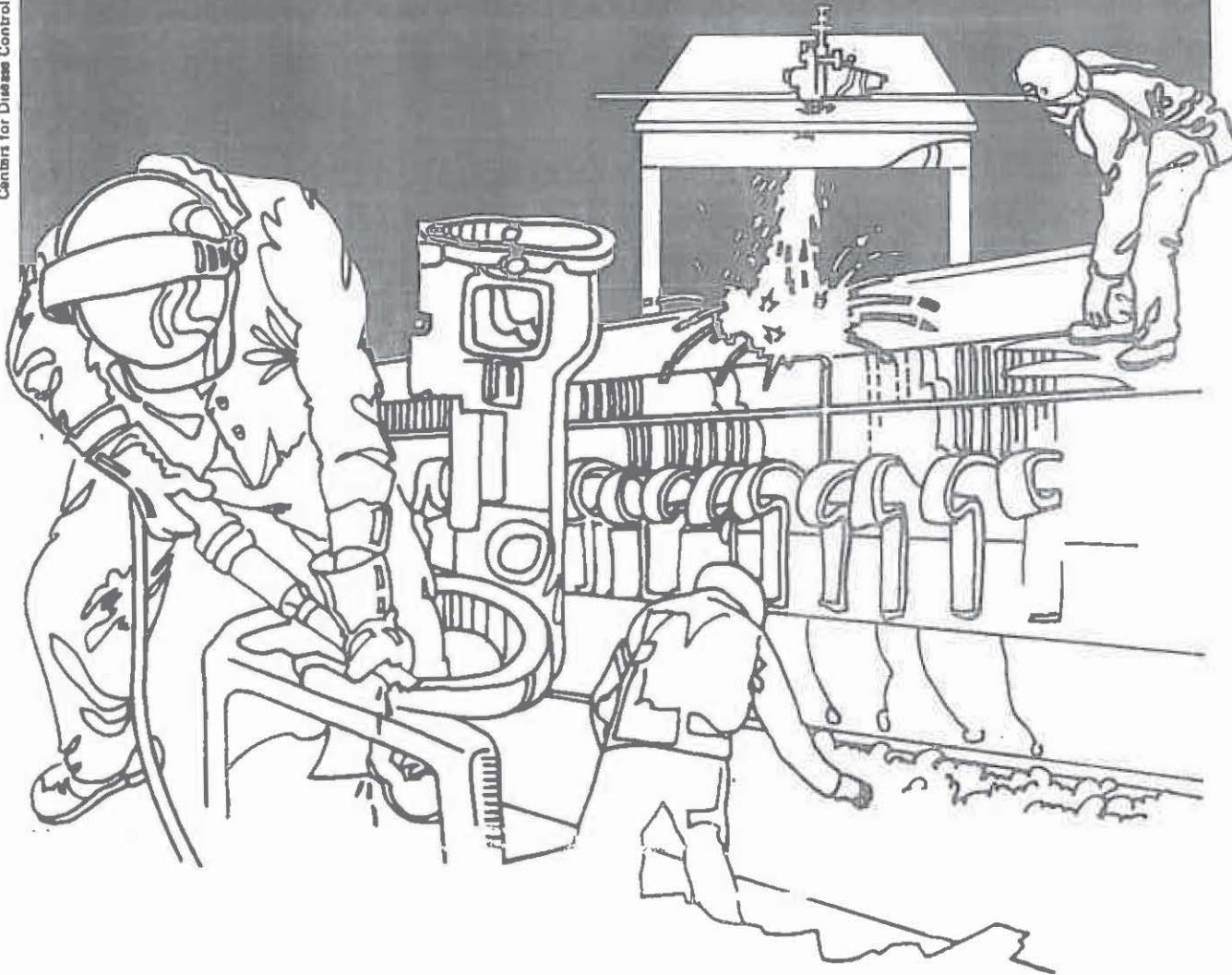


# NIOSH



# Health Hazard Evaluation Report

HETA 80-116-1034  
FERRO CORPORATION  
CLEVELAND, OHIO

## PREFACE

The Hazard Evaluations and Technical Assistance Branch of NIOSH conducts field investigations of possible health hazards in the workplace. These investigations are conducted under the authority of Section 20(a)(6) of the Occupational Safety and Health Act of 1970, 29 U.S.C. 669(a)(6) which authorizes the Secretary of Health and Human Services, following a written request from 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.

The Hazard Evaluations and Technical Assistance Branch also provides, upon request, medical, nursing, and industrial hygiene technical and consultative assistance (TA) to Federal, state, and local agencies; labor; industry and other groups or individuals to control occupational health hazards and to prevent related trauma and disease.

Mention of company names or products does not constitute endorsement by the National Institute for Occupational Safety and Health.

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## I. SUMMARY

On April 24, 1980, the National Institute for Occupational Safety and Health (NIOSH) was requested by Local 1170, United Steelworkers of America to conduct a health hazard evaluation at Ferro Corporation, Cleveland, Ohio. At this plant Ferro Corporation manufactures porcelain enamels (Coatings Division) and metal-based pigments (Color Division). NIOSH was asked to evaluate possible hazards of occupational exposure to lead and cadmium.

On May 23 and 24, 1980, NIOSH conducted a walk-through survey and a preliminary medical evaluation. Inadequate ventilation and poor work practices were noted in several areas. Three (20%) of 15 heavily exposed workers had blood lead levels above 40 ug per deciliter (dl), indicating excessive lead absorption, and 3 (20%) had blood cadmium concentrations above 0.7 ug per dl, indicating increased absorption of cadmium.

