A REPORT OF RECENT MEDICAL STUDIES OF FIVE U.S. LEAD PLANTS

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Summary

Epidemiologic studies conducted in 1975-76 in 5 different lead plants across the U.S. have shown unacceptably high blood lead levels and symptoms of lead poisoning in every plant studied. Hematologic, neurologic, and renal damage due to lead were also encountered. Inappropriate medical practices were noted including the misuse of oral chelating drugs and allowing lead-poisoned workers to continue to be exposed to lead during chelation therapy.

The usefulness of monitoring blood lead and/or protoporphyrin levels as indicators of lead toxicity was clearly shown by the high degree of correlation between these measurements and the signs and symptoms of lead toxicity. The newly developed portable method of zinc protoporphyrin (ZPP) determination was shown to be a promising method of screening lead-exposed workers.

In several locations, home contamination with lead dust from lead plants resulted in lead absorption and in one location lead poisoning among workers' children. These studies illustrate the importance of good work practices which, in addition to minimizing worker exposure, will minimize exposure to his family.

Chronic neurologic and renal effects of lead exposure were demonstrated in this study. Neuropathy was noted at a relatively low blood lead level (81μg/100ml) and after only 2 months of exposure. Abnormal glomerular function (elevated blood urea nitrogen and creatinine levels and decreased glomerular filtration rates) and abnormal tubular function (impaired urinary concentrating ability and reduced lead clearance rates) were noted in lead workers.

These findings emphasize the need for improved industrial hygiene measures in the lead industry and the importance of biological monitoring in preventing lead toxicity. Based on the data contained in this report, a standard of 60µg/100ml is recommended as the "biologic threshold limit value" for lead.

A. Introduction

Occupational lead poisoning has been recognized since the 19th century[1] and methods for its prevention were well developed in Great Britain in the early 20th century[2]. Nevertheless, eradication or even control of this disease is far from a reality in the United States in 1976. The current report summarizes the results of 5 recent lead plant investigations (Table 1) conducted by the National Institute for Occupational Safety and Health and the Bureau of Epidemiology, Center for Disease Control, which clearly demonstrate that lead poisoning is occurring with an alarming frequency in lead plants across the country. (Detailed reports of these investigations are included as appendices.) Investigations of 6 other similar plants are currently in progress; data from these studies will be added to the current report as they become available. In the current report, special attention is given to studies which relate blood lead levels to signs and symptoms of lead toxicity and to studies of the chronic effects of lead exposure.

B. Summary of Investigations:

1. Memphis, Tennessee: Secondary lead smelter*

Following reports of excessive blood lead levels and clinical lead poisoning at this plant, 77 (92%) of 84 current employees and 1 former employee were examined in November 1975[3]. Concurrently, community lead exposure and exposure of workers' families to lead carried home on work clothing[4] was evaluated.** Workers blood levels ranged up to 184 µg/100 ml with 67% ≥60 µg/100 ml (Table 2). Highest blood lead levels were seen in production area workers. Classical symptoms of lead poisoning were evident: abdominal pain (17%), gastrointestinal dysfunction (22%), joint pain (28%), neuromuscular symptoms (27%), and anorexia (23%). Ten workers were found to have lead neuropathy, with weakness of wrist extensor muscles. Anemia (hemoglobin concentration < 14gm/100 ml[5]) was noted in 11 workers and correlated with elevated blood lead levels and erythrocyte protoporphyrin concentrations. Elevated blood urea nitrogen (BUN) levels (greater than 20 mg/100 ml) were seen in 6 workers. These azotemic workers had worked at the plant longer and were older than workers with normal BUN levels. Increased arsenic absorption was noted in 10 (14%) of 72 employees. A survey of 228 children living near the smelter showed no excessive lead absorption. In January 1976, management temporarily closed the smelter and initiated efforts to redesign the production process. Young children of smelter workers were found to have elevated blood lead levels and 8 were hospitalized and treated for lead poisoning. (Details of that investigation are discussed below.)

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* Complete report - Appendix 1

** Complete report - Appendix 2

2. Joplin, Missouri: Lead chemicals plant*

In March and May 1976, medical evaluations of 53 production workers at this plant revealed blood lead levels from 39 to 135 µg/100 ml, with 83% ≥ 60µg/100 ml. Comparable levels of erythrocyte protoporphyrin were noted. Three workers were anemic; wrist weakness was noted on physical examination of 1 worker; ankle drop in 1 worker; and tremor of the outstretched hands in 2 workers. Symptoms compatible with lead intoxication were noted in 20 workers (Table 3).

The most notable finding of this study involved renal disease among these workers: 17 had elevated blood urea nitrogen (BUN) levels (>20 mg/100 ml) further studies revealed significant impairment of renal tubular and/or glomerular function in 7 workers which may represent lead nephropathy. (A more detailed discussion is given below.)

3. Salt Lake City, Utah: Secondary lead smelter**

A medical evaluation[6] of 36 (86%) of the plant's 42 employees was conducted in January 1976 following the hospitalization of 7 workers for lead poisoning. Two of those hospitalized had blood lead levels over 250 µg/100 ml and evidence of possible lead encephalopathy. Nineteen employees had unequivocal lead poisoning*** within the previous 4 months;

* Complete report - Appendix 3

** Complete report - Appendix 4

***Lead poisoning was defined in this study as a blood lead level ≥80µg/100ml and/or urine lead levels ≥250µg/100ml plus symptoms compatible with lead toxicity which could not be otherwise explained. As discussed below, manifestations of lead toxicity are seen at blood lead levels < 80µg/100ml and, therefore, this definition uses criteria which may exclude persons suffering from the toxic effects of lead.

13 workers were anemic.

These cases of acute lead poisoning were related to a change in plant processes: two small rotary furnaces, previously used for antimony production, were used to process lead in December 1975. Inadequate ventilation of these furnaces was felt to be responsible for the elevated air lead levels in the plant. One case of lead poisoning occurred in the company president who was apparently exposed outside the production area: the air intake for the ventilation system for his office was adjacent to the furnace room.

The smelter was closed by the local and state health departments on January 16, 1976, and the plant was ordered to reduce lead exposure to an acceptable level. The plant was reopened in February 1976 after engineering controls were instituted and personal industrial hygiene stressed, but was closed again in June 1976 after blood lead levels of employees were noted to be rising.

4. Eagan, Minnesota: Secondary lead smelter*

Two cases of chronic lead poisoning in employees at this plant were reported to the state health department and medical studies of employees and their families were conducted in May 1976. The median blood lead level of the 38 workers was 72.5 µg/100 ml; 76% had levels ≥60 µg/100 ml. Workers on the night shift had the highest average blood level. Four workers had weakness of wrist and/or ankle extensor muscles; 10 workers had tremor of the outstretched hands. There was no consistent relationship between lead poisoning symptoms and blood lead levels. Five (16%) of

*Complete report - Appendix 5

31 children (ages 1-9) of plant employees had blood lead levels ≥30 µg/100 ml, indicating increased lead absorption.

5. Atlanta, Georgia: Secondary lead smelter

A brief visit to this plant was performed in March 1976 as a follow-up to an earlier NIOSH study performed in 1971. Five workers were interviewed and plant blood testing records reviewed. Seventy percent of the plants' 39 employees had blood lead levels ≥60 µg/100 ml in February 1976, as compared with 74% over 60 µg/100 ml in January 1975. The percentage of workers with blood lead levels over 80 µg/100 ml had fallen during this period from 43% to 26%. Workers at this plant who were treated for lead poisoning with intravenous chelation were allowed to remain on the job during therapy.

Type of Exposure:

The Tennessee and Utah investigations represent studies of relatively acute exposure to lead ; the average duration of exposure to lead was 4 months and 2 months, respectively. In all the other locations, exposures were more chronic, lasting up to 30 years in some instances. This difference in exposure duration was associated with differences in the prevalence of symptoms and signs of lead toxicity. These effects are discussed separately below.

III. Relationship of blood lead and protoporphyrin levels to manifestations of lead toxicity.

The usefulness of blood lead testing in evaluating workers exposed to lead has been extensively debated. Some workers feel that blood lead

and erythrocyte protoporphyrin (EP) levels bear no relationship to symptoms or signs of lead toxicity and, therefore, these tests have no value in screening lead-exposed workers. However, in the currently reported group of studies, blood lead and EP levels were well correlated with the manifestations of lead toxicity.

1. Symptoms:

Blood lead and EP levels were correlated with the classical symptoms consistent with lead intoxication (Table 3). Persons reporting symptoms during the previous year had significantly higher blood lead levels than workers without symptoms (Table 4). Sixty to seventy percent of workers with symptoms during the past year were currently symptomatic at the time of the interview. Furthermore, the higher the blood lead or erythrocyte protoporphyrin level the greater was the proportion of symptomatic workers. The relationship between blood lead levels and symptoms was clearest in a plant (Tennessee) where workers had been employed for a relatively short period (average duration of employment = 5 months). In plants (Minnesota, Missouri) where the average duration of employment was greater (4 and 20 years, respectively) symptoms correlated less clearly with EP and lead levels (Table 5).

Forty-nine percent of workers with blood lead levels from 60 to 79µg/100ml reported symptoms during the past year consistent with lead poisoning. Within this blood lead range, workers with higher EP levels had higher symptom rates (Table 6). Lower symptom rates were noted for each blood lead category when we considered only workers symptomatic at the time of the interview and blood testing (Table 7). Twenty-three percent of workers with blood lead levels from 60 to 79µg/100ml were currently experiencing symptoms

compatible with lead poisoning when tested. Most of those with blood lead levels below 60µg/100ml who had reported symptoms during the previous year were not symptomatic at the time of testing.

Since the symptoms of lead poisoning are nonspecific and may be related to other disease processes, the same questionnaire used to evaluate symptoms in lead workers in Missouri was administered to an age-matched control group without occupational lead exposure. The prevalence of symptoms consistent with lead toxicity was much lower in unexposed workers than in lead workers living in the same town (Table 8).

2. Hematologic Effects:

Blood lead and erythrocyte protoporphyrin levels were closely correlated (r=0.76) especially in the range of blood lead concentrations from 40 to 80 µg/100 ml (Figure 1). Erythrocyte protoporphyrin levels were closely correlated with levels of zinc protoporphyrin (ZPP) measured directly by a new technique[8] using a dedicated portable hematofluorometer (r= .94) and when measured by the standard method of Lamola et al[9] (Figure 2). Thus, the above-mentioned relationships between symptoms, blood lead levels and EP levels would also hold for ZPP determinations as well.

Anemia, defined as a hemoglobin level < 14 gm/100 ml[5], were noted in 14% of workers at the Tennessee plant and in 31% in Utah. Both of these outbreaks represented relatively acute exposure to lead and the rates were higher than in those plants where chronic exposure to lead was occurring (no anemia was noted in Minnesota and 11% of Missouri workers were anemic).

Comparison of blood lead and EP levels with hemoglobin levels shows a pattern similar to that noted previously for symptom rates. The higher the lead or EP level the lower the hemoglobin level (Tables 8 and 9). Lead-related anemia is associated with elevated EP levels and elevated blood lead levels[10]; therefore, to differentiate anemia related to lead toxicity from anemia of other causes, EP levels in anemic and non-anemic workers were compared. Anemic workers had significantly higher EP levels and blood lead than non-anemic workers.

The clinical significance of anemia varies with the degree of hemoglobin depression. Symptoms classically associated with anemia (i.e., tiredness, fatigue, etc.) are not generally encountered at hemoglobin levels above 8 gm/100 ml[11]. Decrements in work performance as measured by standard techniques has been noted at hemoglobin levels of 11 to 13 gm/100 ml[12]. A dose-response effect between hemoglobin level and work performance was noted throughout the range of hemoglobin levels from 3 to 17 gm/100 ml; a threshold effect was not seen. Resistance to infection may also be impaired in anemic persons [13], but the relationship between the degree of anemia and the degree of decreased resistance has not been established. Therefore, the definition of anemia cannot be based on clinical abnormalities but must be related to the concentration of hemoglobin in the blood of in normal persons. The lower limit of normal for U.S. adult males living at sea level has been consistently reported as 14 gm/100 ml[5,12,13] and this level has been used in defining anemia throughout this report.

IV. Relationships of duration of exposure to manifestations of lead toxicity

In the Tennessee study, blood lead levels rose during the first month of employment and reached a plateau 2 to 3 months after beginning work (Figure 3). At that same plant, symptoms did not develop until 2 to 3 months of employment were completed (Table 10). The predominant symptoms varied with duration of exposure: after brief exposure (2 to 3 months), gastrointestinal symptoms predominated whereas constitutional symptoms and joint pains were relatively more common after prolonged exposure (over 1 year).

In other locations, workers acutely exposed to lead experienced relatively more gastrointestinal symptoms than chronically exposed workers. The highest relative rate of gastrointestinal symptoms was noted in lead poisoning cases in Utah (Table 11) where exposures were very brief (3-4 months duration). Workers in the Minnesota plant were chronically exposed and noted more nonspecific, constitutional symptoms (e.g., tiredness, irritability, joint pains, muscle weakness)(Table 12).

Anemia was noted only after 2 months of work with 1 exception (one worker who had completed 1 month of work, was anemic (Hgb: 13.0 gm/100 ml, lead level: 79 µg/100 ml, EP level: 299 µg/100 ml)).

Wrist extensor weakness was noted in 2 workers who had completed only 2 months of employment at the Tennessee plant. They had blood lead levels of 81 and 130 µg/100 ml and EP levels of 281 and 258 µg/100ml. One worker at the Minnesota plant with 2 months of exposure had a blood lead level of 78 µg/100 ml and an EP level of 250µg/100 ml and mild asymmetrical ankle extensor weakness. Thirteen other workers from Tennessee had evidence of either wrist or ankle weakness; all had worked for at least 7 months and had blood lead levels above 80 µg/100 ml and EP levels above 250 µg/100 ml. One former worker in Tennessee who had worked for 3 months and terminated employment 2 months prior to our testing had mild asymmetrical wrist extensor weakness with a blood lead level of 41 and an EP level of 49 µg/100 ml.

Although no definite relationships were noted between duration of exposure and development of overt lead nephropathy, some abnormalities of renal function were correlated with duration of lead exposure. The highest prevalence of abnormal renal function tests was noted in Missouri where 17 (32%) of 53 workers tested had elevated blood urea nitrogen (BUN) levels; workers with abnormal results had worked at the plant for 4.5 to 31 years. Six (8%) of 78 workers in Tennessee had elevated BUN levels; 3 (8%) of 38 workers in Minnesota had elevated serum creatinine levels (> 1.5 mg/100 ml). Except for 3 workers with less than 2 months of lead exposure, no worker with less than 3½ years of exposure had abnormal renal function tests. Thus, no azotemia was noted prior to approximately 4 years of lead exposure.

V. Renal Function Testing - Missouri

Specific functional abnormalities included decreased glomerular filtration rate (< 91 ml/min[13]) in 8 of 19 workers tested, decreased urinary concentrating ability (inability to concentrate the urine below 800 mosm/liter after a 12-hour water fast[14]) in 8 workers, and elevated urinary beta$_2$microglobulin levels (> 370µg/liter[15]) in 2 workers (Table 14). A significant negative correlation was noted between duration of lead exposure and rate of lead clearance* (Figure 4).

Since a modest age effect was noted in relation to lead clearance (i.e., impaired lead clearance in older workers), data from workers aged 45-55 was analyzed and a significant negative relationship was again noted between duration of exposure and lead clearance rate (Figure 5). Decrease in lead clearance was associated in most workers with impaired urinary concentrating ability and decreased glomerular filtration rate. Blood lead levels increased with decreasing lead clearance rate.

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*Lead clearance was calculated by determining the lead concentration in a timed, measured urine sample and the lead concentration of a whole blood sample drawn during the urine collection period; clearance was computed by the standard formula:

$C = \frac{UV}{P}$. where C= clearance of the substance

U= urine concentration of the substance

P= blood concentration of the substance

V= urine flow rate over collection period[16]

Although this observation must be confirmed on a larger series of workers with varying exposure periods, those data suggest that the measurement of lead clearance may be a useful tool in evaluating renal function in lead workers.

