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LEAD POISONING IN URBAN CHILDREN

Final B Paper Submitted By

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TABLE OF CONTENTS

	page
I. INTRODUCTION.....	1
II. OVERVIEW OF THE PROBLEM.....	3
CAUSES OF CHILDHOOD LEAD POISONING.....	3
EFFECTS OF CHILDHOOD LEAD POISONING.....	18
DETECTION AND TREATMENT OF CHILDHOOD LEAD POISONING.....	20
III. APPROACHES TO THE PROBLEM.....	27
LEGISLATION.....	27
MODELS.....	32
CASE STUDIES.....	36
IV. RECOMMENDATIONS.....	42
V. CONCLUSION.....	48
APPENDIX	
FOOTNOTES	
BIBLIOGRAPHY	

LIST OF TABLES

- Table 1. Environmental Exposure of Young Children to Lead in New and Old Urban Housing
- Table 2. Respiratory Exposure to Lead Reflected in the Mean Blood-Lead Values of Various Groups
- Table 3. Lead Paint Regulations
- Table 4. 25 SMSAs With the Greatest Number of EBLs
- Table 5. Current Programs

LIST OF FIGURES

- Figure 1. Seasonal Distribution by Month of Report of Fatal and Nonfatal Cases of Lead Poisoning and Increased Lead Absorption
- Figure 2. Numbers of Cases for Each Diagnostic Classification, Grouped by Months of Age at Time of Diagnosis
- Figure 3. Atmospheric Concentrations of Lead From Vehicles from mid-1967 through mid-1969 along different sections of U.S. Highway 1 in New Jersey
- Figure 4. Predicted Nationwide Incidence of Lead Based Paint poisoning - 25 SMSAs with Greatest Predicted Incidence
- Figure 5. Census Tracts Ranked According to Risk
- Figure 6. City of New York - Reported Cases of Lead Poisoning
- Figure 7. Spot Map of 219 Cases of Lead Poisoning by Philadelphia Residences of Patients; 1956-1960
- Figure 8. Childhood Lead Poisoning Control Program Strategies
- Figure 9. Causes and Consequences of Childhood Lead Poisoning and Potential Sites for Intervention

INTRODUCTION

...cities have shown a unique and enduring propensity to create health problems and then elect to treat the symptoms rather than deal with the causes.

Alonzo S. Yerby, M.D.

One of the most serious urban health problems evident in our cities today, is that of lead poisoning. A health problem of many dimensions, lead poisoning originates from a variety of sources and affects a variety of victims. In children, at least, it is also wholly preventable.

The substance of concern - lead, is an element which occurs naturally in the environment as less than one per cent of the material in the earth's crust. Lead is defined as a pure metal and is used as such for the manufacture of plumbing pipes, as an additive to gasoline, pesticides, and paints, in the processing of many metallic and chemical products and in the making of pottery.¹

In using lead, man tends to overload the environment, particularly the urban environment. Tons of vaporized lead are emitted into the atmosphere daily by constant auto traffic. Many structures are painted internally and externally with lead-based paints. Pottery containing lead is found in many urban households, and various occupations utilize lead as a raw material. Consequently, lead can enter the human body in several ways - breathing polluted air, drinking contaminated water, eating lead-exposed foods or eating from high lead dishes, oc-

cupational exposure, and ingestion of lead-based paint.² Although in recent years, specific concern has been raised with regard to lead emitted by the use of alkyl antiknock compounds in gasoline, when the total environment is considered, two to three times as much lead is added from paint pigments and other lead products as from the use of lead alkyls.³

It can be seen then, that the problem of lead poisoning is indeed extensive, pervasive, and complex. This paper will deal primarily with the most prominent and serious manifestation of this important health hazard - lead poisoning in urban children, resulting from the ingestion of lead-based paint. The effects of airborne lead as a complicating factor will also be considered, but the basic thesis of this paper is the primacy of leaded paint as the cause of childhood lead poisoning in urban children.

In the first section, the causes, consequences, detection and treatment of childhood lead poisoning will be described.

Section II discusses legislative approaches to the problem,

presents some investigative modeling techniques and a brief examination of the experiences of several cities which have attempted to deal with childhood lead poisoning. Section III presents some specific recommendations, followed by Section IV, the concluding section.

OVERVIEW OF THE PROBLEM

CAUSES OF CHILDHOOD LEAD POISONING

Lead poisoning in children, resulting mostly from ingestion of chips of lead-containing paint from walls and woodwork in old, dilapidated housing, remains a unique public health problem. Its etiology, pathogenesis, pathophysiology, and epidemiology are known. Practical methods are available for screening, diagnosis, prevention, and treatment. Yet each year lead poisoning continues to cause the deaths of many children and mental retardation or other neurological handicaps in many other children.⁴

Lead poisoning is not uncommon in the United States. While the precise incidence of lead poisoning is not known, surveys have shown that 10 to 25 per cent of young children living in deteriorated urban slum housing show evidence of increased lead absorption, and 2 to 5 per cent show evidence of poisoning.⁵ Approximately 30 million housing units built before World War II are still in use today. As many as 7 million housing units are deteriorated and contain surfaces covered with lead paint, and it is estimated the 2,500,000 children live in substandard housing where a potential lead hazard exists.⁶ Taken together, these statistics yield a frightening potential.

Slum areas in the larger older cities seem to have the greatest incidence of lead poisoning, concentrated in their so-called "lead belts." The problem seems to arise from the interaction of two primary causative factors: race and the environment. The term race, can be defined as the habitual,

purposeful and compulsive search for and ingestion of nonfood items such as clay, plaster, laundry starch, ashes, nutty, string, paint chips, paper, dirt, crayons, yarn, matches, and cigarette butts. Adults as well as children may develop pica and both are usually selective in the materials they ingest. Studies have shown that nutritional deficiency is not an etiologic factor in urban slum children, and in fact 50 per cent of the children from both middle class and lower socioeconomic groups habitually engage in pica. Pica usually begins at one year of age and disappears between the ages of 3 and 5. Nearly 50 per cent of mothers of children with pica, have pica themselves, and the child with pica frequently stimulates the behavior in younger siblings. The interaction between child and mother is usually the critical factor in the development of pica in children. Significant interactive factors may include: an absent or poorly functioning mother, or an emotionally troubled mother. In such families, pica often goes unobserved by the mother, thereby precluding early detection in the child.⁷

The amount of lead that is received by the child through pica is also significant:

A chip of paint about the size of an adult's thumb nail can contain between 50 and 100 milligrams of lead, and so a child eating a few small chips a day easily ingests 100 or more times the tolerable adult intake of the metal! ⁸

Studies in selected slum areas reveal that 50 to 75 per cent of old houses contain dangerous quantities of flaking lead-based paints on interior surfaces.⁹ Dwellings in these areas may have several coats of paint on walls, woodwork, and

ceilings, and the base coats generally contain significant amounts of lead. Lead containing paint was frequently used for both interior and exterior dwelling surfaces until about 1940. Such housing is usually in bad repair and paint peelings and loosened plaster provide a hazardous source of lead for children with pica.

The usual locations of leaded paint chewed by children developing lead poisoning are window sills, painted plaster, and walls. Outside sources commonly include painted door frames, fences, porches, and house walls.¹⁰

A study in Cleveland clearly substantiates this relationship between childhood lead poisoning and old deteriorating urban housing. In this study, it was found that 27 per cent of 801 pre-school children residing in old housing had absorbed abnormal quantities of lead and 38 (4.7 per cent) had symptoms of lead poisoning. One hundred and five comparable pre-schoolers living in better housing were also tested and of these, only 3 showed evidence of increased lead ingestion and none showed clinical symptoms of poisoning.¹¹ (See Table 1.) Similar studies have been conducted in other cities.

Table 1. Environmental Exposure of Young Children to Lead in New and Old Urban Housing

Location of Home	No. of Children Studied	No. with Abnormal Urine	No. with Plumbism (Lead Poisoning)
Old Housing	801	216 (27.4)	38(4.7%)
New Housing Project	105	3	0

Source: Chisolm and Farlan, p. 944

In Baltimore, 90 per cent of the reported cases of lead poisoning are in children who reside in multiple-dwelling, rented housing units. Surveys in Baltimore, as well as in Philadelphia and London, have revealed that 70 to 80 per cent of interior painted surfaces contain more than 1 per cent lead. Extrapolation of a limited survey in Boston, indicated a 98 per cent probability of houses with positive findings for lead if a greater number of samples had been analyzed. (Investigating actual cases usually requires analysis of 20 to 25 different samples in order to identify all interior surfaces positive for lead.) Geographic spot maps have been kept by the Baltimore City Health Department during the last 30 years and indicate that as old inner-city dwellings are replaced by urban renewal and other new construction, the location of reported cases of lead poisoning moves outward from the center of the city and that cases are now found in older housing in nearly all parts of the city.¹²

As stated earlier, lead poisoning in children is believed to be due almost entirely to the repetitive eating of leaded house paint. Most of the available data are based on retrospective analyses of small groups of cases from cities in which childhood lead poisoning is a reportable disease and in which the municipal health departments have active programs such as New York City, Chicago, Philadelphia, and Baltimore. While the nationwide incidence of the disease in children is unknown, the following nine epidemiological factors are well-established:

1. Areas of "high risk" are primarily centered in the slums as accessibility to flaking paint and broken plaster, high incidence of vice, and lack of adequate parental supervision provide an optimum environment for lead poisoning.

2. While children between 1 and 6 years of age are the main victims, 85 per cent of the cases are children from 1 to 3 years old; more than 50 per cent of the deaths caused by lead poisoning occur in 2 year olds.

3. Childhood lead poisoning is significantly related to pica, as 70 to 90 per cent of lead-poisoned children exhibit a history of this habit.

4. Symptomatic lead poisoning in children varies by season and there is evidence that 80 to 85 per cent of the cases occur in the summer months of June through September. However, more cases are being reported in the winter months as awareness of the problem among health workers increases. (Some winter cases occur when leaded battery cases are burned for fuel and the fumes inhaled, or there is prolonged contact with the ashes.) While lead encephalopathy (clinical lead poisoning or brain injury) is more frequent in the summer, asymptomatic (sub-clinical) lead poisoning is a year-round disease.

5. Negroes and Puerto Ricans exhibit a high incidence apparently because a greater proportion live in "lead belts" and not because of any known genetic, ethnic, or racial factors.

6. There is no significant difference in incidence by sex.

7. A high incidence occurs among siblings with a 30 per cent incidence rate cited by some investigators.

8. There is a high recurrence rate.

9. Lead poisoning associated with pica is a chronic process and from 3 to 6 months of fairly steady ingestion is required in most cases before clinical symptoms appear. 13

(See Figures 1 and 2.)

Estimates of the incidence of lead poisoning and the presence of adverse blood level concentrations are made on shaky foundations. As stated previously, the disease is only reportable in a few of the larger cities. Usually, recognition is based on the case-finding approach, which is dependent on the level of local medical community awareness and the availability of diagnostic laboratory facilities. Prospective mass screening programs are currently available only in Chicago (since October 1966) and in New York City (since January 1970.)¹⁴ Although

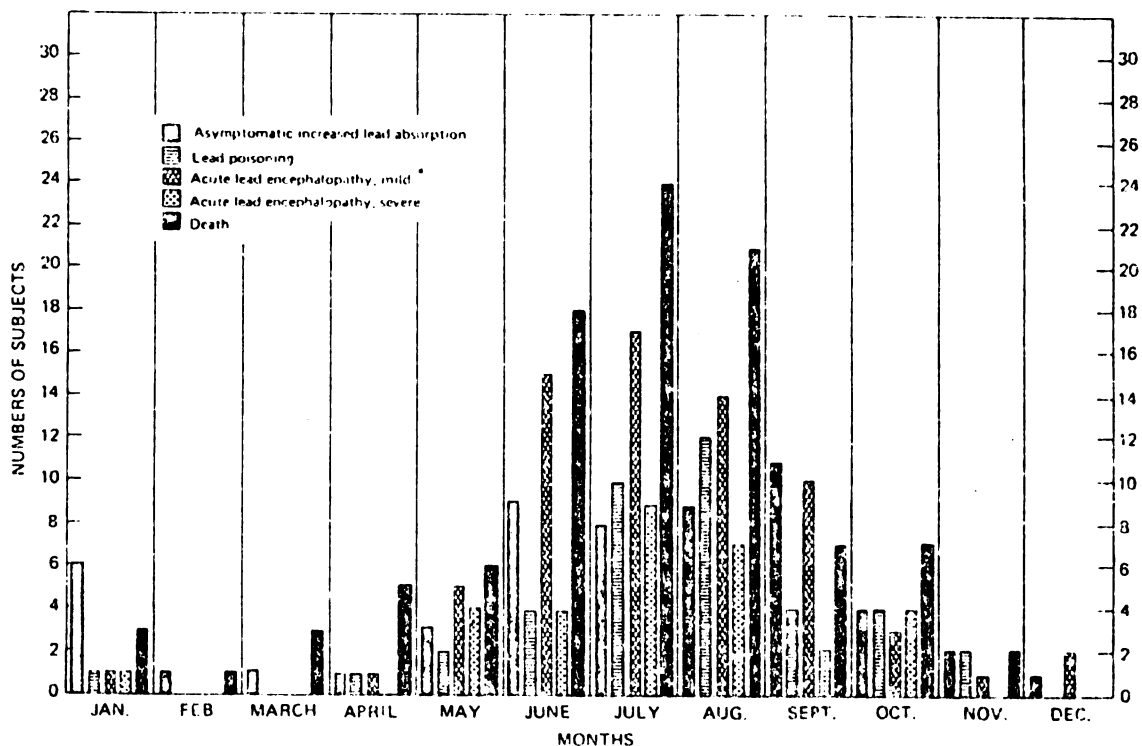


Figure 1. Seasonal Distribution by Month of Report of Fatal and Nonfatal Cases of Lead Poisoning and Increased Lead Absorption.
Source: Committee on Biologic Effects of Atmospheric Pollutants, p. 134

