INTEROFFICE MEMORANDUM

FULLER OBRIEN PAINTS

Please Route: 6/29

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TO: Jerome Crowley, Jr.
Dick Garabedian
Paul McWhirr
Cecilia Stoddard

DISTRIBUTION:

FROM: Mark Allen

LOCATION: SSF

DATE: June 29, 1989

SUBJECT: California Study of Childhood Lead Poisoning

Andy Doyle of NPCA obtained the enclosed report on lead poisoning of northern and southern California school children. This interim report was reported here in the press about a month ago. Even though the response to it seems very quiet, this is a study that we need to consider very seriously. Court cases in Louisiana and New York have given our industy much of the responsibility (and associated negative public relations) to correct this very old problem.

Boston Lead

The report is for your information. However, we should begin planning now a response to this study that considers both the paint industry's view and O'Brien's, if different. Also, we should begin to plan any actions we think are needed in response to these reports.

MA:bsk

Please feel free to copy the report for yourself should you desire, but return original to me when finished. Thank you.

MA

Pursuant to
Chapter 481, Statutes of 1986
Government Code, Section 309.7

Interim Report to the California State Legislature

CHILDHOOD LEAD POISONING IN CALIFORNIA

Causes and Prevention

George Deukmejian
Governor
State of California

Clifford Allenby
Secretary
Health and Welfare Agency

Kenneth W. Kizer, M.D., M.P.H.
Director
Department of Health Services

April 1989

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GLOSSARY OF ABBREVIATIONS

ARB	Air Resources Board
ATSDR	U.S. Agency for Toxic Substances and Disease Registries
CDC	U.S. Centers for Disease Control
CHDP	Child Health and Disability Prevention Program
CLPPP	Childhood Lead Poisoning Prevention Program
COHP	California Occupational Health Program
CPSC	Consumer Products Safety Commission
DHS	Department of Health Services
EP	Erythrocyte Protoporphyrin
EPA	U.S. Environmental Protection Agency
HUD	U.S. Housing and Urban Development
µg/dL	micrograms per deciliter
NHANES	National Health and Nutrition Examination Survey
ppm	parts per million
WIC	Women, Infants and Children Program
XRF	X-ray fluorescence

I. INTRODUCTION

A. Background

Although the hazards of high lead exposure for young children have been well documented for decades, recent research has revealed deleterious health effects at progressively lower concentrations of lead exposure. The U.S. Agency for Toxic Substances and Disease Registries (ATSDR) has estimated three to four million children nationwide are currently at risk from lead exposure at concentrations which place them at risk of adverse health effects (1).

Recognizing the situation, California lawmakers in 1986 enacted a law, commencing with Section 309.7 of the Health and Safety Code (Appendix A) that established the Childhood Lead Poisoning Prevention Program (CLPPP) and mandated that the Department of Health Services (DHS):

- conduct childhood lead screening programs in three different geographical areas of the state (urban northern, urban southern, and rural central valley) that will permit the estimation of the extent and causes of childhood lead poisoning in high-risk target areas in California;

- analyze information collected, design and implement a program of medical follow-up and environmental abatement and follow-up that will reduce the incidence of excessive childhood lead exposures in California;

- register cases of elevated blood lead levels, defined as 25 or more micrograms of lead per deciliter [≥25 μg/dL], and 35 or more μg/dL erythrocyte protoporphyrin (EP) levels [≥ 35 μg/dL] in children and adults reported by law to the DHS by clinical laboratories; and

- submit a policy report containing CLPPP findings to the Legislature with recommendations for the future prevention of childhood lead poisoning in California.

As explained below, the work has not been completed for the final report. Accordingly, this interim report is submitted to the Legislature in compliance with the statute. It contains a summary of the scientific findings and recommendations to date of the Department of Health Services. A final report will be submitted in 1990.

Collection of blood specimens and environmental samples (paint, dust, and soil) has been completed for selected urban neighborhoods of northern (Oakland) and southern (Compton and Wilmington) areas of the state, enabling an assessment of the effects of environmental lead levels on blood lead concentrations in children. Completion of the Oakland study was delayed by a problem with a high rate of false-

positive blood results which required extensive follow-up (described further in Section IV). In addition, a small pilot study was conducted examining water lead levels in one residential area. Since laboratory analysis has not been completed on some environmental samples collected in the southern area, most of this report focuses on analytical results from the Oakland area.

For both areas, the final legislative report in 1990 will examine the effect of residential proximity to freeways, industrial sources, and other factors assessed using a comprehensive questionnaire on childhood blood lead. In addition, field work for the Central Valley study will be conducted in 1989-90. The combined results of all three studies will be submitted in the final report.

The laboratory reporting system called for in the Act has been fully implemented.

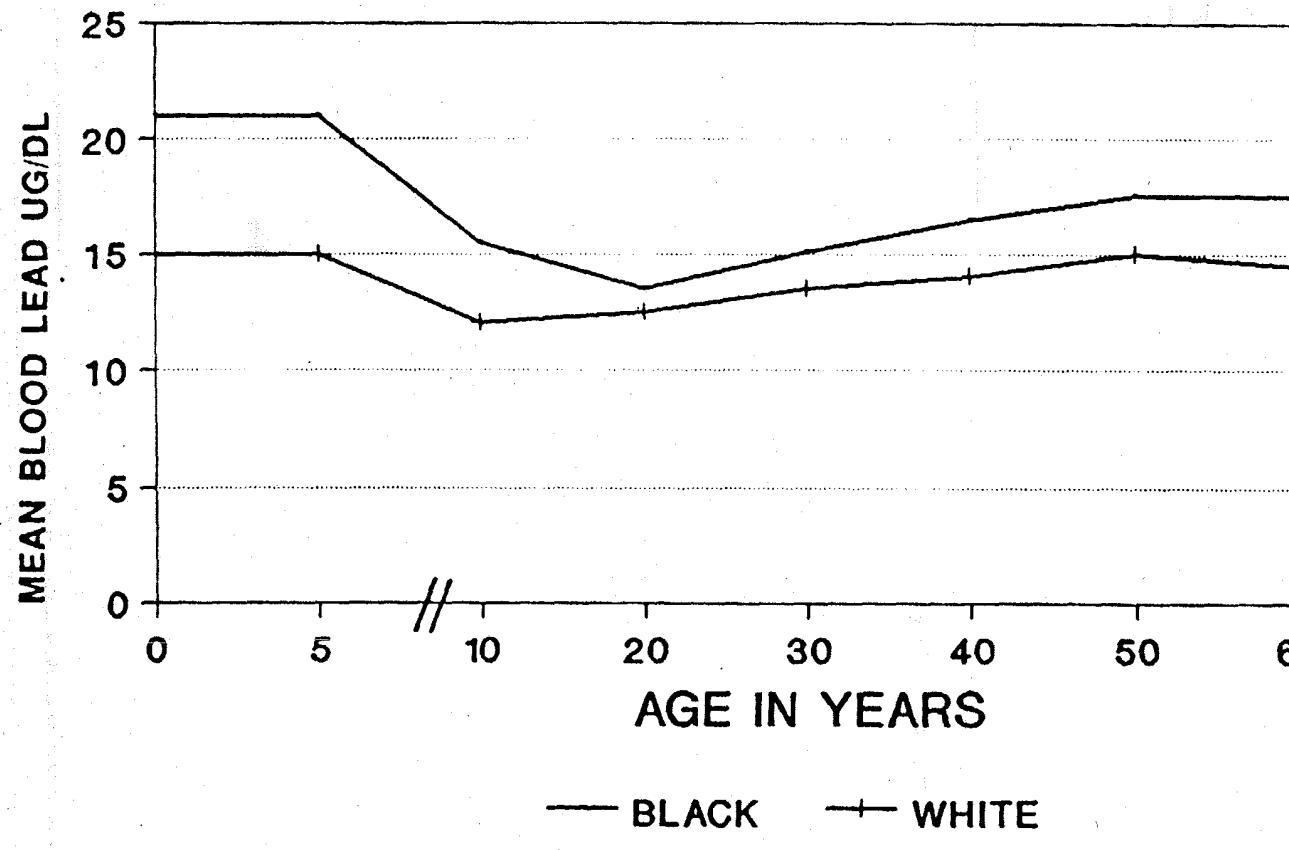
B. Prevalence and Sources of Lead Exposure to Children

1. Who is at Risk?

Although adults are also exposed to environmental lead, young children are at greater health risk for lead exposure because they are able to absorb proportionally more lead from oral ingestion and because normal childhood behavior facilitates contact with lead. Small amounts of lead may be ingested by children via normal hand to mouth behavior. Children absorb about 50% of the lead they ingest compared to a rate of 10% in adults (6). The nutritional status of a child can also affect lead absorption. Research by Mahaffey and others has shown that children who are iron deficient exhibit greater lead absorption and higher mean blood lead levels than children who have normal iron status (15).

Mean (average) blood lead levels vary by age, sex, income, and ethnicity (24). Figure 1 from the National Health and Nutrition Examination Survey (NHANES) shows that blood lead levels in the U.S. population are most elevated among children below the age of 6, are lower between age 6-15 and increase after the age of 15. There is a strong relationship between income and mean blood lead levels among whites, Hispanics, and blacks (13, 24). Figure 2 demonstrates lower-income children in all three groups are at greatest risk for lead poisoning.

