LEAD INDUSTRIES ASSOCIATION, INC.
292 MADISON AVENUE
NEW YORK 17, N. Y.

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LEAD HYGIENE and SAFETY BULLETIN NO. 142

To Members of the Lead Industries Association, Inc.:

CHILDHOOD LEAD POISONING

Illness caused by the ingestion of lead-base paint from interior surfaces by small children is still a grave problem and gives rise to a great deal of publicity in the press.

In order to help solve this serious problem our office maintains a policy of cooperation with Health Departments, Poison Control Centers, Pediatric groups and others. We urge our members, if the occasion should arise, to offer whatever support can be managed for local efforts at controlling the hazard. For instance, one member company gave financial support to a local health department, which support enabled that department to facilitate greatly its program of blood analysis.

For your information we are sending with this bulletin a copy of an article which outlines the experience of the City of Philadelphia. Philadelphia operates one of our better Health Departments and is making a sincere logical effort at controlling this problem.

Don G. Fowler
Director of Health and Safety

DGF:md
Enc: No 6111A

Look Ahead with Lead

LEAD POISONING

IN PHILADELPHIA,

1955 - 1960

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LEAD INDUSTRIES ASSOCIATION, INC.
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Lead Poisoning in Philadelphia, 1955-1960

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Lead poisoning in children has long been recognized to be a serious clinical and public health problem, especially in large and old cities.[1-4] In Philadelphia approximately 50 cases are reported yearly, and from 10% to 20% of diagnosed patients have died in the last 5 years (Table 1). Many of those who survive have permanent damage to the central nervous system and, all too frequently, mental retardation. The chelating agents have marked a significant advance in therapy, and yet the community problem remains much as before. Control has not been accomplished, nor does it seem to rest in early diagnosis and treatment, desirable as that may be from a therapeutic point of view. A comprehensive program of prevention undertaken at a community level gives more promise of success provided that it is accompanied by epidemiologic measures for evaluating accomplishment. Control lies not only in informed medical and public health professions but with other professional groups and official agencies and in informed and determined citizen support.

Childhood lead poisoning differs from other types of accidental poisoning in children because both exposure and intoxication are chronic rather than acute and because methods for both treatment and control differ. For these reasons, a separate program for the control of lead poisoning was established by the Philadelphia Department of Public Health in 1950. In determining the extent to which lead poisoning exists in this city it was first found necessary to make the condition a reportable disease. Moreover, incentives were needed in order to obtain anything like comprehensive reporting. These incentives took the form of services which the Department, together with cooperating private agencies, could provide to reporting hospitals and physicians: the investigation of cases, the collection and analysis of housing, laboratory, and field data, and assistance in the removal of offending paints. To these general ends and toward an ultimate objective of control the Henry Phipps Institute and the Philadelphia Department of Health have developed a joint program of consultation and research with this report marking the first formal analytical accomplishment. However, it is the specific purpose of this paper to go beyond the statistical analysis of data gathered to date in an attempt to begin to localize specific environmental hazards using the classical epidemiologic approach of studying the distribution of cases by "time, place, and person."

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Results

An analysis of lead poisoning in Philadelphia by month of occurrence of acute

Table 1.—*Lead Poisoning in Philadelphia, 1956-1960, by Month of Onset**

	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	Total
Jan	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
Feb	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
March	[illegible]	[illegible]	—	—	—	[illegible]
April	[illegible]	[illegible]	[illegible]	—	[illegible]	[illegible]
May	[illegible]	[illegible]	[illegible]	[illegible]	—	[illegible]
June	[illegible]	[illegible]	[illegible]	[illegible]	—	[illegible]
July	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
Aug	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
Sept	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
Oct	[illegible]	[illegible]	[illegible]	—	—	[illegible]
Nov.	[illegible]	—	[illegible]	[illegible]	[illegible]	[illegible]
Dec.	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
Unknown	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
Total	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
Deaths	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]

* Month of diagnosis used as month of onset.