On March 16-20, 1981, NIOSH conducted a comprehensive environmental and medical evaluation. In the Coatings Division, personal (breathing zone) exposures to airborne lead ranged from zero to 359 ug/M<sup>3</sup> with average exposures (in micrograms lead per cubic meter of air) by job being: smelter-operators, 73; millroom workers, 69; take-off men, 79; mixing, 61; maintenance, 48; and shipping and receiving, 13. Thirty-one (41%) of 75 air samples exceeded the Occupational Safety and Health Administration (OSHA) standard for airborne lead exposure of 50 ug/M<sup>3</sup>. In the Color Division, cadmium exposures ranged from 0 to 384 ug/M<sup>3</sup>. Highest cadmium exposures were in the cadmium department (mean, 229 ug/M<sup>3</sup>); 11 (31%) of 35 air samples exceeded the NIOSH recommended standard for occupational cadmium exposure of 40 ug/M<sup>3</sup>.

Blood lead levels in 23 (21.1%) of 109 workers tested were 40 ug/dl or above; 22 were employed in the Coatings Division. Blood lead levels increased with duration of employment, and were correlated ( $r = 0.54$ ;  $p < 0.01$ ) with air lead exposures. Blood cadmium levels were 0.7 ug/dl or above in 21 (19.3%) workers; 12 were employed in the Color Division. Blood cadmium levels were correlated with airborne cadmium exposures ( $r = 0.43$ ;  $p < 0.01$ ).

Kidney function evaluation found elevated blood urea nitrogen (BUN) concentrations (above 20 mg/dl) in 18 (16.5%) of 109 workers, elevated serum creatinine concentrations (above 1.5 mg/dl) in 8 (7.3%) workers, and elevated serum uric acid levels (above 8.5 mg/dl) in 12 (11.0%) workers. For each of those tests, elevated values would be expected in no more than 3 of the workers tested. Thirty-five workers had one or more abnormal kidney function test results. Uric acid concentrations were correlated positively with blood lead levels ( $r = 0.26$ ;  $p < 0.01$ ). Four workers reported a history of gout.

Chest radiographs of 62 workers with history of exposure to silica dust showed changes compatible with mild pneumoconiosis in three.

On the basis of these results, NIOSH has determined that health hazards existed at Ferro Corporation as the result of excessive occupational exposures to airborne lead, cadmium, and silica dust. Increased lead and cadmium absorption were detected. Occupationally related kidney disease was found, apparently caused by exposure to lead and/or cadmium. X-ray evidence of silicosis was present. Recommendations to reduce environmental exposures at Ferro are presented on Section VII of the report. A special recommendation is made for further evaluation of kidney function in Ferro workers. Medical follow-up is also required for the workers with evidence of pneumoconiosis.

KEYWORDS: SIC 2810 (Industrial Inorganic and Organic Chemicals) lead, cadmium, lead nephropathy, cadmium nephropathy, lead gout, silicosis.

## II. INTRODUCTION

On April 24, 1980 the National Institute for Occupational Safety and Health (NIOSH) received a request from Local 1170, United Steelworkers of America (USWA) to conduct a health hazard evaluation at Ferro Corporation, Cleveland, Ohio. The request stated that occupational health hazards were suspected to exist in the Color Division, Coatings Division, and VEDOC (powder coatings) Division of this pigment and enamel manufacturing complex. Toxic substances of particular concern were lead and cadmium.

An opening conference was held at the plant on May 23, 1980 with representatives of Ferro Corporation, USWA International Headquarters, USWA Local 1170, and NIOSH. At the meeting the general protocol for the study was discussed, and NIOSH requested from Ferro demographic and manufacturing data, environmental and medical monitoring data, and samples of selected raw materials. A walk-through survey was performed, as was a preliminary medical evaluation of blood lead and cadmium levels in 15 workers at high-risk of exposure. The walk-through evaluation disclosed deficiencies in ventilation and also found poor work practices in several areas. Blood lead levels in 3 (20%) of the 15 workers evaluated were above 40 ug/deciliter, indicating increased lead absorption, and blood cadmium levels were above 0.7 ug/deciliter in 3(20%) workers, indicating increased absorption of cadmium. Preliminary review of plant medical records indicated that several workers had chronically increased blood lead or urinary cadmium concentrations.

On the basis of those findings, NIOSH considered that further environmental and medical evaluation of the plant was warranted. However, a strike by Local 1170 delayed the survey until October, 1980. Further delay resulted when Ferro Corporation refused to allow NIOSH right of entry to the plant to perform the environmental and medical surveys and also refused NIOSH access to their medical records. NIOSH obtained a Federal warrant which was, however, not honored by Ferro Corporation. Federal contempt of court proceedings were begun. At that point Ferro Corporation yielded and allowed NIOSH entry to the plant and to their records on March 16, 1981.

The survey was conducted during the week of March 16-20, 1981 following a second opening conference on March 16, 1981. The preliminary results of the surveys were communicated to workers, to Local 1170, to the International Headquarters USWA, and to Ferro Corporation in two interim reports, dated June 1980 and May 1981. Also the 15 workers who participated in the preliminary survey were notified of their test results by personal letter in July 1980.

## III. BACKGROUND

In Cleveland, Ohio, Ferro Corporation produces vitreous (porcelain) enamels and metal-based pigments. The facility evaluated is comprised of three divisions: Coatings Divisions, which produces enamels; Color Division, which produces metal-based pigments; and VEDOC Division, which manufactures dry powder coatings of a non-metallic, generally resinous nature. Coatings and Color were the two divisions examined under the health hazard evaluation.

Coatings Division

Coatings Division produces a series of vitreous enamels, of which the major constituents are silica and clay, and the minor ingredients are inorganic lead, other metals, and pigments. The silica and clay are delivered in rail car quantities, while other materials are delivered by truck. All materials are hopped to the topmost floor of the plant, redistributed into bins, and automatically weighed into a 'charge car'. Minor constituents are added manually. Each 'batch' drops to the lower level and is then mixed in a rotating mill until it is ready to be charged into a furnace; a typical charge weighs about 3000 lbs. After mixing, the 'charge' is taken upstairs again by elevator. It is removed from the elevator by the 'take-off man' and positioned at the entry end of the refractory furnaces by workers using powered hand trucks. The charge is then fed into the furnace by screws which control the rate of entry. In the furnace, the charge melts to form an amorphous glass-like material. Once fused, the molten, glass-like mixture is passed through water cooled rollers which solidify it into a glassy 'frit'. This wafer-thin, glassy frit is broken up mechanically into chips, conveyed to a hopper, and then bagged and shipped, or ground to a powder, remilled for special formulations, and rebagged. Lead oxides often comprise more than 30% of the frit by weight.