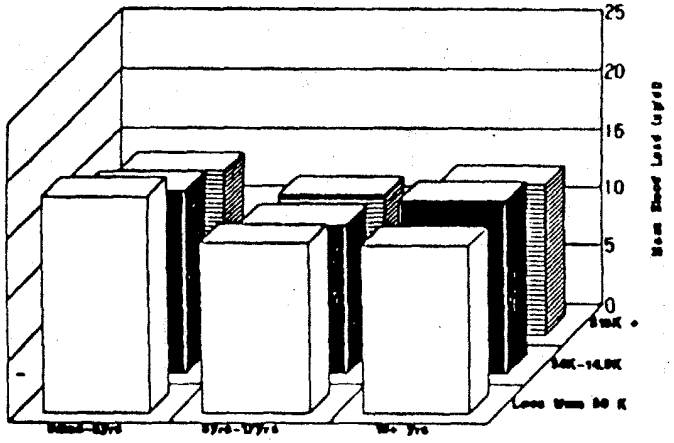
Fig. 1 Blood Lead Levels by Race and Age, United States, 1976-80



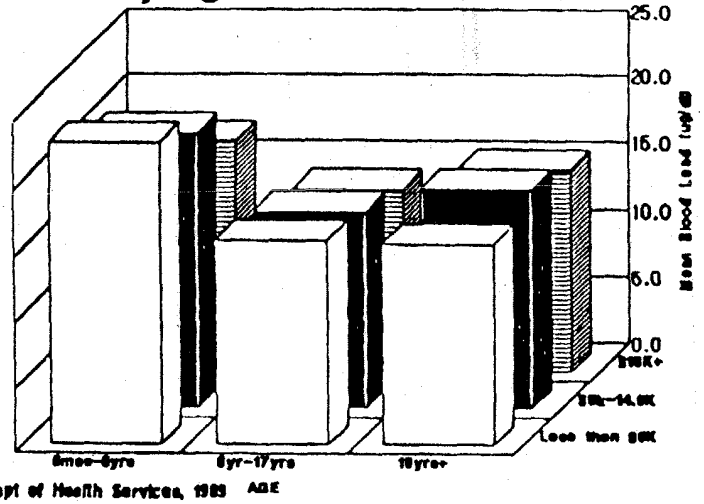
NHANES, 1983

Fig. 2 Mean Blood Lead Levels by Race, Income and Age

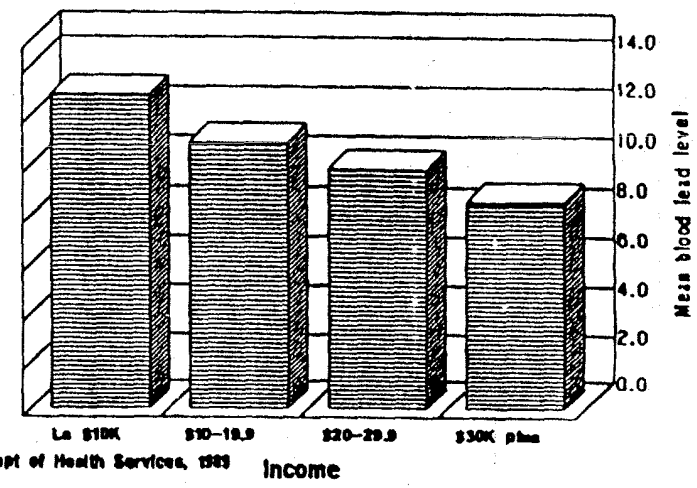
Mean Blood Lead Levels in Whites by Age and Income, NHANES II



Mean Blood Lead Levels in Blacks by Age and Income, NHANES II



Mean Blood Lead Levels by Income for Hispanic Children Age 4-11, HANES



2. What are the Sources?

Unavoidable exposures to lead come from food, water, air, and dust (3). Lead from these sources is called "baseline" exposure. Myriads of other environmental sources pose a potential for human exposure to lead in addition to baseline exposures. Table 1 summarizes potential exposures to lead from a number of common sources for U.S. children.

Table 1: Estimates of Children Under Age 5 Exposed to Lead in the United States

Leaded paint	12.0 million
Dust/Soil	5.9-11.7 million
Leaded gasoline	5.6 million
Water	2.5 million
Primary/Secondary Smelters	0.23 million

(ATSDR, 1988) (1)

- **Lead Paint:** Lead-based paint is a highly concentrated source of lead exposure. Before the amount of lead in paint was controlled by law in 1977, some interior paints contained more than 50% (500,000 parts per million [ppm]) lead (6). When ingested by a toddler, a small paint chip containing 50% lead can produce acute poisoning (1). Children may be exposed to lead in paint directly by ingesting paint chips or by dust contaminated with lead from paint (1, 7, 19, 21). The ATSDR (1) estimates that 3 million tons of lead probably remain in paint accessible to children in the U.S.

  In the late 1950's, manufacturers voluntarily limited the lead content of paint to one per cent (10,000 ppm), but paint in excess of this continued to be produced until 1977, when the amount of lead in household paint was limited to 0.06% (600 ppm) by the Consumer Product Safety Act.

- **Soil and Dust:** Lead in soil and dust is primarily derived from lead paint on houses, automobile exhaust from leaded gasoline, and industrial emissions. It is difficult to estimate the number of children exposed to these sources since no national databases exist. Although there is a wide range of naturally occurring lead in soil, the average lead content in the earth's crust is 15 ppm (12). In soil collected by the U.S. Geological Survey from nearly 1,000 locations throughout the U.S., lead content varied from less than 10 ppm to 700 ppm. Six percent of these samples contained more than 30 ppm of lead (12). Lead is very persistent and relatively immobile in soil under normal pH conditions (normal acidity). In 1979, Doner estimated the mean residence time for lead in California soil to be between 10,000 and 20,000 years (9).

- **Leaded Gasoline:** Data from NHANES (2) found an association between declines in the national blood lead levels and the reduction of lead in gasoline required by U.S Environmental Protection Agency regulations which became effective in 1975. Although leaded gasoline use is restricted, there are still a large number of motor vehicles that use it. In addition, lead deposited in the environment from past leaded gasoline use is still present.

C. Toxicity of Lead

The toxic properties of virtually no other substance have been studied as extensively as those of lead. Lead provides no known physiologic or metabolic benefit. It adversely affects many organs and metabolic systems. The primary target organ for lead toxicity is the brain or central nervous system, and the effects are especially damaging during early childhood development. Relatively low levels of lead in children can produce delayed cognitive development, impaired hearing (22), and reduced IQ scores (4, 5, 10, 11, 16-18, 20, 26, 28). In addition, growth may be inhibited (23). Higher levels of lead poisoning can produce several non-specific symptoms, including fatigue, irritation, sleep disturbance, and constipation. In more severe cases, lead poisoning can cause symptoms of acute abdominal colic, anemia, acute and chronic encephalopathy (brain damage), and chronic nephropathy (kidney damage). Lead has been shown to interfere with hematological (blood) and hepatic (liver) enzymes systems. Acute renal (kidney) effects are seen in severe lead poisoning cases and may lead to chronic nephritis or hypertension. It has long been known that lead can induce stillbirths, spontaneous abortions, neonatal deaths, and sterility.

Undue exposure to lead is undesirable for all, but it is particularly hazardous to children. Children, because of differences in metabolism and excretion of lead, are intrinsically more vulnerable to lead toxicity than are adults. The subtle neuropsychologic effects of lead at low concentrations in children are of serious concern. Presently, the Centers for Disease Control (CDC) consider a blood lead level of 25 µg/dL or greater for children as elevated (6), but a lower level of 15 µg/dL is under consideration. Table 2 outlines lead effects in children at increasing blood lead concentrations.

Table 2

LOWEST OBSERVABLE EFFECT BLOOD LEAD LEVEL (Pb-B) FOR EFFECTS IN CHILDREN[a]

Lowest Effect Blood Lead (µg/dL)	Neurological Effects	Heme Synthesis Effects	Other Effects
10-15 (prenatal & postnatal)	Deficits in neurobehavioral development; electrophysiological changes	ALA-D* inhibition	Reduced gestational age and weight at birth; reduced size up to age 7-8 years
15-20		EP** elevation	Impaired vitamin D metabolism; Py-5-N* inhibition
<25	Lower IQ, slower reaction time (studied cross-sectionally)		
30	Slowed nerve conduction velocity		
40		Reduced hemoglobin; elevated CP* and ALA-U*	
70	Peripheral neuropathies	Severe anemia	
80-100	Encephalopathy		Colic, other GI effects; kidney effects

[a]Adapted from U.S. EPA (25), with updating.
* A metabolic enzyme
** Erythrocyte Protoporphyrin

II. LABORATORY REPORTING OF ELEVATED BLOOD LEAD

A. Reporting of Cases: Children

In accordance with the Childhood Lead Poisoning Prevention Act, DHS established a statewide lead poisoning surveillance system. The surveillance system collects demographic and medical information on California residents with possible blood lead toxicity. The law requires that all laboratories submit a Lead Reporting Form to DHS and the local health department for any blood lead result at or above the level of 25 µg/dL or any EP result at or above 35 µg/dL. The EP test is used to screen for lead toxicity, anemia, and various hematologic (blood) abnormalities. It indicates the toxic effects of lead on heme synthesis (red blood cell production). Blood lead levels measure how much lead is currently in the blood stream.

Childhood lead screening is not required in California, and only one clinic in the state has been identified as doing regular lead screening of children. The CDC has established risk classifications and follow-up guidelines based on the results of blood lead and EP tests (6). The risk classification system is used for interpreting test results and prioritizing cases for follow-up. CDC's risk classifications are used in this report. (These classifications are listed in Table 5.)

