

tact may result in reduced vision or blindness. Dental discoloration and erosion of exposed incisors occur on prolonged exposure to low concentrations. Ingestion may produce fatal effects from esophageal or gastric necrosis.

#### *Systemic—*

The irritant effect of vapors on the respiratory tract may produce laryngitis, glottal edema, bronchitis, pulmonary edema, and death.

#### MEDICAL SURVEILLANCE

Special consideration should be given to the skin, eyes, teeth, and respiratory system. Pulmonary function studies and chest X-rays may be helpful in following recovery from acute overexposure.

#### SPECIAL TESTS

None in common use.

#### PERSONAL PROTECTIVE METHODS

Appropriate gas masks with canister or supplied air respirators should be provided when vapor concentrations are excessive. Acid resistant clothing including gloves, gauntlets, aprons, boots, and goggles or face shield should be provided in all areas where there is likelihood of splash or spill of liquid. Personal hygiene and showering after each shift should be encouraged.

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## METALLIC COMPOUNDS

### *ALUMINUM AND COMPOUNDS*

#### DESCRIPTION

Al, aluminum, is a light, silvery-white, soft, ductile, malleable amphoteric metal, soluble in acids or alkali, insoluble in water. The primary sources are the ores cryolite and bauxite; aluminum is never found in the elemental state.

#### SYNONYMS

None.

#### POTENTIAL OCCUPATIONAL EXPOSURES

Most hazardous exposures to aluminum occur in smelting and refining processes. Aluminum is mostly produced by electrolysis of  $Al_2O_3$  dissolved in molten cryolite ( $Na_3AlF_6$ ). Aluminum is alloyed with

copper, zinc, silicone, magnesium, manganese, and nickel; special additives may include chromium, lead, bismuth, titanium, zirconium, and vanadium. Aluminum and its alloys can be extruded or processed in rolling mills, wireworks, forges, or foundries, and are used in the shipbuilding, electrical, building, aircraft, automobile, light engineering, and jewelry industries. Aluminum foil is widely used in packaging. Powdered aluminum is used in the paints and pyrotechnic industries. Alumina (aluminum oxide,  $Al_2O_3$ ) has been utilized as abrasives, refractories, and catalysts, and in the past in the first firing of china and pottery. Aluminum chloride ( $AlCl_3$ ) is used in petroleum processing and in the rubber industry. Alkyl aluminum compounds find use as catalysts in the production of polyethylene.

A partial list of occupations in which exposure may occur includes:

Aluminum alloy grinders	Foundry workers
Aluminum workers	Petroleum refinery workers
Ammunition makers	Plastic makers
Fireworks makers	Rubber makers

#### PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard specifically for metallic aluminum. It may be considered as a nuisance dust, the applicable standards being: respirable fraction, 15 mppcf or 5 mg/m<sup>3</sup>; total dust, 50 mppcf or 15 mg/m<sup>3</sup>.

#### ROUTE OF ENTRY

Inhalation of dust or fume.

#### HARMFUL EFFECTS

##### *Local—*

Particles of aluminum deposited in the eye may cause necrosis of the cornea. Salts of aluminum may cause dermatoses, eczema, conjunctivitis, and irritation of the mucous membranes of the upper respiratory system by the acid liberated by hydrolysis.

##### *Systemic—*

The effects on the human body caused by the inhalation of aluminum dust and fumes are not known with certainty at this time. Present data suggest that pneumoconiosis might be a possible outcome. In the majority of cases investigated, however, it was found that exposure was not to aluminum dust alone, but to a mixture of aluminum, silica fume, iron dusts, and other materials.

#### MEDICAL SURVEILLANCE

Preemployment and periodic physical examinations should give special consideration to the skin, eyes, and lungs. Lung function should be followed.

#### SPECIAL TESTS

None commonly used.

## PERSONAL PROTECTIVE METHODS

Workers in electrolysis manufacturing plants should be provided with respirators for protection from fluoride fumes. Dust masks are recommended in areas exceeding the nuisance levels. Aluminum workers generally should receive training in the proper use of personal protective equipment. Workers involved with salts of aluminum may require protective clothing, barrier creams, and where heavy concentrations exist, fullface air supplied respirators may be indicated.