Job categories in the Coatings Division in which workers experience significant exposures to dusts include: charge take-off men, who are responsible for removing the charge cars from the elevator; smelter-operators, who tend the smelters and bag the crushed frit; and mill operators, who crush and rebag frits for special applications. Major potential exposures include silica dust for workers unloading and hoppersing bulk silica and lead particulates for workers in the mill room and furnace areas.

Color Division

The Color Division produces metal-based pigments which are used in photographic and other applications. Primary constituents of many pigments produced in the Division are cadmium and selenium. Raw material is initially handled in a manner similar to that used in the Coatings Division. After hoppersing, weighing, and milling, a batch is formed which is fired for a specified length of time in a rotary or other kiln. This firing process fuses the pigments and changes the crystalline structure of the component materials to produce brilliant colors.

Job categories which are considered to be at risk of excessive dust exposures in the Color Division include: mill operators, cadmium renderers, filter-press operators, and batcher/blenders. The major potential airborne exposure was considered to be to cadmium.

#### IV. EVALUATION DESIGN AND METHODS

##### A. Environmental

A total of seven shifts were sampled during the environmental portion of the survey: day shifts on March 17, 18, and 19, 1981; evening shifts on March 16, 17, and 18; and midnight shift on March 18. Seventy-five personal (breathing zone) and 27 area air samples were collected in the Coatings Division, while 35 personal and 11 area samples were taken in the Color Division.

Sampling was performed according to NIOSH Sampling Data Sheets #S 312, Cadmium, and #S 341, Lead. MSA Model G pumps were used at a flow rate of 1.7 liters per minute to collect samples on 37 mm/0.8  $\mu$  MCE membrane filters supported by cellulose back-up pads. Calibration checks were performed approximately every two hours during the sampling period with a Kurz Portable Mass Air Flow Meter.

Work practices and conditions were observed during the survey, and the general ventilation needs of the facility were assessed.

##### B. Metals and Dusts Analyses

Analyses of environmental samples for metals, and for their content of respirable and total dusts were performed by the Utah Biomedical Testing Laboratory. NIOSH Method P&CAM 173 was used to analyze filters for metals. Filters were wet ashed with nitric and perchloric acids to insure their complete oxidation. The ashed samples were then diluted to 25 ml and aspirated into an atomic absorption spectrophotometer.

Respirable particulate fractions in air samples were isolated using a high-volume pump which drew workplace air through a stainless steel cyclone onto a preweighed filter at a rate of 9 liters per minute (lpm). All dust particles of 10  $\mu$ m or less in diameter were thus isolated on the filter. At the same time, a total dust sample was taken in order that the percentage of respirable dust in each total dust sample could be determined. Particulate weights were determined by weighing the samples plus the filters on an electrobalance and then subtracting the weights of the preweighed filters. Tare and gross weighings were done in duplicate.

##### C. Medical

Voluntary participation was solicited from all current and from selected retired Ferro employees in the Coatings and Color Divisions. After a signed consent form had been obtained from each participant, a questionnaire was administered; blood and urine specimens were collected; and a chest x-ray was taken on workers reporting a work history of silica exposure. Urine samples were analyzed for creatinine, beta-2-microglobulin, phosphorus, and cadmium concentrations. Blood and serum samples were analyzed for 1,25-dihydroxy vitamin D (calcitriol), parathyroid hormone, ionized calcium, lead, free erythrocyte protoporphyrin (FEP), and cadmium concentrations as well as for hematocrit and for a general battery of blood assays (SMAC), including blood

urea nitrogen (BUN), serum creatinine, uric acid, calcium, and phosphorus. Lead and cadmium assays were performed by ESA Laboratories, Bedford, Mass., by anodic stripping voltammetry. All other determinations were performed by Consolidated Biomedical Laboratories (CBL), Columbus, Ohio. Reference ranges at CBL for relevant test results at CBL are as follows:

blood urea nitrogen (BUN)	10 - 20 mg/dl
serum creatinine	0.7 - 1.5 mg/dl
uric acid	4.0 - 8.5 mg/dl
serum calcium	9.1 - 10.9 mg/dl
serum phosphorus	2.5 - 4.5 mg/dl
Urine beta-2-microglobulin (by radioimmunoassay)	4 - 370 ng/ml
serum calcitriol (by rachitic chick assay of de Luca et al.)	26 - 44 pg/ml

Those reference ranges were established at CBL on the basis of analyses of samples taken from 100 apparently healthy laboratory workers. The reference range for each test result is defined as the mean result  $\pm$  2 standard deviations. Given that definition, no more than 2.5% of test results in any study population will be expected to exceed the upper reference limit.

## V. EVALUATION CRITERIA

### A. Cadmium

Cadmium is a toxic heavy metal used in the manufacture of batteries, pigments, and jewelry, and as a neutron absorber in nuclear reactors. Cadmium may enter the human body either by ingestion (swallowing) or by inhalation of cadmium metal or oxide.

#### Absorption and Measurement

Approximately 6 to 10 percent of ingested cadmium and 15 to 30 percent of inhaled cadmium is absorbed into the body<sup>1</sup>. Cadmium is transported from the site of absorption by the red blood cells and plasma. It is deposited in organs throughout the body, but major depositions occur in the liver and kidneys. Under normal conditions the kidneys accumulate the greatest concentrations of cadmium. Cadmium is excreted from the body only very slowly, and thus accumulates in the cortex of the kidneys over a lifetime.

