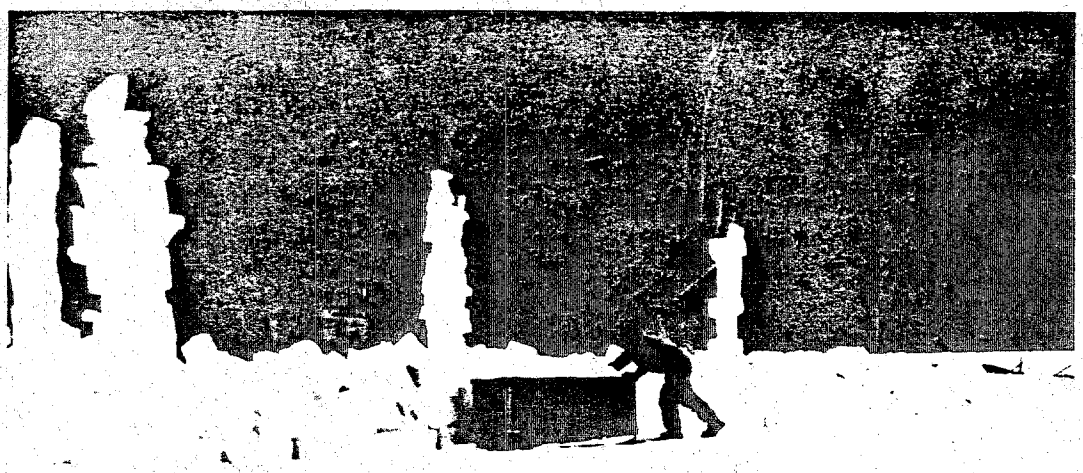
Kehoe[10] and others have studied lead intake and output and established accepted rates of lead absorption in blood and tissue. About ninety-five per cent of ingested lead—that is, lead taken in from food and beverages—is excreted in the feces. Most of the five per cent which remains is absorbed into the blood and later excreted in urine. But a very small portion of this absorbed lead is *retained* in the tissues, mostly in bone, so that over a period of years a *body burden* of lead is accumulated which varies with the changing conditions of lead exposure.

This body burden is a function of lead intake, rate of absorption, and rate of retention of absorbed lead. Of the twenty micrograms that are thought to have been ingested daily by man in an uncontaminated state, only about five per cent, or one microgram, is absorbed into the blood, and most of this absorbed lead is stored by the body; a man in an uncontaminated environment would accumulate a body burden of several milligrams during his lifetime.

Lead exposures are usually expressed today by the amount of lead in the blood. It is necessary, therefore, to convert this estimated natural body burden into an estimated level of lead in the blood. In the absence of other information today, one may suppose that the natural distribution of lead in the body is in



OUT FROM BYRD STATION in search of a remote site where top layers of snow are uncontaminated by the activities of men, the four-man party travelled in a single snowcat pulling a freight sled. Diesel fuel used was low in lead. A tool shed and warming hut were built, a shaft dug, and sample blocks taken from five levels, each representing a different year.



the same proportion which prevails today. In the U.S., the significant range of blood lead is from about 0.05 parts per million to 0.4 parts per million,[10,12,13,14,15] with a mean level of about 0.25 parts per million; while the mean body burden is about 200 milligrams of lead. Following the same proportion, the estimated natural blood level would be about 0.0025 parts per million, or only a hundredth of current typical levels.

There is a possibility that this proportionality may not be quite so linear. That is, in contaminated states the blood may not reveal the full extent of the total body burden of lead, so that changes in blood lead concentrations, in going from natural to contaminated environments, are not so severe as corresponding changes in total body burdens of lead. We may, therefore, be underestimating blood lead levels in the natural state.

All of these estimates of the natural lead state in man are based on a set of relationships which exists in the occurrence of certain subgroups of metals (including lead) in the earth's crust and in man, and on observation of the behavior of these metals in the human body. Details of the geochemical processes involved and full documentation of the studies which contributed to these estimates can be found in my article, "Contaminated and Natural Lead Environments of Man," *Archives of Environmental Health*.[7] Although these estimates cannot carry the weight of direct observation, they represent the only comprehensive attempt yet made to determine natural lead states.

When estimates of natural states are placed together, they give the following perspective of the position of primitive man with respect to lead in his environment:

- about 0.01 parts per million of lead in human foods.
- about 0.5 micrograms of lead per liter of water.
- about 0.0005 micrograms per cubic meter of air.
- total lead intake of about 20.5 micrograms per day for a 150 pound man.
- average body burden of about two milligrams for a 150 pound man.
- average natural blood lead level of about 0.0025 parts per million.

The next step in the process is to survey existing lead states and compare the situation today with these estimates of natural lead states.

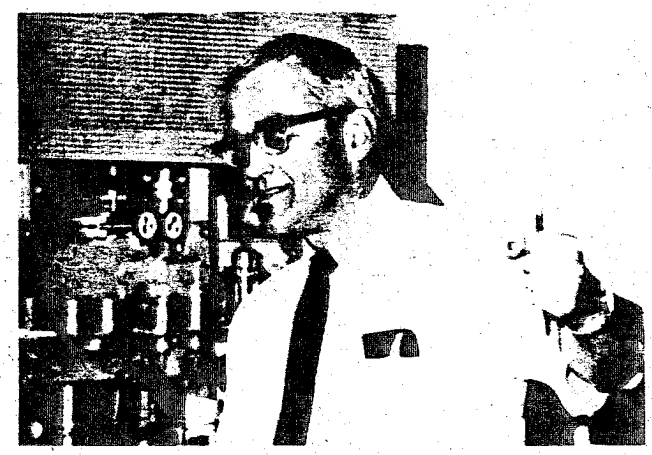
Lead Today

The industrial use of lead today is so massive that the amount of lead mined and introduced into our relatively small urban environments each year is more than a hundred times greater than the amount of natural lead leached each year from soil by streams and added to the oceans over the entire earth.[16] There are indications that about nine-tenths of the lead in the young surface layers of the oceans in the northern hemisphere is industrial in origin,[17,18] and that the atmosphere of the northern hemisphere contains about a thousand times more than natural amounts of lead.[19]

As a result of this contamination, the ratio of industrial lead to natural lead may be very high in common foods and other biological materials, and lead concentrations in typical people and environments far above natural levels. How has the 1,200,000 tons of lead annually consumed by American industry affected man's intake of lead and body burden?

