U. S. DEPARTMENT OF HEALTH, EDUCATION AND WELFARE
CENTER FOR DISEASE CONTROL
NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH
CINCINNATI, OHIO 45226

HEALTH HAZARD EVALUATION DETERMINATION
REPORT NO. 76-36-339

EAGLE PICHER INDUSTRIES, INC.
JOPLIN, MISSOURI

NOVEMBER 1976

## I. TOXICITY DETERMINATION

It has been determined that lead exposure in the Lead Chemicals Department, Chemicals and Fibers Division, Eagle Picher Industries, C and Porter Streets, Joplin, Missouri, has resulted in excessive concentrations of lead in the blood of workers so exposed.

It has been determined that symptoms consistent with lead intoxication as well as signs of lead toxicity including anemia, peripheral neuropathy, and kidney disease are present in workers so exposed. Therefore, lead exposure at this plant is judged to present both an acute and chronic intoxication hazard to workers.

These determinations are based on medical interviews, physical examinations, laboratory testing, and information regarding the toxicity of substances used in this department.

## II. DISTRIBUTION AND AVAILABILITY OF REPORT

Copies of this Determination Report are available upon request from NIOSH Division of Technical Services, Information Resources and Dissemination Section, 4676 Columbia Parkway, Cincinnati, Ohio 45226.

Copies have been sent to:

a) Eagle Picher Industries, Joplin, Missouri
b) Authorized Representative of Employees
c) U. S. Department of Labor - OSHA, Region VII and OSHA, Headquarters
d) NIOSH Region VII

For the purposes of informing the approximately 74 affected employees, the employer will promptly post the Determination Report for a period of 30 calendar days in a prominent place near where the employees work.

## III. INTRODUCTION

Section 20(a)(6) of the Occupational Safety and Health Act of 1970, 29 U.S.C. 669(a)(6), authorizes the Secretary of Health, Education, and Welfare, following a written request by any employer or authorized representative of employees, to determine whether any substance normally found in the place of employment has potentially toxic effects in such concentrations as used or found.

NIOSH received such a request from an authorized representative of employees of the U.R.W. International Union to determine the degree of illness in workers currently employed at the Eagle-Picher Plant (Chemical and Metals Division) in Joplin, Missouri; specifically requested were that biological and clinical evaluations of these workers exposed to lead compounds be carried out.

## IV. HEALTH HAZARD EVALUATION

### Summary

Medical evaluations of 53 production workers at Eagle-Picher Industries' lead chemical plant in Joplin, Missouri, revealed blood lead levels ranging from 39 to 135 µg/100ml, with 44 (83%) ≥60 µg/ml and 19 (36%) ≥80 µg/100ml. Comparable levels of erythrocyte protoporhyrin were noted. Blood urea nitrogen (BUN) levels were elevated in 17 workers. Anemia (hemoglobin <14gm/100ml) was noted in 6 workers. Symptoms compatible with lead intoxication were reported in 20 workers. Wrist weakness was noted on physical examination in 1 worker, ankle drop in 1 worker, and tremor in 2 workers. Discrepant results on blood lead testing were noted between analyses performed by NIOSH, CDC, and the private medical laboratory normally used by the company to test the workers.

### A. Plant Process - Conditions of Use

Eagle-Picher Industries, a large, diversified corporation which manufactures industrial chemicals, machinery, and transportation products, operates a lead chemicals plant in Joplin, Missouri, which produces lead oxides (red lead and letharge), lead peroxide, lead sulfate, lead silicate, and "blue lead". The 74 production workers, currently employed in the lead chemicals area, are either production workers (29 men), plant cleaners (2) bagroom cleaner (1), equipment mechanics (9), dock workers (3), general maintenance mechanics (12), maintenance supervisors (2), production foremen (5), lab technicians (3), janitors (3), truck driver (1), or general supervisors (4). The workers ranged in age from 24-61 years (median 50 years), were white, male, and had worked at the plant for 4.5-34 years (median, 20 years).

Medical surveillance of workers conducted by the plant physician consisted of a monthly physical inspection for the presence of anemia, or symptoms of lead intoxication and semi-monthly blood lead testing. Blood lead samples were analyzed by Upsher Laboratories, Kansas City, Missouri. Workers with acute symptoms of lead poisoning were treated by the plant physician with intravenous EDTA. Confirmatory blood lead measurements had been obtained on some workers treated in this fashion. Oral EDTA therapy had been used, in courses lasting 7-10 days, to treat workers with mild symptoms of lead poisoning; the last recorded treatment was in January 1976. Anemic workers received a hematinic medication and, if hematinic therapy was not successful, a blood lead determination was performed. Some workers were given EDTA in the absence of symptoms, if their blood lead levels were elevated. Workers with significant symptoms of lead intoxication or significantly elevated blood lead levels were removed from exposure; workers with minimal symptoms continued to work.