The blood cadmium concentration is the best biological indicator of recent cadmium exposure and absorption.<sup>2,3</sup> The normal blood cadmium level is below 0.7 micrograms (ug) per deciliter of whole blood. Chronic cadmium exposure can be assessed by measuring the cadmium content of the kidneys through the technique of neutron activation analysis.<sup>4</sup> The urine cadmium concentration, although used widely as an index of exposure, is primarily an indicator of cadmium-induced kidney damage; the urine cadmium concentration does not ordinarily begin to increase until after injury has occurred to the kidneys.<sup>5</sup>

### Acute Toxicity

Acute inhalation exposure to cadmium can cause pneumonia or pulmonary edema,<sup>6</sup> as well as liver and kidney damage.<sup>7</sup> Ingestion of toxic quantities can produce nausea, vomiting, and diarrhea. Exposure to an airborne concentration of cadmium of 40 mg/M<sup>3</sup> is considered immediately dangerous to life.

### Chronic Toxicity

Occupational exposure to cadmium is usually chronic. Chronic occupational exposure to cadmium can produce several toxic effects, of which the most important are emphysema of the lungs and chronic kidney disease.<sup>1</sup> Also occupational cadmium exposure has been associated with cancer of the prostate gland, and there is limited evidence that occupational cadmium exposure may be associated with lung cancer.<sup>8,9</sup>

Apart from malignancy, kidney disease is the toxic effect of chronic cadmium exposure which is of principal concern.<sup>1</sup> Although much remains to be learned about the development of kidney disease in persons exposed to cadmium, the process appears to be a gradual one.<sup>5</sup> Also the process is dose-related; persons with greatest lifetime absorption of cadmium are at greatest risk of kidney disease (nephropathy). The initial signs of cadmium nephropathy are subtle - affected workers will usually have no symptoms in the early stages, and their kidney function test results may still be within the broad range of normal, although their test results will tend over time to move toward the high end of the normal range.

Because the kidney has an enormous reserve capacity, results of the usual renal function tests - the blood urea nitrogen (BUN), the serum creatinine, and the serum uric acid - will not become frankly abnormal until one-third to one-half of kidney function has been destroyed.<sup>10</sup> For that reason, more sensitive screening tests of renal function have been sought. These include measurement of serum concentrations of 1,25-dihydroxy vitamin D (which may be decreased),<sup>11</sup> and measurement of urine concentrations of cadmium and of protein beta-2-microglobulin (both of which are reported to increase in persons with kidney damage caused by cadmium).<sup>12</sup> Also aminoaciduria, renal glycosuria, or hyperphosphaturia may develop.

When any of these test results are abnormal in a person exposed to cadmium, or even when two or more test results are in the high normal range, there exists a possibility of kidney damage. In that circumstance, more complete evaluation of the individual worker by a kidney disease specialist is required.

NIOSH recommends that worker exposures to cadmium dust or fume be limited to not more than 200 ug/M<sup>3</sup> during a 15-minute ceiling period or to a threshold limit value (TLV) of not more than 40 ug/M<sup>3</sup>, as a time-weighted average (TWA) over a ten-hour shift. The Occupational Safety and Health Administration (OSHA) standard for cadmium dust exposure is 200 ug/M<sup>3</sup>, and for cadmium fume exposure 100 ug/M<sup>3</sup>, using an eight-hour TWA for each.

B. Lead

Inhalation of lead dust and fumes is the major route of lead exposure in industry. A secondary source of exposure may be from ingestion of lead dust deposited on food, cigarettes, or other objects. Once absorbed, lead is excreted from the body very slowly. Absorbed lead can damage the kidneys, peripheral and central nervous systems, and the blood forming organs (bone marrow). These effects may be felt as weakness, tiredness, irritability, digestive disturbances, high blood pressure, kidney damage, mental deficiency, or slowed reaction times. Chronic lead exposure is associated with infertility and with fetal damage in pregnant women.

Blood lead levels below 40 ug/deciliter whole blood are considered to be normal levels which may result from daily environmental exposure.<sup>13</sup> However, fetal damage in pregnant women may occur at blood lead levels as low as 30 ug/deciliter. Lead levels between 40-60 ug/deciliter in lead exposed workers indicate excessive absorption of lead and may result in some adverse health effects. Levels of 60 to 100 ug/deciliter represent unacceptable elevations which may cause serious adverse health effects. Levels over 100 ug/deciliter are considered dangerous and often require hospitalization and medical treatment.

The new OSHA standard for lead in air is 50 ug/M<sup>3</sup> calculated as an eight-hour time-weighted average for daily exposure.<sup>13</sup> The standard also dictates that workers with blood lead levels greater than 60 ug/deciliter must be immediately removed from further lead exposure and in some circumstances that workers with lead levels of less than 60 ug/deciliter must also be removed. Removed workers have protection for wage, benefits, and seniority for up to eighteen months until their blood levels decline to below 50 ug/deciliter and they can return to lead exposure areas.

Chronic Toxicity

Lead has been shown in previous studies<sup>14</sup> to cause chronic kidney disease (nephropathy) in persons with long-time occupational exposure. The process is gradual and dose-related. Persons who experience the greatest lifetime risk of manifesting lead-induced kidney disease are those who have experienced the most lead absorption over their working career. The initial signs of lead nephropathy are subtle. Affected workers will usually have no symptoms in the early stages. Their renal function test values may still be within the broad range of normal, although their test results will tend over time to move toward the high end of the normal range.

Because the kidney has an enormous reserve capacity, results of the usual renal function tests - blood urea nitrogen (BUN), serum creatinine, and serum uric acid, will not become frankly abnormal until one-third to one-half of kidney function has been destroyed.<sup>10</sup> For that reason, more sensitive screening tests of renal function have been sought. These include serum measurement of 1,25-dihydroxy vitamin D, which may be decreased in persons with kidney damage caused by lead.<sup>11</sup> Other abnormalities which may also be noted in chronic lead nephropathy include aminoaciduria, renal glycosuria, and

hypercalcuria. Gout is a particularly noteworthy manifestation of lead nephropathy;<sup>15</sup> the elevated serum uric acid concentrations which may occur in lead nephropathy have been associated with the development of gouty arthritis.