VI. Chelation Therapy

In the Missouri plant, oral EDTA was administered to workers with evidence of lead toxicity (anemia) or to workers with elevated blood lead levels; intravenous chelation therapy was also used. During therapy, workers were removed from exposure to lead. In the Georgia plant, intravenous chelation was used but the workers were allowed to continue working at the plant during therapy. In Tennessee, intravenous chelation was reserved for workers with evidence of lead toxicity; no oral therapy was prescribed.

In Missouri, chelated workers had slightly higher BUN levels than non-chelated workers (Table 15); these workers were also older than those without a chelation history and had longer lead exposure.

VII. Illness in Children of Lead Workers

Health effects of occupational lead exposure are not limited to the workers themselves but extend to involve workers' families, especially their children. Lead dust carried home on work clothing has apparently resulted in contamination of the home environment in several locations. Studies in Tennessee, Minnesota, and Vermont have shown elevated lead content in household dust taken from homes of lead workers. Young children living in these homes have been shown to have elevated blood lead levels, some high enough to require hospitalization and chelation therapy.

A study (4) of lead workers' children, 1-6 years of age, and age-matched neighborhood controls showed elevated blood lead and EP levels in the lead workers' children. Thirty-eight (42%) of 91 children tested had blood lead levels ≥30 μg/100 ml, indicating increased lead absorption. Eight children had levels ≥ 80 μg/100 ml and were hospitalized and chelated. Blood levels of workers' children increased as the fathers duration of employment at the smelter increased (Table 16) in a manner similar to that previously noted (Figure 3) in the workers themselves.

The children's blood levels were modestly correlated with their fathers' blood lead levels (Table 17) and closely correlated with the level of lead in household dust ($r = .50$, $p < .0025$) (Table 18). The hypothesized mechanism of exposure was through ingestion of lead-rich household dust through normal hand-to-mouth activity common in young children. This mechanism has been shown to be responsible for lead absorption in children exposed to lead in inner cities (17) and in children living near primary lead smelters (18,19).

VIII. Performance of Blood Lead Analyses

In the Missouri plant, periodic blood lead testing was performed as part of an ongoing medical surveillance program. The blood tests were analyzed in a laboratory which appears to be providing results which are consistently lower than the actual lead content of the sample. A study using blind, split sample analyses showed this laboratory to be approximately 30% lower than the target value determined by the toxicology laboratory of the Center for Disease Control (Table 19).

IX. Biologic Monitoring System

If lead toxicity is to be prevented, a biologic monitoring system should be instituted which removes a worker from exposure prior to the development of illness. Since overt toxicity (i.e., wrist weakness and lead-related anemia) was noted at a lead level of about 80 ug/100 ml, and since anemia and lead-related symptomatology were noted at blood levels below 80 ug/100 ml, this value should not be used as the "biologically safe" value.

In our studies, we encountered no workers with evidence of lead-related anemia, wrist weakness, or lead-related renal disease, with a blood lead level <60 ug/100 ml. Very few workers with blood lead levels <60 ug/100ml had symptoms consistent with lead intoxication. Since the primary reason for biologic monitoring is to prevent the occurrence of overt illness and chronic disability in workers exposed to hazardous materials, a level should be set which provides a margin of safety which will insure that the most susceptible workers will not develop lead toxicity.

The data in this report clearly support the setting of a biologic threshold limit value of 60 ug/100ml for occupationally-exposed populations. Deleterious health effects may occur at lower blood levels and, if these are conclusively demonstrated in the future, this standard should be revised downward.

X. History of Plant Inspections and Citations

Every plant studied had been inspected by OSHA during the 12 month period prior to our medical studies. The Missouri plant had been cited by OSHA in 1975 alleging a situation that could cause serious physical harm. Our medical studies were performed during the 1 year abatement period

which expired September 10, 1976. During the abatement period, workers were required to wear respirators and a program of improved engineering controls and medical and environmental surveillance were instituted.

The Tennessee plant had been inspected by the State of Tennessee's Occupational and Radiological Health Division and found to be in violation of existing standards on numerous occasions since 1951 (Table 19). The company had been cited twice (1972 and 1974) for excessive exposure to airborne lead and once for an inadequate respiratory protection program (1975). Our studies there were performed during an abatement period.

The Minnesota plant had been found to have excessive airborne lead exposure on numerous occasions since 1949 (Table 20). The most recent inspection in November 1975 had documented air lead levels up to 14 times the standard (0.2 mg/m^3).

Despite these numerous inspections and citations, significant health problems were noted in workers employed at each of these plants.

XI. Evaluation of the Usefulness of the Protoporphyrin Test in Screening Lead Workers:

Determination of the protoporphyrin level in blood has been proposed as a method of screening workers for excess lead absorption. The newly-developed portable hematofluorimeter, which measures zinc protoporphyrin directly from a drop of blood, provides a rapid and inexpensive method of performing this analysis.

Measurement of the sensitivity and specificty of a screening test is essential in evaluating its usefulness. "Sensitivity is the extent to which persons who truly manifest a characteristic are so classified; specificity is the extent to which persons who do not manifest a characteristic are correctly classified" (20)(Example, Table 22). Using data from 152 workers from 3 plants (Tennessee, Missouri, and Minnesota), the sensitivity and specificity of the EP test was calculated using different levels of acceptability for EP and blood lead (Table 23). Ninety-six percent of the workers with blood lead levels $\geq$60ug/100ml had EP levels $\geq$100ug/100ml (sensitivity); 60% of workers with blood lead levels less than 60ug/100ml had EP levels < 100ug/100ml (specificity).

The usefulness of a screening test varies with the prevalence of the characteristic for which screening is performed. As the characteristic becomes less common, the number of "false positives" (e.g., workers with "abnormal" EP tests but "normal" blood lead levels) will increase. The predictive value positive (PVP) measurement is a useful indicator of the applicability of screening tests to a given situation, since it incorporates specificity and prevalence into its calculations (21).

$$PVP = \frac{\text{number with positive EP test \& elevated lead level}}{\text{number with positive EP test}}$$

In a plant with a high prevalence of elevated lead levels (plant A-Table 24), the EP test, using 100ug/100ml as the cut off value, would be more useful (i.e., have a higher predictive value) than in plants (Tables 25 & 26) with lower prevalence of elevated lead levels. As seen in

Tables 24-26, as the prevalence of elevated blood lead levels decreases the number of false positives (workers with "abnormal" EP test but "normal" blood lead level) increases.

Table 1. Lead Plant Investigations 1975-1976

Date of Study	Location	Type of Plant	Date Plant Began Lead Processing	Pounds of Lead Processed per Month
November 1975	Memphis, TN	Secondary smelter	1948	2.3 million
March 1976	Joplin, MO	Lead chemicals plant	--	--
May 1976	Eagan, MN	Secondary smelter	1948	1.5 million
January 1976	Salt Lake City, UT	Secondary smelter	1975	30,000-250,000
March 1976	Atlanta, GA	Secondary smelter	--	--

Table 2: Blood lead levels in lead plant workers

Blood lead level (ug/100ml)	Memphis, TN	Joplin, MO
< 40	14 (18%)	1 (2%)
40-59	12 (15%)	7 (17%)
60-79	26 (33%)	21 (50%)
≥ 80	26 (33%)	13 (31%)
Total tested	78	42

Blood lead level (ug/100ml)	Atlanta, GA	Salt Lake City, UT
< 40	6 (15%)	1 (3%)
40-59	6 (15%)	5 (17%)
60-79	17 (44%)	2 (7%)
≥ 80	10 (26%)	21 (72%)
Total tested	39	29

Blood lead level (ug/100ml)	Eagan, MN	TOTAL
< 40	3 (8%)	25 (11%)
40-59	6 (16%)	36 (16%)
60-79	17 (45%)	83 (37%)
≥ 80	12 (32%)	82 (36%)
Total tested	38	226

Table 3. Symptoms of Lead Poisoning

1. Gastrointestinal symptoms (Nausea, vomiting, diarrhea, and/or constipation)
2. Abdominal pain
3. Neuromuscular symptoms (muscular weakness and/or tremor)
4. Anorexia
5. Joint pains
6. Constitution complaints (fatigue, insomnia, irritability, and/or headache).

Table 4. Distribution of symptoms and blood lead levels of employees, Memphis, Tennessee, November 1975

Symptom	Present/ Absent	Number Employees (%)	Blood Lead Level Mean (µg/100ml)
Gastrointestinal symptoms	Present	17 (22%)	101.24*
	Absent	61	65.98
Abdominal pain	Present	13 (17%)	100.77*
	Absent	65	68.25
Neuromuscular symptoms	Present	21 (27%)	94.52*
	Absent	57	65.98
Anorexia	Present	18 (23%)	93.89*
	Absent	60	67.60
Joint pains	Present	22 (28%)	92.59*
	Absent	60	66.23
Constitutional complaints	Present	31 (40%)	89.68*
	Absent	47	63.11

*Significantly greater than level of those without symptoms ($p < .01$, 1-tailed t test)

Table 5

Percentage of Workers with Symptoms* of Lead Poisoning by Blood Lead and Erythrocyte Protoporphyrin Level at Three Lead Plants

Plant Location	Ratio. (%) of Symptomatic Workers by Blood Lead Level (µg/100ml)			
	< 40	40-59	60-79	≥ 80
Tennessee	1/13 (8%)	2/12 (17%)	7/26 (27%)	18/26 (69%)
Missouri	[0/1 (0%)]†	4/7 (57%)	7/21 (67%)	7/13 (54%)
Minnesota	[0/2 (0%)]	2/6 (33%)	9/14 (64%)	5/12 (42%)
	Erythrocyte Protoporphyrin Level (µg/100ml)			
	< 100	100-199	200-299	≥ 300
Tennessee	2/26 (8%)	2/13 (15%)	11/16 (69%)	13/21 (62%)
Missouri	[1/1 (100%)]	4/10 (40%)	10/15 (62%)	9/15 (60%)
Minnesota	[0/2 (0%)]	1/5 (20%)	[0/4 (0%)]	15/23 (65%)

*Two or more of symptoms in Table 3 during past year

†Brackets around cells with 5 members or less

Table 6

Percentage of Symptomatic* Workers by Blood Lead and Erythrocyte Protoporphyrin (EP) Level

Erythrocyte protoporphyrin (ug/100ml blood)	Ratio (%) of Lead Workers With Symptoms By Blood Lead Level (ug/100ml)				
	< 40	40-59	60-79	≥ 80	Total
<100	1/15 (7%)	2/9 (22%)	[0/5 (0%)]	-	3/29 (10%)
100-199	[0/1 (0%)]+	2/10 (20%)	5/15 (33%)	[0/2 (0%)]	7/28 (25%)
200-299	-	[3/3 (100%)]	10/19 (53%)	8/13 (62%)	21/35 (60%)
300-399	-	[1/2 (50%)]	10/14 (71%)	7/14 (50%)	18/20 (60%)
≥ 400	-	-	5/8 (63%)	15/22 (68%)	20/30 (67%)
Total	1/16 (6%)	8/24 (33%)	30/61 (49%)	30/51 (59%)	

*Two or more of the symptoms or symptom categories in Table 3 during past year

+Brackets around cells where 5 or less persons represented.

Table 7

Percentage of Workers Currently Symptomatic* by Blood Lead and EP Level

Erythrocyte Protoporphyrin	Ratio (%) of Symptomatic Workers by Blood Lead Level (µg/100ml)				
	< 40	40-59	60-79	> 80	Total
< 100	0/13 (0%)	0/8 (0%)	[0/5 (0%)]	-	0/26 (0%)
100-199	[0/1 (0%)]	[0/4 (0%)]	3/14 (21%)	[0/2 (0%)]	3/21 (14%)
200-299	-	[2/3 (67%)]	3/15 (20%)	5/12 (42%)	10/30 (33%)
300-399	-	[0/1 (0%)]	[2/5 (40%)]	3/10 (30%)	5/16 (31%)
≥ 400	-	-	[2/5 (40%)]	8/15 (53%)	10/20 (50%)
Total	0/14 (0%)	2/16 (13%)	10/44 (23%)	16/39 (41%)	

*Workers reporting 2 or more symptoms in Table 3 at time of blood sampling.

Table 8. Current symptoms of lead toxicity in lead workers and controls Missouri, 1976

Symptoms	Lead Workers (43)	Controls (15)
Insomnia	5 (12%)	1 (7%)
Excessive Fatigue	19 (44%)	1 (7%)
Dizziness	8 (19%)	0
Muscle Weakness	6 (14%)	0
Joint Pain	10 (23%)	4 (26%)
Abdominal Cramps	1 (2%)	0
Nausea	1 (2%)	0
Constipation	0	0

Table 10. Mean Hemoglobin Level by Erythrocyte Protoporphyrin Range
Missouri 1976

	Erythrocyte Protoporphyrin Level (µg/100ml)		
	< 200	200 - 399	≥ 400
Number tested	11	21	11
Percentage Anemic+	0%	19%	18%
Mean Hemoglobin Level	15.89	15.03*	14.70**
Standard Deviation	0.91	1.03	0.78

*Significantly less than mean level for EP < 200 (t = 2.26, p < .025, one tailed t)

**Significantly less than mean level for EP levels < 200 (t = 3.14, df = 20)
(p < .005, one tailed t)

+ Anemia defined as hemoglobin level < 14.0 gms/100ml

Table 11. Percentage of production workers with symptoms by duration of employment, Tennessee 1976

Symptoms	MONTHS OF EMPLOYMENT COMPLETED 0-1 (N=17)	2-3 (N=12)	4-12 (N=8)	> 12 (N=20)	Total Number Reporting Symptoms
Nausea	0%	33%	63%	25%	14
Constipation	0%	8%	13%	30%	8
Anorexia	6%	42%	63%	35%	18
Abdominal pain	0%	25%	50%	25%	11
Muscular weakness	0%	25%	50%	35%	14
Joint pains	0%	17%	88%	45%	19
Fatigue	0%	25%	75%	30%	15
Insomnia	0%	25%	25%	25%	10
Irritability	0%	25%	63%	15%	11

TABLE 18. Occurrence of Symptoms in Lead Smelting Plant Employees--Minnesota 1976

Symptoms	Number with Symptom	Percent with Symptom
Tiredness	17	52
Loose bowel movements	11	33
Irritability	9	27
Joint pains	9	27
Muscle weakness	9	27
Loss of appetite	7	21
Headache	6	18
Leg cramps	6	18
Constipation	6	18
Nausea*	5	16
Trouble sleeping	4	12
Throwing up*	4	13
Abdominal pain*	3	9
Weight loss**	2	6
Shakes	2	6

* Excluding 1 employee with symptom due to ulcers.
** Excluding 2 employees with symptom due to diet. Of the 2 with weight loss not related to diet, 1 lost 20 pounds in 15 months and 1 lost 22 pounds in 7 months.
\+ 13 of these 14 smoke.

Table 12. Frequency of symptoms among 22 cases of lead poisoning, Utah 1976

Symptom	Number	Percent
Abdominal cramps	19	86
Nausea	18	82
Diarrhea	12	55
Headache	12	55
Dizziness	12	55
Anorexia	12	55
Vomiting	9	41
Paresthesias	9	41
Constipation	8	36
Fatiguability	8	36
Myalgias	7	32
Weight loss	6	27
Irritability	5	23
Chest pain	5	23
Muscle weakness	4	18
Tremor	4	18
Joint pains	4	13

Table 14: Results of Renal Function Testing, Missouri 1976

SUBJECT	AGE	DURATION OF LEAD EXPOSURE (YEARS)	BLOOD LEAD LEVEL (ug/100ml)	CREATININE CLEARANCE (ml/min/1.73 sq.m BSA)	FASTING URINE OSMOLALITY (mosm/liter)	LEAD CLEARANCE RATE* (ml/min)
1	56	7	154	85		.07
2	48	20	66	142		.82
3	37	20	35	82	871	1.46
4	47	23	71	72	588	0.10**
5	45	8	87	128	1020	0.61
6	53	23	61	91	1025	0.97
7	52	26	61	115	650	0.70
8	38	20	123	109	608	0.94
9	60	25	75	75	278	0.30
10	42	21	66	96	1180	0.36
11	47	7	48	103	820	1.09
12	53	29	96	89	708	0.10
13	35	13	56	109	965	1.13
14	62	16	105	97	912	1.01
15	52	25	78	73	286	0.25
16	53	20	92	108	912	0.04**
17	51	7	80	65	704	2.04
18	52	31	55	43	652	0.80
19	29	4.5	58	112	1114	0.92
Normal Range			< 60	>91	800-1300	

*See text for calculation formula

**Urine lead level for these 2 subjects below analytical detection limit (0.01ug/liter); urine concentration estimated to be .005ug/liter (½ detection limit) for these 2 samples.

