

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
HE 79-78-655

BERTOIA STUDIO
BALLY, PENNSYLVANIA

JANUARY 1980

I. TOXICITY DETERMINATION

A health hazard evaluation of the Bertoia Studio art work and wind system production areas was conducted during the periods of May 10, June 20, and August 1, 1979. NIOSH Regional (III) Industrial Hygienist, Frank A. Lewis, carried out a comprehensive walk-through, indirect medical questionnaire interviews and environmental sampling to determine possible employee exposures to beryllium dust, cadmium fumes, and fluorides (soluble).

It is the judgement of this industrial hygienist that the workers were not exposed to potentially toxic concentrations of the aforementioned toxic materials. In fact, all of the air samples taken were below the reliable analytical limit of detection for each of the substances.

II. DISTRIBUTION AND AVAILABILITY OF DETERMINATION REPORT

Copies of this Determination Report are currently available upon request from NIOSH, Division of Technical Services, Information Resources and Dissemination Section, 4676 Columbia Parkway, Cincinnati, Ohio 45226. After 90 days the report will be available through the National Technical Information Service (NTIS), Springfield, Virginia. Information regarding its availability through NTIS can be obtained from NIOSH, Publications Office at the Cincinnati address.

Copies of this report have been sent to:

- a) Bertoia Studio, Bally, Pennsylvania
- b) Employee
- c) NIOSH, Region III
- d) OSHA, Region III

For the purpose of informing the two employees of the results of the Bertoia Studio survey, the employer shall promptly "post" for a period of 30 calendar days the Determination Report in a prominent place(s) for their perusal.

III. INTRODUCTION

Section 20(a)(6) of the Occupational Safety and Health Act of 1970, U.S.C. 669(a)(6), authorizes the Secretary of Health, Education, and Welfare, following a written request by an 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.

On April 16, 1979, NIOSH Region III received a request from Bertoia Studio for a health hazard evaluation of the art work and wind system production areas. The employer requested NIOSH to evaluate the effects of beryllium dust and cadmium fumes upon the health of the workers to make appropriate recommendations where necessary.

NIOSH Regional Industrial Hygienist, Frank A. Lewis, met with the owner and his two assistants for the opening and closing conferences, walk-through survey and environmental sampling on May 10 and August 1, 1979. Discussions with management involved the collection of information concerning process description, engineering controls, personal protective equipment and clothing, work practices, training programs, monitoring, record-keeping and medical surveillance for the areas in question. Employee interviews focused in on the job description, work practices, training programs, and any associated health problems.

IV. HEALTH HAZARD EVALUATION

A. Plant Process

Bertoia Studio consists of two levels - a first floor and a basement area. The first floor operations involve the drilling, cutting, grinding, and welding of steel, aluminum, brass and bronze; the brazing of these metals and beryllium alloys and the acid-spraying of art works with nitric or sulphuric acids are also done here.

Welding operations are done under a large canopy hood (3 ft. X 5 ft.) which operates quite effectively in capturing the welding fumes. Ventilation measurements show a face velocity of 300 feet/minute and a capture velocity of 75 feet/minute at one-half the vertical distance from the work table surface to the hood face (~ 2 ft. maximum distance at which fumes would be generated); smoke tube studies also showed good capture. Very large pieces are welded out in the open shop area (~ 20 ft. HT X 50 ft. W. X 75 ft. L.).

The brazing operation of beryllium/copper alloys is done using a silver solder (cadmium) and fluoride flux without the use of any local exhaust. Adjacent windows, however, are kept open but air movement is turbulent (15-50 feet/minute) at the work operation. The employee wears a double cartridge half face-piece respirator for toxic dusts, mists and fumes (TC21C-142 with R-12 cartridges). This operation is done by one man approximately once a week.

The basement area involves the sanding, drilling, and cutting of all of the aforementioned materials and the arc/TIG welding of aluminum and stainless steel.

Beryllium alloys are cut on a carborundum cut-off wheel by one or two men approximately once a week. A local exhaust attachment is used which operates quite effectively in capturing beryllium dusts. Capture velocities of greater than 800 feet/minute were measured at the two points of capture - behind and below the cut-off wheel. However, this local exhaust unit had only recently been installed. The employees wear a double-cartridge half face-piece respirator for toxic dusts, mists, and fumes (TC21C-142 with R-12 cartridges).

B. Evaluation Design

Personal air samples were taken for the duration of the work operations and will provide a basis for evaluating employee exposures for the eight-hour work day. Air samples were taken for beryllium dust (cutting operations), cadmium fumes (brazing operation), and soluble fluorides. Brass or bronze art pieces are made sporadically. Therefore, it was not possible to sample for lead or copper dusts and fumes during the course of this survey.