In one survey of foods in Britian and the United States,[20] the American beverages were found to have only five times the estimated natural lead content, but in this case and in two other surveys,[9,21] all other foods showed a much higher lead content, ranging up to 4.2 parts per million in British beverages.

It should be pointed out that if a person ingests, for a prolonged interval, food and liquids averaging several parts per million of lead, he will become



Clair C. Patterson, Ph.D. has been a research fellow in the Division of Geological Science at the California Institute of Technology since 1952, is well known for measuring the age of the earth. He says, "I had the good fortune to be exposed after the war at the University of Chicago to the influence of men who originated the new field of nuclear geochemistry, and there I participated with my colleagues in the development of theories of the origin of meteorites, and by means of mass spectrometric techniques, developed methods for dating small quantities of minerals in common rocks and methods for using micro amounts of lead isotope tracers to unravel geological processes." In 1963, Dr. Patterson and his colleagues discovered that the oceans were being substantially polluted with airborne lead. While a visiting professor at the Massachusetts Institute of Technology in 1964, he made a study of the extent of lead pollution in human populations.

TABLE I

Source of Lead	Amount per Day[34]	Lead intake (in micrograms)	Amount of Lead Absorbed by Various Groups (in micrograms per day)			
			Rural Non-Smokers	Rural Smokers	Urban Non-Smokers	Urban Smokers
Food	2 Kg	400	20	20	20	20
Water	1 Kg	10	1	1	1	1
Urban Air	20 cu. meters	26	—	—	10	10
Rural Air	20 cu. meters	1	0.4	0.4	—	—
Tobacco Smoke[31]	1 ½ Packs	24	—	10	—	10

TABLE II

Approx. Number[35-37]	Group	Micrograms of lead taken into the body per day	Micrograms of lead absorbed per day	Parts per million of lead in blood[6]
30,000,000	Urban Smokers	461	41.0	.21
70,000,000	Urban Nonsmokers	437	31.0	.17
30,000,000	Rural Smokers	436	31.4	.17
70,000,000	Rural Nonsmokers	412	21.4	.11

incapacitated with acute classical lead poisoning. It is interesting to note that studies which showed that this was true[10] were being made at about the same time U.S. government health agencies, at Congressional urging, had finished investigations of lead arsenate pesticides in 1939 and had set maximum permissible levels of lead in apples at 7 parts per million. Data pertinent to the ill effects of several parts per million of lead in food are summarized on page 74 of Kehoe's *Harben Lectures*.[10]

In the United States, the average lead concentration in foods is about 0.2 parts per million, about twenty times the inferred natural concentration.

Lead concentration in fresh surface waters and in local water supplies has been the object of many surveys in this country.[22-27] On the basis of these surveys, average concentrations are about 0.008 parts per million of lead in rivers and about 0.011 parts per million in municipal water supplies. The increased lead content in municipal water supplies is probably due to contamination from lead piping in the water systems.

A conservative estimate of average lead concentrations in existing urban atmospheres is about 1.3 micrograms of lead per cubic meter.[28,29] For existing rural atmospheres a similarly conservative estimate would be about 0.05 micrograms per cubic meter.[14,30] These estimates are based on direct measurements of lead concentrations in selected urban and rural atmospheres.

An additional and important contribution to respiratory lead exposure originates in recent decades from tobacco smoke. The lead in tobacco comes from auto exhaust fallout and lead arsenate insecticides. In this country, the average one-and-a-half pack-a-day cigarette smoker used to be exposed to about 24 micrograms of lead per day by inhalation.[31]

All the above data can be summarized to give a fairly accurate estimate of an average person's lead

intake. But most of the lead passes directly through the digestive system and is excreted. The important factor, for our purposes, is the amount of lead that is absorbed into the blood.

As mentioned before, in the discussion of lead intake under natural conditions, only five per cent of the body's lead intake from food and water is absorbed even temporarily into the blood and tissues.[32] In striking contrast, about *forty per cent* of the lead that is inhaled is absorbed by the body.[33]

By grouping the population at large into urban and rural, smokers and non-smokers, the population can be divided into categories according to variations in respiratory exposure, and their respective rates of lead intake and absorption can be compared as shown in Table I.

There is probably very little difference in the average lead content of food and water consumed by people in these four population groups. The large differences in the amounts of lead absorbed by the groups is the result of differences in the amounts of lead they inhale from air or from smoking.

Although there is very little difference in total lead intake among these groups, the differences among these groups in lead absorption and blood lead levels, as shown in Table II, contrast sharply. The total increase in lead intake from lowest to highest is only about twelve per cent, but increases in absorption and blood lead levels are close to 100 per cent. This disproportion is the result of the higher absorption factor of inhaled lead, and serves as another forceful warning of the greater hazards from lead in urban air and in tobacco smoke.