DHS received 550 reports on 466 children in the first year of operation, from April 1987 through April 1988. Most of the reports were submitted because of elevated EP levels, without accompanying blood lead results, and just over half of all reports included blood lead results (Table 3). Of these, almost 75% were below the reporting standard of 25 µg/dL. Because only a few sources in five counties generated 66% of all reports, the geographic distribution of reports is greatly skewed and the populations served by these sources are highly over-represented among the reports (Table 4). According to CDC risk classifications, 2.6% (14) of the reports received had blood lead and EP levels high enough to be classified as being at moderate risk for lead toxicity, 1% (6) were at high risk, and 1% (5) were at urgent risk (Table 5). For reports of children at high and urgent risk of lead toxicity, DHS called local health departments, physicians and families to ensure that appropriate follow-up occurred.

The number of reports submitted to DHS was much lower than anticipated. Even given very low hypothetical prevalence rates for lead exposure in California children, it would be expected that if all children at risk were screened, identified and reported, thousands of children annually would be reported with blood lead at 25 µg/dL or greater. DHS contacted pediatric providers, the Child Health and Disability Prevention (CHDP) program and lead-proficient laboratories and found that no comprehensive population-wide lead screening is currently done for California children. In practice, EP determinations are used by physicians primarily to measure iron deficiency and other hematologic effects rather than to diagnose lead toxicity. EP does not correlate well with blood lead at levels under 40 µg/dL. Only 8.5% of the reports to the reporting system were blood lead levels alone. Forty percent of the reports were for elevated EP's with blood lead levels under 25 µg/dL.

Table 3. Number of Elevated (≥35 µg/dL) Blood Tests Reported by Method of Analysis April 1987 to April 1988

California Department of Health Services, 1989

Tests Done	Number	Percent*
EP** by Hematofluorometer Only	227	41.3
EP** by Extraction Only	21	3.8
Blood Lead Only	56	10.2
Blood Lead & EP** by Hematofluorometer	10	1.8
Blood Lead & EP** by Extraction	36	6.6
EP** by Hematofluorometer & EP** by Extraction	7	1.3
Blood Lead & EP** by Hematofluorometer & EP** by Extraction	193	35.1
TOTAL	550	100.00

Includes multiple reports on the same individuals (466 Children)

* Sums to 100.1 due to round-off error

** EP = Erythrocyte Protoporphyrin

Table 4. Origin of Elevated Lead Case Reports
April 1987 to April 1988
California Department of Health Services, 1989

County	Blood Lead ≥25 µg/dL	EP* > 35 µg/dL & Blood Lead < 25 µg/dL	EP* > 35 µg/dL No Blood Lead Reported	TOTAL
Santa Clara	15	126	9	150
Los Angeles	9	1	65	75
San Diego	5	0	68	73
Alameda	8	37	5	50
Fresno	1	3	15	19
Orange	1	7	6	14
Contra Costa	0	4	4	8
Imperial	0	0	6	6
San Mateo	0	0	6	6
San Francisco	0	0	6	6
Solano	0	0	5	5
All Others	7	11	36	54
TOTAL	46	189	231	466

* EP = Erythrocyte Protoporphyrin

Table 5. Risk Classification of Elevated Blood Tests Reported
April 1987 to April 1988
California Department of Health Services, 1989

CDC* Risk Classifications	Number of Blood Tests	Percent
Not Classified EP** Only - Blood Lead Not Done	255	46.4
Ia - Low Risk Probable Iron Deficiency	216	39.2
Ib - Low Risk Transient, Stable or Changing Blood Lead Levels	51	9.2
Erythropoietic Protoporphyria	3	0.6
II - Moderate Risk	14	2.6
III - High Risk	6	1.1
IV - Urgent Risk	5	0.9
TOTAL	550	100.0

Includes multiple reports on same individual (466 children)

* U.S. Centers for Disease Control

** EP = Erythrocyte Protoporphyrin

B. Reporting of Cases: Adults

Numerous reports on adults have been sent to DHS. The DHS California Occupational Health Program (COHP) has the responsibility for the investigation and follow-up of these reports. Adult lead poisoning has important implications for children because of the possibility of lead from the adult's workplace being brought home on clothing and thereby exposing children and pregnant women.

In the first 18 months of operation (April 1987 through September 1988), nearly 8400 reports on about 2700 adults were received and analyzed by COHP. For the vast majority of these reports (8103, or 95%), elevated blood lead levels were due to occupational exposures (14). 8.9% of these cases had blood lead levels over 50 µg/dL and could be considered urgent medical situations in that there is a risk of permanent disability and, as stated above, of possible household contamination of children and pregnant women from "take-home" exposures from the workplace. An additional 17% of cases had blood lead levels between 40-50 µg/dL.

A blood lead level of 40 µg/dL or greater in adults may cause or contribute to brain and nerve damage, reproductive failure, kidney impairment, high blood pressure, and other adverse health effects. Virtually all of these reports involved individuals with well-known occupational exposures in such worksites as lead battery plants, lead smelters, lead or brass foundries, gun firing ranges, radiator repair shops, and demolition work or painting of structures with lead paint. 79% of reported cases were for men and women in reproductive age groups (under 50). Many (46%) of these cases were Hispanic-surnamed.

In the course of conducting telephone follow-up for cases over 50 µg/dL, COHP staff found that many patients, physicians, and employers were unaware of the health hazards of lead, or of appropriate measures to be taken for case management. There is clearly a potential for "take-home" exposures. However, no one has studied the situation thoroughly enough to know to what extent children and pregnant women are exposed to lead inadvertently brought home on clothing from work, or what their levels of exposure might be.

III. IDENTIFICATION OF HIGH RISK TARGET AREAS IN CALIFORNIA

Pursuant to the CLPP Act, DHS developed a set of criteria which were used in selecting geographical target areas for study. These criteria included:

- proportion of pre-1950 housing in excess of 60%;
- children aged 1-5 living in at least 10% of homes;
- presence of industrial lead emitters;
- proximity to freeways and arterials;
- ethnic factors predisposing to lead exposure (such as the use of home remedies containing lead); and
- household income.

These criteria also have been applied in estimating the numbers of children in California who may be at risk for lead poisoning by virtue of residing in these areas.

The CLPPP has completed two epidemiologic studies thus far in high risk target areas. The first study was conducted in Oakland and the second was conducted in the Wilmington and Compton areas of Los Angeles County. In the Oakland target area, 70% of the housing was built before 1950. Demographic analysis was used to identify these areas using the above criteria. The Alameda County and Los Angeles County health departments were consulted regarding the likelihood of these areas containing sources of lead. Air Resources Board (ARB) data on industrial lead emitters were used to identify a number of major air emission point sources in the Wilmington and Compton areas.

Similar high risk areas have been identified as possible study sites in Sacramento, Bakersfield, and Fresno. It is anticipated that a survey will be conducted in the Central Valley during fiscal year 1989-90.

IV. SCREENING IN TARGET AREAS

A. Methods

1. Sample Selection and Door to Door Survey:

551 children under the age of six in Oakland and 487 children in Los Angeles were tested for lead poisoning in their homes. Prior to testing, a complete census, which enumerated the children in the target areas, was taken to ensure maximum participation. Environmental samples were collected at each home to look at lead levels in paint and soil. For participating children, blood was drawn in the home by capillary fingerstick or by venipuncture and analyzed for lead and EP. Parents were interviewed using a questionnaire to analyze factors that might be related to lead exposure.

2. Environmental Exposure Assessment - Data Collection:

At each surveyed house, samples of soil, interior paint and exterior paint were collected. Soil samples were collected at each house from the front, side, and rear yards as well as from under the rain drain. The front yard and rear yard samples were composited samples taken in an effort to obtain a 'representative' exposure sample. In the Los Angeles study, a sample of housedust was also taken by vacuum cleaner. In the Oakland area, a small study of tap water lead was conducted. Six samples were collected from each of eleven homes. In addition, field staff rated the physical condition of the housing in its interior and exterior as well as any surrounding buildings. Results of soil and paint analysis for Los Angeles are not yet available.

3. Population Characteristics: Ethnicity, Health Care and Income:

In Oakland, 54% of the children studied were Hispanic, 26.3% were Asian, 12.9% were black, and the remaining 6.7% were white and other ethnicities (Figure 3). In Los Angeles, 90% of the subjects were Hispanic. In Oakland, 33% of the study participants were covered by the Women, Infants and Children (WIC) program, 42% were covered by Medi-Cal, and 20% had no health insurance at all. Of the participating families, 70% had total family incomes under $15,000 per year and 20% were unemployed.

4. Methods of Analysis:

All samples collected for lead (blood, soil, and paint) were analyzed using atomic absorption spectrophotometry. EP was analyzed using a hematofluorometer.

Data were analyzed using descriptive univariate and bivariate analyses and multivariate linear regression modelling. The dependent variable of interest was blood lead. Independent variables were chosen based on either past research suggesting an association with blood lead or on researcher interest. The analysis first examined the distributions of variables of interest such as blood lead, EP, paint and soil lead, ethnicity, income, and other demographic and environmental

variables. Further analyses examined the relationship between blood lead levels and other variables of interest.

Using the 1980 census, other census tracts in the state in which at least 60% of housing was built before 1950 were identified. The population of children under age six in these areas was used to project the total number of children in the state with lead exposures due to living in these areas. Projections were done using linear extrapolation.