manifestations (Table 1) reveals what has been shown in other cities. Like the encephalitides of infectious origin, the acute illness is a warm weather disease and even cursory

Fig 1.—Five-year-old boy with chronic lead poisoning standing by a window, the sill of which was the main source of lead ingestion. X-rays of the long bones show metabolic evidence of lead poisoning of over a year's duration, whereas plumbism was not diagnosed until the child was 4 9/12 years of age in August, 1960.

inspection of the 5 year totals reveals that most cases became manifest and were diagnosed in the warmer months. However, this feature of summer prevalence seems to be more closely related to the manifestation of symptoms than to the ingestion of lead. No one knows precisely what seasonal factors are at work to make lead encephalitis a disease of the months of sunshine, but the circulation of lead ions in the blood, like the circulation of calcium ions, has powerful metabolic determinants.

Rapoport and Rubin[6] have advanced experimental evidence to indicate that actinic rays of the summer sun increase the absorption of lead from the intestine, an interpretation consistent with the clinical studies of Chisolm and Harrison.[7] This is a reasonable interpretation, for x-rays of the long bones will often reveal that a high degree of absorption and deposition of lead (Fig. 1) has been going on for many months without recognized clinical manifestations. Factors relating to dosage are surely involved but to an undetermined degree. Thus Dods[8] has emphasized that outdoor paint becomes dry and powdery in the hot sun, and that transfer of lead in smudges and in rain water is favored. On the other hand, the now classical outbreak of lead poisoning in 1933 in Baltimore due to the burning of old battery casings was not brought to light until August

Fig. 2.—Spot map of 219 cases of lead poisoning by Philadelphia residences of patients; 1966–1968

although the coverings had been used as fuel for many months preceding and in most cases during the previous winter.[9]

A spot map (Fig. 2) of cases arranged by residence of patients in Philadelphia suggests that the clustering of some cases and the linear arrangement of others may provide a community index of hazards which will be a useful guide to intensified efforts at control. The linear distributions observed are compatible with the distribution of heavily leaded paints in old houses, on selected streets and in specific areas. Clustering could be a function of tenement housing. However, the over all pattern is such as to raise suspicions that factors other than the distribution of lead in old paints may be involved in selected areas, possibly water supplied through ancient conduits, or localized pollutions of the environment. The facts warrant further analysis of these distributions by age, race, and ethnic groups, by objective evidence of paint chewing (on window sills, cribs, or from the presence of paint flakes), and by environmental factors such as suspect industries and highways—for industrial and automobile exhausts are, as is pointed out elsewhere in this issue of the Archives, other potential sources for the emission of lead into the environment. The intake of lead through the respiratory tract is well known to be the most effective way for the metal to enter quickly into the human organism. In all events a campaign for control might be concentrated in selected streets and districts of the city, thereby avoiding dissipation of money, personnel, and effort.

Apart from the distribution of cases in time and their location in space the third epidemiologic dimension to be routinely examined involves the identification of cases according to person distributions. Age, sex, and race are the first parameters usually examined, although sex (Table 2) may be quickly dismissed here, for while there is a slight excess of cases among males, there is no striking tendency for lead poisoning to

TABLE 2—*Lead Poisoning in Philadelphia, 1956-1960, by Sex*

Yr.	M	F	Total
[illegible]			
[illegible]			
[illegible]			
[illegible]			
[illegible]			
Total	[illegible]	[illegible]	[illegible]

involve boys rather than girls. However, the study of cases by age and race may be more rewarding.

Figure 3 shows in graphic form the distribution of 197 cases in children under

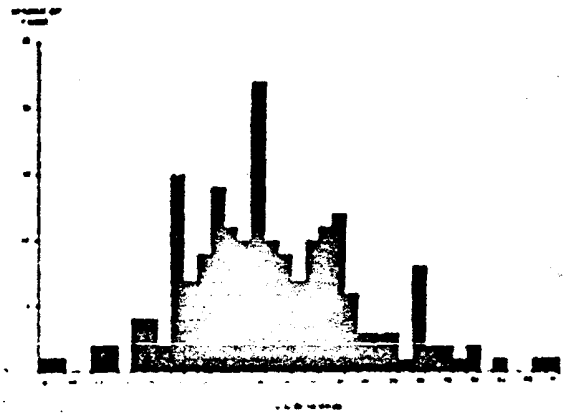

Fig. 3—Lead poisoning cases by age in months; 197 children under 4 years.