In summary, there is no doubt that industrial lead, especially lead from auto exhaust wastes, has greatly increased lead contamination of the atmospheric environment in the modern era. The studies of lead concentrations in northern polar snows and in sea waters of the northern hemisphere indicate a significantly sharp increase in lead contamination in the last few decades. As a result, people in the northern hemisphere, especially city-dwellers, have been exposed to constantly increasing amounts of lead. Most of this increase seems to be in the form of airborne lead aerosols, so that it is now clear that atmospheric lead contamination is, at least in large cities, a hazard of equal or greater magnitude than lead in food and water.

It has been demonstrated that there is a clear dose-response relationship between atmospheric lead contamination at levels common in urban air pollution and the blood lead levels of those exposed.

What will happen if lead contamination continues to accelerate in the future as it has in the last two hundred years? There is good reason to believe that consumption of leaded gasolines will continue to increase as the number of automobiles increases and as auto engines become more powerful.

The public may soon be forced to decide whether the health hazards inherent in contamination from lead are outweighed by the social and economic exigencies of today's patterns of lead consumption. For more than forty years federal and state health authorities failed to recognize this hazard, and my lead contamination study met severe opposition at first, but the climate of opinion has changed. The possibility of danger from current levels of lead pollution is now a respectable viewpoint even in the field of medicine.[38] Federal and state health people are re-examining the situation. It may be that increasing lead contamination will soon necessitate the setting of standards for lead contamination of the atmosphere.

All photographs courtesy of Dr. Patterson.

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LEAD AND HEALTH

A *Scientist and Citizen* adaptation of an article by H. L. Hardy, "What is the Status of Knowledge of Toxic Effect of Lead?" *Clinical Pharmacology & Therapeutics*, 7:713-722, November-December, 1966.

THE MOST OBVIOUS CONCLUSION to be drawn from any review of the available knowledge of lead effects on health is that little is known of *how* lead causes illness except in the event of heavy industrial exposure. An equally important conclusion is that the amount of lead which causes damage to any one person is still in doubt.

Because all recognized effects of lead in the body are harmful and the individual responses varied it is a considerable leap to conclude that there is a threshold below which lead damage does not occur. The threshold may be useful in predicting a point below which certain *clinical symptoms* do not appear, but there is no guarantee that damage does not occur below this level and in the absence of the symptoms of clinical lead poisoning.

Health authorities have been concerned with the prevention of easily recognized acute lead poisoning in lead workers and small children—the two groups most subject to lead poisoning. The most common diagnostic techniques have been blood tests to determine the concentration of lead in the blood and to check for evidence of lead-caused interference with the blood-forming system of the body.

But all the blood tests can do is warn of the danger of acute lead poisoning or verify a clinical impression of lead poisoning. Elevated blood lead levels, in the range of 0.5 to 0.8 parts per million, merely indicate that the subject has been exposed to excessive amounts of lead; but individuals with such high blood lead levels may display no clinical symptoms of poisoning, and acute lead poisoning occurs only in the upper limits of this range.

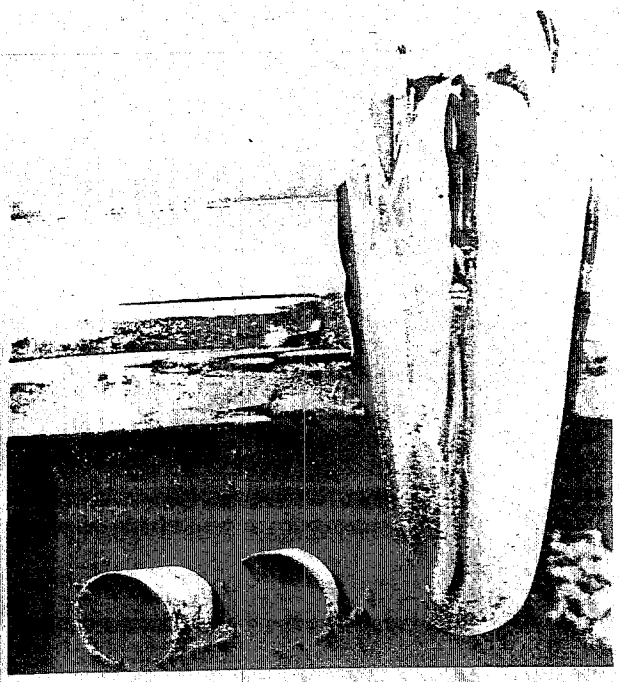
It has recently been suggested that lead may damage health at low levels of exposure — lower than those associated with classical lead poisoning — or that it may be a contributing factor in the development of certain chronic diseases. Little has been done to find out if there is a variety of lead poisoning we have not recognized: a chronic debilitation that results from an intake of lead far lower than has been assumed dangerous. Perhaps damage from low-level lead intake has been overlooked because no one knew what to look for.

If low-level damage from lead exists, it occurs in the range of 0.25 to 0.8 parts per million in the blood, and it is quite a different thing from classical lead poisoning. It cannot be recognized if the signs and symptoms which identify classical lead poisoning are considered necessary for diagnosis.

The basic problem in determining the mechanisms of lead poisoning is the extreme variability of individual response to lead. Amounts of lead that cause serious illness in one person may produce no detectable harm in another. A lead worker with a blood lead level of 0.65 parts per million may become ill, with all the symptoms of lead poisoning, while a fellow worker with a lead level as high or higher may show no ill effects. In industry, some men are able to work under conditions of heavy exposure for twenty-five to thirty-five years, while others, working in the same room under identical conditions, will develop lead poisoning in a few years or even a few months.

Moreover, even subjects reacting to the same exposure at the same time may show symptoms of variable severity or of various types. Some develop colic, others palsy or paralysis, and others brain damage. In some, the kidneys are particularly affected; in others, the nervous system; in others, the blood.