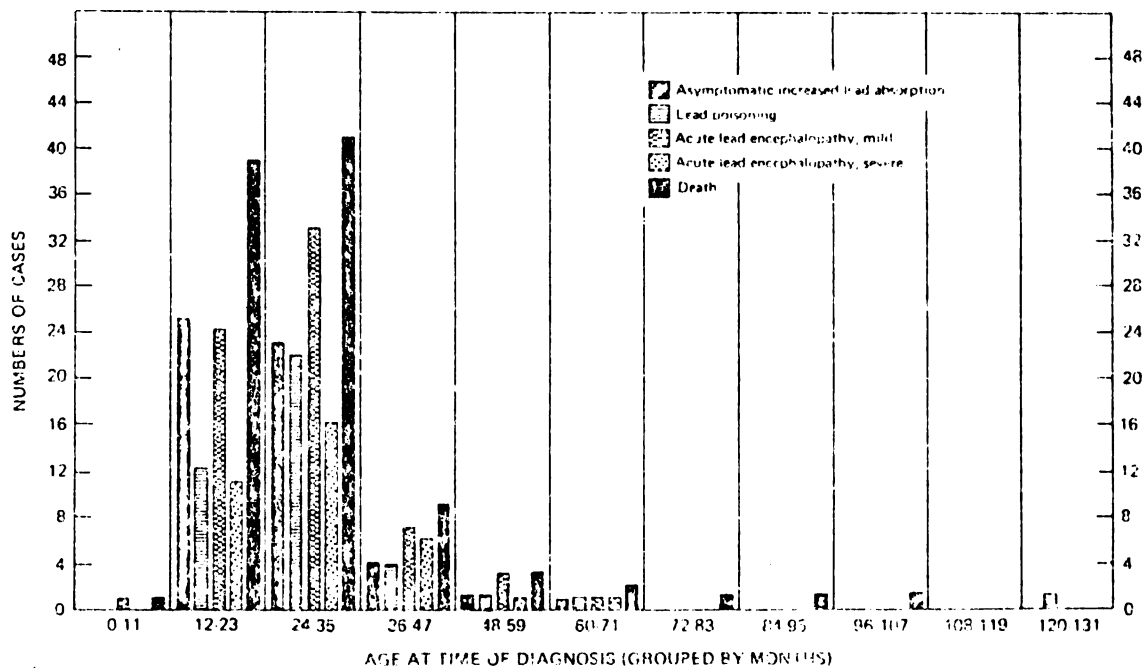


Figure 2. Numbers of Cases for Each Diagnostic Classification, Grouped by Months of Age at Time of Diagnosis.
Source: Committee on Biologic Effects of Atmospheric Pollutants, p. 136

adequate epidemiological data on the distribution of blood lead concentrations in children are largely unavailable, large-scale screening programs in urban areas suggest:

...perhaps 5 to 10 per cent of young children who live in deteriorating old housing have concentrations of lead in their blood that may be associated with adverse metabolic effects, at the very least, and 1 to 2 per cent may have evidence of lead poisoning. Where active screening programs are in operation, the numbers of reported cases increase, while the number of reported deaths decrease to well under 1 per cent of reported cases.¹⁵

In addition to lead-based paint itself as a causative factor, several other factors aggravate and magnify the problem. One such factor is the lack of awareness about the problem among physicians and other health workers. Many physicians are unaware of the existence or the magnitude of childhood lead poisoning, because they seldom encounter cases of it, or because the cases they do encounter are incorrectly diagnosed. Another common misconception is that the manufacture of lead-free paint today has resulted in the extinction of lead poisoning in children, without regard to the fact that old houses often still contain many layers of lead-based paint, paints manufactured for outdoor use still contain lead, and such outdoor paints may be used for interior surfaces by uninformed people. Those physicians and health workers who are aware of the problem of lead poisoning, are often hesitant to make a positive diagnosis, resulting in undue delays in treatment and the loss of valuable time.¹⁶

A poorly informed public is the second complicating factor, as many parents are unaware of the danger associated with pica, or the consequences of paint ingestion. Additionally,

inadequate prevention of reexposure to lead results in a high rate of recurrence of lead poisoning among children. The failure to prevent reexposure to lead contributes greatly to the mortality and morbidity of lead poisoning.¹⁷

In addition to poor housing conditions and the lack of awareness on the part of professionals and the public, inadequate health and housing codes also contribute to the persistence of lead poisoning. In those cities with codes specifically prohibiting lead paint in the interior of dwellings, enforcement of such codes is usually unsatisfactory for reasons such as expense to landlords, the city's fear of abandonment by landlords, a shortage of personnel to carry out the necessary enforcement procedures, and difficulties and confusion in legal procedures and concepts. As Jin-Fu states:

Failure to get rid of lead paint in a house where a child is known to have developed lead poisoning usually means that a treated child returns to the same hazardous environment to be exposed to another episode of poisoning.

Even if the afflicted child is moved to another house, the problem still remains:

If the lead paint in the house is not removed, the lethal heritage will soon pass on to other families with children, and lead poisoning among children multiplies.¹⁸

Although the slum areas of large urban centers exhibit the highest incidence of childhood lead poisoning, the problem is not restricted to poor slum dwellers, as cases of lead poisoning have been reported in children from socially and economically advantaged homes. Urban migration patterns from central core cities to the suburbs have correlated well with an

increase in cases of lead poisoning in previously unreported areas, as older housing is subdivided into apartments. In Baltimore, 48 per cent, and in Philadelphia, 50 per cent of the reported cases of lead poisoning among children occurred outside the inner cities.¹⁹

Rural areas and small towns are not immune to the problem either:

Of 230 rural children 1 to 5 years old, tested in New York's Dutchess County and Connecticut's Litchfield County, 9 per cent had blood levels considered unduly high. Some of these children were from unner- and middle-income families. 20

In a recent study of 6,000 children in 14 Illinois cities with populations ranging from 10,000 to 150,000 persons, 18.6 per cent carried high levels of lead in their blood. (300 micrograms is the daily permissible intake from food and other sources.)²¹

Still another previously unrecognized potential lead poisoning-causing situation, is that associated with the remodeling of old homes. Workmen as well as children and their parents can get lead poisoning simply by breathing in small particles of lead during the process of remodeling and renovating inner city houses with lead-based paints on interior surfaces. In a study by Wolf, of several cases in the Capitol Hill area of Washington D.C., the residue of the heavy sanding and scraping in houses undergoing remodeling was tested. It was found to have a high lead content. Wolf does not suggest that people stop remodeling old houses, but does recommend that certain precautions be taken to avoid lead poisoning including: wearing face masks, using a wet mop after cleaning up

debris, not eating in the same room where work is going on and insuring that plates and utensils are not exposed to lead dust, and having body lead levels checked periodically.²²

From the point of view of contamination of the total environment with lead, lead-using industries, paint pigments, and metallic products have each contributed more than burned leaded fuel. Thus any proposal to rid the environment of lead pollution through the removal of lead from automotive fuels, must take into consideration the existence of other lead products such as paints and manufactured items.

The general picture that emerges from consideration of lead in the environment is of a steep gradient of pollution emanating from the cities in proportion to their size. The absence of significant upward trends in the concentration of lead in rural soils and water and in the food sources of man in the last 30 years indicates that the great increase in the combustion of lead alkyls has not had a large impact on the intake of lead by nonurban people and animals, except perhaps by direct inhalation.²³

The magnitude of transfer of airborne lead to the soil is directly related to the density of auto traffic. In urban areas, the surface soil of parks, street dust, and narrow bands beside major roadways are heavily contaminated with lead.

Only in the urban setting is man possibly exposed to hazardous circumstances relative to atmospheric lead pollution, occupational exposures in the lead-using industries excepted. The high concentration of lead in urban air and on the surfaces of parks and streets constitute a source of intake additional to the usual dietary sources and in special circumstances may be a substantial source.²⁴

(See Figure 3.)

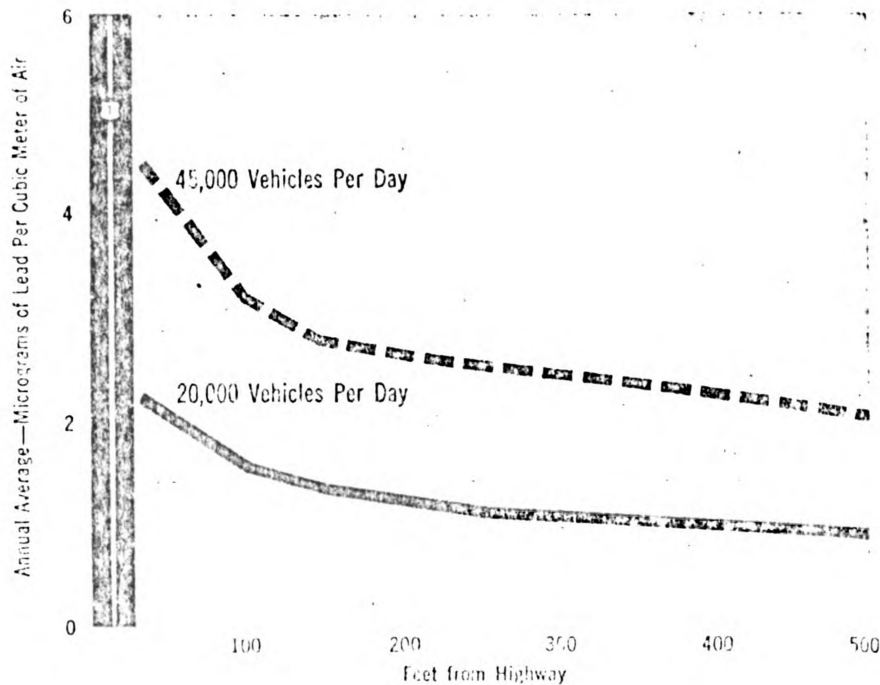


Figure 3. Atmospheric Concentrations of Lead from Vehicles from mid-1967 through mid-1969 along different sections of U.S. Highway 1 in New Jersey.

Source: Craig and Berlin p. 6

While the general urban population faces no identifiable current threat from the concentration of lead in the air, two groups of people are exposed to airborne lead to what seems to be an undesirable degree. The first group is those persons who are exposed to unusually high concentrations of lead in ambient air for occupational reasons - garage workers, traffic police, and lead trades workers. Such exposure can produce blood lead concentrations in excess of 40 μ g. (μ g=micrograms and refers to micrograms per 100 milliliters of whole blood.) However, this level of blood lead concentration is probably only attained by a relatively small proportion of those exposed.²⁵ The other group significantly threatened by lead in ambient air consists of infants and small children. Recent surveys of chil-

dren in large cities indicate that many had blood lead concentrations in the ranges of 40 to 60 μ g. While such high blood lead concentrations cannot be definitely said to arise from the inhalation of lead, it is likely that at least some of the lead burden in children does come from the ingestion of lead-bearing street dust and soil, which frequently reaches lead concentrations in excess of 2000 μ g/g.²⁶ (See Table 2.)

Table 2. Respiratory Exposure to Lead Reflected in the Mean Blood-Lead Values of Various Groups

POPULATION	EXPOSURE (MICROGRAMS PER CUBIC METER OF AIR)	MEAN BLOOD LEAD (MICRO- GRAMS PER 100 GRAMS)
rural U.S.	0.5	16
urban U.S.	1.0	21
downtown Philadelphia	2.4	24
Cincinnati policemen	2.1	25
Cincinnati traffic policemen	3.8	30
Los Angeles traffic policemen	5.2	21
Boston Automobile Tunnel employees	6.3	30

Source: Chisolm, p. 21

One study which attempted to determine the extent to which the ingestion of dirt and dust contaminated with lead exhaust from cars, contributes to the childhood lead problem was conducted by Haer and Aronow. While most lead in dirt appears to be a result of deteriorating lead-based house paint, Haer and Aronow utilized a tracer process to determine the amount of lead a child might receive from eating dust contaminated with lead sources other than paint chips (primarily leaded gasoline.) The tracer, Lead-210, is naturally occurring and is present in

relatively large amounts in dust and particulates, but nearly absent in paint. The results of the study showed that children with pica and other evidence of high lead intake, and normal children both excreted identical amounts of Lead-210. Thus, this study concluded that dust and air-suspended particulates were not the sources of lead in these urban children.²⁷

Another study, however, concluded that street dust was in fact a potential source of ingested lead, particularly for children. In this study, monthly dustfall samples were collected in 77 midwestern cities in 1966. The mean lead concentration in dust from various sectors in each city was calculated after averaging the results for all of the cities in this way:

residential.....	1636	4g/g of dust
commercial.....	2413	4g/g of dust
industrial.....	1512	4g/g of dust

It was determined from this study that "the swallowing of as little as 14g of such dust could result in the oral intake of an amount of lead that exceeds by a factor of 10 or more the estimated mean daily intake of lead from normal food and drink in non-exposed children."²⁸

In another study, conducted in 1971, dentine lead levels were measured from the shed deciduous teeth of children in two Philadelphia school districts. One district, District 5, was considered "high risk" as it was within the acknowledged "lead belt" of the city. The second district, District 8, was considered low risk. District 5 was characterized by a predominantly black population, which while mobile, tended to stay within the district. The houses were older than 40 years and many were in a severe state of deterioration. The western half of district 5 is the acknowledged "lead belt" of the city, from which many

cases of lead poisoning are reported. The eastern portion of the district is highly industrialized with a predominantly white population. Housing is old, but in generally good repair. Diagnosed lead poisoning is rare from this area.