B. Study Results

1. Blood Lead Levels:

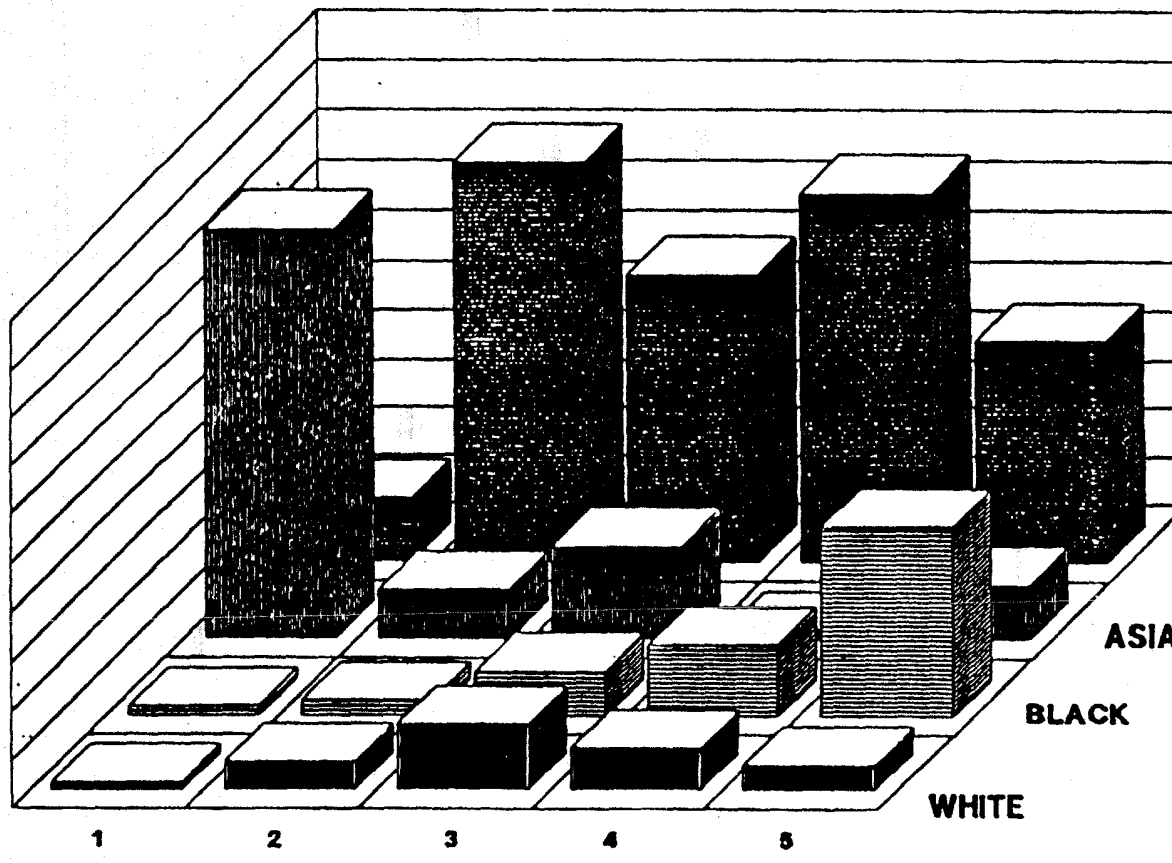
The surveys had response rates of 80% in Oakland and 82% in Los Angeles. The average blood lead level in Oakland was 11.5 µg/dL with a range from 3 to 43 µg/dL. In Wilmington the mean blood lead level was 11.1 µg/dL with a range from 7 to 36 µg/dL. In Compton the mean blood lead level was 10.5 µg/dL, ranging from 7 to 32 µg/dL. Males had significantly higher mean blood lead levels than females, and children age 36-41 months had higher mean blood lead levels than other children (Figure 4). Those with the lowest income and least access to medical care had the highest blood lead levels. Those with no health insurance or with Medi-Cal coverage had higher blood lead levels than those with private health insurance coverage (Figure 5).

Although the standard for intervention on childhood blood lead levels is presently 25 µg/dL and above, evidence in the last 10 years indicates adverse health effects may occur at 15 µg/dL. In both Oakland and Los Angeles, 1.3% of the children examined had blood lead levels ≥25 µg/dL. In Oakland and Los Angeles respectively, 19.1% and 20% of the children had blood lead levels ≥15 µg/dL. The 1980 census shows that there are approximately 250,000 children under age 6 living in tracts where 60% of the housing was built prior to 1950. Applying these rates to this population suggests that statewide there are 3,200 children with blood lead ≥25 µg/dL and 50,000 children with blood lead ≥15 µg/dL.

2. EP as a Screening Test:

Results from the CLPPP studies illustrated in Figure 6 demonstrate that the sensitivity of EP in detecting an elevated blood lead is extremely poor. When an elevated blood lead level is defined as 25 µg/dL or greater, the EP test will detect the elevation only 46% of the time. When an elevated blood lead level is defined as 15 µg/dL or greater, the EP test will detect the elevation only 16% of the time. The specificity of the EP test is higher (80%), but still unacceptable. If the EP test had been used to screen children in these studies, 20% of children with blood lead ≥25 µg/dL would have been falsely misclassified as negative. The rate of false EP negatives for blood lead > 15 µg/dL is about 16%. This finding was consistent with previous studies (15).

Fig. 3 Ethnicity by Census Tract of Residence
Childhood Lead Poisoning Study,
Oakland, California, 1987



Calif Dept of Health Services, 1989

Fig. 4 Blood Lead Levels by Sex in Children Aged 1-5
Childhood Lead Poisoning Study,
Oakland, California, 1987

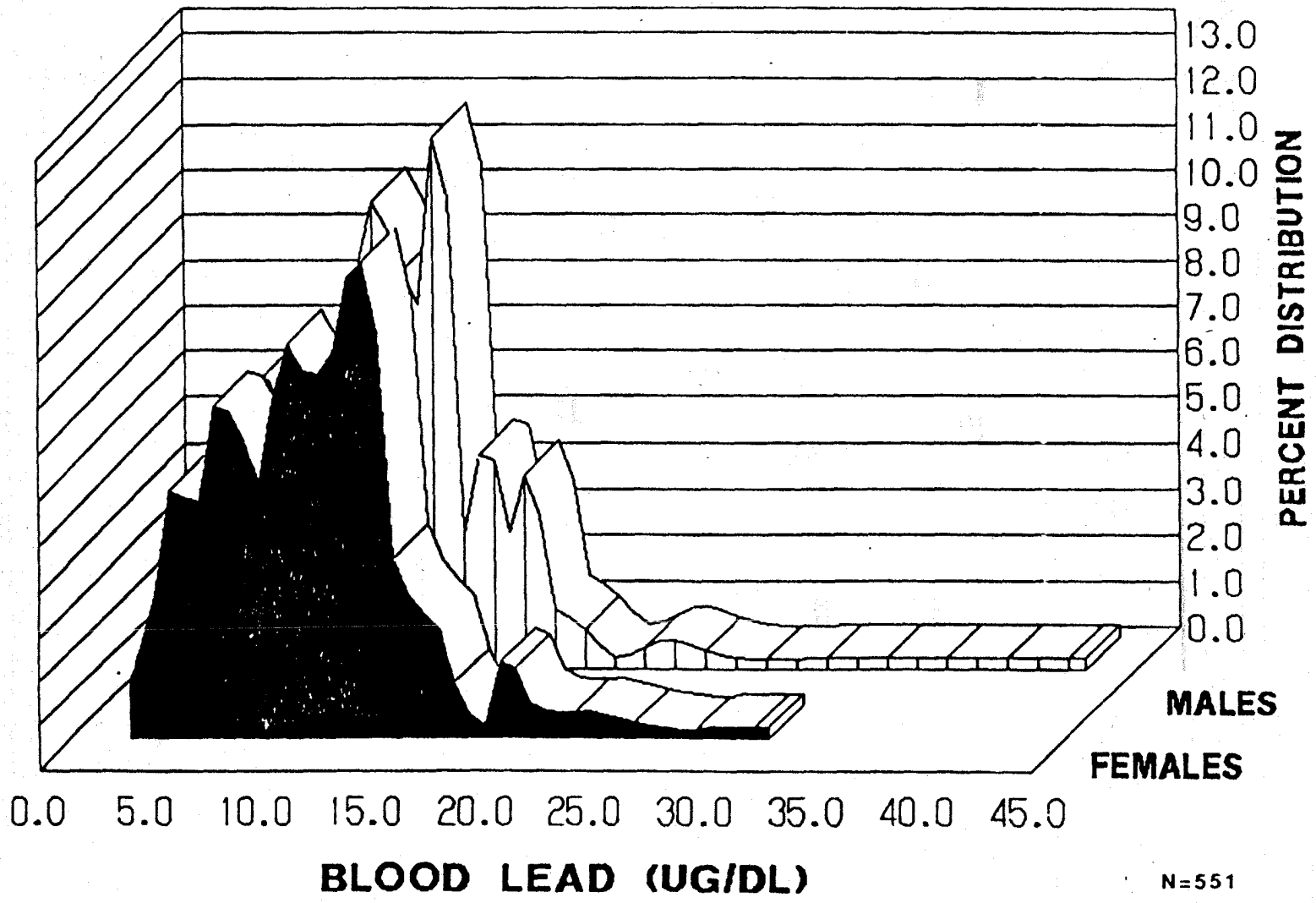
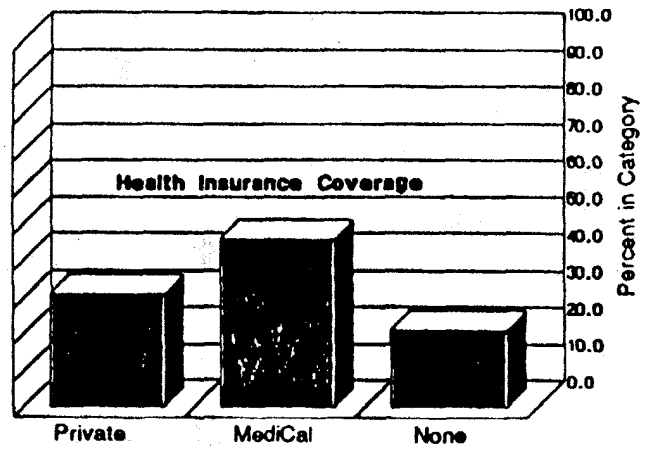