Despite this wide difference in individual response —a difference which also appears in uniform groups of experimental laboratory animals—there is a consistent quantitative relationship between the amount of lead and the kind and intensity of its effect on tissue, for example, blood cells, muscle, intestinal strips, and nerve cells.[1]



THE PRINCIPAL SOURCE of airborne lead is undoubtedly tetraethyl lead, an anti-knock agent added to gasoline since 1924. This lead would offer no hazard if it were burned and retained in crank case oil, but unfortunately, twenty-five to fifty-five per cent of it comes out of the exhaust.

Since the variability of response occurs only when the intact living organism is exposed to lead, the difference in response is probably due to individual differences in factors that control the amount of lead to which tissues in the intact body are exposed. Some such factors are diet, malnutrition, infection, disease (especially blood or kidney diseases), injury, vitamin deficiency and drugs.

The identification of low-level lead damage is complicated by the fact that there is much to be learned about the physiological effects of lead. Several aspects of lead behavior in the body are imperfectly understood. Among these are the action of lead in destroying red blood cells, the effect of lead on certain organs (especially the kidneys and brain), the effect of lead on growth in immature animals, and the cumulative and additive effects of lead retained in the body for years.

Perhaps the most commonly agreed upon effect of lead is its damage to red blood cells. Anemia is an almost universal finding in cases of lead poisoning among children and adults. The damage occurs at two levels: interference with the body's basic process of blood cell formation and injury to mature red blood cells. These circulating cells become more fragile and therefore fail to survive normally. Apparently the lead alters the cellular mechanisms which maintain the cell's structure. As the body fights the anemia resulting from the destruction of red cells in the blood stream, the production of red cells in the bone marrow is accelerated. Since lead interferes with the process of hemoglobin formation, the anemia cannot be fully offset by increased formation of new red blood cells if lead exposure continues.

The damaging effect of lead on the structure and function of the kidneys has been investigated intensively, but there is still considerable controversy about the exact nature of the effects. The most common effect seems to be a deterioration of the arteries of the kidney, which in time can produce a crippling atrophy of the organ. It is still uncertain, however, how great a part other, non-lead related causes may play in this damage.

Researchers in England[2] and Australia[3] have claimed an association between excess mortality from kidney disease and job-related lead exposure, and the Australian studies[3,4] indicate that acute childhood lead poisoning may be related to the development of chronic kidney disease in early middle age.

Not enough is known about the manner in which lead damages the nervous system. There is no question, however, that it produces a variety of changes in practically all portions of the nervous system, some of which are unquestionably harmful. The effect of acute lead poisoning on the brain is extreme. Central nervous system damage caused by lead is marked by destruction of various types of brain cells and degeneration of capillaries and blood vessels throughout the entire structure of the brain.

Lead encephalopathy (as lead injury to the brain is termed) can cause brain hemorrhage, accumulation of fluid, swelling or shrinking of the brain, and atrophy of the convolutions. There are serious and widespread disturbances of blood circulation throughout the brain in acute cases. Lead damage to the brain can cause convulsions, delirium, or coma. It can result

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in severe headaches, blindness, paralysis, mental retardation, or death.

It has been long accepted that acute lead encephalopathy can cause mental retardation. R. K. Byers, a pediatric neurologist, has suggested that mental retardation may also result from lead poisoning in which brain damage had not been diagnosed:[5]

> I can't believe that this isn't in some way related to interference with the brain enzyme systems through amounts of lead which may not in themselves be impressive . . . I originally got interested in lead because of children who had had lead poisoning and been sent home from the hospital as cured, who then turned up in my neurological clinic because they were misbehaving in one way or another, or not learning in school. One kid set the schoolroom on fire, another nice little girl danced around on the desk.

Byers adds that although none had ever had acute brain inflammation, all showed a history of lead poisoning. He continues:

> The point I am trying to make about these children is that though none of them, at two or three years, had acute lead encephalopathy with their acute intoxication, when they reached six or seven they showed evidence of injury. This group of children deteriorates gradually without ever having had acute lead encephalitis . . . I think that lead does something to the growing brain which is different from what it does to the adult brain.

There is no doubt that lead has serious effects on growth and development of immature animals. No consistent relationship has yet been found, however, between the rate and amount of lead intake and interference with growth and development. Studies of the effects of lead on some of the basic chemical processes in animal organs have led some researchers to believe that lead produces profound changes in metabolism as a result of its effect on many basic enzyme systems.[1] Attempts to pursue this course, however, have produced uncertain and often contradictory findings. Again, as in the case of lead effect on blood, brain and kidneys, there is no doubt that lead causes abnormalities and injury, but there is no certainty as to the manner in which it works its harm.

It is also necessary to consider the cumulative and additive effects of lead. It is *possible* that slight but constantly increasing amounts of lead in body tissues may cause damage, whether working alone or with other harmful factors. Because of the non-specificity of signs and symptoms, because of delayed diagnosable damage, because of the possibility of other harmful agents acting like or with lead, sophisticated attention to the potential effect of low doses of lead is required —in much the same manner as low levels of ionizing radiation have been studied since the use of atomic energy for military purposes in 1945.

Studies with isotopes of lead or stable lead compounds as they occur in twentieth-century urban air might tell us how much toxic effect specific organs can tolerate without impaired function or threat to health. Bolder attempts must be made to study the effects of multiple factors acting with lead on an organ or system.

Extension of studies such as those of Byers,[5] Smith,[6] and studies at The Massachusetts Institute of Technology, relating mental retardation and psychic disturbances in children with excess lead (both with and without clinical lead poisoning), will be helpful.