District 8 is the area into which Philadelphia expanded after World War II. Houses are newer and in good repair. The population is predominantly white. Diagnosed lead poisoning is extremely rare in this district.

In all, 151 children with no **p**rior history of lead poisoning were tested for dentine lead levels. The results of the study showed that black children in public schools from areas of deteriorated housing, had marked elevations of dentine lead, with 20 per cent of the children having levels in the range associated with toxicity. In general, the white children in the newer housing had the lowest levels, but as an unexpected result, a group of white children from intact housing, living near and attending school adjacent to a major lead processor also had elevated dentine lead. Because of this proximity to a major lead processor and also because of extremely heavy auto traffic in the area, the authors concluded that for these children, airborne lead was an important vehicle of exposure. In their discussion, the authors state that their study demonstrates that exposure to lead poisoning is more widespread than had been reported, and also suggests sources of lead other than paint. Furthermore, populations other than those traditionally acknowledged are being exposed to biologically important amounts of lead.

The finding of elevated lead levels in urban
black children living in deteriorated housing

and in white children whose housing is generally in good repair, suggests that both paint and airborne lead are operative factors, and that children in deteriorated housing who live in areas of heavy vehicular traffic flow are in fact being exposed to both sources.²⁹

While it has been shown that contaminated street dust alone does not account for clinical lead poisoning in children, the swallowing of such dust may significantly account for the higher mean blood lead content in urban children and the relatively large fraction having a blood lead content of 40 to 60 μ g. Combined with mica for paint, an increased intake of lead from contaminated dust would result in a total lead intake sufficient to cause symptomatic illness.³⁰ However, the direct ingestion of lead-pigment paints is unmistakably the principal environmental source in cases of severe acute lead poisoning in children.³¹

Other studies have attempted to determine whether persons (not necessarily children) in urban communities show an increased absorption of lead as compared to non-urban communities. In 1961, Hofreuter studied the blood lead concentrations of groups of people from 6 American cities and compared them with those of a rural population. Taking a number of factors into consideration such as sex, and smoking habits, Hofreuter and his colleagues were able to show that for each sub-group, urban lead concentrations were in excess of rural values. In 1965, Musbaum attempted to determine whether body burden of lead correlated with the length of time spent in a city (Los Angeles) by studying bone specimens. He found no correlation, however, between bone lead concentrations and length of stay in the area.³²

Another study conducted in 1968-71, determined the concen-

tration of lead in the ambient atmosphere at 59 sampling sites in 8 American communities. (This study was an expansion of and a follow-up to a similar study carried out in 1962, entitled "Survey of Lead in the Atmosphere of Three Cities.") The purpose of the study was to examine the extent to which the blood lead levels of selected population groups reflect exposure to lead at various levels in community atmosphere. The study was conducted in 8 regions reflecting various geographical and climatological characteristics and included the communities of: Cincinnati, Philadelphia, Los Angeles, New York City, metropolitan Washington D.C., Chicago, Houston, and Los Alamos (N.M.). In order to insure as specific and consistent a relationship to known air levels of lead as possible, the populations studied were primarily women volunteers living within a prescribed region surrounding an air sampling instrument. The study was concerned exclusively with absorption of ambient community atmospheric lead through the respiratory system and did not involve pediatric exposures or occupationally-associated exposures of men. The study indicated that while urban levels of blood lead are higher than suburban levels, air concentrations of lead are not clearly reflected in the blood lead levels on a general national basis. Thus, there are factors other than atmospheric lead level which are of relatively greater importance in determining the blood lead levels in population groups. The precise nature of these other variables appeared to differ among the regions studied and was not defined. Between regions, lead from food intake is believed to be significant and climate may also be important, but in urban-suburban comparisons, airborne lead levels seem to be the major determinant. This study did not attempt

to examine the relationship between age and blood lead level.³³

The association of paint with childhood lead poisoning was first postulated by Riddock in 1924.³⁴ Despite the variety of evidence and often conflicting data, it is probably safe to conclude that ingestion of lead-based paint is still the chief source of lead poisoning in children, with inhalation of airborne source aggravating existing symptoms, but probably not initiating them in the absence of lead-paint paint.

EFFECTS OF CHILDHOOD LEAD POISONING

It has been known for over 100 years that lead is an abortifacient and that female lead workers had a high rate of miscarriage. Paternal factors have also been known to be significant for many years. It appears that a man suffering from lead poisoning may pass abnormal sperms to a woman, resulting in children born with lethal malformations. Even those children who appear to be normal, may carry an elevated concentration of lead from birth, and thus may be more susceptible to further contamination from any source.³⁵

Children who are born normal and later develop lead poisoning, are likely to be left with permanent physiological and mental damage. Lead poisoning is cumulative. Symptoms begin to appear some weeks or months following the continued ingestion of small amounts of lead. Early symptoms may include irritability, fretfulness, or disturbed gastrointestinal function, characterized by lack of appetite, constipation, vomiting or cramps. More severe intoxication results in lead encephalitis (brain injury) due to intracranial pressure. The acute stages of the

disease are manifested in changes in mental state, ataxia (irregularities in the functions of the body), persistent vomiting, muscle weakness or paralysis, delirium, stupor, coma, convulsions, and frequently death. Lead poisoning is further known to have an inhibiting effect on red blood cell development and may cause damage to kidneys and liver. The disease in children differs considerably from that in adults. Adults rarely develop central nervous system complications or encephalopathy, while peripheral neuritis, lead line of the gums, and colic, common symptoms in adult cases, are rare in children.³⁶

While increased efficiency of treatment has decreased the mortality rate associated with lead poisoning, many victims of the disease suffer some form of intellectual or behavioral sequelae (complications.) The specific sequelae seem to be determined by length of exposure, speed of diagnosis, and the method of treatment. In cases of reexposure, the probability of permanent brain damage is 100 per cent.

The severity of intellectual sequelae appears to be closely related to whether or not encephalopathy is present. If it is present, the probability of sequelae is at least 40 per cent. The specific capabilities usually affected are perception, form discrimination, and language skills. Determination and diagnosis of emotional and behavioral sequelae is more difficult and subjective than the intellectual complications just described as "the effects of the syndrome are hopelessly confounded with the effects of environmental deprivation, which often characterizes the lives of children in the socio-economic area which produces the largest incidence of lead poisoning."³⁷ Children with lead poisoning usually exhibit hyperactivity, are easily distrac-

ted and emotionally labile. They also tend to be aggressive and have difficulty in establishing "normal" social relationships.

There appears to be a close relationship between severity of intoxication and emotional sequelae. Also, the type of treatment apparently does not have a strong relation to emotional sequelae, while the ability of the parents to provide support does.³⁸

According to a recent Public Health Service Report (1971) approximately 400,000 American children have elevated blood levels and some 10,000 require treatment for lead poisoning. Annually, 200 children die and 800 are so severely injured as to require permanent care. Thirty-two hundred suffer moderate to severe brain damage, requiring substantial specialized care.³⁹ Thus, with sufficient evidence to indicate that lead can produce disturbances in body and brain metabolism, and cause lasting mental impairment, it is essential that every effort be made to reduce the body lead burden of children to as low a level as possible.

DETECTION AND TREATMENT OF CHILDHOOD LEAD POISONING

Evidence of lead poisoning is best obtained through community screening. Five essential factors must be considered in planning such a screening program:

1. The children at risk - which children, by present knowledge should be tested?

As leaded paint best identifies the children at risk, screening should center on those areas where there are older homes in poor repair, having surfaces painted with leaded paint.

2. Access to the children - what organizations can most readily reach these children?

Parents are the key to reaching children at risk, as educated

parents can exert effective pressure to obtain relevant tests for their children. Although several facilities may combine to pool their outreach efforts, screening of all children at risk may not be feasible, and thus it is necessary to pinpoint target areas. One way of determining such target areas is to assure that children already identified as lead poisoned define high risk environments. In some cities, this may be a single localized area, while in other cities, cases may occur over a large part of the city. In the latter situation, the indicator case can be used to determine "clusters" to be screened. Such cluster testing could be done by the health department upon notification from a laboratory testing for elevated blood levels, and would involve visiting the indicator child's residence to test other household members between 1 and 6 years old, and neighborhood children (where housing conditions warrant it.)

3. The technology of testing - what technology is available for doing the screening test on children?

The technique of obtaining specimens has been simplified by the substitution of finger sticks for venipunctures, allowing more people to be trained to take specimens. The success of any screening program is dependent on the accuracy and validity of the laboratory data, as both patient and environmental follow-up rely on laboratory results. Because of the difficulty and expense of creating and maintaining a high quality lab, all but the largest programs generally contract for the work.

4. Follow-up - what are the mechanisms for medical follow-up?

The blood levels at which various actions are taken are somewhat arbitrary and vary from program to program. In Newark, N. J. a blood lead level under 40 μ g, requires only a repeat

test within a year if the child still seems at risk. With a blood lead level between 40 and 59 μg , the child is considered to have evidence of increased absorption and will be tested at 1 to 3 month intervals until the extent of the danger is clarified. If the child exhibits a blood lead level of 60 μg or more, hospitalization and treatment are required. Those children who require re-testing and re-examination must be under a physician's care, although his services may be augmented with the help of para-professionals. Screening programs must be able to anticipate the needs for various services, and the families' ways of paying for outpatient and hospital care should also be considered in the planning process. Additional factors to be dealt with, especially in urban areas, are the problem of missed appointments and the fact that children may be taken to more than one facility for health care.

Each facility, while only incidentally aware of other services to individual patients, tends to assume patients lost to follow-up are receiving care elsewhere. Difficulties are further increased by the mobility of the urban population. In a Newark lead screening survey, 40 per cent of the families had been less than two years at their present address. For lead poisoning, some common registry is essential. In Newark, this function is performed by the Central Lead Registry.⁴⁰

5. Environmental control to eliminate the source of lead poisoning is expensive - what options are there for environmental control and who will set priorities for their application on an ongoing basis?

Something must be done to the walls of the house in which lead poisoning has occurred, and although a variety of methods exist, they all present problems. Wall coverings in houses with lead paints usually hold for only a few months due to ^{the} weak infrastructure of such houses. Stiffer covering such as beaver board works well but is more expensive. Paint may be flamed

off but this involves substantial hazards, and removing paint mechanically requires intense and costly labor. While relocation of the child after treatment is a temporary solution, it offers no protection to new young occupants.

Innovative funding mechanisms, such as arrangements for revolving loans and tax incentives, need exploration. A prerequisite for a forceful program of environmental control is appropriate legislation and regulations to fix and enforce responsibility for rehabilitation (i.e. removal of the surface or making it inaccessible.)⁴¹

Screening is most effectively accomplished through a cooperative effort among existing organizations and agencies, and by building upon the health service structures already in operation within a community. "Centralization and management of the program, will almost certainly require a special office to provide communication, leadership, and access to the facilities needed for the combined efforts toward the common goal of eliminating childhood lead poisoning."⁴²

The important steps in diagnosing lead poisoning as outlined by the U.S. Department of Health, Education, and Welfare (HEW) in a booklet entitled, "The Recognition of Lead Poisoning in the Child" are:

1. Awareness that vice may be associated with lead poisoning.
2. Presence of symptoms such as lack of appetite, listlessness, increased irritability, vague abdominal pain or cramps, constipation, or vomiting.
3. Convulsions or unexplained unconsciousness.
4. Special laboratory tests are indicated when suspicion is present and include tests for the presence of an iron deficiency anemia, abnormal blue spots or "stippling" in the red blood cells, abnormal density of the ends of the bones as seen by x-ray, and special tests for abnormal concentrations of lead in blood and urine.⁴³

The identification of a child with lead poisoning is just the beginning of several preventive and therapeutic measures. First, and most importantly, the child and the source of lead must be separated, initially by hospitalization, if the child is toxic. While in-patient treatment regimens vary, all are based on chelation - the use of chemicals which combine with lead and thus facilitate its urinary excretion. In milder cases, the chemical edathamil calcium disodium (CaEDTA) is used, while in more severe cases the agent is British Anti-Lewisite (BAL). While these drugs are life-saving, they are also toxic.⁴⁴

Long-term care is essential and is the most difficult and most important aspect of treatment. Long-term care is dependent upon the precept that no child ever returns to a leaded house.

This aspect of therapy requires the coordinated efforts of public health authorities to effect the removal of hazardous lead sources, assistance for the mother in her quest for safe housing, and increasingly, mobilization of the community itself. Adequate care often requires brief hospitalization in a convalescent facility or foster home. Thereafter, the child must be followed closely until he reaches school age. Where possible, enrollment in nursery school or Head Start programs is advisable to provide stimulation for the child, which may for the pre-school child, reduce his emotional needs for *pica*.⁴⁵

Because of the many medical and environmental factors involved, it is difficult to make a comprehensive estimate of total medical and related expenses attributable to lead poisoning. Generally speaking, the type of exposure, the age of the patient, and the severity, recurrence, and sequelae of the illness are the important variables influencing total direct medical costs in cases of lead poisoning.

Separation from exposure is the essential component

of lead poisoning therapy and thus hospitalization is frequently indicated. Obviously, costs for treatment fall most heavily and directly on the poor, who are the least able to pay. Treatment costs can be divided into six general categories:

1. direct medical costs for acute and convalescent care
2. after-care and excess school costs for the partially brain-damaged
3. custodial care for the permanently and severely injured
4. correction of hazards in housing
5. preventive health supervision
6. supporting municipal and state health department activities

It is difficult to generalize about the economic impact of childhood lead poisoning as diagnostic and treatment facilities vary widely among communities and few of these have programs which can be considered either comprehensive or wholly adequate.

