INCIDENCE OF HIGH BLOOD LEAD LEVELS IN CHICAGO CHILDREN

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ABSTRACT. The Chicago Board of Health in October 1966 began a mass-screening program using a blood lead test to detect lead poisoning in children. Atomic absorption spectroscopy made it possible to screen 5,000 specimens in 1 month, and to test a total of 68,744 children in 2 years.

The incidence of high blood lead values was variable and seasonal; it was lowest in November through January and highest in June. Control children exhibited the same seasonal variation in lead levels as did the children at-risk for lead poisoning.

As a result of this program, 1,154 children were treated with chelates for lead poisoning in 1967 and 1968 at the Lead Poisoning Clinic, and the incidence of high blood lead levels among children living in the same areas declined from 8.5% in 1967 to 3.8% in 1968. *Pediatrics*, **44**:661, 1969, LEAD POISONING, BLOOD LEAD LEVELS, ATOMIC ABSORPTION SPECTROSCOPY.

LEAD POISONING of children living in substandard conditions is a major public health problem. The size of the problem is unknown because most instances are neither discovered nor identified. Jacobziner[1] compared pediatric lead poisoning to an iceberg: the small visible portion represents the children with encephalopathy, and the larger, submerged portion represents the children with asymptomatic lead poisoning. The incidence in children cannot be assessed clinically because there are no specific clinical signs or symptoms. However, with atomic absorption spectroscopy (AAS) now available, the incidence of increased lead absorption can be established by the precise and accurate measurement of the concentration of lead in blood.

In October 1966 the Chicago Board of Health began a mass-screening program using an AAS blood lead test to detect lead poisoning in children. There were 72,237 children tested by December 1968. The purpose of this paper is to summarize the findings of this screening program for the years 1967 and 1968.

MATERIALS AND METHODS

Subjects

The children screened were from high-risk, low-income Urban Progress Center areas. Figure 1, a map of the City of Chicago, indicates the survey areas. Community representatives visited every family in designated blocks urging them to bring their children to the centers for a lead test.

The majority of the subjects were 1 to 6 years old. Controls were obtained by selecting the oldest children screened, i.e., children 10 to 14 years of age. They were selected because they were over the age group at-risk for lead poisoning. Physicians at the Urban Progress Centers drew blood samples which were heparinized and submitted to the laboratory for lead determination and hematocrit reading.

Lead Analysis

B-D disposable Plastipak 10 cc Luer-Lok syringes,* with 20 gauge, 1½ in. needles,

* Becton, Dickinson and Company, Rutherford, New Jersey.

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TABLE I

BLOOD LEAD SCREENING PROGRAM
1967–1968 POPULATION SUMMARY

Year	*Number of Children Screened*	*Number with Lead Concentration ≧50 μg/100 ml*	*Number Treated with Chelates*
1967	27,959	2,379	582
1968	40,785	1,556	572
Totals	68,744	3,935	1,154

were used for venipunctures. Blood samples were collected in B-D #L3200 10 ml vacutainer tubes* containing 2 drops of heparin. Samples were refrigerated from 12 to 60 hours at the urban progress centers until delivered to the laboratory.

Five milliliters of whole blood were used for analysis. Lead was concentrated by chelation with ammonium pyrrolidine dithiocarbamate and extracted with methyl isobutyl ketone. The lead concentration of the extract was determined by a Beckman atomic absorption unit† equipped with a single element (lead) hollow burner, lamp, air-acetylene laminar flow burner, DB-G spectrophotometer, and recorder. Details of the method used in this laboratory have been published.[2] The limit of sensitivity (A = 0.01) for detection of lead in blood was 1.5 μg per 100 ml blood or 0.015 ppm. The standard deviations of pooled samples with means of 23.4 μg lead per 100 ml blood and 76.5 μg lead per 100 ml blood (with lead added) were ± 0.7 and ± 1.0, respectively. Coefficients of variation were 3.2% and 1.4%, respectively.