4 years of age during the 5 year period 1955-1960 arranged by month of age. Two, possibly 3, peaks of age involvement emerge —the yearlings, the 2 year-olds, and 3-year-old children. One is not sure whether these 3 represent different groups at risk, different causations, or different crops, so to speak, a year apart. These distributions tend to confirm the suggestion of Chisolm and Harrison that if the child isn't recognized in a particular summer he may not be recognized until the following summer.

Where cases are distributed according to race or ethnic group of sick patients (Table 3), Negroes contribute a disproportionately high share of the disease. Assuredly this is basically because they comprise a disproportionately high percentage of the residents in the "high risk" areas. However, there is reason also to suspect the contributary influence of a variety of personal, social, and economic factors which are as difficult to define epidemiologically as is race. Puerto Ricans form an interesting group to look at separately from the others. The age distribution of cases in this group ranges with few exceptions from a year and a half to 2 years of age, the age of greatest risk of lead intoxication from window-sill nibbling. Certainly, if there is one source of lead poisoning about which there is little room for doubt it is heavily leaded paint of the type used 25-50 years or more ago, and experience in this city and in other old cities

TABLE 3—*Lead Poisoning in Philadelphia, 1956-1960, by Race or Ethnic Group*

Yr.	W	N	P.R.	Total
[illegible]				
[illegible]				
[illegible]				
[illegible]				
[illegible]				
Total	[illegible]	[illegible]	[illegible]	[illegible]

such as Baltimore, Boston, and Chicago has established the hazard.

In Philadelphia a survey of leaded paints in 100 dwellings occupied by Puerto Ricans was conducted in 1958. This survey disclosed that 87% of the dwelling units occupied by this group contained at least one room in which the lead content of painted surfaces was above the permissible (1%) limit.

Comment

Much of the kind of data reported here is not new, such as the descriptive epidemiology of lead poisoning as a disease of toddling infants with a history of pica, the manifestations of which are most frequently discovered in the summer months. What is new, we believe, is the attempt to understand and to control lead poisoning in Philadelphia in the same way that epidemic infections were studied and controlled in the past through sustained cooperation and investigation by both private and official public health agencies and hospitals. Tuberculosis, for example, has been a focal point of cooperative effort on the part of the Philadelphia Hospitals, the Department of Health, and the Henry Phipps Institute for 50 years.

To the end of an improved control of the community disease the integrated application of clinical, laboratory, and epidemiologic methods is now being aimed at lead poisoning by a centralized reporting of cases, by spot-mapping and location of the regions and houses of greatest risk, by identification of populations at greatest risk, and finally through a series of epidemiologic and chemical studies being undertaken to determine the seasonal, geographical, socio-economic, and other factors at the back of observed distributions. In other words, the application of epidemiologic methods to the problem of lead poisoning in Philadelphia is being made in order to attempt control on a public health level by preventive measures rather than by relying on clinical efforts only, such as early diagnosis and treatment.

Summary and Conclusion

Enough has been accomplished by the reporting and investigation of cases of childhood lead poisoning to the Philadelphia Department of Health that reasonably clear indices of its endemicity and epidemicity are emerging. Cases are of the order of 50 per annum, and deaths in 1959 alone about doubled those of New Jersey's widely heralded outbreak of eastern equine encephalitis. These deaths are due to lead encephalitis, and, as with other forms of severe encephalitis, neurological sequelae are frequently devastating.

However, despite progress, the identification of specific hazards in an appreciable percentage of cases reported each year requires further study before effective prevention becomes an attainable reality. Lead paint in old dwellings is certainly a major menace. But both spot mapping and field investigation of reported cases suggest that other factors may be involved.

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