When one or more of the standard kidney function tests is in the abnormal range in a worker exposed to lead, or even when two or more of the test results are in the high normal range, there exists the possibility of kidney damage. In that circumstance, more complete evaluation of the individual worker by a kidney disease specialist is required.

C. Dusts

Although NIOSH has not recommended a permissible exposure limit for nuisance dusts, according to the best available data it is prudent to follow the recommendations of the American Conference of Governmental Industrial Hygienist (ACGIH) that workers be exposed to a TWA of not more than 10 mg/M<sup>3</sup> of airborne total nuisance particulates during an 8-hour day. The OSHA standard for nuisance dust exposure is 15 mg/M<sup>3</sup> (TWA).

D. Silica Dust

Crystalline free silica may cause a form of pulmonary fibrosis called silicosis when particles of silica between 0.5 and 5 microns in diameter are deposited in the lungs. Symptoms usually develop slowly with cough, shortness of breath, chest pain, weakness and wheezing developing over a period 10-20 years after initial exposure to moderate levels of crystalline free silica. Higher exposure levels may accelerate the development of the disease. Persons with silicosis may have a greater risk of developing a particularly hazardous form of tuberculosis called silico-tuberculosis. Silicosis is diagnosed by x-ray examination of the chest.

VI. RESULTS AND DISCUSSION

A. Environmental

Cadmium and lead are the two principal occupational exposures at Ferro Corporation. Cadmium exposures exist in both the Color and Coatings Divisions, but are predominant in the Color Division. Lead exposures are confined to the Coatings Division (Tables I and II).

Airborne cadmium exposures in the Color Division were highest in the Cadmium Department, where five samples showed an average concentration of 229 ug/M<sup>3</sup>, and a range of 74-384 ug/M<sup>3</sup>. Average airborne cadmium exposures in categories in Color Division were as follows: Batching/Blending, 42 ug/M<sup>3</sup> (range, 20-75 ug/M<sup>3</sup>); Shipping and Receiving, 33 ug/M<sup>3</sup> (range, 20-75 ug/M<sup>3</sup>); and General Manufacturing, 24 ug/M<sup>3</sup> (range, 3-79 ug/M<sup>3</sup>). The lowest average cadmium exposures in the Color Division were measured in the Maintenance Department ( 5 ug/M<sup>3</sup>, range, 0-9 ug/M<sup>3</sup>). Airborne cadmium exposure in Coatings Division job categories ranged from 0 to 105 ug/M<sup>3</sup>, with an average of 8 ug/M<sup>3</sup>. The recommended NIOSH standard for occupational exposure to cadmium is 40 ug/M<sup>3</sup>. Eleven (31%) of 35 air cadmium exposure measured in the Color Division exceeded that recommended limit.

Airborne lead exposures in Coatings Division job categories ranged on average from 13 to 79 ug/M<sup>3</sup>. Mean exposures in all except Shipping and Receiving were at or above the NIOSH Recommended Permissible Exposure Limit, and OSHA Standard of 50 ug/M<sup>3</sup>. The highest mean exposure to airborne lead was among Take-Off men, 79 ug/M<sup>3</sup> (range, 65-98 ug/M<sup>3</sup>). Smelter-operators had a mean exposure of 73 ug/M<sup>3</sup> (range, 0-359 ug/M<sup>3</sup>). Millroom workers, who crush and rebag specialty frits, had a mean exposure of 69 ug/M<sup>3</sup> (range, 7-113 ug/M<sup>3</sup>). Employees in the Mixing Department of the Coatings Division had a mean exposure of 61 ug/M<sup>3</sup> (range, 21-81 ug/M<sup>3</sup>). Maintenance personnel, who are required to perform many jobs within the Department, had a mean exposure of 49 ug/M<sup>3</sup> (range, 6-207 ug/M<sup>3</sup>). Thirty-one (41%) of 75 air lead exposure measures in the Coating Department exceeded the current OSHA air lead standard.

Silica exposures at Ferro are currently efficiently controlled. Silica transfer operations, which move silica from rail car to charge car utilize automated transfer techniques, including covered conveyors and automatic scales. Because of the containment engineering, and because personnel are not involved in the transfer process, personal silica exposures were not determined. Prior to the installation of the equipment, it is conceivable that exposures may have been of concern, as indicated by the x-ray screening results.

#### B. Medical

The medical evaluation included 119 workers; 79 with current employment in the Coatings Division, 35 from the Color Division, and five others.

The workers evaluated tended to be middle-aged and stable in their employment. (To some extent those observations are the result of lay-offs which preceded the NIOSH evaluation in which younger, less senior workers were terminated.) The average age of the workers evaluated was 48.9 years. On average, they had been employed at the plant for 20.4 years.

Elevated blood lead levels were found in both the Coatings and Color Divisions. Lead levels were, however, significantly greater in the Coatings Division, and the average blood lead level in Coatings Division workers - 35.1 ug/dl (micrograms per deciliter of whole blood) was significantly higher than that in the Color Division - 20.9 ug/dl (p <0.01). Twenty-two workers (31.9%) in the Coatings Division had blood lead levels of 40 ug/dl or above, indicating that they had absorbed excessive amounts of lead into their bodies.<sup>13</sup> No workers in the Color Division had blood lead levels of 40 ug/dl or higher. The highest blood lead level in a Coatings Division worker was 69 ug/dl.

Blood lead levels were found to be highest in those workers with greatest exposure to airborne lead as measured by personal (breathing zone) air lead determinations (correlation coefficient, r = 0.54; p <0.01). Also, blood lead levels tended to increase with the number of years of exposure to lead (r = 0.22; p = 0.04). No correlation was found between workers' blood lead levels and their age.