There is a wealth of knowledge of the biological effects of lead and its compounds. There is no available evidence that lead is useful to the body, and all data support the fact that lead is a heavy metal poison. We must commit ourselves to the task of identifying the damage, if any, that results from low-level exposure to lead.

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By HENRY A. SCHROEDER

AIRBORNE METALS

AIR IS ONLY ONE PART OF A SYSTEM which includes the total physical environment to which man is exposed. When air contains foreign substances, rainfall carries them to the ground. When these substances are soluble in water, they may appear in the water supply; when they fall on the soil, they may appear in plant foods which are eaten by man and domestic animals. Therefore, the problem of air pollution is closely linked to that of soil and water pollution, and its effects can exceed irritation of the lungs from acutely toxic substances.

The scientist's version of the average man — a "standard man" weighing seventy kilograms—inhales and exhales about twenty cubic meters of air every twenty-four hours. This daily volume of air (weighing some 24.1 kilograms) is about one-third the weight of his body and about six times the weight of his daily intake of food and water.

When air contains abnormally high concentrations of trace metals—a trace metal is one which makes up less than 0.01 per cent of the body—they will be inhaled. Once in the lungs, trace metals vary in their behavior: some accumulate in the lungs, in various proportions, more or less permanently; of these, some are absorbed into other body tissues.

We are concerned here with those chemical elements which may produce slowly accumulating effects during years of exposure. We want the answers to several basic questions:

- What metals are in the air?
- What are their principal sources?
- Which metals remain in the lungs and which are absorbed into the rest of the body?
- Which metals are beneficial, which inert, and which harmful?

Eight of these metals—aluminum, chromium, copper, iron, lead, manganese, strontium and zinc—were present in all the lungs tested; barium, tin and titanium were almost as frequent in occurrence in human lungs.

For more than fourteen years, Professor Isabel H. Tipton, Dr. H. Mitchell Perry, Jr. and I have worked with others, to answer these questions. Professor Tipton and her colleagues have provided the basic data on the content of twenty-one trace metals in human tissues from ten cities in the United States[1] and fifteen cities in Europe, Asia and Africa,[2] comprising over 400 cases. Nine years ago E. C. Tabor and V. W. Warren reported the first comprehensive data on metals in the air of American cities.[3] J. J. Balassa and I have been collecting basic data on the lifetime effects of some twenty trace metals on growth, longevity, and lifespan of mice[4] and rats,[5] attempting to duplicate as nearly as possible trace metal exposures and accumulations which occur in humans, in hopes of discovering what diseases, if any, may result.

We have learned that of the various airborne trace metals that accumulate in the lungs and other tissues at least two, lead and cadmium, are known to have injurious effects on humans. Others have demonstrable toxic effects on mice and rats, suggesting additional research to determine the effects on human health of airborne metallic elements known to accumulate in human tissue.

Table I lists some of the metals present in the atmospheres of more than twenty American cities and those present in the lungs of Americans.

These and all the other elements in Table I are found in coal, and most of them in petroleum.

In addition to these, other elements are found in coal: antimony, arsenic, beryllium, boron, gallium, germanium, lanthanum, mercury, rubidium, tungsten, uranium, yttrium, zirconium and probably niobium.[6] Many of these, including arsenic, cadmium, antimony, lead, mercury, zinc, and tellurium, vaporize at the burning temperatures of coal and oil, as do many other elements contained in chemical compounds, and all elements may be found in soot, ash, or smoke. Some elements—calcium, aluminum, silicon and iron —generally remain in ash and are not carried up chimneys into air; they are of little concern in the measurement of air-borne trace metals. There is no doubt, however, that the process of burning coal and oil adds greatly to trace metal contamination of the air as the trace elements in the fuels are vaporized. In fact, one of our most serious experimental problems is preventing trace elements from going up in smoke as we burn our tissue samples into ash for analysis.

In our consideration of possibly harmful trace elements, we can more or less eliminate those known to be essential for normal physiological processes; chromium, manganese, iron, cobalt, copper, zinc, and molybdenum. It is possible that vanadium and selenium also belong in this category. We would expect to find them normally in lung and other tissues, although air-borne contamination may deposit them in a form unusable by the body, thus making them a hazard.

All metals can be toxic to living tissue if they occur in large enough amounts and in the proper form. Those metals which are not essential for life and health, which may or may not be toxic, and which accumulate in lung tissue with age[7] must be carefully scrutinized; these include aluminum, cadmium, lead, titanium, vanadium and tin. We can probably eliminate from our consideration metals which are present in lungs in very small quantities and which do not accumulate with age in body tissues: bismuth, gold, nickel and silver. We know little about the behavior of germanium, tellurium, arsenic, antimony and niobium, which are probably present in air.