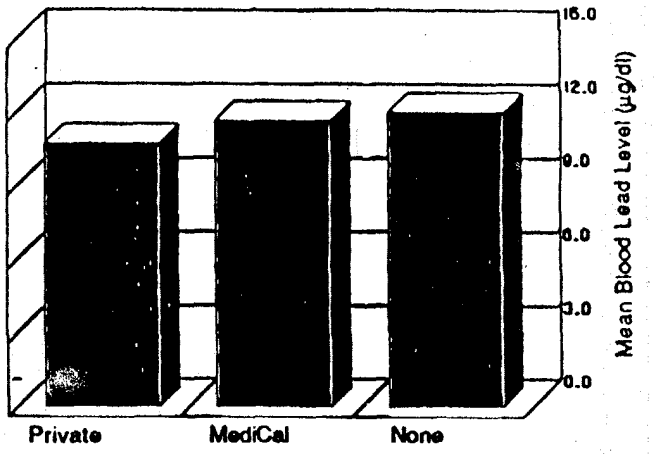
Fig. 5 Blood Lead, Income and In. ;ance Status
Childhood Lead Poisoning Study,Oakland, California, 1987

Health Insurance Coverage



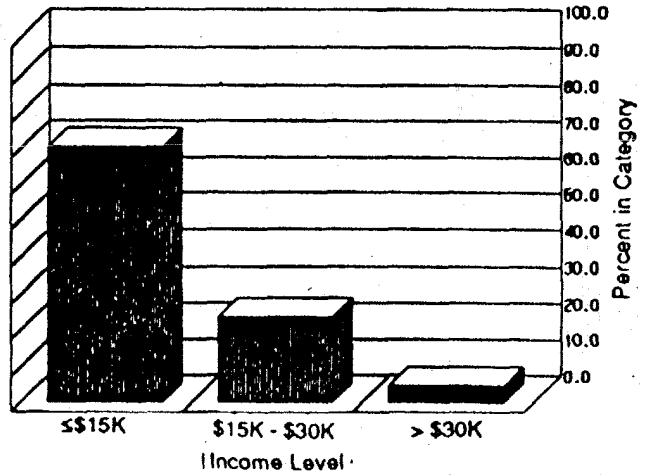
Calif Dept of Health Services, 1989

Mean Blood Lead by Health Insurance Status



Calif Dept of Health Services, 1989

Income Levels



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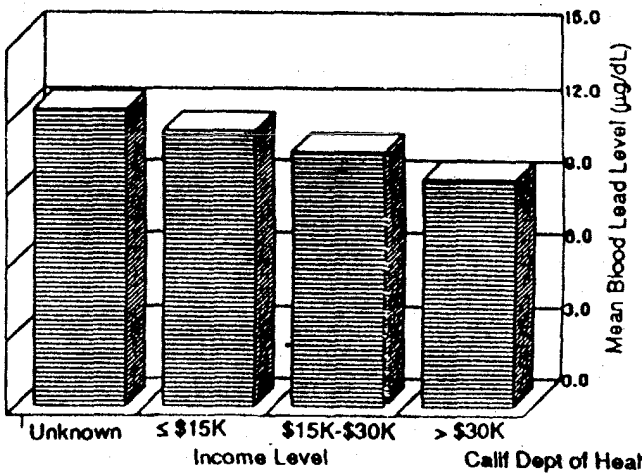
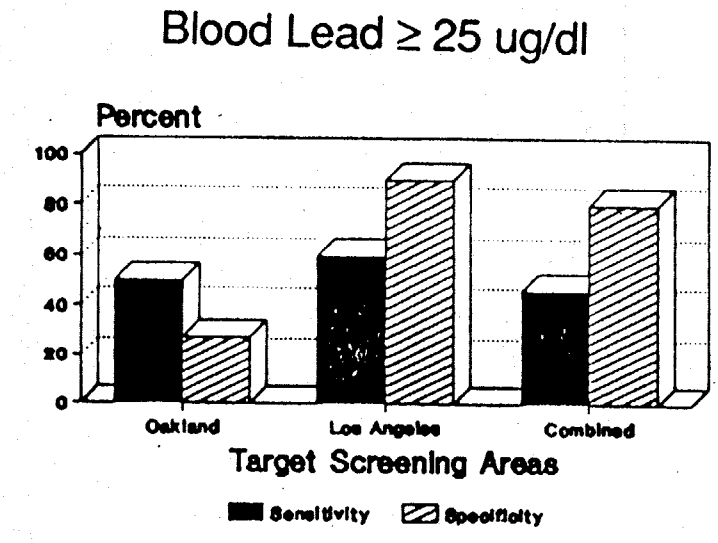
Mean Blood Lead Level by Income

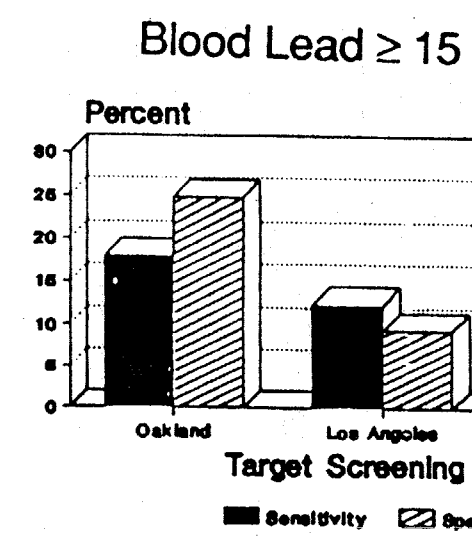


Calif Dept of Health Services, 1989

N=551

Fig. 6 Sensitivity and Specificity of FEP in Detecting Blood Lea
Childhood Lead Poisoning Study,
Oakland and Los Angeles, California, 1987-88





OAKL
LOS

3. Blood Lead Sample Collection:

In the Oakland study, it was found that blood collection using the fingerstick method was extremely inaccurate, even when proper precautions were taken to avoid contamination. The false-positive rate obtained in the Oakland study was over 50%, necessitating retesting of over one-third of the original sample, and adding substantially to the time and effort required to do the study. Since the fingerstick approach was so problematic, blood was drawn by venipuncture in Los Angeles County. This approach had several advantages: (a) a sufficient sample size was easily available; (b) no false positives were found; and (c) the method was easier on the child than the fingerstick method because there was less need to do repeated sampling to obtain sufficient quantity or for follow-up. The response rate in Los Angeles was identical to that found in Oakland, indicating parental acceptance of the method.

4. Soil Lead Levels:

Soil lead levels found in the Oakland target area were high, exceeding State environmental standards in the majority of instances. Figure 7 shows the mean soil lead and paint lead concentrations in parts per million. The average soil lead level in Oakland was 1232 ppm. As a comparison, the California hazardous waste regulations define wastes containing 1000 ppm of lead as a hazardous waste. U.S. background levels range between 10 and 700 ppm.

5. Soil Lead and Blood Lead:

The association between soil lead and blood lead was examined, adding adjustments for age, sex, and ethnicity. Front yard and back yard soil lead were significantly associated with blood lead. These locations are probably where children spend more of their time. Figure 8 shows that for every 500 ppm rise in front yard soil lead, a child's blood lead is predicted to rise an average of 4 µg/dL. For every 500 ppm rise in rear yard soil lead, a child's blood lead rises 5 µg/dL.

6. Paint Lead Levels:

Paint samples were obtained from 75% of the households surveyed in Oakland. Samples were only collected if painted surfaces were peeling or chipping. The mean paint lead level for interior paint was 9,971 ppm, ranging from 32 to 56,554 ppm. The mean lead level in exterior paint was 33,610 ppm, ranging from 5 to 215,572 ppm. As stated above, the U.S. CPSC (Consumer Products Safety Commission) standard for paint sold for interior or exterior residential use is 600 ppm dry film weight.

Mean indoor and outdoor paint lead levels by location of sample are shown in Figure 9. Interior paint taken from walls had the lowest average lead level (11,300 ppm). Outdoor paint samples from window sills and porches had the highest average lead level (46,415 ppm). Indoor paint lead levels greater than or equal to 600 ppm (the Federal standard) were found in 75% of the households. Outdoor paint lead levels greater than or equal to 600 ppm were found in 91% of the households.

Interviewers rated the homes for the condition of the paint and found that almost 10% of the homes had peeling or flaking interior paint and 26% of homes had peeling or flaking exterior paint.

7. Paint Lead and Blood Lead:

Figure 10 shows the association between interior paint and blood lead. Study results showed blood lead was 1.37 times higher in children where lead in paint from doors and windows was high, compared to children exposed to lead paint from interior walls. Other factors influencing exposure to paint lead in children include hours of play indoors, home refinishing, and the condition of the paint. Paint on window sills and doors had a higher mean lead content than paint on interior walls.

To understand the full impact of lead paint exposure in children, it would be useful to study an area where residential paint meets the federal standard.

A significant association was also found between lead in exterior paint and childhood blood lead levels. Children living in houses with exterior paint lead over 16,000 ppm had blood lead levels twice that of children living where levels were under 16,000 ppm. The analyses also showed that paint on exterior walls has a higher mean lead content than paint on interior walls in the homes studied.

8. Tap Water Pilot Study:

For the Oakland homes studied, the average lead concentrations in all the drinking water samples was less than 5 parts per billion (ppb). Lead doses were calculated by multiplying the number of self-reported cups of water consumed per day by the concentration of lead in the drinking water for the household, using different absorption rates. Average lead doses estimated by this method were very low. No conclusions could be made about the blood lead-water lead association from these data because of the extremely small sample size.