C. Evaluation Methods

The personal air samples for beryllium dust exposure were collected on 0.45 μ HA filters using a three-piece cassette. Air was drawn through the filter and back-up pads at a flow rate of 1.5 liters/minute using a MSA Model G personal air sampling pump. These samples were analyzed according to NIOSH method P&CAM 121. Because of the small volume of air sampled, these samples were analyzed with another batch using a graphite furnace to lower the limit of detection⁴.

The personal air sample for cadmium fumes was collected on a 0.8 μ AA filter with back-up pad using a three-piece cassette. Air was drawn through the filter using a Model G MSA pump at a flow rate of 1.5 liters/minute. These samples were analyzed according to NIOSH method of S313 using atomic absorption spectrophotometry¹.

The personal air sample for soluble fluorides was collected in 10 ml of 0.1 N sodium hydroxide solution using a midget impinger. Air was drawn through the impinger at a flow rate of 2.0 liter/minute using a MSA Model G air sampling pump. These samples were analyzed for fluoride using NIOSH method P&CAM 117¹.

The results of these samples are presented in Table I.

D. Evaluation Criteria

1. Environmental Standards

| <u>Substance</u> | <u>OSHA</u> ^{2,5} | <u>ACGIH</u> ³ | <u>NIOSH</u> ^{4,6,7} |
|-------------------|---|--|---|
| Beryllium Dust | 2 $\mu\text{g}/\text{M}^3$ -8hr.TWA* 5 $\mu\text{g}/\text{M}^3$ -acceptable ceiling 25 $\mu\text{g}/\text{M}^3$ -maximum ceiling (30 minutes) | 2 $\mu\text{g}/\text{M}^3$ -8hr.TWA 25 $\mu\text{g}/\text{M}^3$ -STEL** | 0.5 $\mu\text{g}/\text{M}^3$ -(130 minutes) |
| Cadmium Fume | 0.1 mg/M^3 -8hr.TWA 0.3 mg/M^3 -ceiling | 0.05 mg/M^3 -8hr.TWA | 40 $\mu\text{g}/\text{M}^3$ -8hr.TWA 200 $\mu\text{g}/\text{M}^3$ -ceiling (15 minutes) |
| Fluorides | 2.5 mg/M^3 -8hr.TWA | 2.5 mg/M^3 -8hr.TWA | 2.5 mg/M^3 -8hr.TWA |

*TWA = time-weighted average

**STEL = short-term exposure limit

2. Toxicological Data^{3,4,5,6,7}Beryllium - Local

The soluble beryllium salts are cutaneous sensitizers as well as primary irritants. Contact dermatitis of exposed parts of the body are caused by acid salts of beryllium. Onset is generally delayed about two weeks from the time of first exposure. Complete recovery occurs following cessation of exposure. Eye irritation and conjunctivitis can occur. Accidental implantation of beryllium metal or crystals of soluble beryllium compound in areas of broken or abraded skin may cause granulomatous lesions. These are hard lesions with a central nonhealing area. Surgical excision of the lesion is necessary. Exposure to soluble beryllium compounds may cause nasopharyngitis, a condition characterized by swollen and edematous mucous membranes, bleeding points, and ulceration. These symptoms are reversible when exposure is terminated.

Systemic

Beryllium and its compounds are highly toxic substances. Entrance to the body is almost entirely by inhalation. The acute systemic effects of exposure to beryllium primarily involve the respiratory tract and are manifest by a nonproductive cough, substernal pain, moderate shortness of breath, and some weight loss. The character and speed of onset of these symptoms, as well as their severity, are dependent on the type and extent of exposure. An intense exposure, although brief, may result in severe chemical pneumonitis with pulmonary edema.

Chronic beryllium disease is an intoxication arising from inhalation of beryllium compounds, but is not associated with inhalation of the mineral beryl. The chronic form of this disease is manifest primarily by respiratory symptoms, weakness, fatigue, and weight loss (without cough or dyspnea at the onset), followed by non-productive cough and shortness of breath. Frequently, these symptoms and detection of the disease are delayed from five to ten years

following the last beryllium exposure, but they can develop during the time of exposure. The symptoms are persistent and frequently are precipitated by an illness, surgery, or pregnancy. Chronic beryllium disease usually is of long duration with exacerbations and remissions.

Chronic beryllium disease can be classified by its clinical variants according to the disability the disease process produces.