Average blood lead levels by current division and job category were as follows:

<u>JOB CATEGORY</u>	<u>NO. WORKERS TESTED</u>	<u>BLOOD LEAD LEVEL (ug/dl)</u>	<u>Mean (<math>\pm</math> S.D.)</u>
<u>Coatings Division</u>			
Mixing Department;	10	35.1	( $\pm$ 14.4)
Take-off			
Smelter Operator	23	39.8	( $\pm$ 11.5)
Millroom			
Shipping & Receiving	9	25.7	( $\pm$ 9.4)
Maintenance	23	36.1	( $\pm$ 9.7)
Total	65		
<u>Color Division</u>			
Cadmium Department;	19	22.0	( $\pm$ 6.7)
General Manufacturing,			
Blending, Batching			
Shipping & Receiving	5	18.8	( $\pm$ 4.7)
Maintenance	8	22.4	( $\pm$ 8.3)
Total	32		

Smelter operators in the Coatings Division had the highest blood lead levels.

Erythrocyte protoporphyrin (EP) is a chemical compound normally found in small amounts in the red blood cells. The EP concentration rises when the metabolism of red blood cells is disrupted by absorption of lead, and an elevated EP concentrations is an early sign of lead toxicity. Fifteen workers (13.8%) at Ferro had EP levels above the upper normal limit for adults of 100 ug/dl; 14 of those workers were employed in the Coatings Division, and one in the Color Division. EP levels were highly correlated with blood lead levels ( $r = 0.59$ ;  $p < 0.01$ ).

Elevated blood cadmium levels were found in both the Coatings and Color Divisions, but predominated in the Color Division. The average blood cadmium level in Color Division workers - 0.56 ug/dl - was significantly higher than that in Coatings Division workers - 0.37 ug/dl ( $p < 0.01$ ). Twelve workers in the Color Division (34.3%) had blood cadmium levels of 0.70 ug/dl or above, indicating that they had absorbed excessive amounts of cadmium; by contrast, only eight workers (11.6%) in the Coatings Division had blood cadmium levels of 0.70 ug/dl or above). The highest blood cadmium concentration in a Color Division worker was 1.37 ug/dl. Blood cadmium levels were positively correlated with personal (breathing zone) exposures to airborne cadmium ( $r = 0.43$ ;  $p < 0.01$ ).

Highest blood cadmium levels (mean 0.68 ug/dl) were found in the cadmium department of the Color Division.

Kidney Function Evaluation. Blood urea nitrogen (BUN) values in 18 (16.5%) of 109 workers tested at Ferro were above the upper laboratory reference value of 20 mg/dl (Table III). No more than three such elevated values would be expected in a group of adult males of the number examined at Ferro. The highest BUN value was 26 mg/dl.

Serum creatinine values were elevated above the upper reference value of 1.5 mg/dl in 8 (7.3%) of the 109 workers. Three such values would have been expected. The highest serum creatinine value was 2.1 mg/dl.

Serum uric acid concentrations were elevated above the upper reference limit of 8.5 mg/dl in 12 (11.0%) of the 109 workers. Three such elevated values would have been expected. The highest serum uric acid value was 9.9 mg/dl. Serum uric acid concentrations were positively correlated with blood lead levels ( $n = 109$ ;  $r = 0.26$ ;  $p < 0.01$ ), but not with blood or urine cadmium concentrations. Four workers at Ferro (3.9%) reported that they had been diagnosed previously as having gout; two were taking "gout medication" at the time of the survey, and the current uric acid levels of the four were: 9.4, 9.0, 8.7, and 5.9 mg/dl.

Urine beta-2-microglobulin concentrations were elevated above the upper reference limit of 370 nanograms (ng)/ml in five workers at Ferro. All five had always worked in the Coatings Division. No correlation was found between beta-2-microglobulin level and any parameter reflective of exposure to cadmium.

Serum concentrations of 1,25-dihydroxy vitamin D (calcitriol) were depressed below the normal range of 26-44 picograms/ml in nine of 108 workers. No correlation was evident between calcitriol concentrations and blood lead levels, as has been reported previously in children exposed to lead.<sup>11</sup>

Urine cadmium concentrations, which when elevated, are an indicator of kidney damage in cadmium workers,<sup>5</sup> were abnormal (greater than 20 ug/l) in only one Ferro worker (27 ug/liter). Urine cadmium concentrations were however, higher on average in Color Division workers (3.7 ug/l) than in Coatings Division workers (2.8 ug/l). This difference became more obvious on examination of the workers whose whole work experience at Ferro had been confined to one or the other Division (with no crossing over). For life-long Color Division workers, the mean urine cadmium concentration was 4.1 ug/l, and for Coatings Division workers, 2.6 ug/l. Urine cadmium concentrations were slightly correlated with years of cadmium exposure ( $r = 0.22$ ;  $p = 0.10$ ), and with blood cadmium concentrations ( $r = 0.24$ ;  $p < 0.01$ ).

Questionnaire Results - Examination of questionnaire results indicated no cases of symptomatic lead or cadmium poisoning in Ferro workers.

X-ray Results. Chest x-rays were performed on 62 workers who reported that they had a past history of exposure to silica dust at Ferro Corporation. Three had x-ray changes compatible with mild to moderate pneumoconiosis. Their clinical and radiological features are summarized in Table IV.

### C. Discussion

The data collected in this investigation indicate that workers at Ferro Corporation are overexposed to lead and to cadmium. Although symptomatic lead and cadmium poisoning were not encountered, there was clear evidence in exposed workers of (1) increased heavy metal absorption, (2) disrupted heme biosynthesis (as manifest by elevated EP values), and (3) kidney disease. Chronic kidney disease is a toxic effect of great concern in workers exposed to heavy metals. Occupationally induced kidney disease was manifest in Ferro workers by unusually high frequency rates for elevated concentrations of BUN, serum creatinine, and uric acid. Given that these are all relatively insensitive tests of kidney dysfunction,<sup>10,14</sup> the elevation of one or more of these test results in 35 of 109 workers evaluated at the plant suggests a serious degree of occupationally induced kidney disease in Ferro workers.

There was also medical evidence of overexposure to silica dust. Three workers at Ferro with histories of exposure to silica at Ferro had x-ray evidence of pneumoconiosis.