Fig. 7 Indoor Paint, Outdoor Paint and Soil Lead Means by Cen
Childhood Lead Poisoning Study,Oakland, California, 19

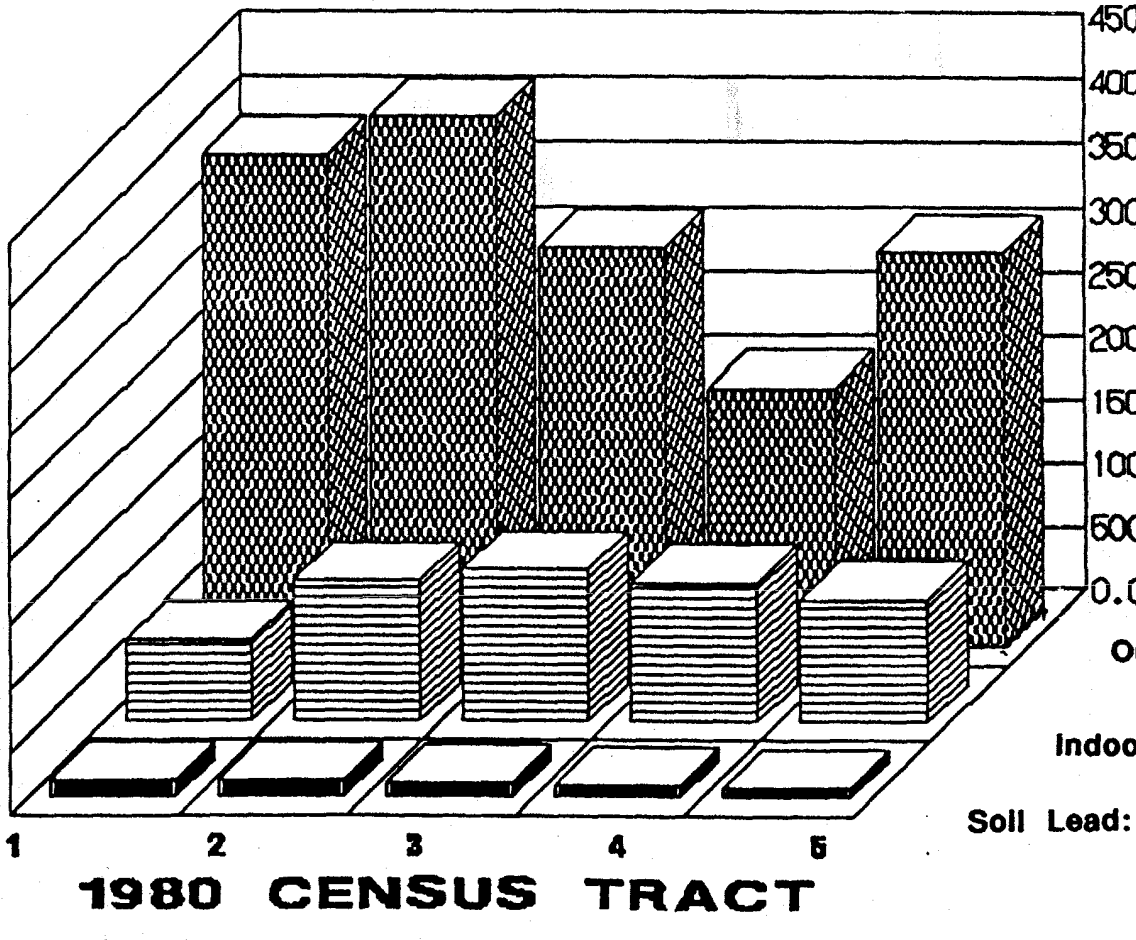


Fig. 8 Blood Lead and Soil Lead
Childhood Lead Poisoning Study,Oakland, Californi

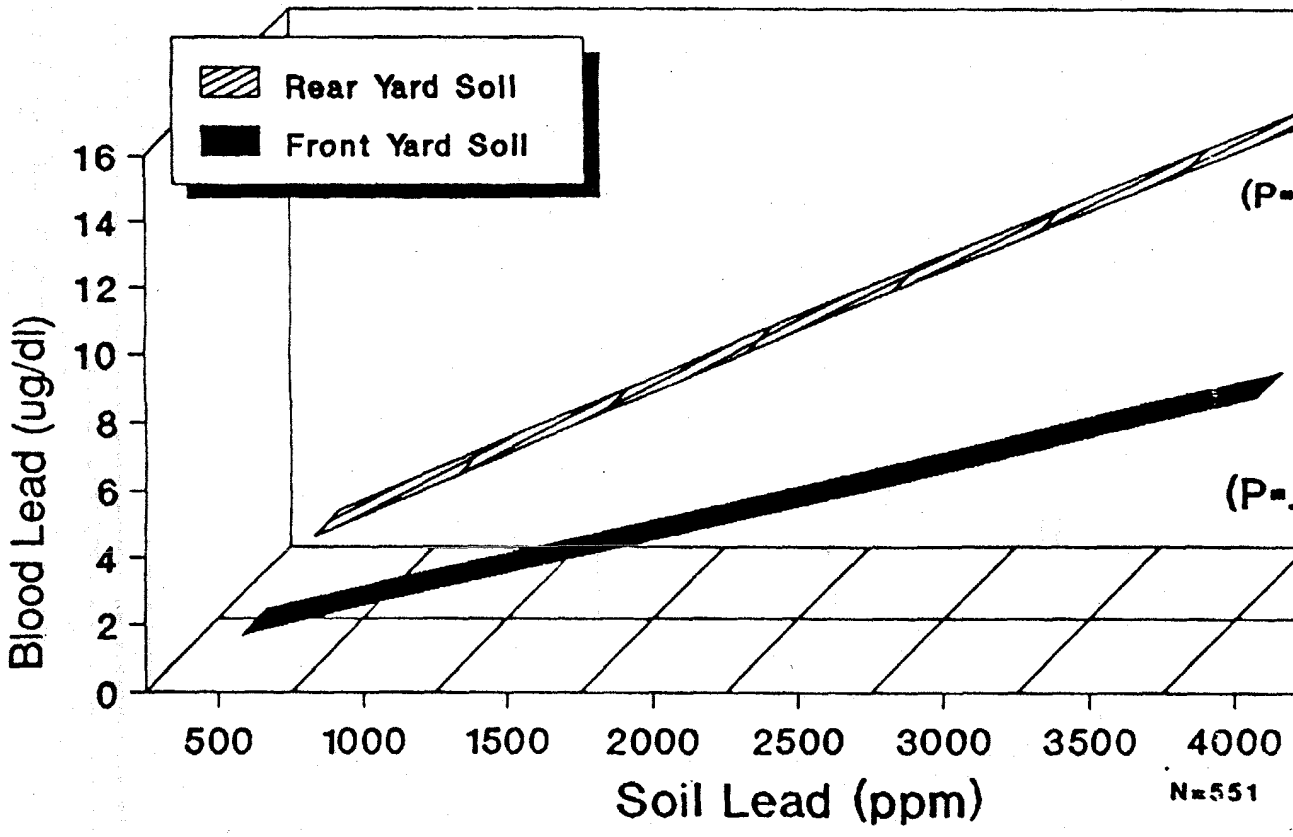


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Fig. 9 Mean Paint Lead Levels by Sample Location
Childhood Lead Poisoning Study,Oakland, California, 1987

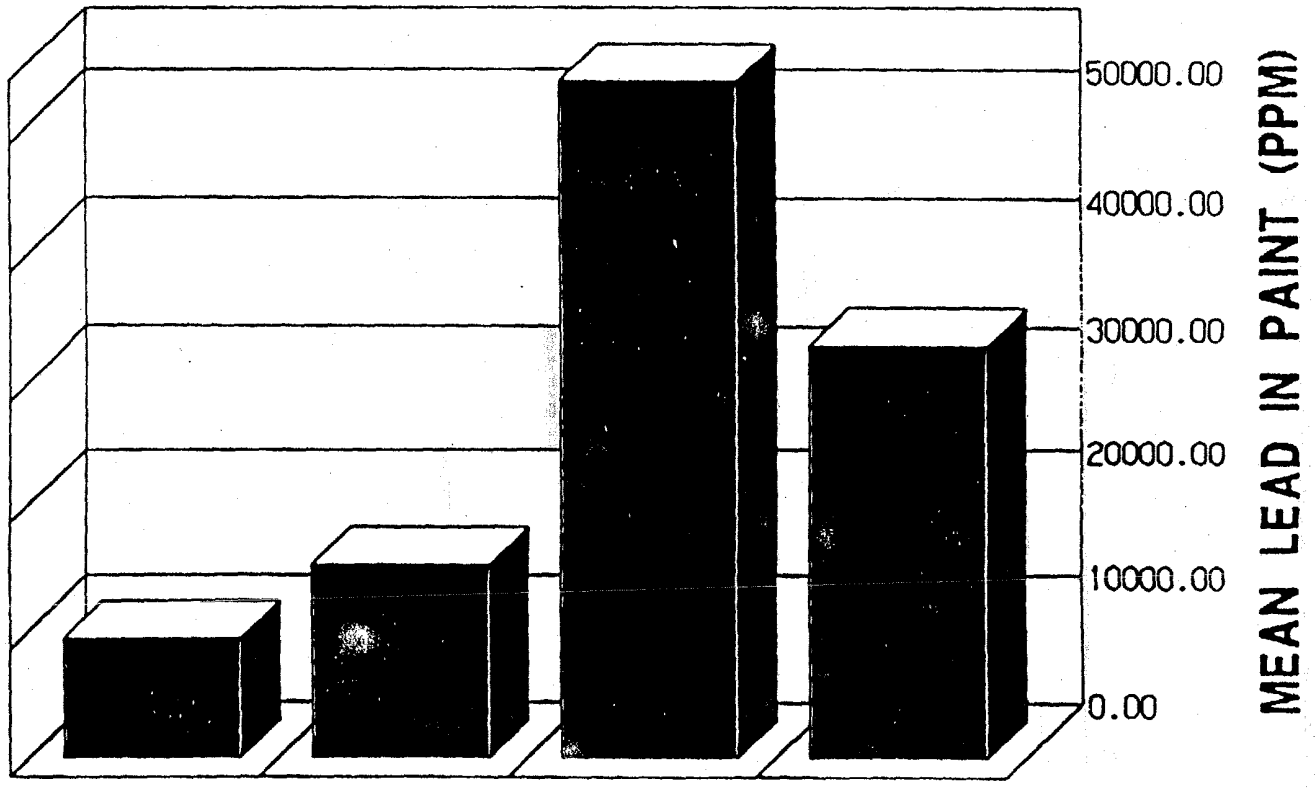
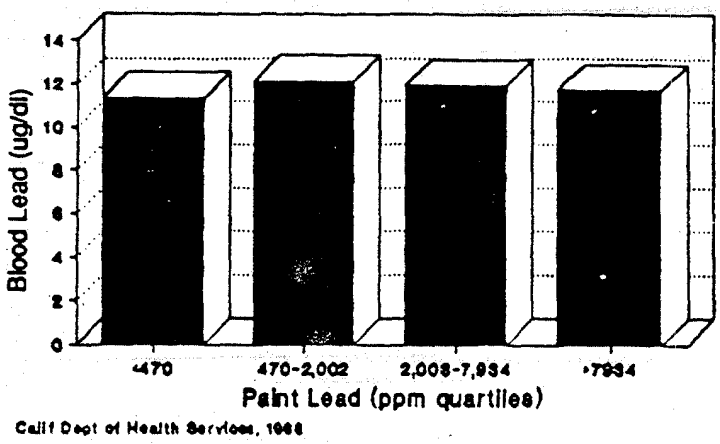