In Baltimore, children are treated under the general medical policy that no child found to have increased lead absorption, with or without symptoms, is returned to a "loaded" home. The child is first treated in a general hospital for a brief period and then placed in a convalescent facility until a safe dwelling (modern public housing or adequately repaired old housing) is found for the family. In a group of 45 Baltimore children treated from 1965-1970, the average total time of acute and convalescent hospitalization was 100 days and the average direct hospitalization cost for 34 patients was \$2746. The two highest hospital bills were over \$6000 each - in one case a result of repeated hospitalization for complications of encephalopathy and in the other case, from an excessively long wait for admission to public housing. Asymptomatic

children can be treated at lower cost and on an out-patient basis in convalescent facilities.⁴⁶

The total direct medical cost of lead poisoning can be roughly estimated according to the final clinical outcome, as follows:

1. asymptomatic increased lead absorption without obvious residual permanent injury: \$1500-2000 per patient
2. moderate permanent brain damage (special schooling required): \$18,000 per patient
3. severe permanent brain damage (institutional care required): \$245,000 per patient (1972 medical costs)

These direct treatment costs can be contrasted with the cost of repairs to substandard housing to eliminate the paint hazard.

In Baltimore, these costs range from \$150-1200 per apartment, while New York City estimates costs at \$1263 per apartment.⁴⁷

Adequate housing repair is therefore comparable in dollars with the direct medical costs of treatment of a single asymptomatic child with increased lead burden. Also repairs to substandard housing can prevent lead poisoning in all children who may live in a given house during the remainder of the house's useful existence. If houses were inspected and repaired before the onset of pica, direct medical costs could be totally eliminated.⁴⁸

APPROACHES TO THE PROBLEM

LEGISLATION

On January 13, 1971, President Nixon signed the Lead-Based Paint Poisoning Prevention Act (P.L. 91-965). It was the first federal law designed to reduce the hazard of lead poisoning for the nation's children. Essentially the Act provided fiscal and legal support for the following:

Title I - Grants up to 75 per cent of total cost for local programs, for detection and treatment of lead-based paint poisoning, including community testing and follow-up and educational programs outlining health dangers.

Title II - Grants in conjunction with those under Title I for the elimination in residential housing of lead-based paint on interior and other surfaces to which children may be commonly exposed. (Both titles call for employing residents of communities or neighborhoods to carry out purposes of the Act.)

Title III - A federal research and demonstration program to determine the extent of lead poisoning and to find more effective ways of controlling lead paint risk.

Title IV - Authority to prohibit future use of lead-based paint in residential structures either constructed or rehabilitated after the effective date of the Act.⁴⁹

(See Appendix)

The Act authorized appropriations of \$10,000,000 in 1971 and \$20,000,000 in 1972 to carry out its provisions. Amendments to the Act were made in 1972 and 1973, with the latter group incorporating most of the elements of the earlier, namely:

1. an increase in federal funding from 75 per cent to 90 per cent of the cost of local programs
2. an expansion of the definition of "lead-based paint" from 1 per cent lead content to 0.06 per cent

3. exclusion of the lead-based paint poisoning problem from the scope of the Public Health Service Act

4. the creation of a National Childhood Lead Based Paint Poisoning Board to advise HEW

5. prohibition of lead-based paint in residential structures receiving federal assistance, and on any toys, furniture, cooking, drinking, and eating utensils.

The amended Act appropriates \$63,000,000 a year for two years of which \$25,000,000 is for detection and treatment of lead poisoning, \$35,000,000 is for elimination of the poisoning and \$3,000,000 is for research.⁵⁰ Under this Act, HEW has provided funds for 40 cities to start or expand efforts to prevent lead-based paint poisoning. In addition to these 40, approximately a dozen other cities have started programs either in the Health Department or the Housing Inspector's Office, or both, with many other cities indicating their desire to start a lead poisoning program as soon as enough public and official interest to allocate funds is generated. In addition to funding, technical help is also available through a National Clearinghouse on Lead Paint Poisoning Prevention established by HEW in the Health Services and Mental Health Administration in Rockville, Maryland. The federal government is also doing research to improve and simplify screening, testing, and treatment methods.⁵¹

In addition to federal legislation, other kinds of local solutions to the problem of lead poisoning need to be considered such as housing code regulation by local governments and landlord-tenant innovations. Several cities have passed ordinances dealing with lead paint poisoning, placing it in the broad context of governmental code enforcement. (See Table 3.)

Table 3. Lead Paint Regulations

Area	Health or Housing	Label Paint for Sale	Set On Interior Surfaces	Can Force Removal	No Peeling Paint	Source
Baltimore	Housing	yes	yes	yes	yes	Baltimore Lead Paint Labeling Ordinance #1501, 6/22/58, Housing Code, Ordinance #322, 1951
Boston	Health		yes	yes		
Chicago	Housing	yes	yes	yes	yes	Municipal Code of Chicago, Section 78-17.2
Cincinnati	Health	yes	yes	yes		Cincinnati Lead Ordinance, 1960
Connecticut	Health, Housing	yes	yes ("accessible surfaces")	yes	yes	Public Act 194 - An Act to Enforce the Elimination of Lead Based Paint in Housing Accommodations, 1971
Jersey City	Health	yes	yes	yes		City Ordinance #C-36 - An Ordinance Regulating the Sale of Lead Paint in the City of Jersey City, 1962
Massachusetts	Health	yes	yes + exterior	yes		Chapter 1081, An Act Providing for a Comprehensive Program of Lead Poisoning Prevention and Control 1-1971
Newark	Health	yes	yes	yes		Ordinance Numbers 80102170, 80102170, 80102170, and 80102170 of the City of Newark, N.J., Nov. 1970.
New Haven	Health, Housing	yes	yes + (accessible exterior)	yes		Amendment of Housing Code Relative to Lead Paint on Dwelling Units 1968 - Lead Paint Ordinance - 1968
New Orleans	Health	yes	yes + (accessible exterior)	yes		Ordinance 828 amended 1971
New York State	Health	yes	yes + (accessible exterior)	yes		Official Compilations of Codes, Rules and Regulations of the State of New York (Health) 1970. An Act to Amend the Public Health Law in Relation to the Prevention and Control of Lead Poisoning
New York City	Health	yes	yes	yes		New York Administrative Code - Lead Paint Regulations 1970
Norfolk	Housing				yes	
Rhode Island	Health			yes		
Philadelphia	Health	yes	yes	yes		Regulations Relating to Labeling, Application, and Removal of Lead Paint 1966
St. Louis	Housing	yes	yes + (historic painting)	yes	yes	Ordinance # 50678 Lead Poisoning Ordinance 1970
Washington, D.C.	Health, Housing		yes	yes	yes	Health Regulations, Part 1 Use of Lead Paint, 1970, Housing Regulations Section 2605, 1970
Wilmington	Health	yes	yes	yes		Ordinance Regulating and Governing the Sale of Housing and Installing a Housing Code in Wilmington, Del., Section 1A, Lead Paint, 1969

Source: Gilsinn, p. 11

However, a variety of problems arise in attempting to enforce such a municipal ordinance. Some of these problems are related to the manner in which code violations are detected. Voluntary reporting by occupants is inexpensive, but may not be satisfactory as occupants may refuse to disclose violations of which they are aware. By reporting an infraction, a tenant may bring about retaliation, eviction, condemnation of the building, or rent increases. Periodic survey inspections by the municipality are both costly and may cause administrative problems. Occupants may deny city inspectors access to units. Owners may obstruct code enforcement when the code is too strict and if compliance requires large expenditures.

A second type of problem associated with codes, concerns the type of enforcement itself. Strict enforcement may compel some occupants to vacate condemned or abandoned substandard housing and seek shelter in tighter market, whereas loose enforcement, while possibly maintaining the housing market, may involve and permit the occupancy of unsafe and unsanitary units.

Owner's response to a municipal code program is a third kind of problem. Response is affected by the substance of the code and the location of holdings, tax considerations, and overall profit picture. Landlords are hesitant to improve in hard-core slum areas where property values are reduced as buildings age, and the area's socio-economic status declines. Also, if the cost of improvements is passed on to the tenants via rent increases, the owner may have difficulty finding occupants or present tenants may vacate. Regarding tax consider-

ations, although removing the lead hazard from a dwelling unit may not be an assessable improvement, general tax pressures on the owner may affect his investment scheme so as to preclude voluntary repair expenditures. Some owners may simply be unable to pay for building improvements, and even if long-term financing were available for improvements, many might still refrain believing that the building does not merit investment, or the fear of going into debt. The alternatives facing an owner who does not consider his return on improvements to be adequate are: continuing operation in violation of the code, allowing the city to make the necessary repairs, or abandonment.

The final set of problems concerns those legal methods which code enforcement agencies may be authorized to use. Criminal prosecution is the foremost method of enforcement, with fines serving to deter infractions and induce repairs. However, this approach has often been criticized. First, determining the fine is difficult as it must be large enough to be considered more than a negligible cost of business and small enough to leave capital for the needed repairs. Also, many owners may be willing to risk apprehension if enforcement efforts seldom result in prosecution, or when the cost of compliance is greater than the amount of the probable fine. A fixed per diem civil penalty for each day a violation persists might encourage rapid repair to avoid a large total fine, but many landlords might continue to risk prosecution as that proposal would appear not to increase the number of violations caught and penalized. Equitable remedies such as injunctions and receivership are effective, but require large commitments from the enforcement agencies.

Injunctions require the owner to repair or cease operating his building, with equity providing needed supervision of the compliance effort and the availability of contempt sanctions. In the receivership process, when basically sound buildings are not repaired, the court-appointed receiver makes repairs and manages the building until the amount of rent collected equals the cost of repair. Despite the substantial enforcement efforts involved, the use of these two methods in controlling the problem of lead poisoning can be desirable. A direct municipal emergency repair program is a final alternative when the owner does not repair and immediate work is necessary for occupant welfare.⁵²

When one considers the unwillingness of many owners to repair, the inefficiency of criminal sanctions, and fiscal limitations on city governments, the prospect of solving the lead poisoning problem is poor. It has been suggested that the frustrations of a code enforcement program lead to "a tendency to negotiate the tenant's rights away with the hope that sooner or later and adequately financed rent-subsidy or public-housing program will eliminate the slums. In consequence, a vicious circle of non-action is created."⁵³

In addition to municipal codes and laws, the existence of a landlord's obligation to keep his tenant's premises free of lead paint hazards may be derived from specific state legislative enactments, more general statutory duties, or judicial interpretations of the common law. Massachusetts, Maryland, and New York have passed statutes which impose a duty not to apply lead paint to the interior surfaces of dwellings. Thirteen states have statutes which establish a landlord's duty to repair those units in which such paint has already been applied and is allowed to peel - creating a lead hazard. A few juris-

dictions provide that in some circumstances, a tenant may repair certain deficits in his dwelling and deduct the cost incurred from his rental payment. Statutes in Massachusetts and New York permit rent abatement, suspending the tenant's obligation to pay rent until repairs are made. Other provisions permit rent withholding - in which a court collects and holds the tenant's rent until repairs are made.⁵⁴

Finally, in addition to the maintenance of painted surfaces, the nature of the paint itself has also been subject to legislation. In 1955, the American Standards Association developed a standard (Standard Z 66.1) specifying that paints for toys, furniture and dwelling unit interiors should not contain harmful quantities of lead, limiting the lead content to less than 1 per cent in the final dried solids of fresh paint. This excluded lead pigments but did not necessarily eliminate other lead additives in the total paint formulation. Additionally, this standard as well as the Federal Hazardous Substances (Labeling) Act of 1960, required the paint industry to comply with certain labeling provisions on cans of manufactured paint.⁵⁵

MODELS

Because the costs involved in dealing with the problem of lead poisoning are substantial, the use of models may prove to be an effective and efficient approach. One set of two mathematical models devised in 1972, by staff at the National Bureau of Standards, was used as an aid to determine the magnitude and extent of lead poisoning in the U.S. in order to ascertain what level of commitment of resources would be most appropriate to alleviate the problem, and where those resources could be

most effectively applied. The model relates the magnitude of lead poisoning in an area to characteristics of the people and housing in the area. The model is based on the assumption that those areas now reporting lead poisoning can be characterized well enough to predict which other areas are similar and thus, (on the average) should have the same levels of poisoning. The primary purpose of the model was to estimate the nationwide magnitude and extent of lead poisoning, by estimating the number of children with elevated blood lead levels (EBL: 40 μ g/100 ml or more) separately for 241 metropolitan areas of the country. The initial model was not capable of predicting EBLs for separate neighborhoods within a city.

The procedure followed in the model to obtain estimates of the nationwide magnitude and extent of lead poisoning consisted of 4 steps common to most modeling efforts:

1. data acquisition
2. model construction
3. model validation
4. application of the model

The output of the model consisted of several tables of statistical information including:

- * the number of children 6 years of age and under, the estimated number of high risk children (those living in dilapidated or deteriorated housing) and the number of EBLs estimated by the model

Of the 241 SMSAs listed, there were 17 million children 6 years of age and under, an estimated 2,000,000 of which were high risk, with 600,000 EBLs to be expected in these SMSAs.