1. Asymptomatic nondisabling disease is usually diagnosed only by routine chest X-ray changes and supported by urinary or tissue assay.
2. In its mildly disabling form, the disease results in some nonproductive cough and dyspnea following unusual levels of exertion. Joint pain and weakness are common complaints. Diagnosis is by X-ray changes. Renal calculi containing beryllium may be a complication. Usually, the patient remains stable for years, but eventually shows evidence of pulmonary or myocardial failure.
3. In its moderately severe disabling form, the disease produces symptoms of distressing cough and shortness of breath, with marked X-ray changes. The liver and spleen are frequently affected, and spontaneous pneumothorax may occur. There is generally weight loss, bone and joint pain, oxygen desaturation, increase in hematocrit, disturbed liver function, hypercalciuria, and spontaneous skin lesions similar to those of Boeck's sarcoid. Lung function studies show measurable decreases in diffusing capacity. Many people in this group survive for years with proper therapy. Bouts of chills and fever carry a bad prognosis.
4. The severely disabling disease will show all of the above mentioned signs and symptoms in addition to severe physical wasting and negative nitrogen balance. Right heart failure may appear causing a severe nonproductive cough which leads to vomiting after meals. Severe lack of oxygen is the predominant problem, and spontaneous pneumothorax can be a serious complication. Death is usually due to pulmonary insufficiency or right heart failure.

Beryllium is also suspected of having cancer-causing potential for humans. Lung and bone malignancies from exposures to beryllium compounds have been reported in laboratory animals and although epidemiological studies in humans are not definitely conclusive, evidence appears to be consistent with test animal findings.

Cadmium - Local

Cadmium is an irritant to the respiratory tract. Prolonged exposure can cause anosmia and a yellow stain or ring that gradually appears on the necks of the teeth. Cadmium compounds are poorly absorbed from the intestinal tract, but relatively well absorbed by inhalation. Skin absorption appears negligible. Once absorbed cadmium has a very long biological half-life and is retained in the kidney and liver.

Systemic

Acute toxicity is almost always caused by inhalation of cadmium fumes or dust which are produced when cadmium is heated. There is generally a latent period of a few hours after exposure before symptoms develop. During the ensuing period, symptoms may appear progressively. The earliest symptom is slight irritation of the upper respiratory tract. This may be followed over the next few hours by cough, pain in the chest, sweating, and chills which resemble the symptoms of nonspecific upper respiratory infection. Eight to 24 hours following acute exposure severe pulmonary irritation may develop, with pain in the chest, dyspnea, cough, and generalized weakness. Dyspnea may become more pronounced as pulmonary edema develops. The mortality rate in acute cases is about 15%. Patients who survive may develop emphysema and cor pulmonale; recovery can be prolonged.

Chronic cadmium poisoning has been reported after prolonged exposure to cadmium oxide fumes, cadmium oxide dust, cadmium sulfides, and cadmium stearates. Heavy smoking has been reported to considerably increase tissue cadmium levels. In some cases, only the respiratory tract is affected. In others the effects may be systemic due to absorption of the cadmium. Lung damage often results in a characteristic form of emphysema which in some instances is not preceded by a history of chronic bronchitis or coughing. This type of emphysema can be extremely disabling. Some studies have not shown these effects.

Systemic changes due to cadmium absorption include damage to the kidneys with proteinuria, anemia, and elevated sedimentation rate. Of these, proteinuria (low molecular weight) is the most typical. In advanced stages of the disease, there may be increased urinary excretion of amino acids, glucose, calcium, and phosphates. These changes may lead to the formation of renal calculi. If the exposure is discontinued, there is usually no progression of the kidney damage. Mild hypochromic anemia is another systemic condition sometimes found in chronic exposure to cadmium.

In studies with experimental animals, cadmium has produced damage to the liver and central nervous system, testicular atrophy, teratogenic effects in rodents after intravenous injection of cadmium, decrease in total red cells, sarcomata, and testicular neoplasms. Hypertensive effects have also been produced. None of these conditions, however, has been found in man resulting from occupational exposure to cadmium. Heavy smoking would appear to increase the risk of cumulative toxic effects.

Fluorides - Local

Fluorine and some of its compounds are primary irritants of skin, eyes, mucous membranes, and lungs. Thermal or chemical burns may result from contact; the chemical burns cause deep tissue destruction and may not become symptomatic until several hours after contact, depending on dilution. Nosebleeds and sinus trouble may develop on chronic exposure to low concentration of fluoride or fluorine in air. Accidental fluoride burns, even when they involve small body areas (less than 3%), can cause systemic effects of fluoride poisoning by absorption of the fluoride through the skin.