A single blood or urine sample requires approximately 15 minutes for processing; but, after the sample is extracted, the result is available in 20 seconds. One technician operating an AAS unit can determine the lead content of 900 sample extracts in 8 hours; however, in addition to the technician operating the AAS unit, the personnel required for the entire laboratory procedure includes four technicians to process the samples and to read and record results, one clerk, and one dishwasher. The cost of the materials is about 25¢ per test.

† Model 979, Beckman Instruments, Inc., Fullerton, California.

TABLE II

DATA OF SURVEY AREAS FOR CHICAGO BLOOD LEAD SCREENING PROGRAM—1967–1968

Urban Area	*A*	*1967*				*1968*				*Totals for 1967–1968*			
	Number of Children Under 5 Years (1960 Census)	*B Number Tested**	*% of A*	*Number with high lead†*	*% of B*	*C Number Tested**	*% of A*	*Number with High Lead†*	*% of C*	*D Number Tested**	*% of A*	*Number with High Lead†*	*% of D*
1	10,000	2,500	25%	157	6.3%	3,100	31%	53	1.7%	5,600	56%	210	3.8%
2	15,100	1,700	11%	73	4.3%	1,800	12%	56	3.1%	3,500	23%	129	3.7%
3	22,000	2,000	9%	262	13.1%	3,000	14%	166	5.5%	5,000	23%	428	8.6%
4	22,300	5,200	23%	509	9.8%	5,500	25%	270	4.9%	10,700	48%	779	7.3%
5	13,700	4,100	30%	316	7.7%	4,900	36%	142	2.9%	9,000	66%	458	5.1%
6	24,500	6,600	27%	393	6.0%	7,700	31%	277	3.6%	14,300	58%	670	4.7%
7	20,500	3,400	17%	432	12.7%	7,000	34%	356	5.1%	10,400	51%	788	7.6%
8	15,300	2,200	14%	218	9.9%	6,100	40%	227	3.7%	8,300	54%	445	5.4%
9	4,500	300	7%	19	6.3%	1,700	38%	9	0.5%	2,000	44%	28	1.4%
Totals	147,900*	28,000*	19%	2,379 = 8.5%		40,800*	28%	1,556 = 3.8%		68,800*	47%	3,935 = 5.7%	

* Figures given to the nearest hundred.
† High lead=50 μg lead/100 ml whole blood or higher.