- * a ranking of the 25 SMSAs having the greatest predicted incidence of EBL, in order to illustrate any possible geographic tendencies. (See Figure 4 and Table 4)

Figure 4. Predicted Nationwide Incidence of Lead Based Paint Poisoning - 25 MSAs With Greatest Predicted Incidence

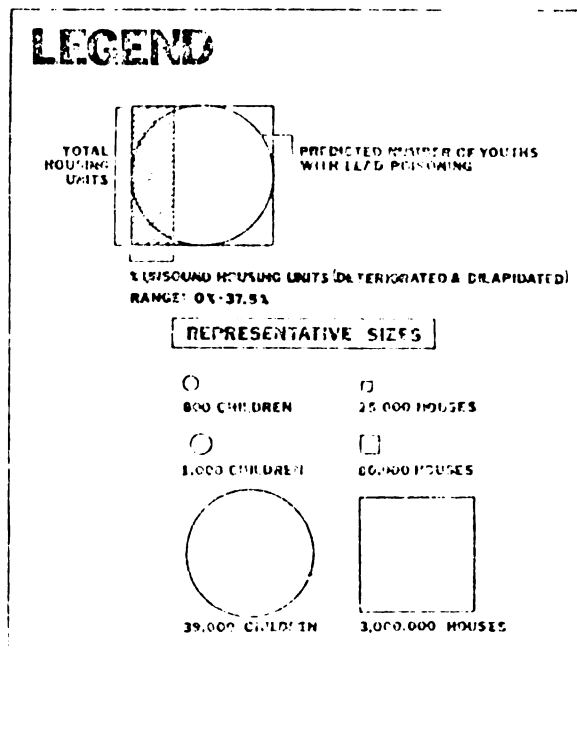
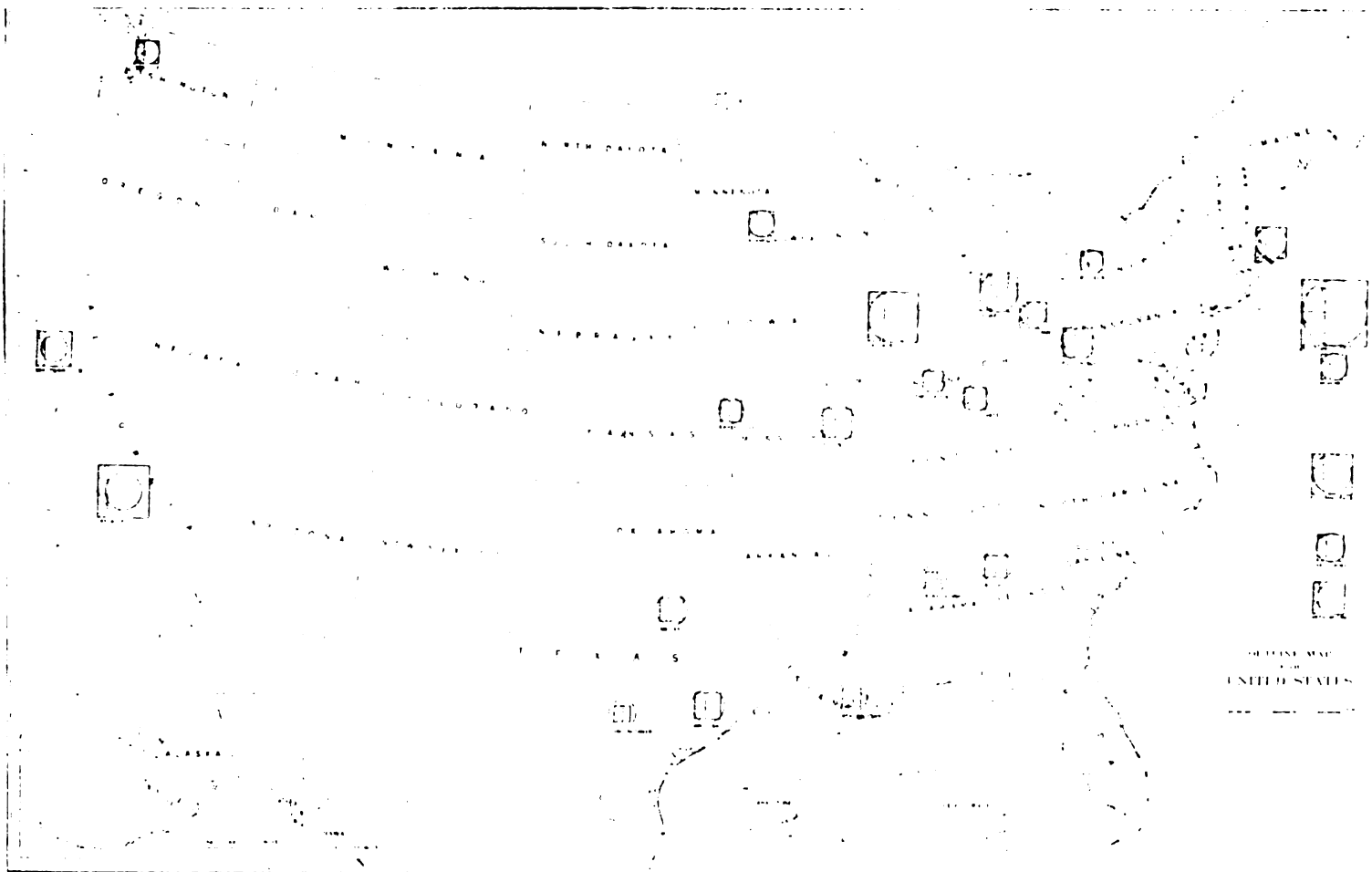


Table 4. 25 SMSAs with the Greatest Number of EBLs

SMSA	EBL	Population Rank
1. New York, N.Y.	40600	1
2. Chicago, Ill.	24100	3
3. Los Angeles - Long Beach, Cal.	14600	2
4. Philadelphia, Pa.	14400	4
5. Detroit, Mich.	14200	5
6. St. Louis, Mo.-Ill.	11500	10
7. Pittsburgh, Pa.	10500	9
8. Houston, Tex.	9700	13
9. Boston, Mass.	9400	8
10. Dallas, Tex.	8600	16
11. Atlanta, Ga.	8100	19
12. Baltimore, Md.	7800	11
13. New Orleans, La.	7200	30
14. San Francisco-Oakland, Cal.	7000	6
15. Newark, N.J.	6300	14
16. Minneapolis-St. Paul, Minn.	6700	15
17. Cincinnati, Ohio-Ky.-Ind.	6400	20
18. Cleveland, Ohio	6400	12
19. Washington, D.C.-Md.-Va.	6300	7
20. San Antonio, Tex.	6000	37
21. Kansas City, Kan.-Mo.	5800	25
22. Birmingham, Ala.	5500	43
23. Seattle-Everett, Wash.	5300	18
24. Indianapolis, Ind.	5300	28
25. Buffalo, N.Y.	5200	23
Total	253400	

Source: Gilsinn, p. 114

- * estimates of the number of children in each WMSA, with blood lead levels of 40~~ug~~ or more, 50 or more, 60 or more, and 70 or more

The estimates yielded by the NBS model, confirmed that pediatric lead poisoning is a major urban health problem in this country, as they suggested that some 600,000 children if tested, would show undue absorption of lead.⁵⁶ The authors of the study caution:

...it is believed that the problem will persist as long as peeling lead painted surfaces are accessible to young children. Discovering and treating children with EBIs will partially alleviate the problem, but its full solution requires the removal of lead paint on all surfaces accessible to children.⁵⁷

A second model, devised in 1972 by Juanita Gaston (MSU Master of Arts, Department of Geography) takes a different approach. It is a spatial rather than mathematical model, and it is to be used on a community-wide rather than nationwide scale. The purpose of the model is to ascertain which children are at greatest risk to lead poisoning by defining and then mapping by census tracts the areas of a city (in this model Lansing was used) with low incomes and old deteriorated pre-World War II housing. The model was intended to address the disadvantages posed by the three most commonly used risk-determination approaches:

1. The random selection of patients from pediatric hospital wards or patients from neighborhood health clinics - The major disadvantage of this approach is that the most deprived and disorganized families do not use health centers, thus causing the child living in a high risk area who does not come to the health center to be the most likely affected.

2. The analysis of paint from the child's environment - The major disadvantages of this approach are first, a diseased child moving from a house containing lead paint to a house free of such paint would not be screened, and secondly, the machine used to do such screening is quite

extensive and thus uneconomical for mass screening.

3. Neighborhood door-to-door lead poisoning educational campaigns in which families are urged to bring their children to health centers for lead poisoning tests - The disadvantage of this approach is that some families may not have transportation, or cannot afford to take time off from work to have their children tested.

Gaston's model is basically constructed by pinpointing in space the residents at risk. (See Figure 5) The steps involved include:

1. constructing tables and maps of the percentage of residential structures built before 1940 and the median income by census tract, for the whole city

2. compilation of maps in terms of age of the residential structure and the condition of the structure for all structures in each of the census tracts screened intensively in the high risk areas

3. the use of certain variables called predictors to increase the predictive value of the model including:

- a. a history of nica
- b. existence of anemia
- c. number of siblings living at home
- d. marital status
- e. supervision of child
- f. source of family income
- g. amount of time child is left in the care of others

The advantages of the spatial model as proposed by Gaston are: it includes all children within a pre-selected area, not just those visiting clinics; the children may be tested in the home by a finger stick blood test, saving a family transportation costs and valuable work time; the approach has only a minimal cost, making it economical for massive screening.⁵⁸

Although detection is only "half the battle" against lead poisoning, models such as these can aid states, cities and communities in developing and conducting intensive local programs to accomplish this.

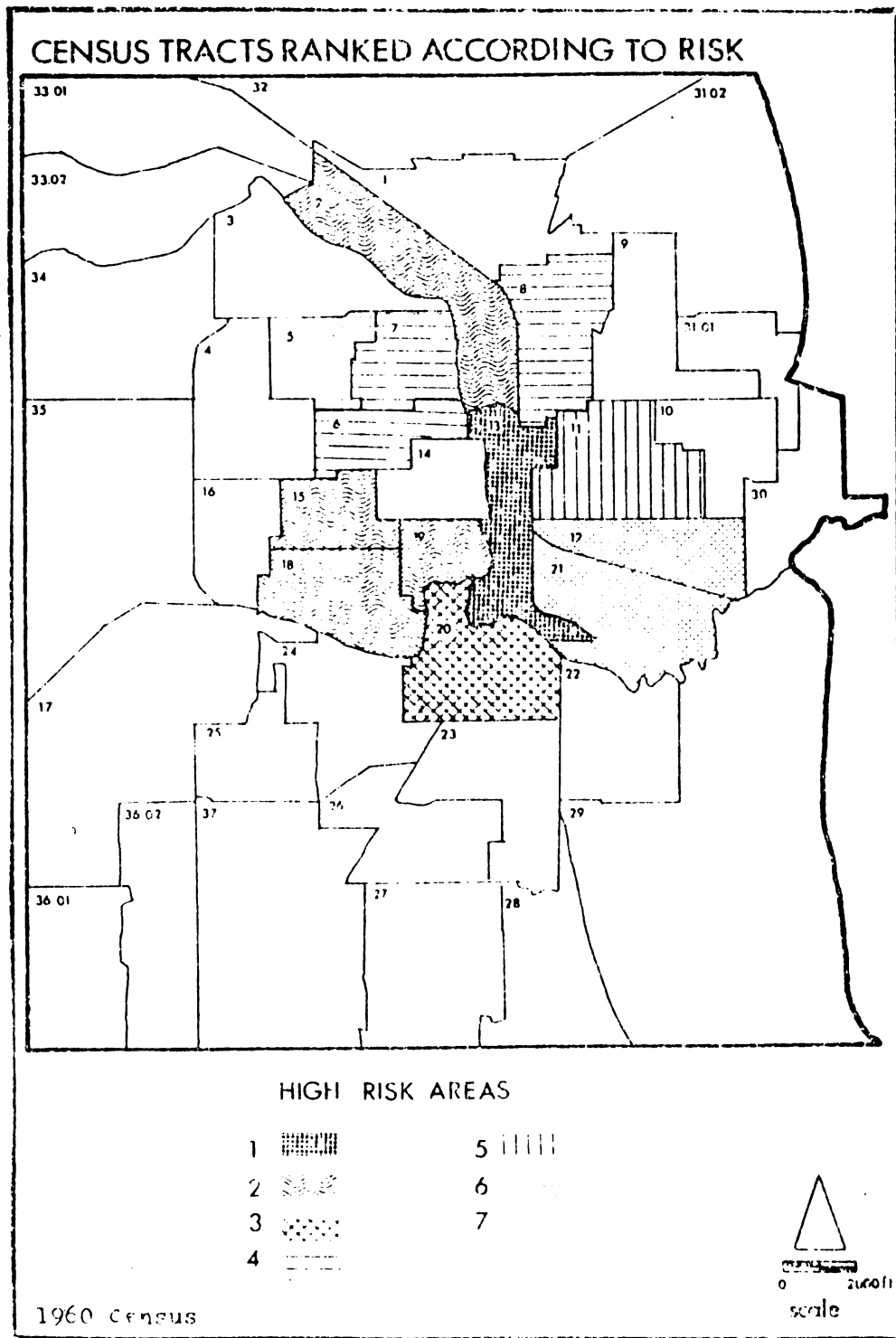


Figure 5. Census Tracts Ranked According to Risk

source: Gaston, p. 60

CASE STUDIES

Essentially, a given city's program for dealing with lead hazards consists of three parts: identification, including casefinding; environmental control, including treatment of victims of lead intoxication ; and elimination of sources.