Fig. 10 Blood Lead & Interior Paint
Childhood Lead Poisoning Study,
Oakland, California, 1987



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C. Conclusions

1. Prevalence of Lead Exposure to Children in High Risk Areas:

Approximately 250,000 children under age 6 live in California census tracts in which, like those studied, 60% of the housing was built before 1950. In the tracts in the study, 1.3% of the children had blood lead levels of 25 µg/dL or greater. Based on that finding, DHS projects that 3200 California children under age 6 have lead poisoning. Twenty percent of the children had blood lead levels of at least 15 µg/dL, the level which is being considered by CDC as the new standard; projected statewide, 50,000 children in the state under age 6 might be at risk.

The number of children at such risk over time could be even greater, since lead-based paint and soil contamination remain an exposure source for successive generations of children who occupy the housing -- both newborns in the current household and children of new families that move in. California families with children under age 6 are highly mobile: in any 15 month period, nearly 40% of these families move to a new home (29).

It is estimated (based on the 1980 census) there are over 2 million housing units in California that were constructed before 1950 and therefore may contain lead-based paint. CLPPP studies completed to date address only high-risk populations. Subsequent studies to estimate the prevalence of elevated blood lead levels in lower and moderate risk areas such as census tracts with less than 60% pre-1950 housing, or higher household income, would provide data for comparison purposes.

2. EP alone is not an adequate screening test for lead toxicity:

EP has been used as an inexpensive screening test for lead poisoning in children for many decades, and as a preliminary test for lead exposure. If an elevated EP (>35 µg/dL) was found, testing for blood lead levels was recommended. In these studies, the use of EP alone as a screening tool would have failed to detect large numbers of children with mildly elevated blood lead levels. The medical test of preference for screening must therefore be blood lead, with EP as an additional measure to assess severity of toxicity in children with elevated blood leads.

3. Effect of Proposal to Lower Blood Lead Standard:

If the CDC intervention level is lowered to 15 µg/dL, more than 10 times as many children residing in high risk target areas will have blood lead levels defined as elevated. As Figure 11 shows, the CDC recommended level has been steadily lowered since 1960. Lowering the standard to 15 would increase the target population for Oakland for all age groups studied (Figure 12).

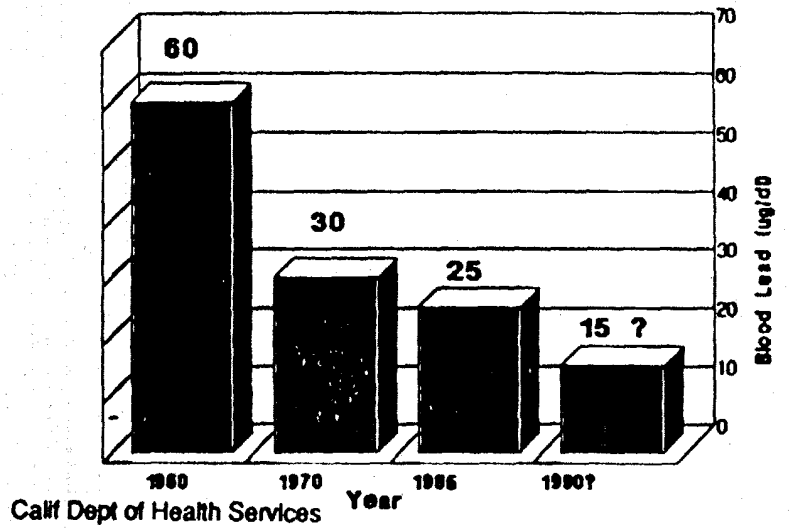
4. Venipuncture is the Method of Choice for Collecting a Blood Lead Sample:

In these studies, there were high rates of false positives for capillary blood leads which led to high follow-up costs in Oakland. To avoid this problem, DHS attempted to collect blood by venipuncture in Los Angeles. The method was feasible, effective, and acceptable to parents.

5. Sources of Lead Exposure to California Children:

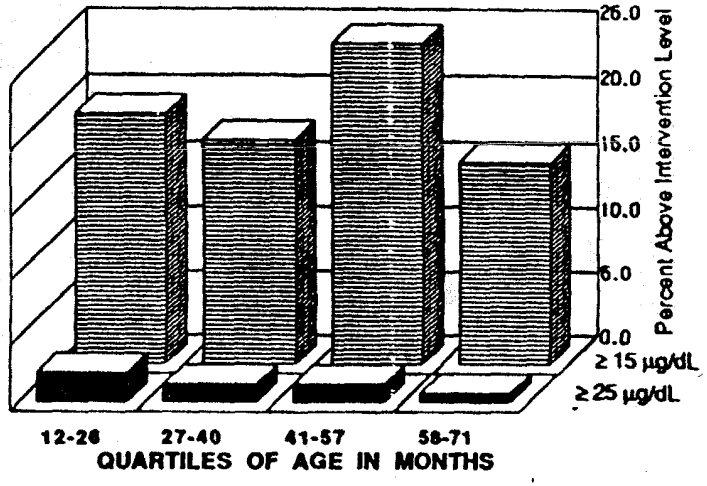
Lead levels in paint in high risk areas were frequently in excess of current Federal requirements. Levels in soil and paint correlated with blood lead levels of children living in the housing. 75% of the interior paint and 91% of the exterior paint sampled in these studies exceeded Federal standards. In addition, 10% of the interior paint was in poor physical condition, making the lead in paint accessible to active young children.

Fig. 11 CDC Recommended Intervention Level for Blood Lead



Calif Dept of Health Services

Fig. 12 Percentage of Children with Elevated Blood Lead Level.
Current & Proposed CDC Intervention Levels.
Childhood Lead Poisoning Study, Oakland, California, 1987.



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V. ABATEMENT TECHNOLOGIES

A. Household Paint

1. Regulations for Paint in California:

There are no statewide regulations limiting the allowable level of lead in paint on surfaces in housing in California. The CPSC only limits the amount of lead in residential paint for sale but not the past application of lead paint on homes. Current federal paint inspection activities are limited to testing U.S. Housing and Urban Development (HUD) housing when a federally-assisted housing unit is upgraded under their modernization program. A 1987 HUD regulation was passed that requires testing and abatement of surfaces coated with lead paint. HUD plans to implement the program pending the results of a study of testing and abatement techniques and funding. California has no programs to identify and abate lead-based paint used on private housing. Local authorities may act under their general mandate to protect the public health, but, with one exception, there are no specific local mandates related to lead-based paint abatement. Only in Los Angeles is there a specific mandate to require the removal of lead bearing substances from any dwelling when a child is identified with an elevated blood lead level. In conducting follow-ups of laboratory reports, DHS staff were frequently told by county personnel of the need for a lead paint regulation in California.

2. Creating a Hazard: Use of Improper Lead Abatement:

Of primary concern is the recognition that improper abatement can be extremely hazardous. Rather than providing a remedy for a lead hazard, abatement can increase the risks to more people if it is done improperly. For example, sanding lead painted surfaces can disperse lead dust throughout the environment. Workers can be severely poisoned if not adequately protected during abatement. Children and other family members can inhale and ingest lead dust created by abatement. Lead poisoned children who have returned home from the hospital to improperly abated homes have become repoisoned (8) due to high lead dust levels left behind. Furthermore, improper disposal of lead-contaminated debris generated during abatement can pose a risk to other community members who come in contact with it.