Systemic

Inhalation of excessive concentration of elemental fluorine or of hydrogen fluoride can produce bronchospasm, laryngospasm, and pulmonary edema. Gastrointestinal symptoms may be present. A brief exposure to 25 ppm has caused sore throat and chest pain, irreparable damage to the lungs, and death.

Most cases of acute fluoride intoxication result from ingestion of fluoride compounds. The severity of systemic effects is directly proportional to the irritating properties and the amount of the compound that has been ingested. Gastrointestinal symptoms of nausea, vomiting, diffuse abdominal cramps, and diarrhea can be expected. Large doses produce central nervous system involvement with twitching of muscle groups, tonic and clonic convulsions, and coma.

The systemic effects of prolonged absorption of fluorides from either dusts or vapors have long been a source of some uncertainty. Fluorides are retained preferentially in bone, and excessive intake may result in an osteosclerosis that is recognizable by X-ray. The first signs of changes in density appear in the lumbar spine and pelvis. Usually some ossification of ligaments occurs. Recent investigation suggest that rather severe skeletal fluorosis can exist in workers without any untoward physiological effects, detrimental effects on their general health, or physical impairment.

Fluorides occur in nature and enter the human body through inhalation or ingestion (natural dusts and water). In children, mottling of the dental enamel may occur from increased water concentrations. These exposures are usually minimal and occur over extended periods.

E. Evaluation Results and Recommendations

1. The personal air samples taken for beryllium dust, cadmium fume and fluorides (soluble) were found to be below their reliable analytical limit of detection which is considerable below the environmental criteria for these substances. Therefore, these employees would not be exposed to potentially toxic concentrations of these substances under existing conditions.
2. Safety glasses with side-shields should be worn when cutting, drilling, or grinding.
3. Throw-away coveralls, booties, and gloves should be used when cutting beryllium alloys and cleaning out the local exhaust ventilation unit and filters.
4. Throw-away coveralls, booties, and gloves and local exhaust ventilation filters should be disposed of in "labeled" plastic bags for "proper" disposal (contact the local EPA or DER authorities).
5. Smoking, eating or storage of foods should not be allowed in the work areas.
6. Employees should wash-up thoroughly (hands, neck and face) after cutting, grinding, or drilling beryllium alloys, bronzes and brasses.

7. Chemical face shields, acid-resistant gloves, apron and booties should be worn by employees spraying nitric and sulphuric acids on art pieces.
8. An eye-wash located "close-by" and accessible to the employees should be installed in case of acid-splash to the eyes and/or face.

V. REFERENCES

1. NIOSH Manual of Sampling Data Sheets, 1977 Edition, DHEW, PHS, CDC, NIOSH, March 1977.
2. General Industrial, OSHA Safety and Health Standards, 29 CFR OSHA 2206, Revised January 1976.
3. Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1978, American Conference of Governmental Industrial Hygienists, Cincinnati, Ohio.
4. Criteria for a Recommended Standard...Occupational Exposure to Beryllium, Publication No. 72-10268, NIOSH.
5. Beryllium - Proposed Occupational Safety and Health Standard, DOL, OSHA, October 1975, Federal Register, 29 CFR 1910.
6. Criteria for a Recommended Standard...Occupational Exposure to Cadmium, Publication No. 76-192, NIOSH.
7. Criteria for a Recommended Standard...Occupational Exposure to Inorganic Fluoride, Publication No. 76-103, NIOSH.

VI. AUTHORSHIP AND ACKNOWLEDGEMENTS

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Table I
 Results of Personal Air Sampling*
 Bertoia Studio
 Bally, Pennsylvania
 August 1, 1979

| <u>Substance</u> | <u>Sample Number</u> | <u>Sampling Time**</u> | <u>Job Operation</u> | <u>Concentration</u> |
|---------------------|----------------------|------------------------|--------------------------------------|-------------------------------------|
| Beryllium Dust | BS-1 | 12 minutes | Cutting (Beryllium/Copper alloys) | < 0.2 µg/sample < 0.04 µg/sample |
| Cadmium Fume | BS-3 | 11 minutes | Brazing (Silver Solder) | < 2.0 µg/sample |
| Fluorides (soluble) | BS-5 | 11 minutes | Brazing (Flux) | < 2.0 µg/sample |

*All samples and blanks were below the reliable analytical limit of detection, which is 0.2 µg/sample (aspiration) and 0.04 µg/sample (graphite furnace) for beryllium; and 2 µg/sample for cadmium and fluoride.

**In general, the work operations are of extremely short duration; this accounts for the short sampling time and low volume as no other exposures occur throughout the remainder of the work day.