There are six necessary policies to be included when initiating a lead-control program. First, the chief executive of the city government must actively support the concept, as the progression of activities will inevitably lead to involvement of a number of intra-city governmental departments. Second, it must be understood that successful lead control measures will initially increase in cost as more lead hazards are identified. Third, there is little value in appropriating funds for casefinding of one age group for one year, and eliminating the funds in the next year. The fourth policy is that specific goals must be established at the outset, so that the evaluation process used will give a true picture of the program in progress. Short range goals would include: organization and staffing of the agency, linking neighborhood organizations and other community departments, beginning community education and screening efforts, ^{and} the systematic selection of geographic areas to be tested. Long range goals might be: legislation and firm deleading and renovation programs. The establishment of clear-cut objectives and review of priorities is the fifth essential policy, as if such objectives are limited and realistic, the probability of continuing fiscal support is greater. Finally, each city must develop and follow a plan of attack designed specifically for its individual needs and resources.

A specific sequence of steps are necessary in planning and

executing a lead control program:

1. The "prime movers" in the city, including ghetto residents, must become concerned about the problem and must first convince the local government that the economic benefits of dealing with the problem, warrant the costs. Additionally, the director of the lead-control program must have access to the city's chief executive.

2. The establishment of a staff for the lead program, the size of which depends on the size of the city, and the goals set for the program. A time-phased total plan should be finalized within three months by this staff.

3. Mobilize an educational effort concerning the problem of lead.

4. Casefinding, keeping in mind that it is but a tool to attack the larger problem of lead in the environment.

5. Obtaining legislative and fiscal support for a total effort.⁵⁹

The eradication of lead poisoning is not an impossible task. While slum clearance combined with provision of adequate housing for the poor is the most effective means of elimination, lead poisoning can be successfully reduced through education, early detection, treatment and follow-up programs that include removal of lead from houses wherever it is found. A few cities have demonstrated the value of such methods.

In Chicago, there is a massive screening program for lead poisoning operated through the coordinated effort of local officials, health workers, and the community. The program was begun in 1965 and by 1969 over 120,000 children had been tested and over 1500 treated. Because of this program, the incidence of elevated blood lead levels among children declined from 8.5 per cent in 1967 to 3.8 per cent in 1968. Along with the rise in the number of cases detected, came a decline in the fatality rate. In 1962, the first year the disease was made reportable, 203 cases of lead poisoning were reported with a fatality

rate of 2.9 per cent. In 1968, the number of reported cases rose to 702, while the fatality rate dropped to 1.3 per cent. The Chicago program is concentrated in nine areas in the city and includes the operation of an ambulatory treatment center.⁶⁰

Similar figures have been reported from New York City, through a program in which physicians from all medical agencies in the city are encouraged to send blood specimens on all suspected cases of lead poisoning to the city health department laboratory for prompt and accurate blood lead analysis. In the "lead belt" areas of the city, public health sanitarians visit every house in designated blocks. (See Figure 6) They take screenings for lead determinations, inquire about pica, take urine samples from all children under 6 years of age, and advise parents about the dangers of pica and its association with lead poisoning. From January to July 1970, 64,644 blood specimens were analyzed by the New York City Health Department, 2010 children with blood lead concentrations of 60~~45~~ or greater were found, and 2 lead poisonings caused deaths were reported.⁶¹

In Philadelphia, lead poisoning was made a reportable disease in 1959, but with little effect. In 1966, the Philadelphia City Council took action and regulations were issued regarding the labeling, application, and removal of lead paint. In 1968, a urine screening program for lead poisoning among children 1 to 3 years old was initiated in Philadelphia's high risk areas. (See Figure 7) Through these efforts, numerous properties have been made safe, reported cases of lead poisoning have increased, and the number of lead poisoning deaths has decreased.

Baltimore was one of the first cities to recognize lead poisoning in children as a public health problem. For over

CITY OF NEW YORK

Reported Cases Of

LEAD POISONING

By Place Of Residence

New York City - 1963

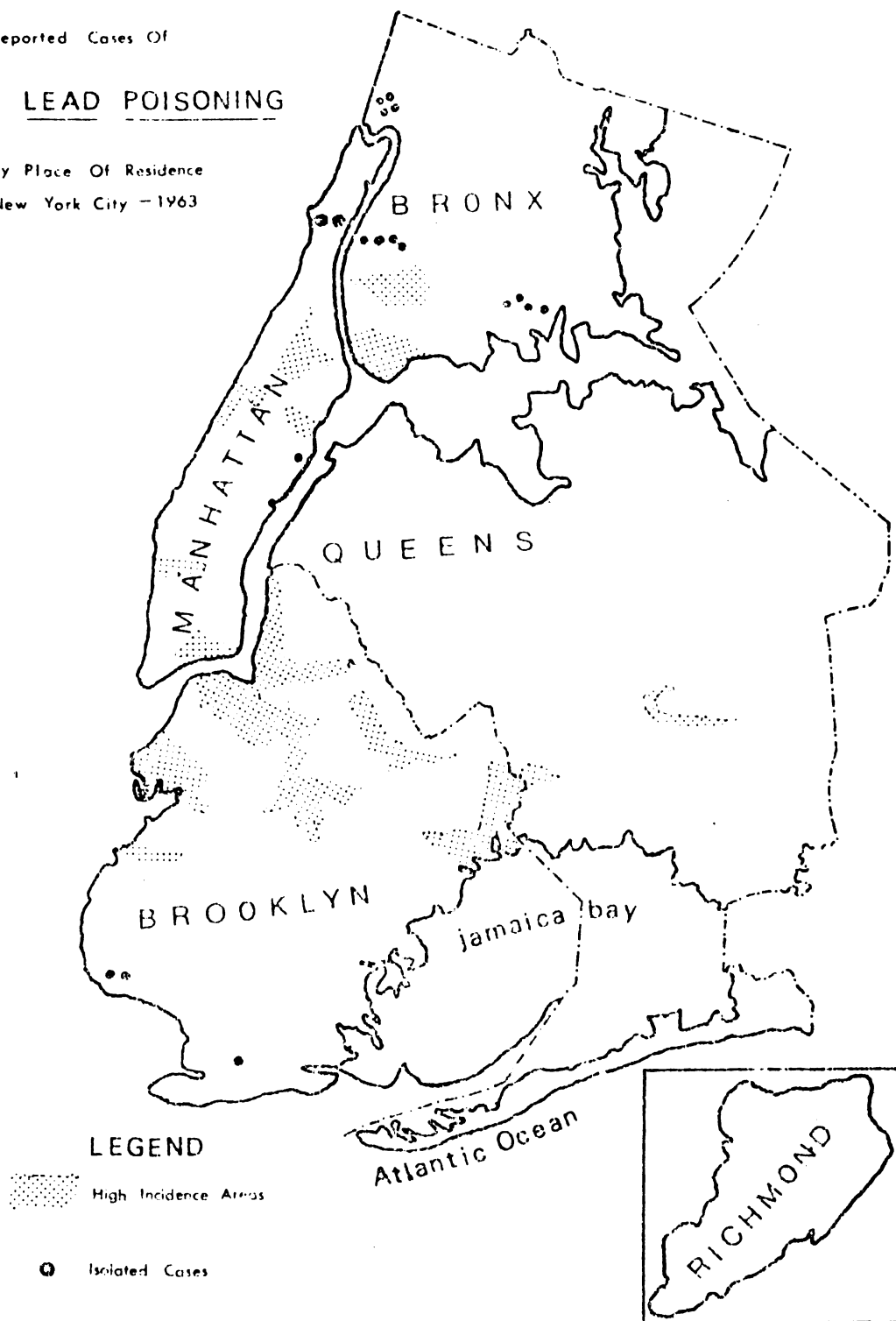
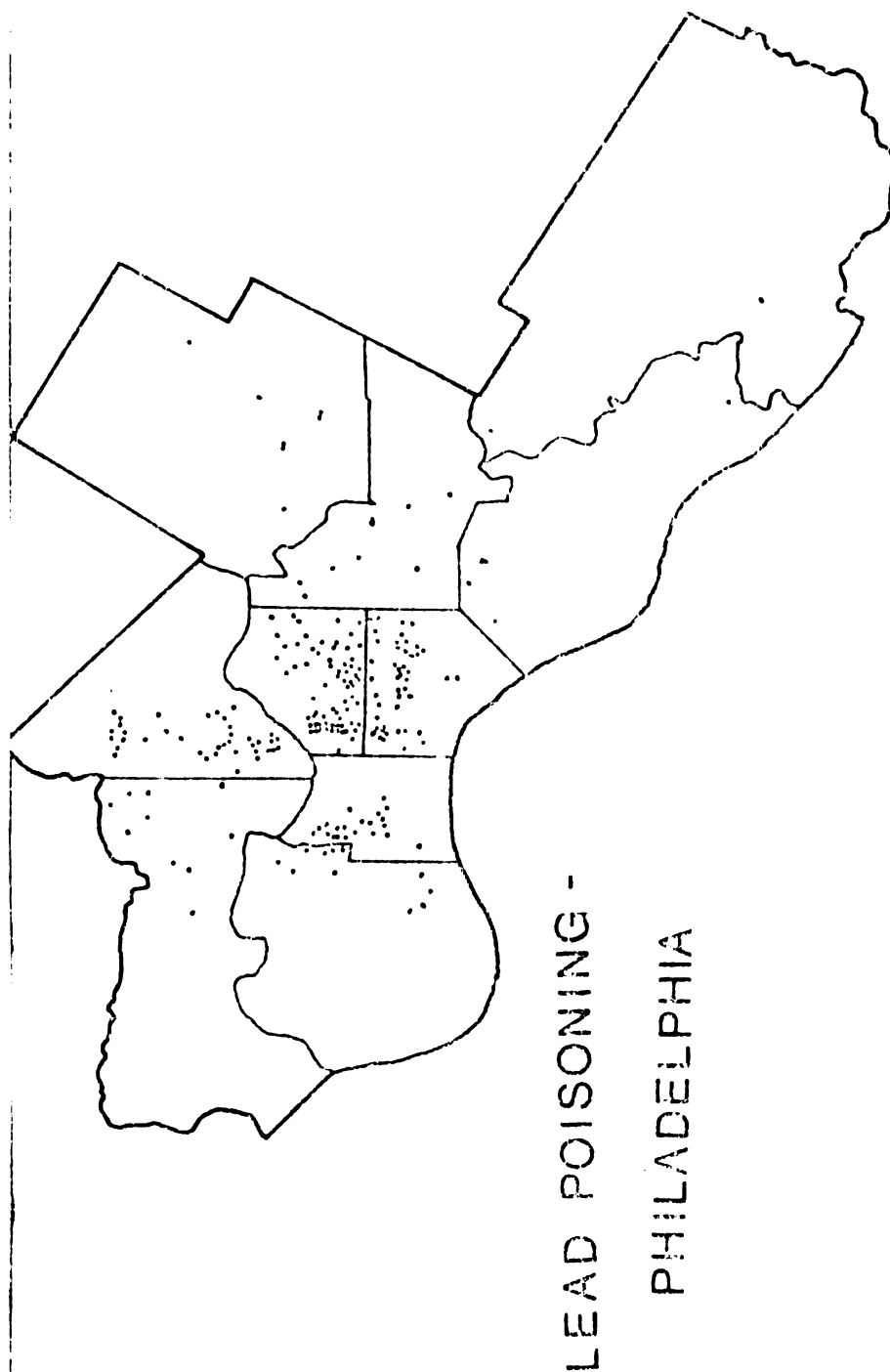


Figure 6. City of New York - Reported Cases of Lead Poisoning

Source: Gaston, p. 25



Spot map of 219 cases of lead poisoning by Philadelphia residences of patients; 1956 - 1960

AFTER INGALLS, 1961

Figure 7. Spot Map of 219 Cases of Lead Poisoning by Philadelphia Residences of Patients; 1956-1960

Source: Gaston, p. 28

30 years, the city has dealt with the problem through continuous detection and prevention programs, that include enforcement of health and housing codes, epidemiological surveys, and intensive educational campaigns. With the help of the Property Owner's Association, the city health department has received cooperation from landlords. The continued existence of the various programs along with paint removal from many dwellings, has yielded encouraging results. Another key to Baltimore's apparent progress is that no child is ever returned to a dangerous environment. After hospitalization, the child goes to a convalescent home and is not returned to his family until all hazardous sources have been removed, or the family has been helped to find lead-free housing.⁶²

Cincinnati has attempted to deal with the complexities of case-finding, prevention, public and professional education, by the establishment of a Lead Control Center, a cooperative project of various city agencies, institutions, professionals in many fields, and a variety of other interested individuals. The Center maintains a close relationship with Cincinnati's Poison Information Center, and is responsible for reporting all cases of diagnosed lead poisoning to the health department, for insuring adequate inspection of victim's homes, for providing temporary custodial care, and for maintaining proper records of known cases and insuring adequate follow-up care.⁶³

Voluntary citizen action in several cities has also played a significant role in the fight to eradicate lead poisoning. In 1968, the Urban League of Rochester, New York, persuaded a group of youth from its Project Unlift Youth Incentives Program to assist the Rochester Committee for Scientific Information in

its study of lead poisoning, through the collection of paint samples from slum homes. The program has continued and the youths now work with the Rochester Neighborhood Health Center and with doctors from the University of Rochester's Strong Memorial Hospital Department of Pediatrics. In addition to collecting paint samples, the young people distribute educational materials and collect urine samples as indicated by paint sampling. Through this program, nearly 7 per cent of the children tested so far have been found to have dangerous levels of lead in their systems.⁶⁴

lay volunteers were also used in a New York City ghetto community in a four day door-to-door lead education and case-finding program in 1966. The goal of the program, which was coordinated by several physicians and the New York City Department of Health, was to reach those high risk families who have limited contact with professional medical personnel. The group of volunteers were parents and all were residents of the community. Some spoke Spanish as well as English. The volunteers were instructed in all aspects of lead poisoning - methods of prevention, importance of early detection, and etiology and sequelae of the disease, and then conducted a four day door-to-door campaign to educate neighborhood residents about lead poisoning. In addition, posters, press and radio announcements, and a volunteer-manned sound truck were also utilized. As a result of this volunteer effort, 116 children were screened, of which 8 were found to have elevated blood lead levels. In evaluating this program, the organizing physicians commented:

The health services in the ghetto slum areas require increased manpower. Such manpower is present and willing, although it needs to be uncovered and mobilized. Our volunteer help

was secured without difficulty and their enthusiasm and spirit were excellent.... This neighbor-to-neighbor type of approach lends itself particularly well to the dissemination of public health information and may have advantages over more authoritarian techniques particularly in deprived areas. A successful program of health education must have some foundation within the community if it is to reach the people and stimulate a positive response.⁶⁸

RECOMMENDATIONS

Numerous programs, plans and guidelines have been proposed by a variety of people in many fields to solve the problem of childhood lead poisoning. (See Table 5) While most incorporate many of the same key principles and recommendations, some of the differences in their scope and focus are worth noting. (See Figure 8)

Some approaches to lead poisoning prevention are rather narrow and specific. Scherz proposes a six-step program which focuses on preventing the exposed child from ingesting paint:

1. All occupants should be warned.
2. Remove leaded paint or cover it.
3. Keep a child suspected of eating lead from further exposure.
4. Alert health workers and those who come into contact with children from hazardous dwellings to early symptoms, and test all suspects.
5. When a case is found, test other children in the house.
6. Treatment should start immediately after diagnosis.⁶⁶

Most approaches, however, are broader and more encompassing. Burd  and Reames view the problem of lead poisoning as a cycle which, in order to prevent serious consequences, must be broken at an early juncture. (See Figure 9) In their view, the most vital step is the removal of substandard housing where layers of lead-containing flaking paint and crumbling plaster are present. During the time span necessary for effective housing replacement, emphasis should be given to the prevention of pica

Table 5. Current Programs

Area	Number Screened	Year	Description of Program
Baltimore	718	1966	Mostly by physicians or in hospitals
	850	1970	
Buffalo			Small screening effort
Chicago	25,000	1967	Mass screening
	40,000	1968	
	17,727	1969	
	44,047	1970	
	39,975	1971	
Cincinnati			Small screening effort, test children with symptoms
Columbus			Test children with symptoms
Connecticut			Using primarily the MA urine test
Bridgeport			2 cases reported, small screening, also test in clinics
Hartford	147		Test in hospital and test children with symptoms
New Haven	1897	1970	Mass screening in 1970, not funded in 1971, now screening primarily in hospitals and clinics
Stamford	150		Small screening effort
Waterbury	500		Small screening effort
Delaware			
Milford			PCM survey, small screening, in hospitals
Denver			Small screening effort.
Illinois			
Aurora	1763	1971	Mass screening, 12% of all children screened
Springfield	670	1971	
Peoria	87	1971	
El. St. Louis	376	1971	
Peru	763	1971	
Joliet	383	1971	
Rock Island	285	1971	
St. Meline	192	1971	
Eldridge	103	1971	
Piquette	226	1971	
Carbondale	264	1971	
Indianapolis			PCM survey, small screening effort
Kansas City			Testing in hospitals
Massachusetts			
Boston			Pilot project, initiated by community groups
Cambridge			In hospitals, planning future efforts
Dorset			In clinics
Worcester			Screening in Model Cities areas

Table 5. Continued

Area	Number Screened	Year	Description of Program
Milwaukee			Test children with symptoms
Minneapolis			Test children with symptoms
Nashua, N.H.			BCIM survey
Nashville	97		Pilot project
New Jersey:			
Jersey City			Testing in hospitals, testing children with symptoms
Newark	3043	1971	Some mass screening and also in hospitals
Paterson			Testing children with symptoms
Trenton			Testing in hospitals
New Orleans	727		Using a finger prick test
New York City	1643	1969] Mass screening
	84368	1970	
	87559	1971	
Norfolk	About 1260	1971	Grant from BCIM to conduct small screening
Ocala			Small screening effort
Philadelphia	About 5000		Screening in Model Cities Areas, testing in hospitals and clinics, not using blood lead test
Portland, Maine	About 1000	1970	Small screening effort, testing in hospitals, using ALA urine test
Portland, Ore.			BCIM survey
Rhode Island	1500		Screening using hair samples
Rochester, N.Y.			Screening, also test children with symptoms
Sacramento			BCIM survey, small screening effort
St. Louis	4027	1971	Mass screening in selected areas
San Antonio			Test children with symptoms
San Francisco			Small screening effort
Salt Lake City			BCIM survey
South Bend			Small screening effort
Syracuse			Small screening effort, test children with symptoms
Washington, D.C.	808	1970] Small screening effort and testing in clinics
	1821		
Yonkers			Small screening effort, test children with symptoms

Source: Gilsinn, pp. 26, 27

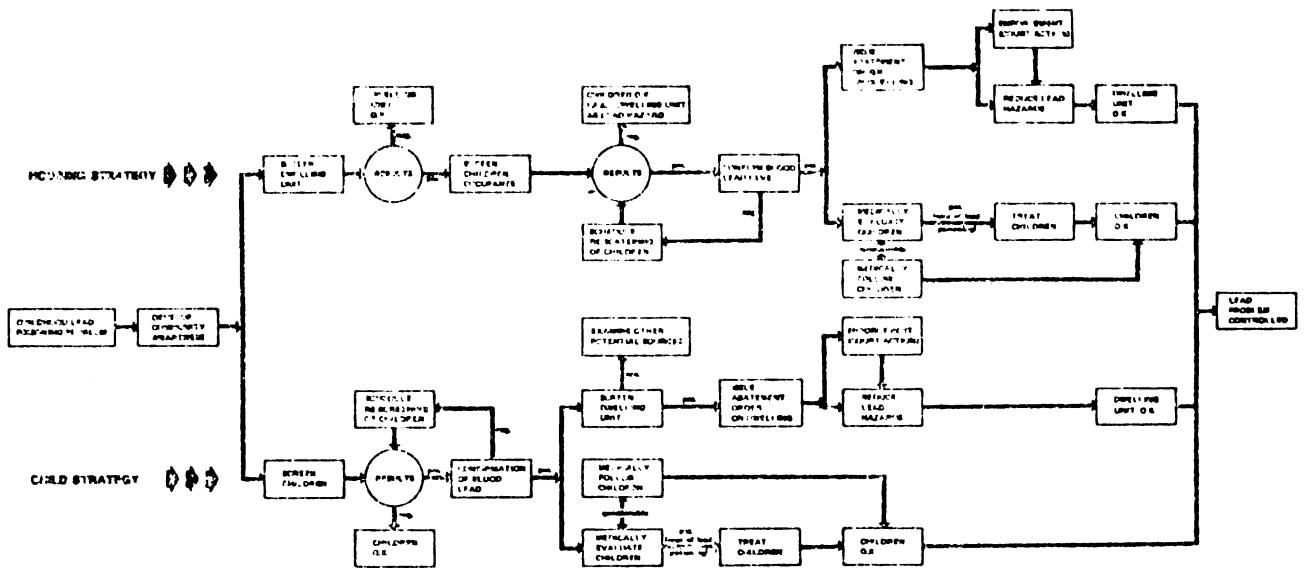


Figure 8. Childhood Lead Poisoning Control Program Strategies

Source: Hardy et al, p. 102

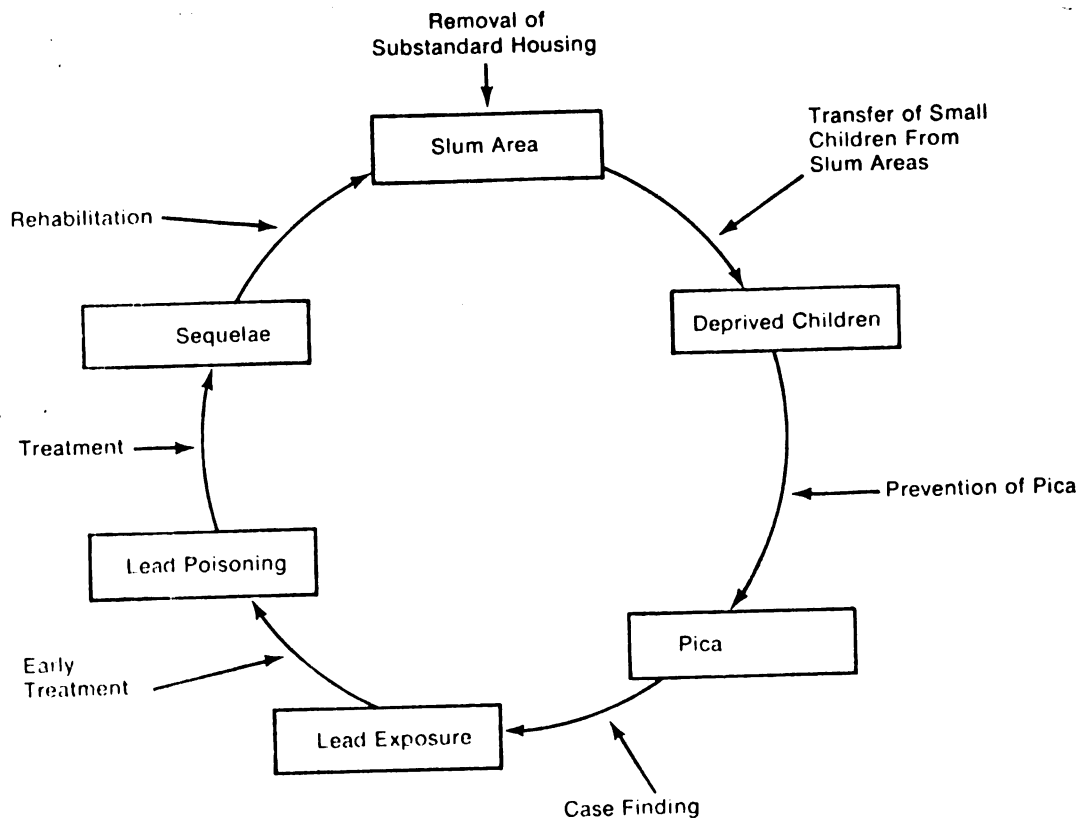


Figure 9. Causes and Consequences of Childhood Lead Poisoning and Potential Sites for Intervention

Source: Burdette and Reames, p. 738

in those children who must remain in hazardous surroundings. Primary responsibility for control of lead lies with the family, and thus parents must be informed of the dangers associated with this habit. Mass media, school parent groups, and community meetings should all be utilized in this educational effort. Burd  and Reames also recommend for smaller communities, the establishment of "lead centers" - special clinics where high risk children receive evaluation and care. These centers should be comprised of an interdisciplinary staff to help families cope with environmental situations which produce the exposure hazards and provide consistent education of the family through personal contact. The efficacy of such centers will ultimately depend upon the location and ease with which parents can obtain help.⁶⁷

According to Challon, a two-fold task exists for any community that is considering the establishment of an active lead poisoning control program. First, the children at greatest risk must be located, screened, and treated when indicated. Secondly, and perhaps more importantly, as far as the overall elimination of lead poisoning is concerned, the source of the lead-based paint must be located and made unavailable for further ingestion. "It is obvious that any program designed to control or eliminate lead-based paint poisoning in a community must be based on a thorough understanding of the epidemiology of the lead problem in that community."⁶⁸ Challon also emphasizes the need for an inter-disciplinary approach to the problem and stresses that the medical profession alone cannot "cure" lead poisoning. According to Challon, while medical input is needed in designing and operating screening and treatment facilities, lead

poisoning is basically a housing and political problem:

The time is now right to broaden the fight, to extend the responsibility for the physical and mental well-being of 2½ million children below the age of 6 living in deteriorating housing to the other professions that can effect change. The time is right for lawyers and judges to enforce housing codes and protect those children who are too poor to protect their constitutional rights; for politicians and office holders, to seek passage of new, more workable ordinances and appropriate measures; for sanitarians, housing technologists, unemployed NASA engineers to lend their skills; and most important, for parents and community groups to become involved in their own destiny. If this problem is to be solved, it is at the community level that the action will occur.69

Chisolm and Kaplan point out that comprehensive health clinics for disadvantaged children in many cities provide a suitable module for more effective child and parent care in the area of lead poisoning:

In high-risk areas, the incorporation of a screening test for excessive lead ingestion into the regular laboratory procedures in such clinics, together with the development of interview techniques designed to identify the child with incipient vice offer the best current hope for the prevention of childhood plumbism...[additionally] what families in in such neighborhoods require are the imaginative and continuous efforts of health and social action teams directed at parent participation and education.70

Hin-Fu states that the ideal solution to childhood lead poisoning is slum clearance and urban renewal with the provision of adequate housing for families of low incomes. However, as this goal cannot be achieved quickly, control and prevention must depend on other means including:

1. Public education through all channels and a media of communication to point out the danger of paint eating, the symptoms of lead poisoning, and urge parents to seek help whenever lead poisoning is suspected.

2. Education of physicians, nurses, social workers, and all other health workers on the prevalence of lead poisoning among children, so as to serve as an index for suspicion.

3. Mass screening programs in "lead belts" for all children between 1 and 6 years of age, using blood lead determination.

4. Immediate referral of children found to have elevated blood lead levels to a medical center for diagnosis and treatment if necessary; prevention of re-exposure; follow-up and retesting of all treated children who continue to be exposed. Health workers must work closely with housing authorities to insure that lead paint is removed from every dwelling where poisoning has occurred.

5. The establishment of effective health and housing codes pertaining to lead and lead poisoning and the diligent enforcement of these codes.