Table 15. Blood Urea Nitrogen (BUN) Levels and Serum Creatinine by Treatment History, March 1976, Missouri.

No. of Courses** of Oral EDTA	Total Number	Mean BUN	Mean Creatinine	Number (%) Elevated *
None	28	17.28	1.01	6 (21)
1-3	10	19.30	1.08	3 (30)
4-13	5	21.00	1.12	2 (40)

*Greater than 20 mg/100 ml

**Courses consisted of 4 gms EDTA orally for 5-7 days

Table 16. Relationship of children's blood lead level to duration of father's employment at smelter--Memphis, November 1975

Months of Employment Completed by Father	Children's Blood Lead Level (ug/100ml)		
	Number	Mean ±	S.D.
0 - 2	5	26.4 ±	5.0
3 - 4	8	43.7 ±	21.1
5 - 12	4	59.5 ±	13.4

Table 17 Blood lead levels of current workers' children by parental blood lead level--Memphis, November 1975

Father's blood lead level	Total No. of Children	No. (%) of Children with blood lead levels	
		30-79µg/100ml	> 80µg/100ml
< 60µg/100ml	12	1 (8.3%)	0
60-79µg/100ml	35	16 (45.7%)	0
80-142µg/100ml	25	12 (48.0%)	4 (16%)

Table 18. Comparison of lead concentrations in household dust with children's blood lead level--Memphis, November 1975

Dust Lead Concentration (ppm)	Children's Blood Lead Level (µg/100ml)	
	Number	Mean ± S.D.
0 - 1000	18	21.3 ± 7.8
1000-2000	5	41.6* ± 11.1
2000-3000	5	46.2* ± 21.1
5000-6000	4	65.0* ± 10.4
7000-80,000	6	73.3* ± 34.0

*Significantly greater than mean level of children exposed to 0 - 1000 ppm level of lead in dust ($p < .001$).

Table 19. Lead Concentration in Split Blood Samples, May 1976

Lab	a CDC	b NIOSH Contract Lab	c Private Lab
Sample #			
1	112	112	80.1
2	95	88	58.3
3	98	110	74.5
4	68	69	49.3
5	71	72	46.2
6	62	62	42.9
7	88	85	63.1
8	92	92	63.9
9	88	92	64.8
10	73	70	47.8
11	112	115	80.9
12	72	80	53.4
13	78	81	61.5
14	83	76	51.8
15	79	91	64.8
16	64	66	46.1
17	71	87	57.5
18	82	80	50.2
19	176	157	103.6
20	54	77	50.2
21	56	54	34.8
22	76	76	51.0
23	96	96	68.8
24	130	144	87.4
25	74	74	48.6
26	82	85	57.5
27	78	82	56.7
28	72	77	52.6
29	96	101	66.4
30	84	91	60.7

Mean Percent difference from CDC result: NIOSH Contract Lab + 4.0%
Private Lab - 29.2%

Number (Percent) with acceptable result*: NIOSH Contract Lab 27/30 (90%)
Private Lab 1/30 (3.3%)

*Acceptable result: ± 15% for blood level ≥40.
± 6 µg/100ml for blood level < 40.

Table 20. History of Inspections and Citations at a Secondary Lead Smelter, Tennessee

Excessive Airborne Lead Concentration Noted	Enforcement Letter or Citation Issued	
Year	Year	Number of Letters/ Citations
1952	1951	1
1953	1953	1
1955	1955	1
1956	1958	2
1958	1961	1
1962	1962	4
1965	1965	1
1972	1967	3
1974	1970	1
	1971	1
	1972	1
	1974	1
	1975	1

Table 21. Minnesota Secondary Lead Smelter: Record of Inspections and Citations by State and Federal Agencies

Excessive air lead concentrations noted	Compliance order or citation issued
1949	1963
1950	1970
1955	1973
1956	1974
1957	1975
1960	
1963	
1964	
1966	
1972	
1973	
1974	
1975	

Table 22

Calculation of Sensitivity and Specificity

Blood Lead Level	Number of Workers with Erythrocyte Protoporphyrin Levels ≥50	<50	Total
≥ 40	130	7	137
< 40	2	13	15
TOTAL	132	20	152

Sensitivity = $\frac{\text{Number correctly identified as positive*}}{\text{Total number positive}}$

130/137 = 95%

Specificity = $\frac{\text{Number correctly identified as negative**}}{\text{Total number negative}}$

13/15 = 87%

* positive = blood lead level ≥40µg/100ml
**negative = blood lead level <40µg/100ml

Table 23

Sensitivity and Specificity of Erythrocyte Protoporphyrin (EP) Screening Test in Detecting Workers with Elevated Lead Levels.

Sensitivity

Acceptable blood lead level	Acceptable EP Levels		
	50µg/100ml blood	100 µg/100ml	200µg/100ml
40 µg/100ml	95%	90%	70%
60 µg/100ml	99%	96%	78%
80 µg/100ml	100%	100%	98%

Specificity

Acceptable blood lead level	Acceptable EP Levels		
	50µg/100ml blood	100 µg/100ml	200µg/100ml
40µg/100ml	87%	94%	100%
60µg/100ml	50%	60%	88%
80µg/100ml	21%	29%	55%

Table 24

Effect of Screening Hypothetical Plant A Using Different Levels of EP Acceptability

1) Assume plant with 100 workers;
2) 75 with blood lead levels ≥60µg/100ml
3) 25 with blood lead levels < 60µg/100ml

Using EP cut off of 100 µg/100 ml whole blood (sensitivity 96%, specificity 60%)

1) Correctly positive*: 72 workers
2) Fail to identify 3 workers with elevated blood lead levels
3) Correctly identify 15 workers with "normal" blood lead levels
4) Incorrectly label 10 workers with "normal" levels as having increased lead absorption

Using EP cut off of 200 µg/100 ml blood (sensitivity 78%, specificity 88%)

10 Correctly positive: 58 workers
2) Fail to identify 17 workers with elevated blood lead levels
3) Correctly identify 22 workers with "normal" blood lead levels
4) Incorrectly label 3 workers with "normal" levels as having increased lead absorption

*Workers with elevated EP who in fact have elevated blood lead level (≥60µg/100ml).

Table 25

Effect of Screening Hypothetical Plant B Using Different Levels of EP Acceptability

1) Assume plant with 100 workers;
2) 50 with blood lead $\geq$60µg/100ml
3) 50 with blood lead $<$60µg/100ml

Using EP cut off of 100µg/100ml

1) Correctly identify 48 workers wtih elevated blood lead levels
2) Fail to identify 2 workers with elevated blood lead levels
3) Correctly identify 30 workers with "normal" blood lead levels
4) Incorrectly label 20 workers with "normal" blood lead levels as having increased lead absorption

Using EP cut off of 200µg/100ml

1) Correctly identify 39 workers with elevated blood lead levels
2) Fail to identify 11 workers with elevated lead levels
3) Correctly identify 44 workers with "normal" blood lead levels
4) Incorrectly label 6 workers with "normal" blood lead levels as having increased lead absorption

Table 26

Effect of Screening Hypothetical Plant C Using Different Levels of EP Acceptability

1) Assume plant with 100 workers
2) 20 with blood lead level $\geq$60µg/100ml
3) 80 with blood lead level $<$60µg/100ml

Using EP cut off of 100 µg/100ml:

1) Correctly identify 19 workers with elevated blood lead levels
2) Fail to identify 1 worker with elevated blood lead levels
3) Correctly identify 48 workers with "normal" blood lead levels
4) Incorrectly label 32 workers with "normal" blood lead levels as having increased lead absorption

Using EP cut off of 200 µg/100ml:

1) Correctly identify 16 workers with elevated blood lead levels
2) Fail to identify 4 workers with elevated lead levels
3) Correctly identify 70 workers with "normal" blood lead levels
4) Incorrectly label 10 workers with "normal" blood lead levels as having increased lead absorption

Table 27

Predictive Value Positive of EP Test Using Different Levels of Acceptability in Plants with Varying Prevalence of Elevated Blood Lead Levels.

Plant	Prevalence of Elevated Lead* Levels	Using EP Cut Off of 100µg/100ml blood	Using EP Cut Off of 200µg/100ml
A	75%	88%	95%
B	50%	71%	87%
C	20%	37%	62%

* ≥ 60µg/100ml

Fig. 1. BLOOD LEAD AND ERYTHROCYTE PROTOPORPHYRIN LEVELS IN WORKERS, MEMPHIS, TENNESSEE, 1975

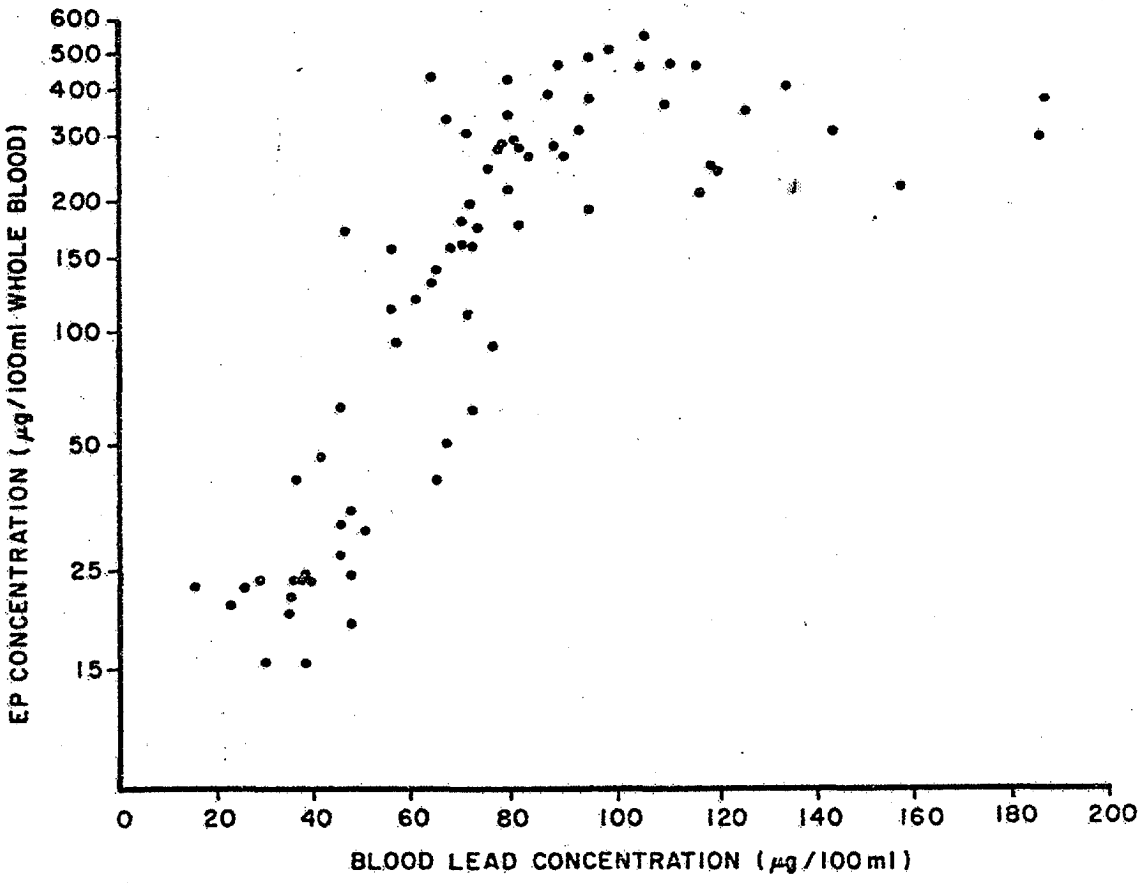


Fig. 2

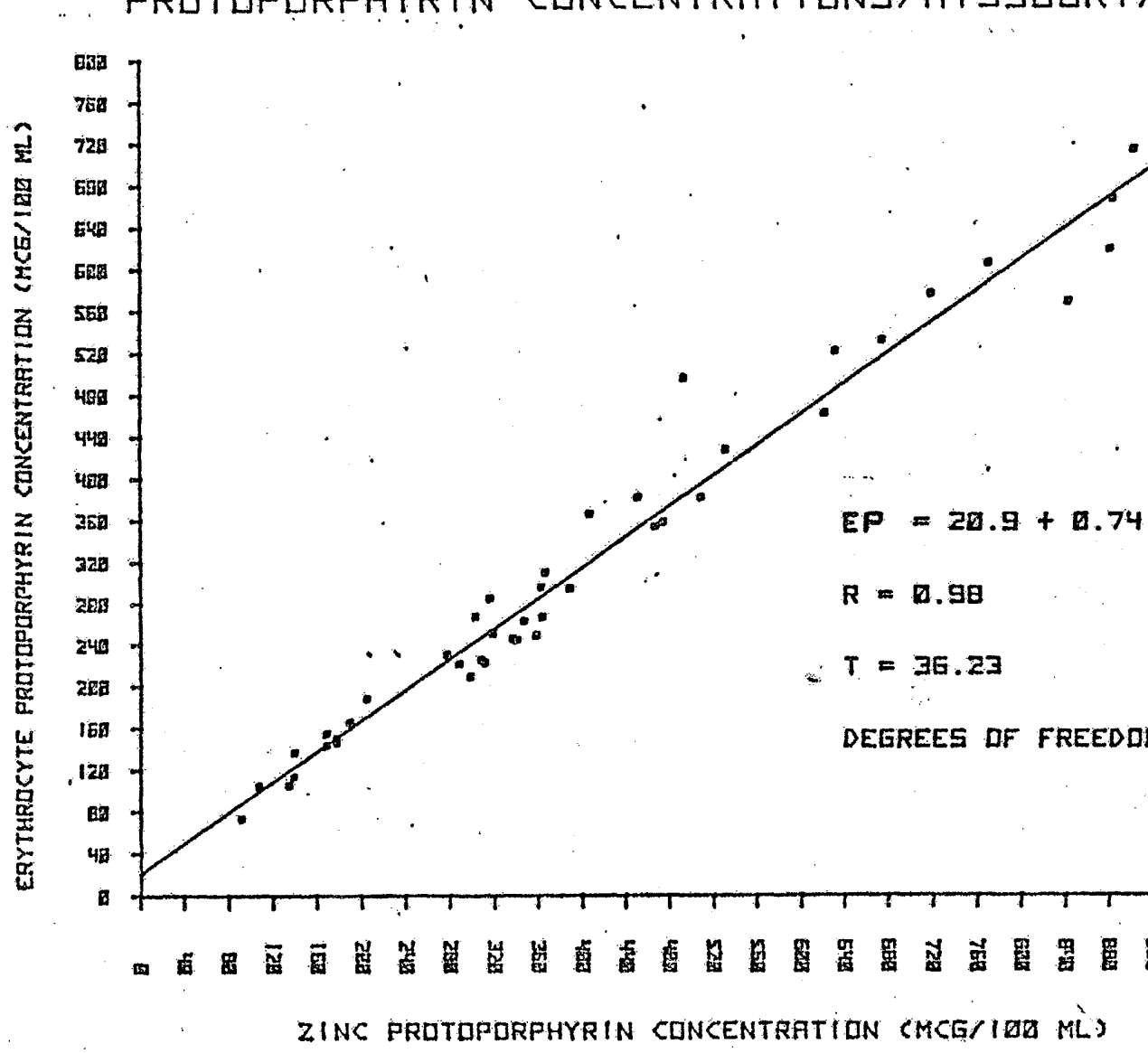
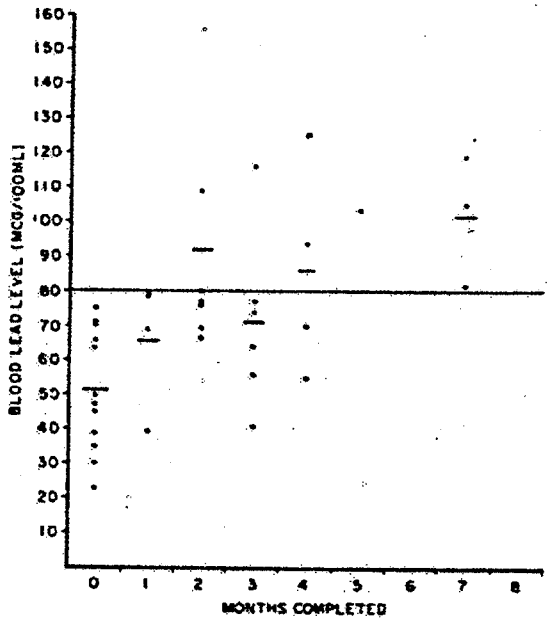