## VII. RECOMMENDATIONS

### A. General Recommendations

1. The need exists at Ferro Corporation for the permanent assignment of personnel whose activity is centered around the enhancement of the health and safety of workers in both Divisions. An effort to coordinate the concerns of all parts of the plant would greatly enhance any program of control measures.
2. Personnel should be assigned, and specially trained, to do environmental monitoring and to interpret results.
3. Ferro Corporation should utilize their present engineering talent to redesign ventilation and materials handling systems which are inadequate in controlling hazardous or toxic exposures.
4. Personal protective equipment is not an acceptable substitute for controlling employee exposures in permanent work station/continuous process situations when engineering controls are feasible.

### B. Specific Recommendations for Coatings Division

1. Since the millroom and blending room experience the most problems with hazardous exposures, it would seem prudent to begin control efforts there. It will be particularly important to reduce airborne lead exposures to levels below the current legal standard of 50 ug/M<sup>3</sup> air.

Knowledge gained in this area by Ferro would enable more rapid and directed efforts in other areas of the plant. Although the Racial Airstream helmets are probably the best possible type of respiratory protection available, it is important to reiterate that personal protective equipment is not a satisfactory method of controlling exposures. As was stated in Interim Report I, the method of designating safe and unsafe exposure areas of the millroom and other rooms in the facility by yellow squares is archaic, and has no basis in scientific fact; contaminated air in the plant does not respect painted lines on the floor.

2. Although vacuuming is evident in the millroom as a method of dust retrieval, a good deal of sweeping was still observed. Closed-circuit vacuum systems should be repaired and expanded and sweeping eliminated.

3. General repair is necessary on the existing local exhaust ventilation system. Some redesign is necessary for the collectors which empty by trap-door into barrels. A skirt arrangement attached to the barrels around the emptying port would probably suffice.

4. Reduce all lead exposures to below the OSHA standard of 50 ug/M<sup>3</sup>.

#### C. Specific Recommendations for the Color Division

1. The jet mill, micropulverizer, and batch blending operations generate appreciable amounts of metal-containing dust. Consideration should be directed towards reducing fugitive dusts from these processes. The addition of hoods to existing flexible duct-work will improve the collection efficiency of these systems.

2. The high exposures to cadmium in the filter-press workers warrant that the process be scrutinized by the management at Ferro to ascertain the aspects of the procedure which generate the excessive amounts of contaminant. Reduction of exposure levels is absolutely essential to maintain the health and safety of those workers.

3. Reduce all cadmium exposures to below the NIOSH recommended level of 40 ug/M<sup>3</sup>.

#### D. Medical Recommendations

1. Specialist follow-up is required for all workers with elevated BUN, serum creatinine, or uric acid concentrations or with excessive excretion in urine of beta-2-microglobulin and also for any other workers at Ferro with more than 2 years' exposure to lead and/or cadmium. NIOSH is currently making arrangements with kidney disease specialists for such follow-up.

2. Specialist follow-up is also required for the three workers who had x-ray evidence of pneumoconiosis.

3. Plant management should establish a screening program for silicosis among those workers who are exposed to silica dust. Guidelines for such a screening program may be found in the NIOSH criteria document on silica.

VIII. REFERENCES

1. Webb M: Cadmium. *Brit Med Bull* 31:246-250, 1975.
2. Lauwerys R, Roels H, Regniers M, et al: Significance of cadmium concentration in blood and in urine in workers exposed to cadmium. *Environ Research* 20:375-391, 1979.
3. Kjellstrom T: Exposure and accumulation of cadmium in populations from Japan, the United States, and Sweden. *Environ Health Perspec* 28:169-197, 1979.
4. Ellis KJ, Vartsky D, Zanzi I, et al: Cadmium: in vivo measurement in smokers and nonsmokers. *Science* 205:323-325, 1979.
5. Friberg L, Piscator M, Nordberg GF, et al: Cadmium in the Environment. Cleveland: CRC Press, 1974.
6. Baker EL, Peterson WA, Holtz J, et al: Subacute cadmium intoxication in jewelry workers: an evaluation of diagnostic procedures. *Arch Environ Health* 34:173-177, 1979.
7. Scott R, Paterson PJ, Mills EA, et al: Clinical and biochemical abnormalities in coppersmiths exposed to cadmium. *Lancet* 2:396-398, 1976.
8. Lemen RA, Lee JS, Wagoner JK, et al: Cancer mortality among cadmium production workers. *Ann NY Acad Sci* 271:273-279, 1976.
9. Piscator M: Role of cadmium in carcinogenesis with special reference to cancer of the prostate. *Environ Health Perspec* 40:107-120, 1981.
10. Page LB, Culver PJ: A Syllabus of Laboratory Examinations in Clinical Diagnosis. Cambridge, Harvard University Press, 1962.
11. Rosen JF, Chesney R, Hamstra A, et al: Reduction in 1,25-dihydroxy vitamin D in children with increased lead absorption. *New Engl J Med* 302:128-131, 1980.
12. Kazantzis G: Renal tubular dysfunction and abnormalities of calcium metabolism in cadmium workers. *Environ Health Perspec* 28:155-159, 1979.

13. Occupational Safety and Health Administration: U.S. Department of Labor: Occupational exposure to lead - final standard. *Federal Register* 43:
14. Wedeen RP, Maesaica JK, Weiner B, et al: Occupational lead nephropathy. *Am J Med* 59:630, 1975.
15. Ball GV, Sorensen LB: Pathogenesis of hyperuricemia in saturnine gout. *New Engl J Med* 280:119, 1969.

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X. DISTRIBUTION AND AVAILABILITY OF REPORT

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1. President, Local 1170, United Steelworkers of America, Ferro Corporation, Cleveland, Ohio
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4. OSHA, Region V

For the purpose of informing affected employees, copies of this report shall be posted by the employer in a prominent place accessible to the employees for a period of 30 calendar days.