6. A concerted effort by research institutions to develop a simple, practical and relatively inexpensive method for paint removal.⁷¹

Moore sees the solution to the lead poisoning problem as lying in the improved interaction of key sub-groups in the community. One such sub-group is composed of agencies and institutions concerned with health, housing, education, and social services. Such institutions have a tendency to operate unilaterally and in isolation from one another, with defined and specific tasks and alignments. The child with lead poisoning may be bounced between agencies with none taking any real action. A second sub-group is that comprised of business and industry, particularly real estate owners and the housing industry. Current real estate and investment practices are often discriminatory and racist, and tend to lock blacks and other minorities into ghettos characterized by poverty, overcrowding, and life-threatening health hazards such as lead poisoning. The final sub-group are the community residents themselves. Few communities have invited resident participation in formulating plans

and programs to combat lead poisoning. Additionally, few agencies develop community input to work-training leaders into the health professions, and fewer still have added trained community organization staff as community liaisons or advocates. According to Moore, agencies and community residents must begin to work together to develop methods of identifying poor housing and lead-infested units. Legal channels and community pressure must combine to develop methods of removal, and the forcing of landlords to uphold codes adequately designed to include testing of paint layers. Relocation services can serve as a basic tool but only if combined with adequate methods of screening and securing the new unit. Unfair housing and investment practices must be abolished, and the behavior manifested in governmental and other structures that perpetuates such conditions must be changed.⁷²

Finally, the role of all levels of government is also key to any efforts which seek to eradicate lead poisoning. In 1974, a group of 200 government officials and civic leaders met in Washington D.C. to draw up proposals for preventive measures. The group's recommendations were:

1. Establishing closer working relationships between federal government agencies and private hospitals.
2. Increasing the number of housing officials who inspect for lead hazards and requiring inspections of all units in an apartment house in which a lead poisoning victim lives.
3. Starting educational campaigns through schools, day-care centers and radio-television to alert parents and older children to lead hazards and poisoning symptoms.
4. Increasing federal funding for private research of all possible lead poisoning factors.
5. Inspecting day care centers for lead hazards as part of the regular licensing procedure.

6. Training teams of public health nurses and social workers who can assist parents of victims or potential victims.⁷³

In addition to the development of programs and strategies, research is also urgently needed to accomplish a number of objectives:

1. Development of an accurate and simple portable device for lead detection in order to systematically identify houses containing lead paint, to replace the more time-consuming collection and chemical analysis of paint samples.
2. A uniform reporting system for all screening programs to facilitate data collection, information exchange, and comparison of results.
3. Improved methods of lead poisoning treatment.
4. Increased knowledge about the causes and cures of poisoning.
5. Determination of the "subtle" or not immediately apparent effects of lead.
6. Development of a reliable, simple, and inexpensive method to determine blood lead level.⁷⁴

CONCLUSION

Although we know a great deal about the medical aspects of childhood lead poisoning and have made much progress in the diagnosis and treatment of this disease, "our society has not developed social and political technologies to keep pace with the medical ones." That is, we can treat lead poisoning successfully, if detected early enough, but we have no medical means by which to **prevent** the occurrence and recurrence of the disease:

Not infrequently, children treated for lead intoxication are returned to the same old environment which caused their illness in the first place. In this regard, intervention by health practitioners in the matter of childhood lead poisoning has much in common with U.S. health delivery in general - the system is weighed heavily in the direction of treatment after-the-fact while preventive strategies remain undeveloped.⁷⁵

In addition to health practitioners - landlords, parents, public officials, community groups, and the general public all bear responsibility for the persistence of lead poisoning. However, existing literature on the subject provides little as to how concerned parties might proceed most effectively, based on the fact that public resources to deal with the problem are not likely to substantially increase in the near future. A variety of matters need to be studied and understood:

- * What kinds of community action strategies will produce the greatest probability for dealing successfully with the lead poisoning hazard?
- * Given limited resources, what are the benefits of public awareness campaigns, parental counseling, housing detoxification efforts, housing code enforcement and court action, and research into the causes and prevention of

childhood mice?

- * How are effective coalitions of community groups committed to action against lead poisoning built?
- * Why have some communities accomplished more than others in dealing with the problem?

Mounting comparative, cross-city studies might be one way of answering some of these questions:

To date, little if any public money has been available for the kind of research that would help to develop effective social and political technologies for combating the lead paint problem. Hopefully, this situation will be rectified in the near future as more attention is devoted to careful study and comparison of diverse approaches utilized in different cities throughout the country.⁷⁶

For all its complexity and severity, and the atmosphere of of ignorance, bureaucratic red tape, and often simple unconcern which surrounds it, and despite the fact that it kills and permanently impairs hundreds of children each year, lead poisoning is still an eradicable disease. As René Dubos, Pulitzer Prize winning writer on man and his environment warned in his speech at a conference on lead poisoning held in 1969:

The problem is so well-defined, so neatly packaged, with both causes and cures known, that if we don't eliminate this social crime, our society deserves all the disasters that have been forecast for it.⁷⁷

APPENDIX

9235

IN THE SENATE OF THE UNITED STATES

[illegible]

Mr. KENNEDY (for himself, Mr. ANTONIO, Mr. BARBER, Mr. CASE, Mr. FORD, Mr. LARSEN, Mr. MONTANA, Mr. CROFT, Mr. HARRIS, Mr. JONES, Mr. JAMES, Mr. ALABAMA, Mr. MASON, Mr. HENRY, Mr. MOORE, Mr. NIXON, Mr. PETER, Mr. TUCKER, Mr. VANDERKAM, Mr. YOUNG, and Mr. YOUNG) introduced the following bill, which was read twice and referred to the Committee on Labor and Public Welfare:

110

To encourage cities and communities to develop intensive local programs to eliminate the health hazards of lead-based paint poisoning.

- 1 *Be it enacted by the Senate and House of Representatives*
- 2 *of the United States of America in Congress assembled,*
- 3 That this Act may be cited as the "Lead Paint Poisoning
- 4 Prevention Act";

**TITLE I—GRANT FOR THE DETECTION AND
TREATMENT OF LEAD-BASED PAINT POI-
SONING**

GRANTS FOR LOCAL DETECTION AND TREATMENT OF

LEAD-BASED PAINT POISONING

SEC. 101. (a) The Secretary of Health, Education, and Welfare (hereafter referred to in this title as the "Secretary") is authorized to make grants to units of general local government in any State for the purpose of assisting such units in developing and carrying out local programs to detect and treat incidents of lead-based paint poisoning.

(b) The amount of any such grant shall not exceed 75 per centum of the cost of developing and carrying out a local program, as approved by the Secretary, during a period of three years.

(c) A local program should include—

(1) educational programs intended to communicate the health danger and prevalence of lead-based paint poisoning among children of inner city areas, to parents, educators, and local health officials;

(2) development and carrying out of intensive community testing programs designed to detect incidents of lead-based paint poisoning among community residents, and to insure prompt medical treatment for such afflicted individuals;

(3) development and carrying out of intensive followup programs to insure that identified cases of lead-based paint poisoning are protected against further exposure to lead-based paints in their living environment; and

(4) any other actions which will reduce or eliminate lead-based paint poisoning.

(d) Each local program shall afford opportunities for employing the residents of communities or neighborhoods affected by lead-based paint poisoning, and for providing appropriate training, education, and any information which may be necessary to inform such residents of opportunities for employment in lead-based paint poisoning elimination programs.

**TITLE II—GRANTS FOR THE ELIMINATION OF
LEAD-BASED PAINT POISONING**

SEC. 201. The Secretary of Health, Education, and Welfare is authorized to make grants to units of general local government in any State for the purpose of assisting such units in developing and carrying out programs that identify those areas that present a high risk to the health of residents because of the presence of lead-based paints on interior surfaces, and then to develop and carry out programs to eliminate the hazards of lead-based paint poisoning.

(a) A local program should include:

- (1) development and carrying out of comprehensive testing programs to detect the presence of lead-based paints in interior surfaces of residential housing;
- (2) the development and carrying out of a comprehensive program requiring the prompt elimination of lead-based paints from all physical structures or interior surfaces on which lead-based paints have been used as a surface covering, including those structures or interior surfaces on which non-lead-based paints have been used to cover surfaces to which lead-based paints were previously applied; and
- (3) any other actions which will reduce or eliminate lead-based paint poisoning.

(b) Each such program shall—

- (1) be consistent with workable programs for community improvement referred to in section 5; and
- (2) afford, to the maximum extent feasible, opportunities for employing the residents of communities or neighborhoods affected by lead-based paint poisoning, and for providing appropriate training, education, and any information which may be necessary to inform such residents of opportunities for employment in lead-based paint elimination programs.

TITLE III—GENERAL

DEFINITIONS

SEC. 301. As used in this Act—

(1) the term "State" means the several States, the District of Columbia, the Commonwealth of Puerto Rico, and the territories and possessions of the United States; and

(2) the term "units of general local government" means (A) any city, county, township, town, borough, parish, village, or other general purpose political subdivision of a State, (B) and combination of units of general local government in one or more States, (C) an Indian tribe, or (D) with respect to lead-based paint poisoning elimination activities in their urban areas, the territories and possessions of the United States.

CONSULTATION WITH OTHER DEPARTMENTS AND

AGENCIES

SEC. 302. In carrying out the authority under this Act, the Secretary of Health, Education, and Welfare shall cooperate with and seek the advice of the heads of any other departments or agencies regarding any programs under their respective responsibilities which are related to, or would be affected by, such authority.

2 Sec. 303. (a) There is hereby authorized to be appro-
3 priated to carry out the provisions of title I of this Act not
4 to exceed \$7,500,000 for the fiscal year 1970 and for each
5 of the two succeeding fiscal years.

6 (b) There is hereby authorized to be appropriated to
7 carry out the provisions of title II of this Act not to exceed
8 \$15,500,000 for the fiscal year 1970 and for each of the
9 two succeeding fiscal years.

10 (c) Any amounts appropriated under this section shall
11 remain available until expended when so provided in ap-
12 propriation Acts; and any amounts authorized for the fiscal
13 year 1970 but not appropriated may be appropriated for
14 the fiscal year 1971. Any amounts authorized for the fiscal
15 years 1970 and 1971 but not appropriated may be appro-
16 priated for the fiscal year 1972.

FOOTNOTES

1. White, pp. 1-2
2. Ibid., p. 2
3. Haar (in Coulston and Korte), p. 76
4. Lin-Fu, p. 1
5. Committee on Environmental Hazards, p. 950
6. Greer, p. 248
7. Subcommittee on Accidental Poisoning, p. 292
8. Chisolm, p. 21
9. Subcommittee on Accidental Poisoning, p. 292
10. Ibid.
11. Ibid.
12. Committee on Biologic Effects of Atmospheric Pollutants, p. 135
13. Lin-Fu, pp. 5-6
14. Committee on Biologic Effects of Atmospheric Pollutants, p. 135
15. Ibid., pp. 139-140
16. Lin-Fu, p. 12-13
17. Ibid., pp. 14-15
18. Lin-Fu (in Children Jan-Feb 1970), p. 5
19. Graham and Graham, p. 39
20. Ibid.
21. Ibid., p. 38
22. Barnes, pp. 8-9
23. Committee on Biologic Effects of Atmospheric Pollutants, pp. 206-207
24. Ibid., pp. 206, 209
25. Ibid., p. 209
26. Ibid., p. 210
27. Haar and Aronow (in Coulston and Korte), pp. 197-200
28. Committee on Biologic Effects of Atmospheric Pollutants, p. 139
29. Needleman et al, pp. 245-248
30. Committee on Biologic Effects of Atmospheric Pollutants, p. 139
31. Ibid., p. 140
32. Waldron and Stöfen, pp. 119-120
33. Tenner and Levin (in Coulston and Korte), pp. 152, 164, 169, 189

34. Waldron and Stöfen, n. 121
35. Ibid., n. 125
36. Williams et al, pp. 230-231
37. Fulwiler and Wright, n. 374
38. Ibid.
39. Barnoko, pp. 1737-1738
40. Browder et al, n. 47
41. Ibid., n. 48
42. Ibid.
43. Allen, n. 90
44. Browder, p. 207, 209, 211
45. Subcommittee on Accidental Poisoning, n. 297
46. Committee on Biologic Effects of Atmospheric Pollutants, pp. 264-267
47. Ibid., pp. 267-268
48. Ibid., n. 268
49. Wolman, pp. 692-693
50. Greer, pp. 260-263
51. HEW, pp. 4, 6
52. Greer, pp. 249-255
53. Ibid., n. 255
54. Ibid., pp. 256-259
55. Committee on Biologic Effects of Atmospheric Pollutants, pp. 75, 78
56. Gilsinn, pp. 13-20, 102-105, 111-122
57. Ibid., p. 122
58. Gaston, pp. 53-61, 91-92
59. Subcommittee on Health, pp. 76-78
60. Committee on Biologic Effects of Atmospheric Pollutants, pp. 135-137
61. Ibid.
62. Lin-Fu (in Children Jan-Feb 1970), n. 7
63. Allen, n. 92
64. Lin-Fu (in Children Jan-Feb 1970), pp. 7-8
65. Thomas et al, n. 106
66. Schera, pp. 720-721
67. BurdÉ and Leones, n. 742
68. Challon et al, n. 655
69. Challon, pp. 561-562
70. Chisolm and Kaplan, n. 649

71. Lin-Fu (in Children Jan-Feb 1970), pp. 8-9
72. Moore, pp. 1430-1434
73. Graham and Graham, n. 41
74. Lin-Fu (in Children Jan-Feb 1970), p. 9
75. Quinn, Duncan, and Cox, pp. 4-5
76. Ibid., n. 6
77. Graham and Graham, n. 41

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