Fig. 3 LEAD LEVEL IN PRODUCTION WORKERS, BY LENGTH OF EMPLOYMENT



Fig

LEAD CLEARANCE RATE BY DURATION OF LEAD

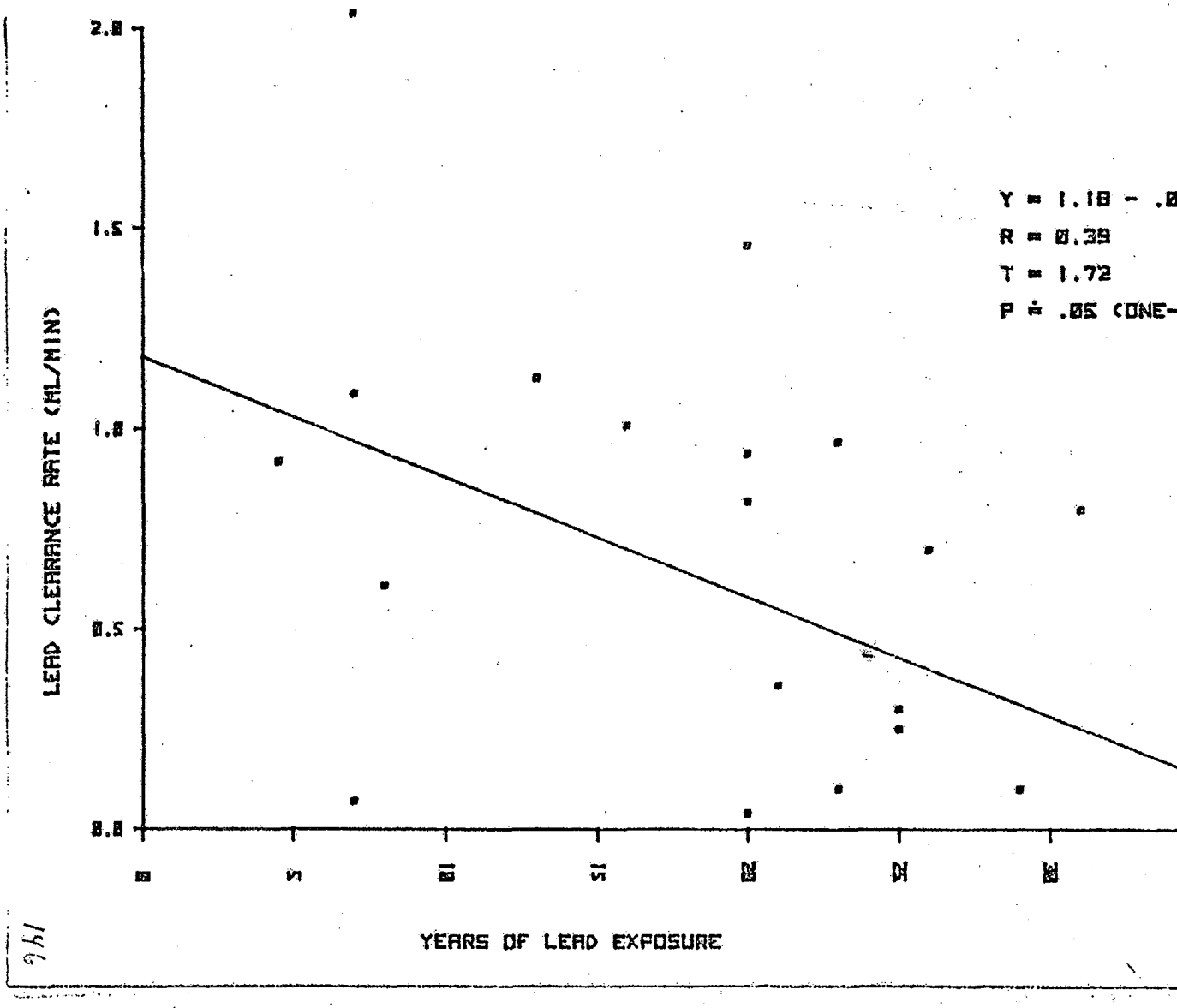
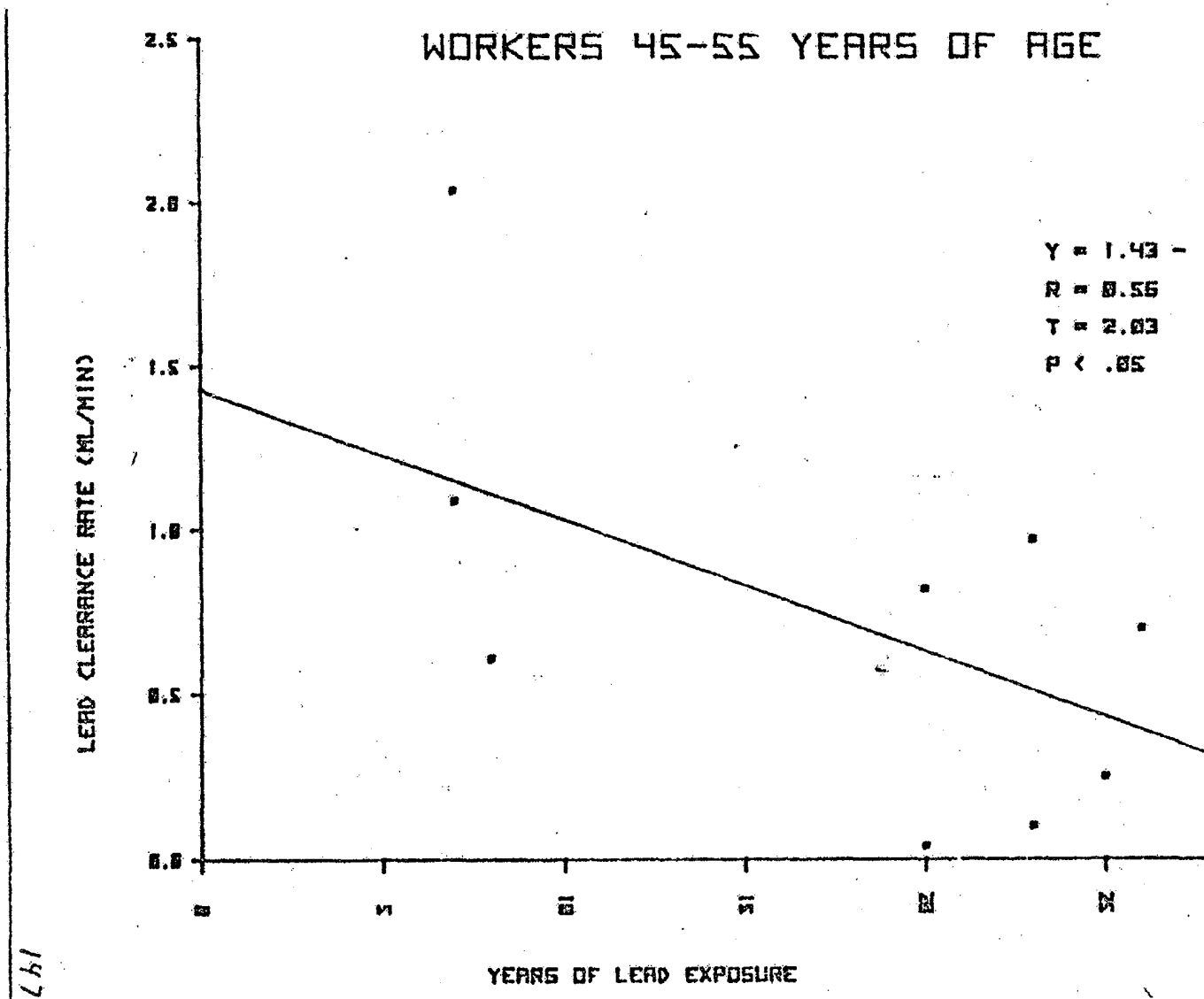


Fig. 5

LEAD CLEARANCE RATE BY DURATION OF LEAD E

WORKERS 45-55 YEARS OF AGE



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Appendix 1

FOR ADMINISTRATIVE USE
LIMITED DISTRIBUTION
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PUBLIC HEALTH SERVICE-CDC-Atlanta
EPI-76-33-2 March 5, 1976

TO : Director, Center for Disease Control

FROM : Cancer and Birth Defects Division
Bureau of Epidemiology

SUBJECT: Occupational Lead Poisoning in Tennessee

SUMMARY

Following reports of excessive blood lead levels and illness in workers at a secondary lead smelter in Memphis, Tennessee, 77 (92%) of 84 current employees and 1 former employee were examined in November 1975. Blood lead levels ranged to 184µg/100ml. with 33% >80µg/100ml. Elevated blood lead levels were seen in all areas of the plant, especially in production workers. Classical symptoms of lead poisoning were evident: abdominal pain (17%), gastrointestinal dysfunction (22%), joint pain (28%), neuromuscular symptoms (27%), and anorexia (23%). Ten workers were found to have lead neuropathy, with weakness of wrist extensor muscles. Anemia was noted in 11 workers and correlated with elevated blood lead levels and erythrocyte protoporphyrin concentrations. There was also suggestive evidence of lead-related renal disease. Increased arsenic absorption was noted in 10 (14%) of 72 employees. Thirty-two (68%) of 47 children of smelter workers had blood lead levels ≥30µg/100ml; 4 of these children with levels ≥80µg/100ml were hospitalized and treated for lead poisoning. A survey of 228 children living near the smelter showed no excessive lead absorption. In January 1976, management temporarily closed the smelter and initiated efforts to redesign the production process.

INTRODUCTION

On November 14, 1975, Philip J. Landrigan, M.D., Chief, Environmental Hazards Activity, Cancer and Birth Efects Division, Bureau of Epidemiology, was notified by David S. Folland, M.D., EIS Officer located in Tennessee, of a problem of lead poisoning in workers at a secondary lead smelter in Memphis. Preliminary evaluation of the plant by Myron Frank, M.P.H., Staff Epidemiologist, Division of Communicable Disease Control, Tennessee Department of Public Health, had indicated that blood lead levels were elevated to >60µg/100ml in 46 of 67 employees tested in early November 1975: 4 had levels ≥200µg/100ml, 16 between 100 and 199µg/100ml, and 26 between 60 and 95µg/100ml. An industrial hygiene evaluation by the Department of Public Health's Division of Occupational and Radiological Health in November 1974 had noted excessive exposure to airborne lead within the plant, and a visit in October 1975 revealed an inadequate respiratory protective program.

In discussions with Dr. Folland and Robert H. Hutcheson, Jr., M.D., Tennessee State Epidemiologist; George Lovejoy, M.D., Director, Shelby County Health Department, Memphis;

Gordon D. Nifong, Ph.D., HEW Region IV, Division of Prevention; Robert Ligo, M.D., Chief, Medical Services Branch, Division of Technical Services, NIOSH; Edward L. Baker, Jr., M.D., EIS Officer; and Dr. Lindrigan, it was decided that further studies were indicated. A preliminary visit on November 21 by Dr. Baker; T.A. Taylor, Memphis-Shelby County Health Department; Myron Frank; and Ken Kronoveter, Industrial Hygienist, Division of Technical Services, NIOSH, revealed that in view of the excessive blood lead levels in the workers, extensive medical evaluations should begin without delay. Dr. Baker and Wendy A. Peterson, epidemiology-elective student, Bureau of Epidemiology, left for Memphis on November 24 to join Dr. Folland and begin the investigation.

BACKGROUND

The secondary smelter in southwest Memphis, Tennessee, recovers lead from scrap storage batteries and combines the purified metal with other substances (such as iron, tin, arsenic, or antimony) for resale. The smelter was opened in 1948 and has undergone several structural modifications, including most recently the installation, in 1974, of bag houses designed to control atmospheric lead emissions. In the 11 months preceeding this investigation (December 1974-October 1975), 25.5 million pounds of lead were smelted.

Flow of materials through the plant begins in the battery wrecking area, where plates and terminals are removed and conveyed to the reverberatory furnace. The reverberatory furnace yields pure "soft" lead, which is transported to the casting and refining area, and slag, which is refined in the blast furnace along with iron to make "hard" lead. In the casting and refining department, the lead is melted, combined with other substances, and cast into "pigs" for delivery to the customer. The area surrounding the furnaces and casting department, called the yard, is used for storing and transporting raw and processed metal. Bag houses are used to collect emissions from the reverberatory furnace, thus reducing environmental contamination and permitting recycling of lead contained in the effluent. Malfunctioning of this system in late 1974 and 1975 resulted in the accumulation of several tons of finely divided particulate lead which is stored in an open area of the yard. Maintenance men circulate throughout the work areas to repair malfunctioning equipment. The office and laboratory are separated from the production area in a cleaner section of the company's property.

This plant has been monitored by the State of Tennessee since shortly after opening and has been found to be in violation of standards on many occasions (Table 1). Over the past 4 years, the company has been cited 3 times by the Tennessee Department of Public Health Occupational and Radiological Health Division--twice for excessive exposure to airborne lead (1972 and October 1974) and once (October 1975) for an inadequate respiratory protection program. In October 1974, airborne lead levels ranged from 0.3-2.0 mg/m^3 calculated as an 8-hour time-weighted average* (twa). The inspections which resulted in the last 2 of these citations were prompted by reports of lead poisoning in workers at the plant. Company records of blood lead testing of 12 long-term workers showed a rise in their average blood lead levels over the 12 months preceeding this investigation.

PRELIMINARY INVESTIGATIONS

On October 31, Myron Frank, M.P.H., Staff Epidemiologist, Epidemiology Section, Tennessee Department of Public Health (TDPH), visited the smelter at the request of the Occupational and Radiological Division, TDPH. He interviewed and obtained venous blood samples on 67 workers. Of these, 53.7% were found to have blood lead levels above 80μg/100ml, a level indicative of excessive absorption of lead requiring immediate medical evaluation (1). Fifty-four and one-half percent had significantly elevated levels (≥190μg/100ml blood) of erythrocyte protoporphyrin, indicative of toxic inhi-

*The Occupational Safety and Health Administration has established 0.2mg/m^3 as the 8-hour average allowable exposure for lead in air in the workplace.

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bition of hemoglobin metabolism by lead (2). Accordingly, more comprehensive examinations were planned to assess the health consequences of these elevated blood levels.