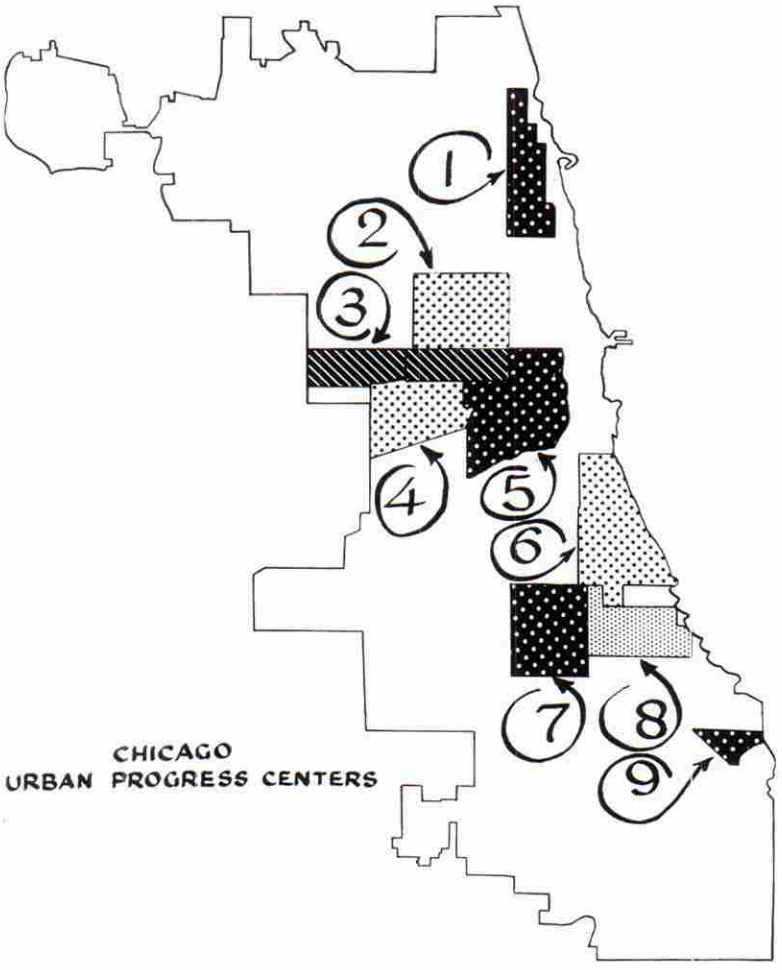

FIG. 1. Areas screened for lead poisoning in Chicago.

Hematocrit Reading

A hematocrit reading is necessary for the interpretation of the lead result because more than 90% of the lead is bound to the erythrocytes. Fire-sealed capillary tubes were centrifuged in microhematocrit centrifuges and read in a hematocrit capillary tube reader.

RESULTS

Blood lead determinations were performed on 68,744 slum area children: 27,959 in 1967 and 40,785 in 1968 (Table I). Of the 68,744 children, 5.7% (3,935) had lead values over 49 μg per 100 ml blood. Children with elevated lead values were referred to a special clinic established by the Chicago Board of Health for the diagnosis and treatment of lead poisoning. Twenty-nine percent (1,154) of the referrals were treated with chelates; the remainder were followed with repeat blood lead determinations until their lead level returned to normal. There were no deaths from lead poisoning among the 68,744 children tested in 1967 and 1968.

The number of samples sent to the laboratory each month varied from about 2,000 to 5,000. The percentage of blood samples with lead levels over 49 μg per 100 ml each month is given in Figure 2. During 1967 the percentages of high lead values rose from 5.7% in January, with small peaks in February and April, to a high peak of

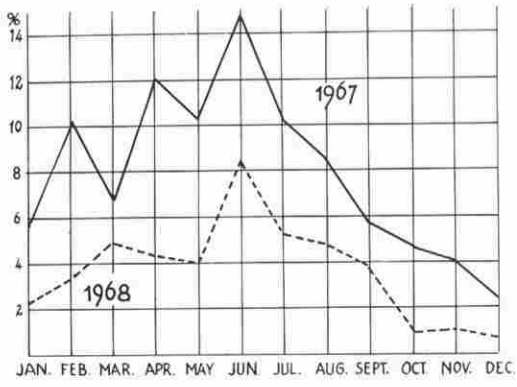

FIG. 2. Percent of blood samples with lead ≧ 50 μg/100 ml.

14.8% in June; they then declined to a low of 2.3% in December. Percentages for 1968 followed a similar seasonal pattern, with a high peak in June. In general, 1968 percentages were down more than 50% from those in 1967.

Figure 3 shows that the mean blood lead levels in 1967 also followed a seasonal pattern. Various authors[3-7] state that the upper limit of "normal" blood lead concentration is 80, 60, 40, 36, or 20 μg per 100 ml. This range of figures indicates that norms should be established, if possible, for our own population. The oldest children screened (10 to 14 years) were selected as a control group for our population because they are above the age at-risk for lead poisoning. There were 746 subjects in the 10- to 14-year range. Figure 3 compares the 1967 monthly mean lead levels of the 746 children in the older control group with the averages found for approximately 21,000 children under 6 years of age, i.e., the age group at-risk for lead poisoning. Both groups had high means in February and June, with low means in November, December, and January. In contrast to the variations in lead content, hematocrit readings did not show a seasonal pattern; the hematocrit means were nearly identical each month.