The company supplied work clothing which was not worn home and was changed daily. Respirators were provided for each worker and were washed daily at the plant. Eating and smoking in production areas was prohibited.

B. Evaluation Design and Methods

March Survey

Following a preliminary plant visit on March 18, 1976, medical evaluations of workers were conducted on March 23, 1976. Of 66 workers listed by the company as non-salaried chemical employees, 43 (65%) participated in the evaluation which consisted of a medical interview, a brief physical examination and determination of blood lead, erythrocyte protoporphyrin, zinc protoporphyrin, hemoglobin, hematocrit, routine blood chemistry (including BUN, creatinine, uric acid, SGOT, and LDH), and urine protein concentrations. Clinic records were reviewed to determine frequency of treatment with oral EDTA. The 23 nonparticipants had worked at the plant longer and were significantly older than participants. Blood lead analyses were performed on all samples by the Western Area Occupational Health Laboratory,*Salt Lake City, Utah. Split samples from 9 workers were analyzed for lead concentration using an atomic absorption technique by Upsher Laboratories, Kansas City, Missouri. Protoporphyrin analyses were performed by Environmental Sciences Associates, Burlington, Massachusetts. All other tests were performed by Eastern Hills Laboratories, Cincinnati, Ohio.

C. Evaluation Criteria

Exposure to lead may cause weakness, weight loss, tiredness and anemia. It may also cause gastrointestinal problems with constipation and abdominal cramping. A black line of the gums may be produced. Tremors may occur. Weakness or paralysis of the muscles of the wrists or ankles may be present. Encephalopathy may develop with headache, vomiting and convulsions. This occurs mainly in children--rarely in adults. Kidney damage may also occur. Lead can pass from an exposed mother to her unborn child and cause lead poisoning and, in some cases, death.

*NIOSH Contractor, Utah Biomedical Test Laboratory

D. March Survey Results

Blood lead levels were obtained on 42 workers and ranged from 39 to 135 µg/100ml, with 41 (98%) ≥40 µg/100ml, 34 (81%) ≥60µg/100ml, and 13 (31%) ≥80µg/100ml. Erythrocyte protophyrin (EP) levels ranged from 74 to 716 µg/100ml blood. Normal ranges for protoporphyrin levels are less well established than for lead levels; however, an EP level of 50 µg/100ml is felt to correspond roughly with a lead level of 40 ug/100ml and a level of 100 µg/100ml corresponds roughly with a lead level of 60 µg/100ml; 42 (98%) of 43 workers had EP levels over 100 µg/100ml.

Symptoms consistent with lead intoxication were noted during the past year in a number of workers. Twenty (47%) of the workers interviewed had neuromuscular symptoms (tremor, weakness, or muscle cramps), 18 (42%) reported gastrointestinal symptoms (abdominal pain, nausea, vomiting, diarrhea, or constipation), 18 (42%) noted joint pain, and 6 (14%) noted decreased appetite or weight loss. Significantly fewer reported having those symptoms currently; 12 (28%) noted neuromuscular symptoms; 4 (9%) noted gastrointestinal symptoms; 10 (23%) noted joint pains; and 4 (9%) noted decreased appetite or weight loss. Symptoms showed little correlation with blood lead or EP levels. Joint symptoms were unrelated to serum uric acid level.

Elevated blood urea nitrogen (BUN) levels (>20mg/100ml) were noted in 11 (26%). Blood creatinine levels above 1.5 mg/100ml, the upper limit of normal were not encountered. Creatinine and BUN were correlated ($r = .42$); the difference in the prevalence of abnormal readings was apparently related to differences in the normal ranges for the two determinations. History of treatment with EDTA appeared to be associated with slightly elevated BUN levels (Table 1). A confounding age effect contributed to the association.

Treatment with oral EDTA was recorded for 18 currently employed workers during the period June 1, 1966 to January 19, 1976. Courses of EDTA consisted of 4 gm of EDTA daily for 5-7 days; individual workers received from 1 to 13 such courses during the period mentioned.

Anemia (defined as a hemoglobin level less than 14 gm/100ml) was present in 3 workers. Physical examination revealed wrist weakness in 1 worker, asymmetrical ankle drop in 1 worker, and tremor in 2 workers. Physical findings were counted as positive only if confirmed by two physicians.

Inter-Laboratory Comparison

Marked discrepancies were noted on blood lead determinations performed on split samples by Upsher Laboratories and the Western Area Occupational Health Laboratory (WAOHL). Values from Upsher were consistently lower than the values obtained by WAOHL (Table 2).