TABLE 1

History of Lead Exposure At A Secondary Lead Smelter

Excessive Airborne Lead Concentration Noted	Enforcement Letter or Citation Issued	
Year	Year	Number of Letters/ Citations
1952	1951	1
1953	1953	1
1955	1955	1
1956	1958	2
1958	1961	1
1962	1962	4
1965	1965	1
1972	1967	3
1974	1970	1
	1971	1
	1972	1
	1974	1
	1975	1

METHODS

On November 24 and 25, workers were requested to participate in a comprehensive medical examination designed to evaluate the health effects of lead exposure at the plant. Participation was voluntary, and workers were advised of the provisions of the Privacy Act of 1974. Attempts were made to contact former workers who had terminated employment within the past 3 months. Those agreeing to participate were interviewed by 2 representatives of the Memphis-Shelby County Health Department regarding their work histories, symptoms during the past year consistent with lead poisoning, and certain demographic information. Two physicians who were not aware of the workers' exposure histories performed brief physical examinations, with special attention given to the nervous system (Appendix I). Blood pressure was measured by an automated system. Laboratory tests included blood sampling for determinations of lead, erythrocyte protoporphyrin (EP), hemoglobin, urea nitrogen, glucose, cholesterol, and uric acid concentrations; urine sampling for arsenic concentration; chest x-ray; and, on selected individuals (those examined on the second day and those with elevated blood lead levels on earlier testing), measurement of conduction velocity of the right ulnar nerve. Blood sampling, chest x-ray, and physical exams were performed in a mobile unit supplied by the Memphis-Shelby County Health Department with the assistance of county health department personnel. Nerve conduction tests were performed by Richard Gilmartin, M.D., staff neurologist, LeBonheur Children's Hospital, who used cutaneous electrodes without sedation. Blood and urine analyses were performed in the laboratories of the Memphis-Shelby County Health Department; the Clinical Chemistry Division, Bureau of Laboratories, CDC; and the National Institute for Occupational Safety and Health.

A concurrent industrial hygiene evaluation was conducted by John Baker, Industrial Hygienist, Occupational and Radiological Health Division, TDPH. This evaluation included sampling of personnel in each area of the plant to determine individual levels of exposure to airborne lead.

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Children of workers and of families living near the plant were tested for lead absorption by the Childhood Lead Poisoning Prevention Program, Memphis-Shelby County Health Department, under the leadership of T.A. Taylor, Director.

Seventy-seven current employees and 1 former employee participated in these evaluations, representing 92% of the work force employed on November 24, 1975. All workers were interviewed and tested for blood lead and EP concentration and urine arsenic level. Complete physical examinations, blood chemistry testing, and chest x-rays were performed on all except 7 workers, who were not tested because of a shortage of personnel on the second afternoon. Ulnar nerve conduction velocities were measured on 41 workers.

RESULTS

Blood Lead Testing - Blood lead levels ranged from 16 to 184µg/100ml, with 26 workers (33%) having levels ≥80µg/100ml, 52 (67%) having levels ≥60µg/100ml, and 64 (82%) having levels ≥40µg/100ml.* Elevated blood lead levels were seen in all areas of the plant, especially in production workers (Table 2). Elevated levels in men in battery wrecking and the yard (considered by the company to be relatively low exposure areas) were related in part to transfer of personnel to these areas from high exposure jobs after high lead levels or illness had occurred. These areas of the plant also exceeded the OSHA standard for lead in air. Significantly elevated lead levels were noted in workers who had worked less than 1 month (mean = 51µg/100ml). Blood lead levels ≥80µg/100ml were noted only in those working more than 2 months (Figure 1).

Symptoms - Classical symptoms of lead poisoning were closely related to elevated blood levels. The percentage of workers with 2 or more of these symptoms increased with increasing lead levels (Table 3). Workers with symptoms had significantly higher lead levels than those without symptoms (Table 4).

TABLE 2

Job Category of Employees and Associated Findings,
Memphis, Tennessee, November 1975

Job Category	Number of Employees	Blood Lead Levels (µg/100ml) Mean (± S.E.M.)	Median Months of Employment	Race (Percent Black)
Battery wrecking	10	104.00 (± 14.66)	2	90
Maintenance	9	87.22 (± 9.14)	11	11
Yard	7	85.86 (± 16.26)	2	71
Furnaces	24	77.29 (± 5.35)	5.5	88
Truck driver	1	72.00 -	70	100
Casting	8	70.00 (± 9.55)	3.5	100
All Production	59	89.35 (± 4.15)	4	76
Supervisor	5	49.60 (± 5.67)	1	0
Laboratory	2	47.50 (± 12.50)	10	0
Office	12	40.83 (± 5.12)	17.5	0
Nonproduction	19	43.84 (± 3.71)	12	0

*A blood lead level ≥60µg/100ml is currently felt to represent excessive absorption of lead requiring immediate medical attention. A level ≥40µg/100ml indicates increased lead absorption (1).

Fig. 1 LEAD LEVEL IN PRODUCTION WORKERS BY LENGTH OF EMPLOYMENT

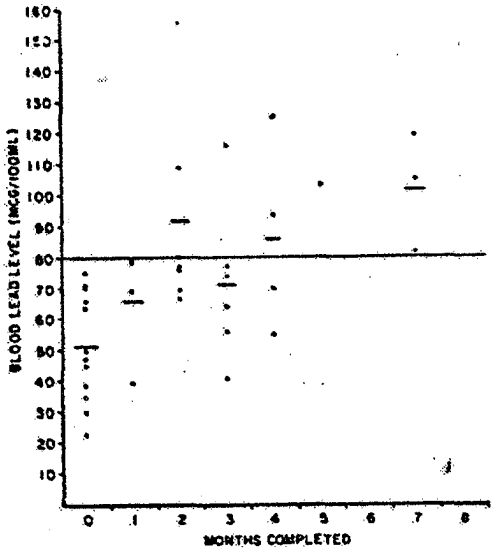


TABLE 3

Blood Lead Levels of Employees in a Secondary Lead Smelter, Memphis, Tennessee, November 1975

	< 40ug/100ml	40–79ug/100ml	≥ 80ug/100ml
Number of Employees	14 (18%)	38 (49%)	26 (33%)
Number with 2 or more symptoms of lead poisoning*	2 (14%)	11 (29%)	17 (65%)

*1. Gastrointestinal symptoms (nausea, vomiting, diarrhea, and/or constipation)
2. Abdominal pain
3. Muscular weakness and/or tremor
4. Anorexia
5. Joint pains
6. Consitutional complaints (fatigue, insomnia, irritability, and/or headache)

TABLE 4

Distribution of Symptoms and Blood Lead Levels of Employees, Memphis, Tennessee November 1975

Symptom	Present/ Absent	Number Employees (%)	Blood Lead Level Mean (µg/100ml)
Gastrointestinal symptoms	Present	17 (22%)	101.24*
	Absent	61	65.98
Abdominal pain	Present	13 (17%)	100.77*
	Absent	65	68.25
Neuromuscular symptoms	Present	21 (27%)	94.52*
	Absent	57	65.98
Anorexia	Present	18 (23%)	93.89*
	Absent	60	67.60
Joint pains	Present	22 (28%)	92.59*
	Absent	60	66.23
Constitutional complaints	Present	31 (40%)	89.68*
	Absent	47	63.11

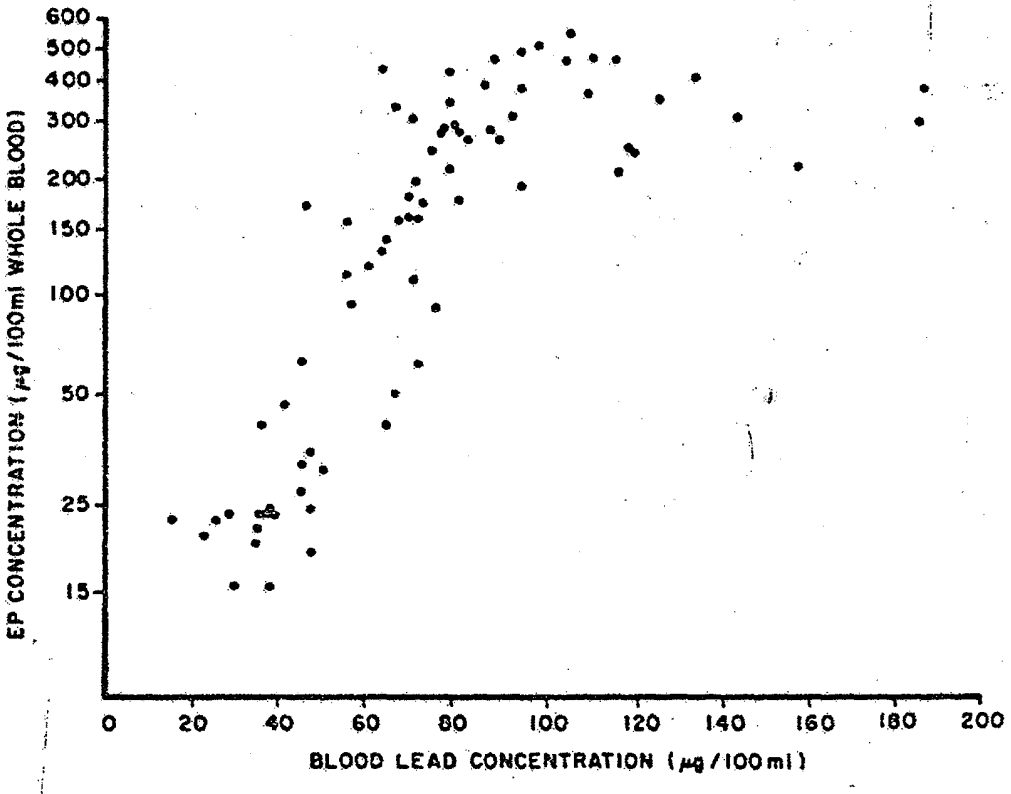
*Significantly greater than level of those without symptoms ($p < .01$, 1-tailed t test)

Hematologic Effects - Inhibition of hemoglobin synthesis by lead was demonstrated by a close relationship ($r = .76$) between blood lead levels and blood levels of erythrocyte protoporphyrin (EP), a hemoglobin precursor which accumulates because of enzymatic inhibition of lead (Figure 2). Anemia, defined as a hemoglobin concentration less than 14 g/100ml (3), was seen in 11 workers. Anemic workers had significantly higher blood lead and EP levels than non-anemic workers (lead: 108.2 vs 86.0µg/100ml, $p < .01$; EP: 309.4 vs 182.3µg/100ml blood, $p < .01$).

Neurological Effects - Peripheral neuropathy was seen in 10 workers, manifested by weakness of wrist extensor muscles tested by physical examination. Workers with weakness had significantly higher lead and EP levels than those not effected (lead: 97.2 vs 69.8µg/100ml, $p < .025$; EP: 326.5 vs 176.9µg/100ml blood, $p < .01$). Nerve conduction velocity did not correlate with the presence of wrist weakness or with lead or EP levels.

Renal Effects - Elevated blood urea nitrogen (BUN) levels (greater than 20mg/100ml) were seen in 6 workers (range 22-40mg/100ml). Mean lead levels for azotemic and nonazotemic workers were 95.00 and 71.89µg/100ml, respectively; these means are not significantly different. Azotemic patients had worked at the plant for a longer time and were older than those with normal BUN levels.

Fig. 2 BLOOD LEAD AND ERYTHROCYTE PROTOPORPHYRIN LEVELS IN WORKERS, MEMPHIS, TENNESSEE, 1975



Arsenic Analyses - Elevated urine arsenic concentrations (greater than 100μg/liter, when corrected to a specific gravity of 1.024) were noted in 10 of 72 (14%) workers tested. Highest levels were seen in the casting area, where arsenic is added to molten lead (Table 5).

Other Results - Abnormalities other than wrist weakness which were detected on physical examination and were suggestive of lead toxicity included tremor of the hands (2 workers), diminished or asymmetrical deep tendon reflexes (4), and possible lead lines on the gums (2). Hypertension was noted in 3 workers. Serum uric acid levels were normal in all workers tested. Blood glucose and cholesterol levels bore no relationship to lead or arsenic levels. Chest x-rays were normal in all but 2 workers--1 with pleural scarring and the other with a nonspecific process requiring repeat studies.

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TABLE 5

Arsenic Levels of Employees, by Job Category,
Memphis, Tennessee, November 1975

Category	Number	Urine Arsenic Levels* (µg/liter) Mean (± S.E.M.)
Casting	8	107.0 (± 22.7)
Maintenance	8	74.5 (± 20.7)
Battery Wrecking	9	58.9 (± 18.3)
Yard	6	31.1 (± 7.2)
Furnaces	21	39.7 (± 6.2)
Supervisor	5	67.7 (± 36.8)
Office and Lab	14	16.7 (± 1.6)

*Corrected to a specific gravity of 1.024 by the formula:

$$\text{Reported Concentration} = \frac{(1.024 - 1.0)}{(\text{Sample specific gravity} - 1.0)} \times \text{Measured Concentration}$$

Community and Family Testing - The Memphis-Shelby County Health Department's Childhood Lead Screening Program tested 228 children under 6 years of age in census tract 55, which borders the smelter and extends 1 mile to the north and east. Thirteen children (6%) had positive tests, i.e., a blood lead level greater than 30µg/100ml and an EP level over 59µg/100ml blood.

The homes of these children were located throughout the census tract. In a census tract of comparable socioeconomic composition located in an industrial area without a lead smelter, 29 of 307 (9%) children tested had positive tests.

Children of smelter workers were also tested in November and December 1975. Preliminary results on 47 children revealed 32 (68%) with lead levels ≥30µg/100ml, 20 (43%) over 39µg/100ml, and 5 (11%) with a lead level over 80µg/100ml or an EP over 190µg/100ml blood.* Four children were hospitalized and chelated during the week of January 5, 1976. Two infants were found to have elevated levels: a 4-month-old boy with a lead level of 50 and an EP of 185, and another 4-month-old boy with a lead level of 52 and an EP of 92.

Correlation of Exposure with Signs and Symptoms - Manifestations of lead poisoning are proportional to the exposure to lead, 2 important determinants of which are duration of exposure and dose. These parameters can be roughly evaluated by relating length of employment and blood lead level to the presence of signs or symptoms of lead poisoning.

None of the workers who had worked at the plant less than 2 months had experienced gastrointestinal symptoms (or abdominal pain) or were found to have wrist drop (Figure 3). One worker, who had completed 1 month of work, was anemic (Hgb 13.0 gm/100ml with a lead level of 79µg/100ml and an EP of 299µg/100ml).

*A lead level ≥30µg/100ml in children is indicative of excessive absorption; a lead level ≥80µg/100ml or an EP level ≥190µg/100ml blood represents excessive absorption which necessitates immediate medical attention (4).

Fig 3 PERCENTAGE OF PRODUCTION WORKERS WITH SYMPTOMS AND SIGNS, BY LENGTH OF EMPLOYMENT

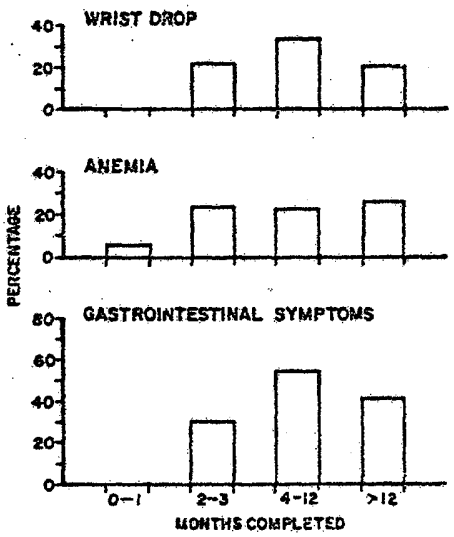
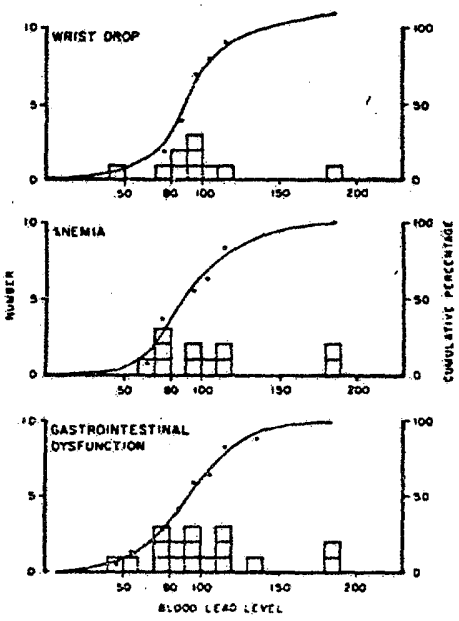


Fig 4 NUMBER AND CUMULATIVE PERCENTAGE OF WORKERS WITH SYMPTOMS AND SIGNS, BY BLOOD LEAD LEVEL



Much higher rates of symptoms and signs appeared in those working for 2-3 months and for 4-12 months. For 5 of 6 symptom categories, those working over a year reported fewer symptoms than those working for 4-12 months.