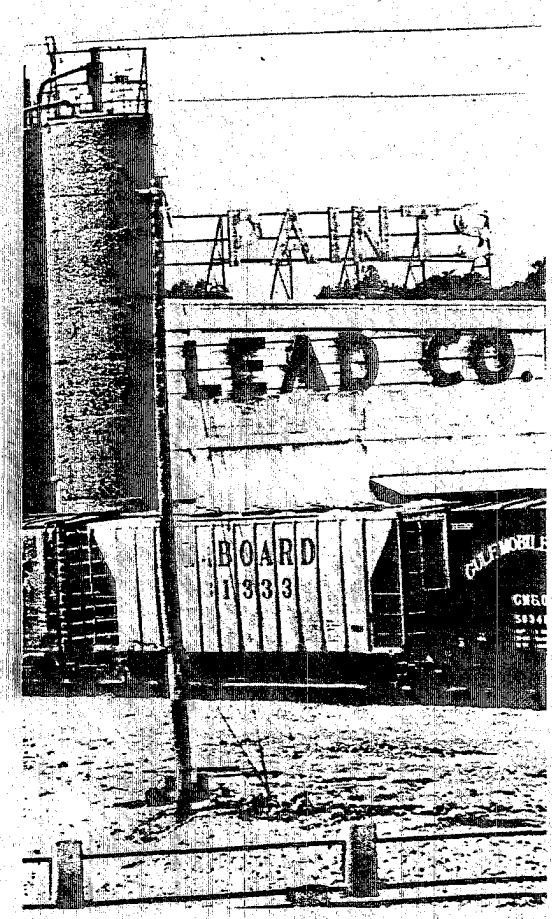
We can get some idea of the relative exposures of Americans to airborne metals by comparing the concentrations of these metals in their lungs with those of persons from other areas of the world. In Table II are the mean concentrations of trace elements in American, African, Near Eastern and Far Eastern lungs of males aged twenty through fifty-nine years. This table omits the trace metals essential for nutrition. It is apparent that there is less cadmium, lead and tin in African lungs than in American and Oriental. Aluminum, barium, strontium and titanium can enter the air in dust from soil, and their accumulation in African and Near Eastern lungs can be accounted for on this basis. The frequency of cadmium, lead, and tin in all but African lungs suggests industrial contamination as the source, for the lung tissues of Africans came from relatively non-industrial areas.

Study of the behavior and effects of cadmium and lead is especially important; these are the most common of the elements which accumulate in the lungs and which are also absorbed into the rest of the body. According to Tipton and Schafer,[7] aluminum, titanium, and vanadium, which accumulate with age, remain largely in the lung as insoluble contaminants, while lead "appears to enter the lung in soluble form and to be quickly carried to other parts of the body."

Lead and cadmium are among the elements known to shorten the life-spans of mice and rats. Lead poisoning in human beings is, of course, a long-recognized problem, and cadmium has been linked to at least one chronic disease of human beings, high blood pressure.[8] R. E. Carroll has shown that the death rate from cardiovascular disease, which includes high blood pressure and coronary heart disease, is significantly higher in areas where the concentration of cadmium in air is high, and significantly lower where the concentration of cadmium in air is low.[9] Most of the cadmium in the body, however, comes from food and water.

We know little about the sources of airborne cadmium, but it is likely that it enters the air as a by-product of industrial processing of zinc. Cadmium is a constant contaminant of zinc, and has a relatively low boiling point, 765 degrees C. Whenever zinc is heated to its boiling point, 906 degrees C., cadmium fumes can enter the air.

The principal source of airborne lead is undoubtedly tetra-ethyl lead, an anti-knock agent added to gasoline since 1924. The amount of mined lead annually used for gasoline additives amounts to about two pounds for each person in the United States (in 1963 the total was 192,811 tons).[10] This lead would offer no hazard if it were burned and retained in crank case oil, but, unfortunately, twenty-five to fifty-five per cent of it comes out of the exhaust, half to two-thirds in particles[11] and the remainder in gaseous form. The smaller particles can be blown by winds to surrounding areas and contaminate food crops; the larger particles may remain on the road; the gaseous forms enter the upper atmosphere and can be precipitated by rain and snow at far distant locations. All this lead accumulates in the environment year after year. We have detected lead in snow at ground level (but not at roof level) 200 yards from a suburban road within four hours after a snowfall. On the other hand, it took



EXPOSURE to low levels of lead is now common in our cities from industrial and traffic sources. It has recently been suggested that lead may damage health at low levels of exposure.

eight days for lead to be detected in snow in a mountain forest near a back road. Analyses of various sections of one-hundred year old elm trees have demonstrated rapidly increasing concentrations of lead since about 1937.[12]

Although *average* lead concentrations in the air are not considered high, varying from 0.2 micrograms per cubic meter on the Great Plains and Rocky Mountain areas to 0.7 micrograms per cubic meter in New England and the Pacific Coastal states, concentrations of six micrograms per cubic meter,[13] and in extreme cases seventeen to forty-five micrograms or more, have been measured in cities, especially on traffic-crowded streets or highways.[14]

At a concentration of one microgram of lead per cubic meter of air, a man would take into his lungs twenty micrograms of lead per day. Most of this lead is in time excreted by the body; if it were not, a man would accumulate in fifteen years of his life 110 milligrams of lead *by inhalation alone*, an amount almost equal to the average total body content of most Americans (estimated at 120 milligrams by the International Commission on Radiation Protection; other estimates are higher, ranging up to 220 milligrams).

But airborne lead is not the sole source of lead in the body. The average American also consumes about 300 micrograms of lead daily in foods and beverages. A man living in an area where lead concentration in air was fifteen micrograms per cubic meter would take in an additional 300 micrograms of lead by inhalation alone, roughly doubling his total lead intake. Lead poisoning would almost certainly develop in time, for the amount of lead absorbed by blood and tissues from the respiratory system is about five times as high as the lead absorbed from the alimentary tract. The blood and tissues absorb about fifty per cent of inhaled organic lead, and only about five to ten per cent of the lead intake from food and beverages (see Patterson article this issue). About ninety per cent of lead retained in the body is stored in bone.

An increase in airborne lead concentration from one microgram per cubic meter to fifteen micrograms per cubic meter would increase lead absorption, for the average man, from about twenty-three micrograms per day to 165 micrograms per day of lead absorbed by blood and tissue, based on the above estimates of absorption rates for inhaled lead and lead in foods and beverages.

Fifteen micrograms of lead per cubic meter of air is an extremely high concentration and relatively uncommon today, found only on or near heavily travelled streets and highways during rush-hour traffic;

but if the number of cars on U.S. roads doubles by 1980, as has been predicted, a concentration of fifteen micrograms of lead per cubic meter of air probably will be commonplace in American cities.