The lead concentration of the 746 controls for the entire year ranged from 5 to 55 μg per 100 ml, with a mean of 23.5 ± 7.8 μg lead per 100 ml blood. Control children appear to exhibit the same seasonal variation in lead values as children in the age group at-risk for lead poisoning.

Table II summarizes the data of the nine communities illustrated in Figure 1. The total number of children living in these areas of the age groups at-risk for lead poisoning is about 150,000. Table II shows that 19% of the 150,000 were screened in 1967 and 28% in 1968. Because of the small percentage of children tested in 1967, there was no re-screening program. The number of "repeaters" was insignificant, so a total of 47% of the children living in these sections were screened in 2 years. The residence and sex distributions were similar for both years, but the age distribution of the children tested in 1968 was more strictly limited to children under 6 years of age.

The incidences of elevated lead levels differed among the various areas (Table II). They ranged from 6.0% (area 6) to 13.1% (area 3) during 1967 and from 0.5% (area 9) to 5.5% (area 3) during 1968. Percentages for the 2-year period by areas were: area 1, 3.8%; area 2, 3.7%; area 3, 8.6%; area 4, 7.3%; area 5, 5.1%; area 6, 4.7%; area 7, 7.6%; area 8, 5.4%; area 9, 1.4%.

Other diagnostic tools were not as satisfactory for detecting lead absorption as the AAS blood lead determination. Of the 582 children treated with chelates in 1967, the percentages of positive results were: blood lead content 50 μg/100 ml or higher (100%), skeletal x-ray (79%), abdominal x-ray (35%), history of pica (79%), and presence of symptoms of lead poisoning (12%).

DISCUSSION

Jacobziner[1] has shown that a successful search for children with lead poisoning requires the routine use of blood lead determinations and that the number of diagnosed cases increases in proportion to the number of blood lead determinations performed. Until atomic absorption spectroscopy (AAS) became available, there was no rapid, specific method for the routine determination of lead in large numbers of biological samples. The simplicity and spec-

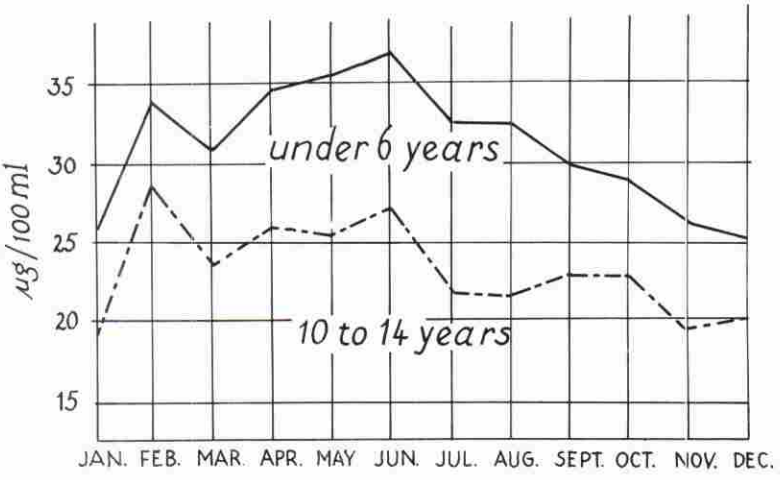

FIG. 3. Mean lead values in children in 1967.

ificity of AAS for the determination of the lead content of blood and urine makes it highly suitable for mass-screening programs. AAS makes it possible to test more specimens in 1 month than other methods could test in 1 year, e.g., the New York Department of Health using a method other than AAS, performed 3,113 blood lead estimations in 1964;[1] our laboratory, with AAS analyzed 5,600 specimens in the month of July 1968.

Although there are 3,000 atomic absorption spectrophotometers in use in the United States and about 10 million determinations were done in 1968 by AAS,[8] we do not know if these determinations are being used for mass-screening programs. As far as we know, the Chicago program is the largest mass-screening lead detection program in the United States.