Gastrointestinal symptoms, wrist drop, and anemia tended to occur at comparable blood lead levels (Figure 4). Virtually no workers had these manifestations with a blood level below 50µg/100ml. Approximately 20%-25% of workers with either gastrointestional symptoms, anemia, or wrist drop had blood lead levels less than 80µg/100ml. A cumulative percentage of 50% with a given sign or symptom was reached at approximately 90µg/100ml.

Relationship of Air Levels to Blood Levels- There were 8 individuals who had both blood testing and air sampling performed on November 25; all of these individuals had worked for at least 2 months in the job which they held on that day. There was a correlation ($r = .56$, $n = 8$) between individual blood and air levels. Mean area air lead concentrations, however, were poorly correlated with mean blood levels of employees working in that area (Table 6). In the battery wrecking area, where air lead levels averaged .12 µg/m^3, worker blood levels ranged from 66 to 184µg/100ml, with the lowest levels being in those working for less than 2 months (66, 75, and 79µg/100ml).

DISCUSSION

Employment at this lead smelter poses a significant risk to the health of production workers and their children. Anemia, peripheral neuropathy, and possible kidney damage occurred in workers as probable sequelae of occupational exposures to lead. Hospitalization of smelter workers' children for treatment of lead poisoning occurred as an apparent result of their exposure to dust from their fathers' contaminated work clothing. Moderately elevated blood lead levels have been noted previously (5,6) in children of lead smelter workers, but none of the levels were high enough to require hospitalization or treatment. In December 1975, 12 plant workers received outpatient chelation with intramuscular injections of calcium EDTA as treatment for signs and symptoms of lead poisoning.

TABLE 6

Mean Air Lead Concentrations and Blood Lead Levels of Employees, by Job Category, Memphis, Tennessee, November 1975

Job Category	Mean Air Lead Concentrations (mg/m^3, 8-hr. TWA)	Number of Workers Sampled	Mean Blood Levels for Workers (ug/100ml)	Number
Yard	.39	2	69.7	5
Smelter	.35	5	72.4	20
Casting	.35	4	88.9	8
Maintenance	.27	2	89.5	11
Battery Wrecking	.12	3	110.1	7

Only workers who had not been transferred from another job category during the previous 2 months.

The most alarming finding of this investigation is the occurrence of lead poisoning in children of lead workers. Preliminary data from a study of these children revealed elevated concentrations of lead (up to 80,000 ppm) in household dust in their homes, most of which did not contain lead paint. This finding underscores the importance of personal hygiene in preventing family members' exposure to lead originating in the workplace and of surveillance of family members. This plant is not unique among secondary lead smelters. An investigation by CDC (7) of a similar smelter in Troy, Alabama, revealed that 81% of workers had blood lead levels over 80ug/100ml, and 8 had been hospitalized for lead poisoning. Since the initial investigation, 21 additional employees of the Alabama smelter have been diagnosed as having lead poisoning, and 6 have been hospitalized. Environmental contamination that has caused human and animal illness has been documented around other secondary lead smelters (8,9).

One of the most significant findings in this study was that approximately 25% of workers with signs and/or symptoms of lead poisoning had lead levels in a range previously presumed to be safe (below 80ug/100ml). While there was good evidence for a dose-response relation in these symptoms, there was no evidence of a threshold of effect. Other recent work has also demonstrated the presence of neurologic (10) and renal (11) toxicity in lead workers with blood lead levels below 80ug/100m. Inhibition of heme biosynthesis has likewise been shown in this and in previous studies to occur at blood lead levels well below 80ug/100ml. Such findings support the current initiative to revise the acceptable level for lead in the blood of workers downward to 60ug/100ml.

An unresolved issue is whether or not the pathologic processes caused by lead absorption are reversible. Anemia, when related to lead-induced enzymatic inhibition, is reversible upon cessation of lead exposure. Levels of protoporphyrin in the blood and urine also tend to return to normal, though more slowly than the blood lead level. Neurotoxicity of lead may be irreversible if the extent or severity of exposure is great enough (12); however, peripheral neuropathies with wrist drop are often reversible

in their early stages. The status of renal toxicity is more controversial: an early effect of lead on the kidney is a reversible tubular dysfunction (Fanconi's syndrome) with little glomerular involvement. Chronic "end-stage" renal disease due to lead is felt by some to follow prolonged severe exposures. Furthermore, recent studies have shown increased rates of lung cancer in workers with prolonged exposure to arsenic (13) and lead (14).

RECOMMENDATIONS

1. Occupational exposure to lead at this plant must be terminated immediately to prevent further disease among this seriously affected group of workers. Temporary closure of the plant is the only way to assure cessation of exposure. Interim measures, such as respirator use and improved personal hygiene should reduce lead exposures but may delay meaningful improvements. Specific hygiene procedures are discussed in the NIOSH Criteria Document on Occupational Exposure to Inorganic Lead (1). Engineering controls are the only way in which a permanent reduction in exposure can be assured.

The plant was closed temporarily on January 19, 1976; it reopened 1 week later at a reduced level of production. Improved personal hygiene and respiratory protective measures have been instituted.

2. Workers with symptoms of lead poisoning, evidence of systemic effects of lead, or elevated blood lead levels should be removed from exposure and should be referred immediately to a physician for evaluation.

Maury Bronstein, M.D., internist, examined all referred workers December 1975-January 1976, treating several of them.

3. A controlled study of children of lead workers should be performed to evaluate sources of lead in the home and to relate these sources to blood lead level elevations.

The study is currently being conducted in cooperation with the Memphis Childhood Lead Screening Program.

4. A more comprehensive evaluation of neurologic and renal toxicity of lead exposure in these workers should be undertaken by specialists in the Memphis area. Workers could be referred through the county health department for such studies.

Richard Gilmartin, M.D., Staff Neurologist, LeBonhaur Children's Hospital, and Fred Hatch, M.D., Chief, Division of Nephrology, University of Tennessee Medical Center, began seeing referred patients in mid-January 1976.

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APPENDIX 1

Form Approved
OMB No. 68-R1009

I.D. #_____

NAME_____ AGE _____ SEX _____

MAILING ADDRESS _____ RACE: _____

PHONE # _____ DATE HIRED _____

JOB DESCRIPTIONS AT SMELTER (MOST RECENT FIRST)	INCLUSIVE DATES

SYMPTOMS DURING THE PAST YEAR:

	YES	NO	DURATION		YES	NO	DURATION
Loss of appetite				Tremors			
Headache				Insomnia			
Muscular Weakness				Irritability			
Nausea				Joint Pains			
Vomiting				Fatigue			
Diarrhea				Leg Cramps			
Constipation				Abdominal Pain			
Cough							

ONSET DATE OF 1st SYMPTOM _____

HAVE YOU SEEN A PHYSICIAN ABOUT ANY OF THESE SYMPTOMS? _____

RECEIVED TREATMENT FOR THESE SYMPTOMS? _____

HOSPITALIZED? _____

ALCOHOLIC BEVERAGE CONSUMPTION? _____

NONOCCUPATIONAL ACTIVITIES WITH POSSIBLE EXPOSURE TO LEAD OR TO DUST OR FUMES? _____

PHYSICAL EXAM:

SKIN _____

CHEST _____

NEUROLOGICAL
- CRANIAL (INCLUDING FUNDI) _____
- MOTOR (WRIST OR ANKLE DROP) _____
- (GRIP STRENGTH) _____
- SENSORY _____
- CEREBELLAR _____
- TREMOR _____

TF 4.240 (CDC)
11-75

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Appendix 4

April 29, 1976

MEMORANDUM TO: Harry L. Gibbons, M.D., M.P.H., Director
Salt Lake City-County Health Department

FROM: Alan G. Barbour, M.D., E.I.S. Officer
Dan Peterson, Occupational Health, Salt Lake City-County Health Department
Jeff Throckmorton, Radiation-Occupational Health, Bureau of Environmental Health

SUBJECT: Epidemic of Lead Poisoning at a Secondary Smelter

INTRODUCTION

In late December 1975 the Salt Lake City-County Health Department was notified by a local physician of 2 recently hospitalized cases of lead poisoning. Investigation revealed another 3 men who had been admitted in December and January to the same hospital and who had been diagnosed as having plumbism. The 5 men worked at a small secondary smelter in Salt Lake City. Members of the Salt Lake City-County Health Department, and of the Bureaus of Environmental Health, of Disease Prevention and of Laboratories of the Utah State Division of Health began an investigation of this outbreak of lead poisoning among the smelter workers.

BACKGROUND

The secondary smelter is located in an industrial area of Salt Lake City but there are clusters of private residences within 1,000 meters of the smelter stack (See Map 1).

The work force usually consists of 20 people: a manager and one part-time secretary in the office, two chemists, two supervisors, two general cleanup and maintenance men, one part-time custodian and 11 furnace workers on three, 3-man shifts per 24 hours. Employee turnover is greatest among the furnace workers; 36 men filled those 11 positions between September 1975 and January 1976.

Prior to 1971 the smelter processed lead. When it changed ownership in 1971, antimony was smelted. From January 1 to August 6, 1975 they produced 92,000 pounds of antimony. On August 6 the smelter operation was again changed to produce lead using the process of direct reduction of oxide material. Into the stationary reverbatory furnace, the furnace workers shovel lead dross shipped in from primary smelters in the Los Angeles, California and Portland, Oregon. The charging material has average concentrations of 73% lead (Pb), 4.5% sulfur, 1.3% antimony (Sb), 1.1% tin, 0.7% copper, 0.8% iron, 0.3% arsenic (As), and 0.2% zinc. The final product averages 87% lead and 13% antimony with traces of tin and copper. The exhaust of the furnace is to a bag house and then through the stack. (See Map 2)

The smelter has a changing room with showers and a small lunch room for employees.

METHODS

Case definitions of lead poisoning and probable lead poisoning are listed in Table 1.

Questionnaires eliciting information on length of employment, job duties, work and social habits, symptoms and onsets were administered to available workers who were both employed at the secondary smelter at some time in period September 1975 - January 1976 and had blood and/or urine lead levels performed between September and January.

Whole blood lead and urine lead and arsenic levels were performed at the Bureau of Laboratories, Utah State Division of Health. Blood samples were drawn into 15 ml heparinized tubes; these tubes had a background of 0.25 μg of lead per tube at maximum. If 10 ml of blood had

been drawn into one tube, the background of the blank tube would contribute at most 2.5 μg to the test result. The colorimetric method of Keenan was used for blood lead determination. Fifteen duplicate blood samples were also tested at the Western Area Occupational Health Laboratory, National Institute for Occupational Safety and Health for whole blood lead, arsenic, and antimony by atomic absorption. Unless otherwise indicated all lead levels used are those reported by the State Laboratory.

Air samples were taken from both personal and stationary samplers on three days during different activities. They were analyzed for lead, arsenic, and antimony using a 0.45μ millipore filter on a M.S.A. permissible portable pump. Ceiling deposits, settled dust, and floor dirt in the furnace room and other locations were also analyzed.

Hospital and physician records of the workers were reviewed after obtaining appropriate releases.

INVESTIGATION

Forty-two persons (41 males, 1 female) both worked at the smelter in the period September to January and had had a lead determination performed. Eleven asymptomatic men had worked only in September but had not been tested. Of the 42, 36 (86%) were interviewed. Six had either left the state or gave no forwarding address when they moved.

Nineteen cases of definite lead poisoning and 3 cases of probable lead poisoning occurred among the 36. The distribution in time of the definite and probable cases by date of onset of symptoms is shown in Figure 1A. Seven men were hospitalized; their admission dates are shown in Figure 1B. The mean duration of hospitalization was 9 days with range of 4 to 14 days.

The incidence of lead poisoning when adjusted for exposure (worker-months) was 75 per 100 worker-months during December and 3 per 100 worker-months for the period of August to November (Table 2).

CLINICAL

The frequency of symptoms of the 22 cases are listed in Table 3. Specific muscle weakness was not a prominent symptom in this outbreak; no worker had wrist drop either subjectively or when examined at time of first treatment. Two hospitalized cases who were tested had mild peroneal nerve slowing on nerve conduction velocity. Mean duration of symptoms was 20.5 days. One worker was ill for 90 days before diagnosis and chelation therapy started. He had been treated for "peptic ulcer". Two workers may have had encephalopathy; one presented with a history of periodic confusion; another had a grand mal seizure after being seizure-free on medication for 3 years. A total of 19 of the cases received chelation therapy.

In Figure 2 are the distribution of blood lead levels (State Laboratory values) for asymptomatic and symptomatic workers. The symptomatic worker who had a blood lead of 20 μg% had at the same time a urine concentration of 0.44 mg/L, elevated urine coproporphyrins (0.9 mg/L) and ΔALA (22 mg/L), and anemia (Hemoglobin 13.0 Gms).

Twenty-one workers who had blood lead determinations also had hemoglobins performed. Figure 3 shows these hemoglobin levels by blood lead level (50-79, 80-120, and >120 μg%). Twelve of 13 men with blood Pb $\geq$ 80 μg% had hemoglobins less than 14.5 Gms. When blood lead levels were adjusted for a hemoglobin of 15.0 Gms, the mean values for the 50-79, 80-120, and >120 μg% groups were respectively 15.2, 13.5, and 13.5 Gms hemoglobin. The worker with a 10.9 Gms hemoglobin, highest determined blood lead level

was 80 μg% (110 μg% when corrected to 15.0 Gm Hgb). His urine ΔALA was 59 mg/L (normal < 6 mg/L), his erythrocyte protoporphyrin was 100 μg%/100 ml, urine coproporphyrin was 607 μg/24 hrs. (normal < 200), and his bone marrow showed increased iron stores, increased sideroblasts, and ringed sideroblasts. Of those workers with blood Pb levels of ≥80 μg%, 7 of 12 with more than 6 months employment at the smelter had hemoglobins of less than 14.5 Gms. Only 2 of 7 workers with 6 or less months of smelter employment and blood Pb levels of ≥80 μg% had hemoglobins below that level.

Serum transaminase (SGOT) levels were elevated in 4 of 8 workers with Pb levels ≥80 μg% (mean 105 units, range 66-156, with normal ≤40 units); six workers with Pb levels of 50-79 μg% all had normal SGOT levels. Alkaline phosphatase, LDH, and bilirubin levels were normal in all 12. Four definite cases had BUN's over 20 but the highest was only 28 and creatinine levels were normal. Tests for reducing substances in the urine of 16 cases were negative.

Duplicate samples tested for blood lead at N.I.O.S.H. laboratory had higher levels than the state laboratory in 13 of the 15 samples. Agreement was closest in the range of values 55 to 168; the mean difference between the two laboratories was 10.3 μg% (Table 6). Whole blood arsenic levels of 15 samples all were below the detectable level by atomic absorption. Arsenic levels on 20 workers' urine samples, 14 (70%) of which had elevated concentrations of lead (>150 μg/L), were in normal range (10-60 μg/L).

Blood antimony levels in 7 of the 15 workers were above the limit of detection (Table 6). Five of these 7 had the highest blood lead levels among all the cases. A 24 hour urine for antimony was less than 200 μg/L in hospitalized case whose urine lead on the same sample was 480 μg/L. Urine antimony levels >1000 μg/L indicate significant absorption.

EPIDEMIOLOGY

The cluster of 20 cases with onset in or around December suggested an intense exposure of limited duration. Among these 20 cases were two refractory repairmen on contract to maintain the equipment. These 2 plus 4 other repairmen from the same contractor worked at the smelter for short periods in December. Blood lead levels were drawn on the 6 repairmen on December 23. Work logs were examined to determine the timing and amount of exposure. Table 4 shows that it was not the total hours of exposure in December but specifically the hours worked during the week of December 8-14 that best identified cases and elevated lead levels.