In one study of the effects of cadmium and lead on rats,[5] we attempted to achieve, by carefully controlled laboratory methods and diet, tissue concentrations within human ranges. All the experimental groups were kept in a low metal environment: the specially-built laboratory is situated on a hilltop more than a mile from the nearest road; the air is filtered and particulate contaminants are electrostatically precipitated.

The rats were fed a special diet of rye, dried skim milk and corn oil, devoid of cadmium and as low in other trace metals as is possible under laboratory conditions. The sole source of fluids was purified spring water to which was added vitamins and essential nutritive elements such as manganese, cobalt, copper and so forth. Five parts per million of cadmium or lead was added to the water of the test groups from the time of weaning until death; a control group received neither of these metals.

Briefly summaried, the experiments showed that cadmium and lead tissue concentrations in human ranges significantly decreased the life span of rats; in addition, the animals fed lead showed a higher mortality rate under exposure to infection. In one series of experiments, the median age at death of rats fed lead was 728 days, while the median age at death of control rats was 961 days; the median for cadmium-fed rats was 814 days. Of 249 rats fed cadmium or lead, forty died before the age of three months; none of the 104 control rats died before the age of three months. Autopsies showed neurological disease to be almost unknown in control groups but frequent in rats fed cadmium or lead. A majority of the cadmium fed rats displayed abnormally high blood pressure and hardening of the arteries in kidney, heart, lung, and liver, as well as enlargement of the left ventricle of the heart.

Table III is a partial summary of our experience in testing trace metals for innate toxic effects in rats and mice, as measured by a reduction in life span or the production of a chronic disease similar to those which humans suffer. It is noted that in addition to cadmium and lead, germanium, arsenic, tellurium and selenite decrease life span in mice or rats or both, and selenite appears to inhibit growth in rats. In most of these cases, the tissue concentrations were within the range of normal human levels. In no case was the incidence of cancers increased.

Many trace metals have been found in the air. Some come from soil dusts, others from the burning of fossil fuels (especially coal), others from industrial processing of metals. One, lead, comes primarily from gasoline additive wastes in auto exhausts.

Most of the trace metals, in the concentrations found, are not necessarily toxic to human beings; nor is there any evidence that they can cause cancer or any other disease when ingested, even though some of them accumulate in human lungs with age.

Two elements, however, cadmium and lead, appear to be toxic in the concentrations or distributions now found, either because of inhalation or because of ingestion of contaminated water supplies or agricultural soils.

As lead additives in gasoline are not absolutely necessary, the solution to the increasing lead burden in the atmosphere, soils and water is to discontinue the use of tetraethyl lead and related compounds, and to substitute for it some more inert metal compound, such as nickel or tin, which will have a similar anti-knock property and permit octane levels comparable to those now achieved. It will be much more difficult for industrialized societies to avoid cadmium contamination, but a program of industrial safeguards against release of cadmium fumes into the open air should help prevent contamination of food and water and result in a significant decrease in accumulation of cadmium in human tissues.

There is a need for the continued study of the effects of trace metals on the living organism. Our sixteen years of research in this field has not touched upon many questions which need answers. Little is known about such trace substances as germanium, tellurium, selenite, arsenic and antimony in their airborne forms. Whether or not they accumulate in lungs, whether or not they are toxic to humans in these forms, and in what concentrations, remain the objects for further study. Knowledge gained in these areas may have important implications for the future treatment of several major chronic diseases.

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Table I

Some Metals in the Air of American Cities and in American Lungs

Metals	Percentage of samples containing the Metal.[3]	Percentage of lungs containing the metal.[7]	Accumulates* in lungs with age.[7]	Remarks
Aluminum	100	100	Yes	In soil
Barium	57	98	No	In soil
Bismuth	43	4	No	
Cadmium	79	58	Yes	Industrial contaminant
Chromium	63	100	Yes	Essential element
Cobalt	14	20	No	Essential element
Copper	94	100	No	Essential element
Gold	-	2	-	
Iron	91	100	Yes	Essential element
Lead	99	100	Yes	From gasoline waste
Manganese	75	100	No	Essential element
Molybdenum	12	17	No	Essential element
Nickel	91	58	No	
Silver	-	39	No	
Strontium	100	100	Yes	In soil
Tin	67	98	Yes	Industrial contaminant
Titanium	77	99	Yes	In soil
Vanadium	20	57	Yes	In soil
Zinc	83	100	-	Essential element

* In other respiratory organs, trachea and larynx, iron, lead, barium and strontium accumulate with age. Accumulation, where shown, was statistically significant at the 0.1% level of confidence, or less.

Table II

Trace Elements in Human Lung (Ash) by Geographical Location*

	AMERICAN		AFRICAN		NEAR EASTERN		FAR EASTERN	
Metals	Parts per million	Percentage of lungs containing	Parts per million	Percentage of lungs containing	Parts per million	Percentage of lungs containing	Parts per million	Percentage of lungs containing
Aluminum	1800	100	3100*	100	3100	100	2500	100
Barium	13	98	22	100	28†	100	15	100
Cadmium	50	58	N.D.†	0	50	23	50	49
Chromium	14	100	16	100	22	100	23	100
Lead	51	100	26†	98	47	100	48	99
Nickel	5	58	7	57	22	56	10	64
Strontium	8	100	10	100	12†	100	9	100
Tin	32	98	5†	59	16	85	29	90
Titanium	200	99	270	98	390	100	140	99
Vanadium	1	57	4	75	10†	79	2	70

† Value significantly different from American at the 0.1% level of confidence or less.

* Values given in parts per million (ppm) ashed lung. About 1.1% of the lung is ash, or minerals.