Review of reports of recent performance of the WAOHL laboratory on the CDC blood lead proficiency testing program (Table 3) revealed that the WAOHL Laboratory was correct in 3 of 3 instances. A correct lead determination is one which is ±15% of the target value, when the target value is ≥40 µg/100ml or ±6µg/100ml of the target value when it is <40 µg/100ml. The protoporphyrin results noted previously further support the reliability of the WAOHL, as do results from internal quality control samples which were analyzed by WAOHL. Seven of 9 pairs of internal split samples analyzed "blind", had blood lead determinations within 10% of each other.

E. Evaluation Design Methods May Survey

In view of the discrepancies noted between the two laboratories and the apparent severity of lead exposure at this plant, blood lead, erythrocyte protoporphyrin, and BUN levels were repeated on certain employees on May 17 and 18, 1976. On these days, the routine semi-monthly blood samples were being drawn and additional blood tubes were collected and analyzed for lead concentration by the CDC Toxicology Laboratory, a reference laboratory for lead analyses for the country, as well as by the 2 labs which analyzed the March samples. BUN levels were repeated on all workers with elevated BUN levels in the March testing or with a history of EDTA therapy.

F. May Survey Results

Close correlation was noted between the blood samples analyzed for lead by CDC and by the Western Area Occupational Health Laboratory; significantly lower values were again reported by Upsher Laboratories (Table 4). The discrepancies noted in March between WAOHL and Upsher appear to be unrelated to the type of anticoagulant used in the sample, since value on split samples using different anticoagulants showed very close correlation.

Elevated BUN levels (>22mg/100ml*) were detected in 3 workers not tested in March. Three workers with normal levels in March had elevated levels in May. Two thirds of workers with elevated BUN levels in March had persistent elevations in May. Seventeen workers had an elevated BUN level on one of the two test periods (Table 5).

Ten workers tested in May who were not tested in March had blood lead levels from 72-96 µg/100ml. Adding this group to the results of the March testing revealed that 52 (98%) had levels ≥40 µg/100ml, 44 (83%) ≥60 µg/100ml, and 19 (36%) ≥80 µg/100ml. Sixteen adult white males without occupational lead exposure were interviewed and tested in May. These men were employed either in

other areas of the Eagle Picher facility (9 men) or in various occupations throughout the Joplin area (7 men). The group age (mean = 44.6 years) was comparable to that of the worker group tested. Blood lead, EP and BUN levels for this group (Table 6) were within the normal range with one exception (control #7: BUN = 25). Current symptom rates were generally lower for the control group than for the lead workers (Table 7).

## V. CONCLUSIONS

(1) Excessive lead absorption was noted in a significant number of workers examined at this plant.

(2) Findings consistent with those noted in lead intoxication were noted in the exposed workers examined. These included symptoms of lead toxicity, anemia, peripheral neuropathy and renal disease.

(3) Apparent deficiencies were identified in the proficiency of Upsher Laboratories in performing blood lead analyses.

(4) The above findings were not identified by the company's medical surveillance program prior to this investigation.

## VI. RECOMMENDATIONS

(1) Every worker with blood lead level >60 µg/100ml should be removed from lead exposure until his level drops below 60 µg/100ml. Chelation should not be used to lower blood lead levels, but should be reserved only for those workers with significant symptoms or signs of lead poisoning. Chelation with oral EDTA should not be used for this purpose.

(2) The results of additional studies are need to determine if signficiant kidney disease exists in these workers and if it is related to occupational lead exposure or to EDTA therapy. These studies are currently in process.

(3) A request has been sent to CDC Laboratory Proficiency Testing Program that the Upsher Laboratory be reevaluated for certification in this area. In the interim, another laboratory should be identified which could perform blood lead analyses with greater accuracy.

(4) An attempt should be made to evaluate those workers who did not participate in the testing; these workers were older and would be expected to have comparable or possibly even greater cumulative lead exposures.

(5) In plant air exposures should be reduced to below the permissible exposure limit and all possible efforts made to prevent ingestion of lead.

(6) Until the air levels are reduced to the required level the respiratory protection program should be required to identify possible problem areas (workers are still receiving excessive exposures either orally or by inhalation

as evidenced by the elevated blood lead values). This respiratory protection program should be rigidly enforced.

(7) Improved medical record keeping in the plant infirmary is recommended to insure proper follow-up of workers: each worker should have a separate medical file with records of his lead testing, treatment history, examinations, and other pertinent medical information. The worker should have access to his file.