When blood lead determinations are done on a neighborhood, door-to-door, mass-screening program, several goals may be achieved:

1. Discovery of children with incipient intoxication before they reach severe, symptomatic stages. A blood lead determination can detect these children before the onset of encephalopathy because they ingest lead-containing paint and plaster several weeks or months before overt symptoms develop. Early recognition and treatment of these asymptomatic children will reduce the mortality rate from lead poisoning.

2. Identification of children who are mild or temporary lead ingesters. These children can be followed with repeat lead determinations until their blood lead content returns to normal or rises to a point where chelation therapy is needed.

3. Reduction of the incidence of lead poisoning through participation and education in mass-screening programs.

Children with values between 40 to 60 µg per 100 ml may be: (1) beginning ingesters whose lead concentration is increasing, or, (2) previously poisoned children whose lead concentration is declining towards normal. The problem of increasing or decreasing lead content is solved by two techniques: obtaining evidence of increased body burden with the EDTA mobilization test[9] and following the blood-lead content at intervals with blood lead determinations.

All procedures, including the EDTA mobilization test and treatment, were managed in the Lead Clinic on an outpatient basis. Details of the clinic operation will be discussed in a subsequent article.

The mean blood-lead concentration of children screened in 1967 who were 10 to 14 years old was 23.5 ± 7.8 µg lead per 100 ml blood. This is similar to the figures given by Chisolm[10] for blood-lead concentrations

found in persons without abnormal or excessive exposure to lead, i.e., the median value is approximately 27 μg and the upper limit is 40 μg lead per 100 gm of whole blood.

While the incidence of high lead values was expected to be variable and seasonal, it was not anticipated that the control groups would exhibit the same seasonal pattern. If this variation is confirmed by future studies, it may be that the "normal" blood lead concentration varies seasonally—perhaps 19 μg per 100 ml from October through January, 23 μg per 100 ml from February through May, a peak of 26 μg per 100 ml in June, and a steady decline to the lower winter values.

The incidences of elevated blood lead values found in the present study are not strictly comparable to those reported by other groups[1,5,6,11,12] for two reasons:

1. the large number of children (68,800) who had a blood lead determination is unique, and,

2. the lead determinations were done without prior screening, i.e., the children were not tested because they had a history of pica or abnormal urinary findings, or because they were suspected cases of lead poisoning. The incidences of elevated blood lead values reported by Moncrieff, *et al.*[5] and Gibson, *et al.*[6] (45% of 122 children and 15% of 40 children, respectively) were in mentally defective children. Bradley and his associates[11] (44% in 333 children), Griggs, *et al.*[12] (34% in 83 children), and Jacobziner[1] (37% of 914 children in 1963 and 16% of 3,113 children in 1964) found these incidences in children with clinical or laboratory evidence or a history suggestive of increased lead exposure.

It is interesting that the percentage of children with high lead values in New York decreased from 37% in 1963 to 16% in 1964,[1] a drop of 57%. This is similar to the decrease in incidence in Chicago children from 8.5% in 1967 to 3.8% in 1968, a drop of 55%. Griggs, *et al.*[12] comment that there was a marked decrease in the number of positive histories of paint ingestion in Cleveland between the first and second years of their study.

SUMMARY

In 1967 and 1968 the Chicago Board of Health tested 68,744 slum area children in a mass-screening program to detect lead poisoning. The blood lead concentration was determined by atomic absorption spectroscopy. Three thousand nine hundred and thirty-five (5.7%) of the children tested had elevated blood lead values, and 1,154 (29%) of these children were treated with chelates.

There was an incidence of high blood lead values in every month, but it was variable and seasonal. The values were lowest in November through January and highest in June.

The control group, from the same areas as the children with high values, had a mean of 23.5 ± 7.8 μg lead per 100 ml whole blood, and they appeared to exhibit the same seasonal variation in blood lead levels as children who were at-risk for lead poisoning.

The incidence of high blood lead levels among children living in the same areas declined from 8.5% in 1967 to 3.8% in 1968.

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