3. Improper Test Methods Can Waste Money:

XRF (X-ray fluorescence) equipment has been used for decades to test for lead in paint. XRF is less expensive than sampling paint and getting a 'wet lab' (chemical) analysis. The XRF can, when properly used, be useful as a screening device. However, improper use or failure to confirm the presence of lead can result in inappropriate intervention. The state of Maryland identified two units in a large housing complex containing lead paint. A contractor surveyed the entire complex with XRF equipment. The result of this survey was that $500,000 was spent on abating lead in 150 units. No laboratory confirmation was done to verify the presence of lead. Had a 'wet lab' analysis been done, it would have revealed that the lead level in the paint was exceedingly low. As it was, subsequent follow-up

revealed no lead was present in the units and one-half million dollars had been expended unnecessarily. Due to the unreliable nature of current, state-of-the-art XRF testing, it is necessary to verify XRF sampling and laboratory measures with wet lab analysis prior to abatement.

4. Residential Soil:

Children are exposed to lead from multiple sources in addition to soil around homes. This fact makes it difficult to assess the contribution of soil lead to the total burden of lead exposure for a child. The CLPPP conducted an extensive literature search and consulted many experts but could not find definitive research indicating that the removal of lead contaminated soils can lower blood lead levels. Based on CLPPP studies, background levels in urban soils and the opinions of experts consulted by CLPPP, it appears a reasonable goal for residential soil lead abatement is to achieve a level under 500 ppm, although lower levels would afford greater protection and safety margins. This level is within the range of "background" concentrations for an urban setting. However, further research is needed to fully understand this problem.

VI. RECOMMENDATIONS FOR FURTHER ACTIONS

A. Epidemiologic and Surveillance Activities (funded)

1. Complete the data analyses for the northern and southern California population based surveys.

2. Perform the Central Valley survey (as mandated) to provide more accurate population estimates for this area of California (1989-90).

3. Perform a population based survey in an area selected as being "lower risk" in terms of age of housing and poverty, in order to provide more accurate prevalence estimates for those groups and a better assessment of the role played by lead-based paint (1990-91).

4. Continue the laboratory reporting system.

B. Lead Screening and Medical Follow-up

1. Recommend that providers screen "high-risk" children in their practices. "High-risk" children are those between the ages of 1 and 5 living in older, dilapidated housing. Recommendations to providers should include:

   a. Annual screening, beginning at age 12 months and continuing through the fifth year, for "high-risk" children.

   b. Use blood lead as the primary test for lead toxicity. According to CDC recommendations (6), the erythrocyte protoporphyrin (EP) test should be used when blood lead is excessive because it can show whether the hematopoeitic system has been affected.

   c. Use a venous blood sample instead of the fingerstick test because of the high false-positive rate associated with the fingerstick method.

2. Medical Follow-Up:

   a. A protocol for medical and environmental follow-up should be developed to aid local health departments in managing lead cases. The CLPPP, with the assistance of other branches in DHS (see recommendation E) should coordinate development of the protocol. The protocol would include a classification system which would make it possible to prioritize lab results (urgent, moderate risk, low risk), and a timetable for the various interventions (telephone call to the physician, home visit, repeat blood lead test, etc.) for each risk category.

   b. Health care providers should be educated to recognize lead poisoning as a public health problem which exists in California and requires

routine screening. Such education could be carried out by the CLPPP and CHDP program.

C. Regulations for Abatement of Lead in Paint.

DHS should continue its leadership role in this issue by supplying background information and data to enable the appropriate agencies to establish standards for lead levels and condition of paint on houses.

D. Study abatement of soil lead:

The CLPPP would evaluate whether soil lead abatement is effective in reducing blood lead levels in children.

E. Task force on Childhood Lead Poisoning:

A task force on childhood lead poisoning shall be convened within DHS to include representatives from all relevant branches. The mission of the group will be to work toward coordination of environmental monitoring and abatement of lead hazards, and to improve screening and surveillance of children with lead poisoning.

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APPENDIX A. ENABLING LEGISLATION

Assembly Bill No. 2977

CHAPTER 481

An act to repeal Section 300.5 of, and to add Article 3.1 (commencing with Section 309.7) to Chapter 2 of Part 1 of Division 1 of the Health and Safety Code, relating to health, making an appropriation therefor, and declaring the urgency thereof, to take effect immediately.

[Approved by Governor July 24, 1986. Filed with Secretary of State July 24, 1986.]

LEGISLATIVE COUNSEL'S DIGEST

AB 2977, Connelly. Health: Childhood Lead Prevention Poisoning Program.

Under existing law, the State Department of Health Services is required, as a part of the program of maternal and child health, to develop a program to prevent and identify disabilities and debilitating diseases caused by lead poisoning for purposes of reducing infant mortality and improving the health of mothers and children. Existing law also requires the governing body of each county or counties, or the department in case of counties which contract with the state for health services, to establish a community child health and disability prevention program which is required to include where appropriate testing for lead poisoning.

This bill would delete the provisions relating to the department program which is part of the program of maternal and child health. It would, instead, establish a state Childhood Lead Poisoning Prevention Program within the department, to compile and analyze information about where high childhood blood lead levels are occurring, to target areas of the state where childhood lead exposures are especially significant, and to design and implement a program of medical followup and environmental abatement and followup that will reduce the incidents of excessive childhood lead exposures in California.

The bill would appropriate $175,000 to the department for the purpose of providing 1st-year funding for the program established by this bill.

The bill would declare that it is to take effect immediately as an urgency statute.

Appropriation: yes.

The people of the State of California do enact as follows:

SECTION 1. Section 300.5 of the Health and Safety Code is repealed.

SEC. 2. Article 3.1 (commencing with Section 309.7) is added to

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Chapter 2 of Part 1 of Division 1 of the Health and Safety Code, to read:

Article 3.1. Childhood Lead Poisoning Prevention Act

309.7. The Legislature hereby finds and declares that childhood lead exposure represents the most significant childhood environmental health problem in the state today; that too little is known about the prevalence, long-term health care costs, severity, and location of these problems in California; that it is well known that the environment is widely contaminated with lead; that excessive lead exposure causes acute and chronic damage to a child's renal system, red blood cells, and developing brain and nervous system; that at least one in every 25 children in the nation has an elevated blood lead level; and that the cost to society of neglecting this problem may be enormous.

The Legislature further finds and declares that knowledge about where and to what extent harmful childhood lead exposures are occurring in the state could lead to the prevention of these exposures, and to the betterment of the health of California's future citizens. Therefore, it is the intent of the Legislature in enacting this article to establish a state Childhood Lead Poisoning Prevention Program within the State Department of Health Services to accomplish all of the following:

(a) To compile information concerning the prevalence, causes, and geographic occurrence of high childhood blood lead levels.

(b) To identify and target areas of the state where childhood lead exposures are especially significant.

(c) To analyze information collected pursuant to this article and, where indicated, design and implement a program of medical followup and environmental abatement and followup that will reduce the incidence of excessive childhood lead exposures in California.

309.71. (a) All medical laboratories shall report to the department each detected case of a blood lead level greater than 25 micrograms of lead per deciliter of human blood or the equivalent standard as measured in micrograms of protoporphyrin per gram of hemoglobin. The blood lead findings, the names, ages, and addresses of the patients involved in each detected case and any additional information necessary to implement the provisions of this article shall be reported to the department in a manner prescribed by the director.

(b) All information reported pursuant to this section shall be confidential, as provided in Section 211.5.

(c) All medical laboratories testing for blood lead levels shall participate in a blood lead and free erythrocyte protoporphyrin (FEP) proficiency testing program.

(d) Laboratories which fail to meet reporting requirements will

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be assessed fines of up to five hundred dollars ($500) at the discretion of the director.

309.72. (a) By July 1, 1987, the department shall identify target areas in which to conduct a childhood lead screening program.

(b) The targeted areas shall include at least one area within the urban San Francisco/Alameda County area, one area within, the urban Los Angeles/Orange County/San Diego area, and one area within the Central Valley Sacramento/Fresno area, and other areas if scientifically indicated as determined by the director.

(c) These target areas shall be described by census tract and shall be selected based on the prevalence of the following factors:

(1) Older housing.

(2) Lead-emitting industry.

(3) History of heavy automobile traffic.

(4) Use or disposal of hazardous materials or waste.

(5) Populations where cultural or ethnic factors or both may result in a higher risk of ingestion of lead.

(6) Population of children between the ages of 12 months and 6 years.

309.73. By October 1, 1988, the department shall complete a screening program for childhood lead in the targeted areas identified pursuant to Section 309.72, and in other areas where scientifically indicated. Further, where environmental abatement is found to be indicated, the department shall carry out field trials of alternative abatement technologies.

309.74. On January 1, 1989, the department shall submit a report to the relevant legislative policy committees, and to the relevant legislative budget subcommittees for their review, describing the results of the screening program, the significance of the results, and the department's recommendations for further actions, where indicated.

SEC. 3. (a) The sum of one hundred seventy-five thousand dollars ($175,000) is appropriated from the General Fund to the State Department of Health Services for the purpose of providing first-year funding to implement the Childhood Lead Poisoning Prevention Program pursuant to Article 3.1 (commencing with Section 309.7) of Chapter 2 of Part 1 of Division 1 of the Health and Safety Code.

(b) It is the intent of the Legislature that future funding for the Childhood Lead Poisoning Prevention Program shall be appropriated through the annual budget process.

SEC. 4. This act is an urgency statute necessary for the immediate preservation of the public peace, health, or safety within the meaning of Article IV of the Constitution and shall go into immediate effect. The facts constituting the necessity are:

In order to have the maximum impact on the reduction of the incidence of childhood lead toxicity in the State of California, and in order for the funding for the Childhood Lead Poisoning Prevention

Program to be synchronized with the fiscal year, it is necessary for this act to take effect immediately.

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