We then looked at all those employed at the smelter in December. Because only 7 workers (controls) were not cases during this time, the 22 cases were divided into those with blood lead levels ≤ 120 μg% and those with levels >120 μg% for stratified comparisons of types and amounts of exposure. Table 5 summarizes these comparisons. Among the 22 cases there was greater exposure to the furnace room than controls. The cases (both $\leq$ and >120 μg%) tended to have a higher percentage of smokers and to spend more of their day at smelter (full-time vs. part-time) than controls. The 9 workers with level >120 μg% all worked full-time. The sub-group with levels >120 μg% when compared with other sub-group of cases and controls tended to be older, to have worked at the smelter for more than 6 months, and to eat more than 3 meals a week at the smelter. There was no significant difference or trend when the following parameters were examined: frequency of respirator use, frequency of shower and change of clothes after work, alcohol consumption, timing of 8 hour shift, possession of earthenware pottery at home, or milk consumption. The

workers denied taking any lead metal home with them. Only 3 of the 22 cases had any children less than 8 years old at home; the majority of workers were either single or had grown children.

One symptomatic case with onset in December was the company president; his highest blood level was 167 μg%. He spent less than 10% of his time in the furnace room, did mostly office work, and ate all of his meals outside of the plant. A female secretary worked 3-4 hours a day in an adjacent office (see map). She never went into the furnace room. The secretary was asymptomatic but had a blood lead level of 42 μg%. What was their exposure? We found that the intake for the ventilation-heating system supplying their offices was located in the warehouse room next to the furnace room.

The question of why 20 workers became ill in December remained. When the amount of lead smelted by month was tabulated (Figure 4) we saw that no lead was processed in November and relatively little in December. The two cases with onsets in early October followed a month when 249,000 pounds were processed. In November the furnace was closed down for cleaning; the workers did maintenance work around the smelter. In December, although little lead was actually processed, an attempt was made to increase output. Two smaller capacity rotary furnaces were utilized for two weeks (December 2-15) in addition to the large stationary furnace. These rotary furnaces were not adequately ventilated, and fumes accumulated in the furnace room. Several workers described the air inside of the furnace room as being like a "thick fog" for one week.

ENVIRONMENTAL SAMPLING

Both personal and stationary air sampling showed excessive air lead concentrations especially during charging activities (Table 7). The sampling was done when only the reverbatory furnace was being used; the 2 rotatory furnaces were not in operation. Breathing zone samples showed that employees were being exposed to up to 21 times the O.S.H.A. 8-hour time weighted average for lead and 64 times the O.S.H.A. exposure limit for a 2-hour 38 minute sample.

Although all employees said that they used their respirators "frequently" or "always", several were observed to be inconsistent in their use. However, one full-time employee, who spent less than 50% of time in the furnace room, had antimony hypersensitivity reactions and wore his respirator religiously; his blood lead level was 143 μg%.

Area samples of the smelter revealed the following: (1) Ceiling deposits in the furnace room were 17% lead, 1% antimony, and 0.5% arsenic; (2) Dust on the furnace room floor was 13% lead, 0.25% antimony, and 1% arsenic; (3) A wipe sample off 2.5 sq. feet of the lunch room table yielded 0.46 mg lead, 1.45 mg antimony, and 0.25 mg arsenic; and (4) 2.5 sq. feet wipe sample off the secretary's desk contained 0.41 mg lead, 1.25 mg antimony, and 0.35 mg arsenic.

DISCUSSION

We found that the outbreak was in major part due to the operation of the rotatory furnaces without adequate ventilation in early December. Two part-time workers became ill after only 2 days exposure in the furnace room. The distribution in time and clinical presentation of the cases was more consistent with a high intensity exposure than chronic lead poisoning.

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There is, though, some evidence of excessive lead exposures before and since the limited operation of the rotatory furnaces: (1) Two of the cases had onsets of illness in October; (2) Air lead concentrations were excessively high during normal operation of the reverbatory furnaces; and (3) long term employees tended to have lower hemoglobins than employees of shorter duration. While, the main route of intoxication was undoubtedly inhalation of the inorganic lead fumes and particles from the furnaces, the considerable environmental contamination of work and eating areas could have contributed to the worker's lead burdens.

Arsenic (As) and antimony (Sb) were present in excessive concentrations in some of the air samples and in substantial amounts in dust and deposits. But there was no evidence of a significant contribution of either of these metals to the illnesses seen. Arsenic urine levels were never elevated and while blood arsenic levels do not correlate as well with clinical status as urine As concentrations, blood As levels were not elevated in specimens from patients at their most symptomatic times. The blood Sb levels seem to vary directly with blood Pb levels but they were 1 to 2 orders of magnitude less than the corresponding Pb concentrations. This was an epidemic of inhalation lead poisoning in an industrial setting. Such occurrences were common in the earlier part of this century and before, but at primarily, lead poisoning is primarily an endemic problem in most industries. Most attention has been focused on setting exact tolerable limits of exposure-both air concentrations and blood concentrations. In this dramatic outbreak such subtleties are less relevant. A small industry operated with little comprehension of the toxicity of lead and with excessive exposures of lead for all its employees. Such an industry should not

have even begun their lead smelting without prior consultation on proposed process, engineering and safety programs.

CONTROL MEASURES

(1) After initial evaluation imminent danger notices were posted, and the management was advised to inform their employees as to the danger cited by this notice.

(2) When air sampling should excessive lead concentrations during normal operation and when the deficiencies were not corrected, the smelter was closed.

(3) When more adequate furnace ventilation systems were installed for exhaust of fumes, when methods of charging that minimized dust emissions were instituted, and when housekeeping had improved, the smelter was re-opened.

(4) Surveillance by air sampling and periodic blood lead levels of workers will continue.

We wish to acknowledge the laboratory assistance of Dick Issacs and Jack Omar at the Bureau of Laboratories, Utah State Division of Health.