N.D. None discovered.

Table III

Innate toxicity of several elements in terms of growth and life span of rats and mice exposed to traces equivalent to the human experience

Element	Mice		Rats		Remarks
	Growth	Life Span	Growth	Life Span	
Titanium	+	0			Rats not tested
Vanadium	0	0	0	0	
Chromium	+	+	+	+	Diabetes with deficiency
Nickel	0	0	0		Rats in process
Germanium	0	-	0		Slightly toxic
Arsenic	0	-	0	0	Tumors depressed in mice
Zirconium	0	0	0	0	
Niobium	0	-	0	0	
Cadmium	0	-	+	-	High blood pressure
Tin	0	0	0	0	
Antimony	0	-	0	-	Toxic
Tellurium	0	-	0		
Lead	0	-	+	-	Toxic at all ages
Selenite	0	-	-	-	Toxic
Selenate	0		0		In process

0 = No effect
+ = Increased
- = Shortened or depressed

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Lead in Ancient and Modern Bones

PERUVIANS WHO LIVED some six centuries ago had much less lead in their bodies than does the modern American. The theory that a rise in lead in the contemporary environment is resulting in a rise in the lead content of the human body received some confirmation from a study just published in *The Journal of Bone and Joint Surgery*, March, 1968, by Robert O. Becker, Joseph A. Spadaro and Edward W. Berg of Syracuse, New York.

Becker and his colleagues used bones from burial grounds in the vincinity of Chancay, Peru. The bodies had been buried in dry sand, and the bones were exceptionally well preserved. Five Peruvian samples were compared with five modern samples.

All the Peruvian samples showed less than five parts per million of lead. Lead in the modern bones ranged from five parts per million to 110, with intermediate values of 16, 45, and 75. A lesser increase was detected in vanadium. In contrast, there was little difference between the ancient and modern bones in concentrations of zinc and copper.

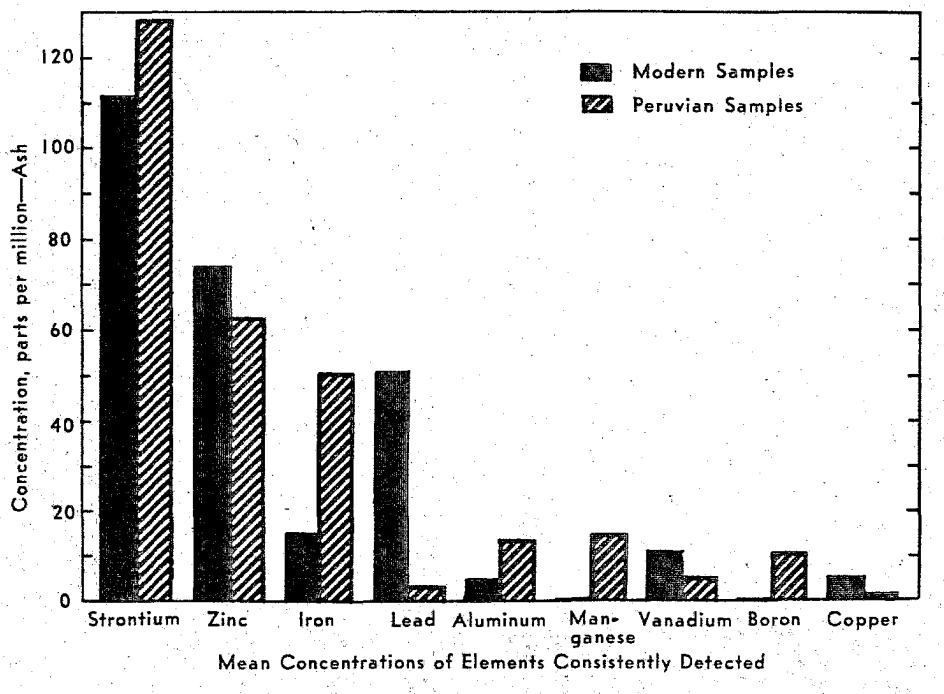
One of the modern samples had less than five parts per million of copper, the others had five; one of the ancient samples had less than five, the others less than three. Zinc concentrations ranged from 60 to 85 in the modern samples and from 25 to 98 in the ancient samples.

Some other elements appeared in higher concentrations in the Peruvian bones than in the modern bones, but this can be explained by the fact that these elements (strontium, iron, silicon, manganese and aluminum) were also found in high concentrations in the sand in which the Peruvian bodies were buried.

The chart below shows the mean concentration of the elements consistently detected in both groups. The elements are arranged in dscending ordr of abundance.

The authors conclude:

Increasing contamination of the modern Western environment with lead, derived particularly from automobile fumes, and the wide use of vanadium in metallic alloys may well be contributing factors to the increased concentration of these elements in modern bone. Although the modern dietary intake of lead is considerably higher than the respiratory intake, only about ten per cent of ingested lead is absorbed. Thus, the quantity of lead inhaled (most of which is absorbed) is a significant source of contamination in man. If we assume a dietary intake for the Peruvian Indians roughly similar to that of twentieth-century Americans, the ten-fold increase in the modern bone examined is a striking reflection of contemporary air pollution. To account for such high lead levels by ingestion alone, the modern dietary intake would have to be one hundred times that of the Peruvian Indians.



ELEMENTS ROUTINELY detected in all specimens of ancient Peruvian and modern bone, arranged in descending order of abundance. The mean concentration in parts per million is depicted. The rather close similarity in concentrations of copper and zinc in both the ancient and modern samples is evident. The remarkable increase in lead in modern bone is also evident. (Reprinted with permission, The Journal of Bone and Joint Surgery)

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