TABLE I

PERSONAL (BREATHING ZONE) AIR  
 LEAD AND CADMIUM EXPOSURES BY DIVISION AND JOB CATEGORY  
 (Exposures given in micrograms per cubic meter of air)

FERRO CORPORATION, CLEVELAND, OHIO

March 16-20, 1981

HE 80-116

JOB CATEGORY	NO. OF SAMPLES	LEAD		CADMIUM	
		MEAN	RANGE	MEAN	RANGE
<u>Coatings Division</u>					
Mixing	11	61	21-81	9	2-23
Take-Off	6	79	65-98	8	3-14
Smelter-Op	29	73	0-359	11	0-105
Millroom	11	69	7-113	8	0-21
Ship-Rec.	1	13	---	4	---
Maintenance	17	49	6-207	5	0-25
<u>Color Division</u>					
Cadmium Dept.	5	0	---	229	74-384
Maintenance	2	--	---	5	0-9
General Mfg.	21	2	---	24	3-79
Ship-Rec.	2	--	---	33	20-45
Batch-Blend	5	--	---	42	20-75
NIOSH Recommended Standard		50		40(10 Hr. TWA)	
OSHA Standard		50		200	
Limit of detection per sample		3.0		1.0	

TABLE II  
PARTICULATE CONTENT OF AIR SAMPLES - RESPIRABLE AND TOTAL FRACTIONS

FERRO CORPORATION, CLEVELAND, OHIO  
 MARCH 16-20, 1981  
 HE 80-116

LOCATION	Sample Vol(1)	Total Part. mg/M <sup>3</sup>	Total Lead ug/M <sup>3</sup>	Resp. Part. mg/M <sup>3</sup>	Resp. Lead ug/M <sup>3</sup>	% Resp. Part.	% Resp. Lead
<u>COATINGS DIVISION</u>							
Near control room & weighing sta- tion	2925	2.0	30.6	0.6	13.6	28	44
Blending Room	603	16.4	232.4	7.5	101.3	46	44
Continuous Mills	1566	0.2	19.2	0.4	15.4	130	80
Take-off area/ charging floor	2763	3.0	104.4	1.0	54.0	34	52
Near Smelter clean-up	2097	7.5	86.4	3.9	76.8	54	89
Millroom (lead- free milling)	2223	0.2	ND	0.1	ND	29	--
<u>COLOR DIVISION</u>							
Shipping Area	1890	0.3	2.7	0.2	2.1	69	80
Second floor	1908	0.5	--	0.5	--	102	--
NIOSH Recommends (8-hour TWA)		10	50				
OSHA Standard		15	50				
Limit of detection		0.01	0.00				

TABLE III  
 RENAL FUNCTION TEST (RFT) RESULTS IN WORKERS WITH ONE OR MORE RFT ABNORMALITIES  
 FERRO CORPORATION, CLEVELAND, OHIO  
 MARCH 16-20, 1981  
 HETA 80-116

BUN (mg/dl)	SERUM CREATININE (mg/dl)	URIC ACID (mg/dl)	URINE BETA-2- MICROGLOBULIN (mg/ml)	HYPOPHOS- PHATEMIA*	HYPO- CALCI- TRIOL**	HYPER- PARATHY- ROIDISM***	YEARS EXPOSED TO LEAD	YEARS EXPOSED TO CADMIUM
22	1.3	6.3	99				.	.
17	1.2	9.0	13				2.0	15.0
19	1.3	8.9	54		+		6.0	21.0
22	1.4	8.0	44				1.0	6.0
16	1.1	9.0	30		+		2.0	11.0
21	1.2	6.0	72		+		5.0	2.0
25	1.1	5.7	196		+		13.0	0.2
18	1.1	9.2	87				1.0	3.0
24	1.2	7.2	247				11.0	5.0
22	1.1	4.9	30				11.0	0.3
13	1.0	9.4	41				0.5	11.0
26	1.5	9.9	30				8.0	2.0
21	1.3	5.8	49				0.4	12.0
15	1.1	6.5	960				12.0	.
18	1.8	8.4	950				.	.
13	1.6	8.6	68				8.0	.
23	1.4	8.9	30		+		34.0	.
12	1.0	3.0	960	+			15.0	.
10	1.0	8.7	115				5.0	.
14	1.4	9.9	56				26.8	.
14	1.0	6.0	960				5.0	.
24	1.4	6.4	50				21.0	.
26	1.2	6.1	125				10.0	.
21	1.1	5.5	81				5.0	.
11	1.1	8.9	119				23.0	.
19	2.1	6.6	30				23.0	.
22	1.2	5.3	543				12.0	.
25	1.4	6.3	67				13.0	.
16	1.6	6.2	81				13.0	.
17	1.6	7.0	30				.	15.0
21	1.0	5.9	69				.	.
25	1.3	8.3	30				.	20.0
22	1.9	7.8	93				.	13.5
25	1.7	9.9	228		+		.	.
17	1.7	6.6	30				.	26.0

Normal range at CBL  
 (10-20) (0.7-1.5) (4.0-8.5) (4-370)

\*Hypophosphatemia = serum phosphorus concentration below 2.5 mg/dl

\*\* Hypercalcuria = urine calcitriol content above 26 pg/ml

\*\*\*Hyperparathyroidism = serum parathormone concentration above 2000 pg/ml

TABLE IV  
 CLINICAL FEATURES OF WORKERS WITH X-RAY EVIDENCE OF PNEUMOCONIOSIS  
 FERRO CORPORATION, CLEVELAND, OHIO  
 MARCH 16-20, 1981  
 HETA 80-116

<u>YEARS AT FERRO</u>	<u>CLINICAL FINDINGS</u>	<u>SMOKING HISTORY</u>	<u>PER CENT RESPIRATOR USE</u>	<u>X-RAY FINDINGS</u>
29	Wheezing, shortness of breath, chest tightness	Never smoked	100	1/1 small rounded opacities
37	Asymptomatic	Smoker-47 yrs.	25-50	1/0 small irregular opacities
12	Shortness of breath, cough	Never smoked	50-75	0/1 small rounded opacities

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