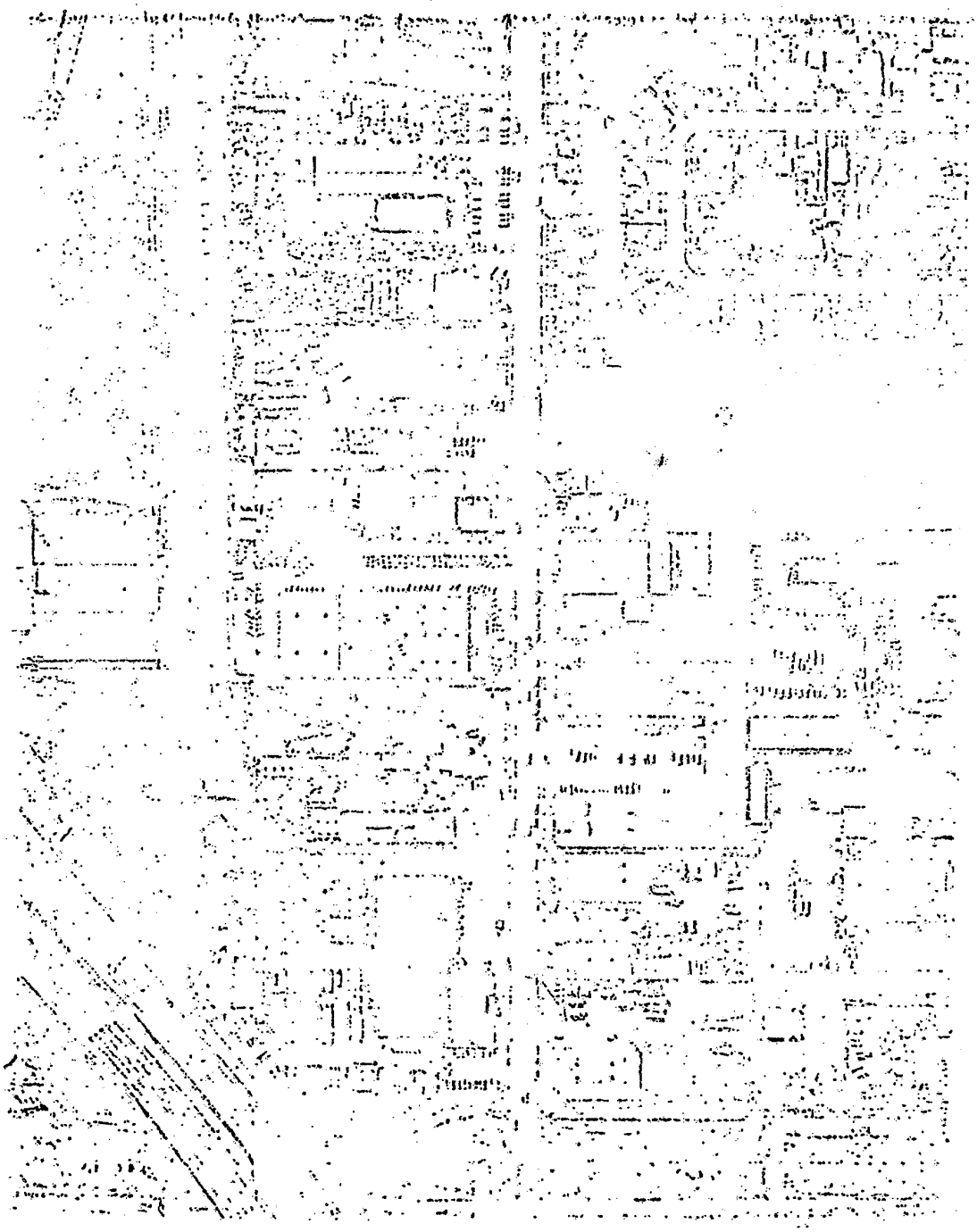

FIGURE 1A

LEAD POISONING- CASES IN SMELTER WORKERS BY WEEK OF ONSET

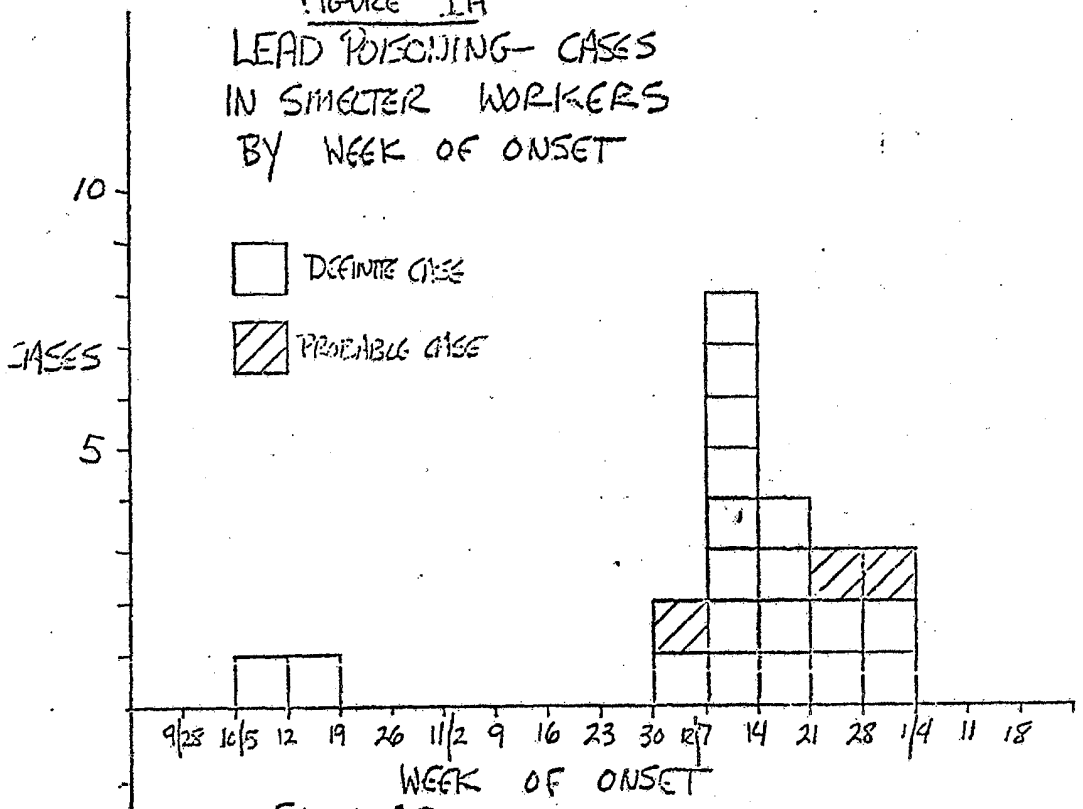

FIGURE 1B

HOSPITALIZED CASES BY WEEK OF ADMISSION

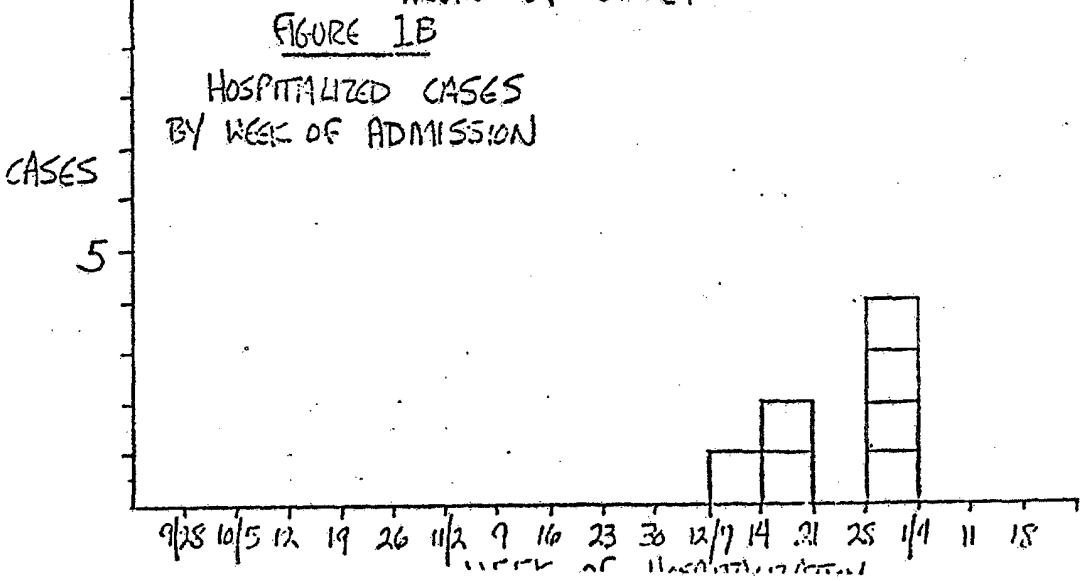

FIGURE 2

BLOOD LEAD LEVEL BY CLINICAL STATUS

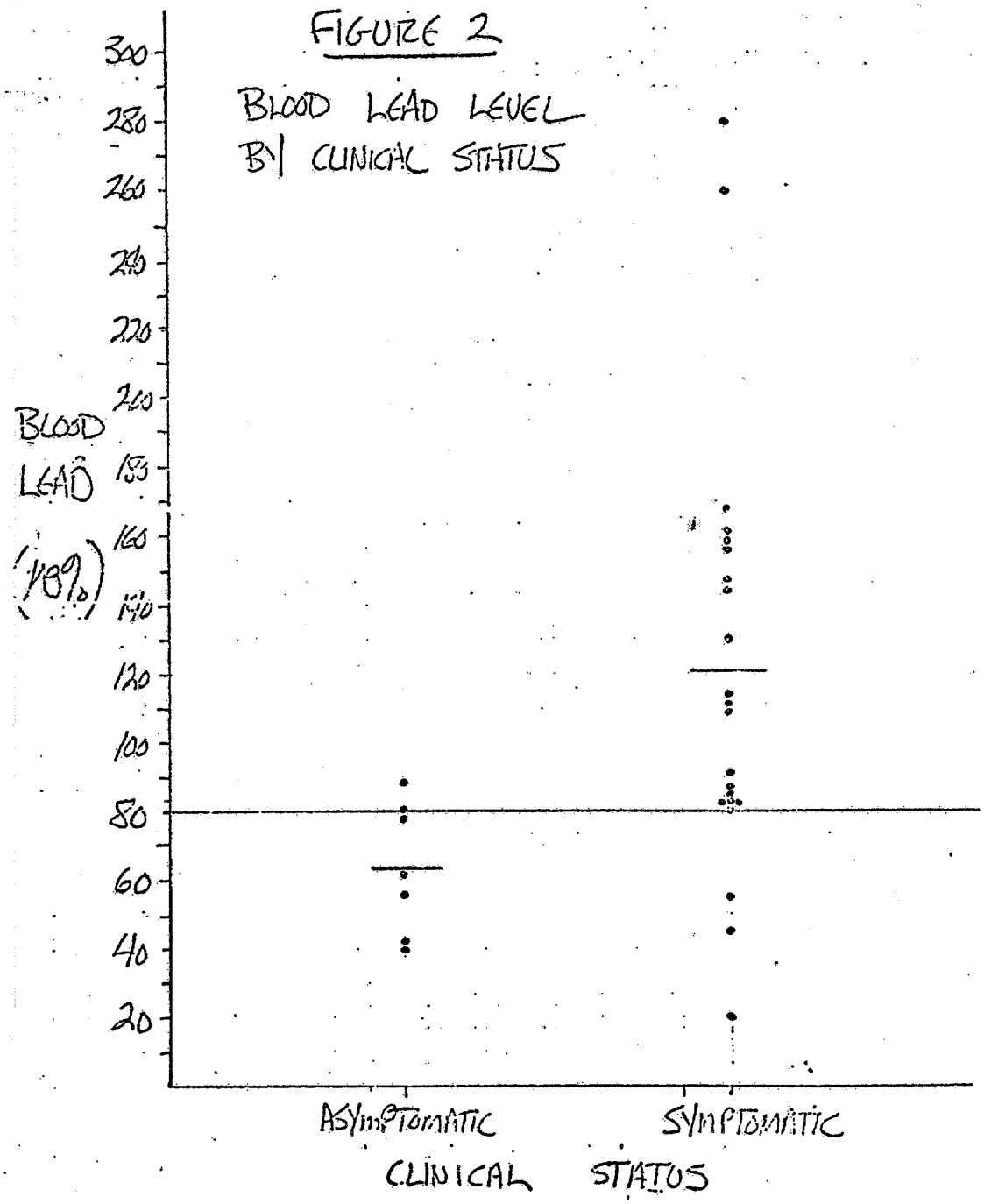

FIGURE 3

HEMOGLOBINS AMONG SMELTER WORKERS BY BLOOD LEAD LEVEL

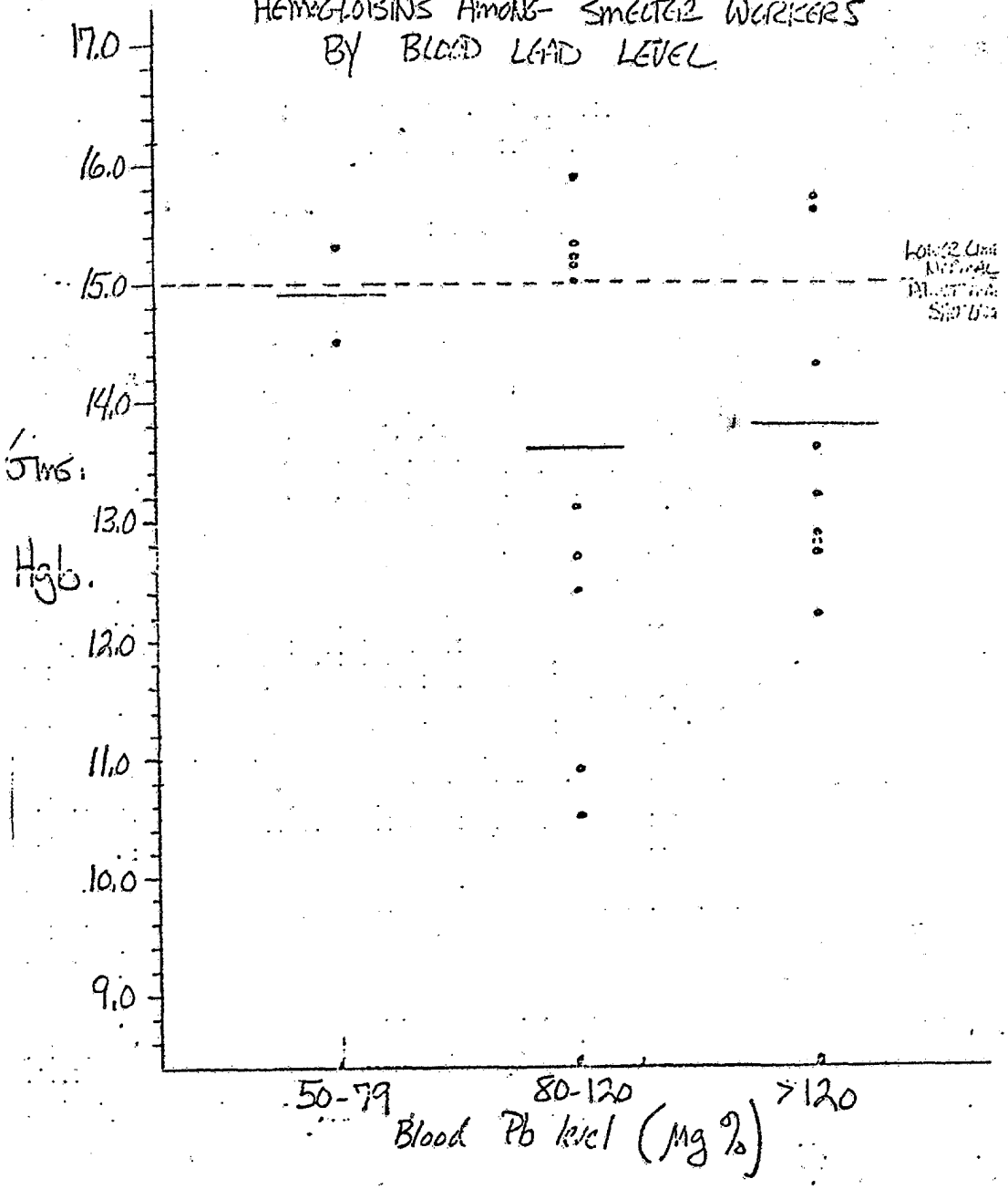

FIGURE 4

AMOUNT OF LEAD PROCESSED BY MONTH JULY 75 - JAN 76

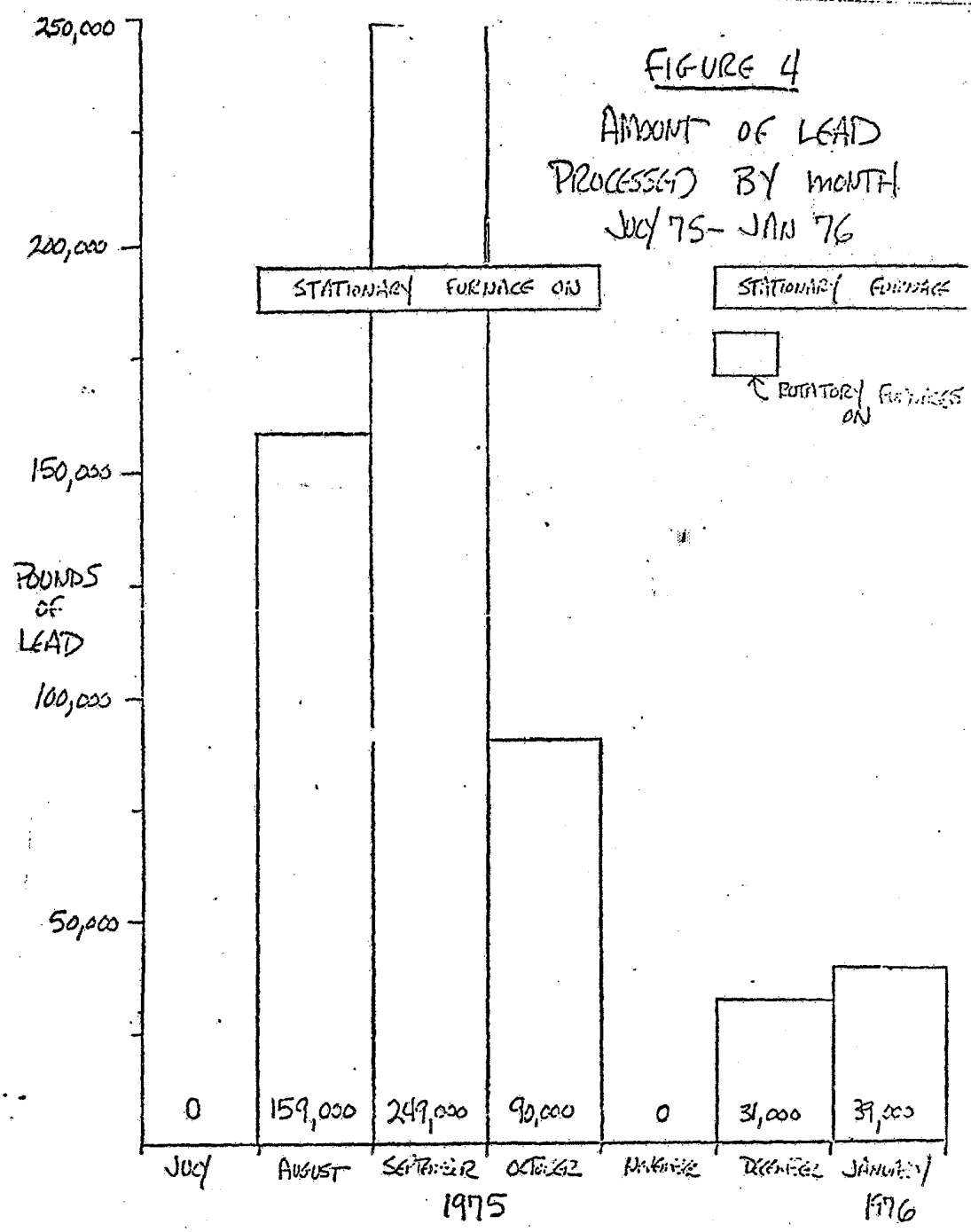

TABLE 1

CASE DEFINITIONS

A. Definite Lead Poisoning:

Required either: (1) Two of the following criteria plus blood lead level of >120 μg/100 ml (μg%); (2) The presence of three of the following criteria plus blood lead of ≥ 80 μg/100 ml (μg%) blood or urine lead of > 200 μg/L.

B. Probable Lead Poisoning:

This diagnosis required either: (1) The presence of one of the following criteria plus blood lead of ≥ 80 μg/100 ml blood or urine lead of > 200 μg/L; or (2) the presence of two of the following criteria plus blood lead of 50-79 μg/100 ml or urine lead of 150-200 μg/L.

CRITERIA

1. Abdominal pain or cramps.
2. Constitutional symptoms (at least two of the following: headache, general weakness, myalgias, dizziness, fatigueability, irritability, and/or insomnia).
3. Gastrointestinal dysfunction (nausea, anorexia, vomiting, diarrhea, and/or constipation).
4. Neuromuscular symptoms (specific muscular weakness, tremor, and/or paresthesias).
5. Joint pains and/or chest pains.
6. Anemia (hemoglobin <14.5 Gms for adult male at 4500 feet above sea level).
7. Elevated free erythrocyte protoporphyrin (FEP) level (≥ 110 μg/100 ml blood) and/or elevated urinary delta amino levulinic acid (ΔALA) level of ≥ 20 mg/L.

TABLE 2

Incidence of Lead
Poisoning at Secondary Smelter
August - December 1975

	Number of Worker-Months Exposure*	Number Of Cases	Cases/100 Worker-Months
August-November	78	2	3
December	24	18	75

* Excludes 6 refractory repairmen who worked <7 days in December.

TABLE 3

Frequency of Symptoms Among
22 Cases of Lead Poisoning*

Symptom	Number Reporting	Percent
Abdominal cramps	19	86
Nausea	18	82
Diarrhea	12	55
Headache	12	55
Dizziness	12	55
Anorexia	12	55
Vomiting	9	41
Paresthesias	9	41
Constipation	8	36
Fatigueability	8	36
General weakness	8	36
Myalgias	7	32
Weight loss (>2 Kg)	6	27
Irritability	5	23
Chest pain	5	23
Specific muscle weakness	4	18
Tremor	4	18
Insomnia	4	18
Joint pains	4	18

* 19 definite and 3 probable cases.

TABLE 4

Exposure of Refractory Repairmen to Smelter
December 1975

Worker No.	Blood Pb Level (12/23)	Onset of Symptoms	Hours Exposure/Week			Total Hrs. Exposure
			Dec 1-7	Dec 8-14	Dec 15-21	
33	87	12/10	0	16	7	23
32	81	12/10	0	9	0	9
31	62	NONE	18	1	19	38
34	40	NONE	1	0	8	9
35	40	NONE	0	0	13	13
36	40	NONE	0	0	0	4

TABLE 5

Characteristics of Controls and Cases

	Control N=7	Case (Blood Pb ≤ 120 μg) N=12	Case (Blood Pb > 120 μg) N=9
Mean Age (range)	35 (17-60)	27 (18-48)	41 (20-61)
Length of employment			
≤6 months	4	7	3
>6 months	3	5	6
Daily work			
Part-time	4	5	0
Full-time	3	7	9
Cigarette smoking			
Yes	1	8	7
No	6	4	2
Alcohol consumption			
Never or occasional	5	8	5
Moderate to heavy	2	4	4
Furnace room exposure*			
50% of day	5	2	4
50% of day	0	8	5
Meals at smelter*			
0-2/week	2	6	2
3-5+/week	3	4	7

* Excludes 6 Refractory Repairmen.

TABLE 6

Blood Lead, Arsenic, and Antimony Levels in Smelter Workers

Worker #	Date of Onset of Symptoms	Date of Treatment	Date of Sample	Bureau of Laboratories	N.I.O.S.H. Laboratories		
				Lead level (μg%)	Lead	Arsenic (μg%)	Antimony (μg%)
33	12/10	1/5	12/23	87	90	<5*	<2.5*
18	12/12	12/18	12/14	220	262	<5	3.0
6	12/9	12/29	1/2	83	98	<5	<2.5
9	12/20	12/23	12/15	250	285	<5	4.8
4	12/20	12/23	12/30	280	338	<5	5.1
8	12/31	1/6	1/2	82	116	<5	3.8
11	12/28	2/1	12/15	55	59	<5	<2.5
26	10/10	1/4	12/23	144	152	<5	4.3
7	12/19	12/23	12/18	168	167	<5	4.0
12	12/17	12/23	12/15	110	132	<5	<2.5
2	12/5	12/24	12/15	160	167	<5	<2.5
3	12/9	12/23	12/29	71	73	<5	5.6
23	12/21	12/30	1/2	40	46	<5	<2.5
1	ASYMPTOMATIC	-----	12/15	30	87	<5	<2.5
15	ASYMPTOMATIC	-----	12/18	42	28	<5	<2.5

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TABLE 7

AIR SAMPLE CONCENTRATIONS ON THREE DAYS

Activity for Day	Location	Duration	Sample Results (mg/m^3) Pb	Sb	As
General cleanup	furnace worker	242	.060	.586	.0025
	office	330	.0318	.000	.0021
	welder #1	214	.564	.915	.0169
	welder #2	339	.262	.454	.006
	furnace room	317	.0965	.107	.0043
	chemist	244	.168	.223	.0034
	lunch room	261	.152	.487	.0043
Tapping furnace	furnace worker #1	164	1.61	2.93	
	furnace worker #2	165	.586	.909	
	furnace room	182	1.21	1.65	
	office	170	.0.96	.000	
	welder	180	.729	.912	
Charging furnace	furnace worker	197	8.40		
	furnace room	380	2.65		
	welder #1	357	1.39		
	welder #2	188	.574		
	office	344	.121		
	lunch room	322	.029		

Distribution: ·Jeff Throckmorton, Radiation-Occupational Health
Dan Peterson, Salt Lake City-County Health Department
Rich Ranck, Salt Lake City-County Health Department
·Lynn Thatcher, Bureau of Environmental Health
·Dennis Dally, Bureau of Environmental Health
·Dick Isaacs
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