(8) Workers identified as having significant impairment of kidney or nervous system function, regardless of whether lead exposure is felt to have caused such impairment, should be removed from further exposure to lead since such exposure might aggravate these conditions.

## VII. AUTHORSHIP

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Table 1. Blood Urea Nitrogen (BUN) Levels and Serum Creatinine by Treatment History, March 1976

| 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/100ml

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

Table 2. Results of Split Samples: Upsher Laboratory and Western Area Occupational Health Laboratory Analyses of Lead in Blood, March 1976

| Sample Number | Upsher Result (µg/100ml) | WAOHL Result (µg/100ml) |
|---|---|---|
| 1 | 63.3 | 84 |
| 2 | 62.5 | 79 |
| 3 | 60.8 | 64 |
| 4 | 64.1 | 73 |
| 5 | 63.3 | 75 |
| 6 | 81.0 | 121 |
| 7 | 56.5 | 75 |
| 8 | 68.4 | 75 |
| 9 | 55.0 | 39 |
| Mean | 63.9 | 76.1 |

Table 3. Lead Proficiency Testing Results: April 1976, Western Area Occupational Health Laboratory

APRIL RESULTS

| Sample # | Target Value | Acceptable Range | WAOHL Result |
|---|---|---|---|
| 1 | 96.0 | 81.6 - 110.4 | 107 |
| 2 | 79.4 | 67.5 - 91.3 | 84.1 |
| 3 | 65.9 | 56.0 - 75.8 | 65.6 |

* All values are µg/100ml

Table 4. Lead Concentration in Split Blood Samples, May 1976

| Lab | a CDC | b WAOHL | c UPSHER |
|---|---|---|---|
| Anticoagulant | EDTA | Heparin | Heparin |
| 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: WAOHL + 4.0%
UPSHER -29.2%

Number (Percent) with acceptable result: WAOHL 27/30 (90%)
UPSHER 1/30 (3.3%)

Table 5. Workers with Elevated BUN Levels

| Worker Numbers | BUN | | Creatinine | Years Employed | No. of Courses of EDTA |
|---|---|---|---|---|---|
| | March | May | | | |
| 1 | -- | 44 | -- | -- | 8 |
| 2 | -- | 30 | -- | -- | 0 |
| 3 | 28 | 30 | 1.1 | 23 | 1 |
| 4 | 30 | 28 | 1.2 | 23 | 4 |
| 5 | 26 | 24 | 1.3 | 7 | 2 |
| 6 | 21 | 27 | 0.9 | 16 | 9 |
| 7 | 23 | 27 | 1.0 | 13 | 0 |
| 8 | 21 | 23 | 1.2 | 25 | 0 |
| 9 | -- | 26 | -- | -- | 0 |
| 10 | 25 | -- | 1.3 | 20 | 0 |
| 11 | 23 | -- | 1.0 | 20 | 0 |
| 12 | 21 | 18 | 1.0 | 21 | 0 |
| 13 | 23 | 18 | 1.0 | 7 | 0 |
| 14 | 23 | 17 | 1.2 | 29 | 1 |
| 15 | 17 | 28 | 0.9 | 4.5 | 1 |
| 16 | 20 | 23 | 1.4 | 31 | 13 |
| 17 | 18 | 24 | 1.2 | 20 | 4 |

Table 6. Control Workers Lab Results: 5/18/76

| | Age | Blood Lead Level | E.P. | BUN* |
|---|---|---|---|---|
| 1. | – | 13 | 20 | 14 |
| 2. | 55 | 20 | 18 | 20 |
| 3. | 46 | 17 | 28 | 15 |
| 4. | 52 | 14 | 30 | 17 |
| 5. | 48 | 22 | 14 | 17 |
| 6. | 30 | 15 | 19 | 12 |
| 7. | 61 | 18 | 19 | 25 |
| 8. | 42 | 30 | 32 | 16 |
| 9. | 36 | 14 | 15 | 18 |
| 10. | 42 | 16 | 23 | 21 |
| 11. | 59 | 21 | 20 | 17 |
| 12. | 44 | 18 | 14 | 20 |
| 13. | 41 | 20 | 19 | 11 |
| 14. | 29 | 12 | 23 | 15 |
| 15. | – | 17 | 21 | 10 |
| 16. | 40 | 13 | 34 | 20 |
| Mean | 44.6 | 17.5 | 21.8 | 16.8 |
| S.D. | 9.7 | 4.5 | 6.2 | 4.0 |

* normal 8-22 mg/100ml

Table 7. Current Symptoms of Lead Toxicity

| | 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 | |
| Diarrhea | 2 | (4%) | 0 | |
| Constipation | 0 | | 0 | |