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## ARSENIC

### DESCRIPTION

As, elemental arsenic, occurs to a limited extent in nature as a steel gray metal that is insoluble in water. Arsenic in this discussion includes the element and any of its inorganic compounds excluding arsine. Arsenic trioxide ( $\text{As}_2\text{O}_3$ ), the principal form in which the element is used, is frequently designated as arsenic, white arsenic, or arsenous oxide. Arsenic is present as an impurity in many other metal ores and is generally produced as arsenic trioxide as a by-product in the smelting of these ores, particularly copper. Most other arsenic compounds are produced from the trioxide.

### SYNONYMS

None.

### POTENTIAL OCCUPATIONAL EXPOSURES

Arsenic compounds have a variety of uses. Arsenates and arsenites are used in agriculture as insecticides, herbicides, larvicides, and pesticides. Arsenic trichloride is used primarily in the manufacture of pharmaceuticals. Other arsenic compounds are used in pigment production, the manufacture of glass as a bronzing or decolorizing agent, the manufacture of opal glass and enamels, textile printing, tanning, taxidermy, and antifouling paints. They are also used to control sludge formation in lubricating oils. Metallic arsenic is used as an alloying agent to harden lead shot and in lead-base bearing materials. It is also alloyed with copper to improve its toughness and corrosion resistance.

A partial list of occupations in which exposure may occur includes:

Alloy makers	Lead shot makers
Aniline color makers	Lead smelters
Arsenic workers	Leather workers
Babbitt metal workers	Painters
Brass makers	Paint makers
Bronze makers	Petroleum refinery workers
Ceramic enamel makers	Pigment makers
Ceramic makers	Printing ink workers
Copper smelters	Rodenticide makers
Drug makers	Semiconductor compound makers
Dye makers	Silver refiners
Enamelers	Taxidermists
Fireworks makers	Textile printers
Gold refiners	Tree sprayers
Herbicide makers	Type metal workers
Hide preservers	Water weed controllers
Insecticide makers	Weed sprayers

#### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for arsenic and its compounds is 0.5 mg/m<sup>3</sup> of air as As. NIOSH has recommended 0.002 mg/m<sup>3</sup> of air as As based on its carcinogenic effects.

#### ROUTES OF ENTRY

Inhalation and ingestion of dust and fumes.

#### HARMFUL EFFECTS

##### *Local—*

Trivalent arsenic compounds are corrosive to the skin. Brief contact has no effect, but prolonged contact results in a local hyperemia and later vesicular or pustular eruption. The moist mucous membranes are most sensitive to the irritant action. Conjunctiva, moist and macerated areas of skin, the eyelids, the angles of the ears, nose, mouth, and respiratory mucosa are also vulnerable to the irritant effects. The wrists are common sites of dermatitis, as are the genitalia if personal hygiene is poor. Perforations of the nasal septum may occur. Arsenic trioxide and pentoxide are capable of producing skin sensitization and contact dermatitis. Arsenic is also capable of producing keratoses, especially of the palms and soles. Arsenic has been cited as a cause of skin cancer, but the incidence is low.

##### *Systemic—*

The acute toxic effects of arsenic are generally seen following ingestion of inorganic arsenical compounds. This rarely occurs in an industrial setting. Symptoms develop within ½ to 4 hours following ingestion and are usually characterized by constriction of the throat followed by dysphagia, epigastric pain, vomiting, and watery diarrhea. Blood may appear in vomitus and stools. If the amount ingested is suf-

ficiently high, shock may develop due to severe fluid loss, and death may ensue in 24 hours. If the acute effects are survived, exfoliative dermatitis and peripheral neuritis may develop.

Cases of acute arsenical poisoning due to inhalation are exceedingly rare in industry. When it does occur, respiratory tract symptoms—cough, chest pain, dyspnea—giddiness, headache, and extreme general weakness precede gastrointestinal symptoms. The acute toxic symptoms of trivalent arsenical poisoning are due to severe inflammation of the mucous membranes and greatly increased permeability of the blood capillaries.

Chronic arsenical poisoning due to ingestion is rare and generally confined to patients taking prescribed medications. However, it can be a concomitant of inhaled inorganic arsenic from swallowed sputum and improper eating habits. Symptoms are weight loss, nausea and diarrhea alternating with constipation, pigmentation and eruption of the skin, loss of hair, and peripheral neuritis. Chronic hepatitis and cirrhosis have been described. Polyneuritis may be the salient feature, but more frequently there are numbness and parasthenias of "glove and stocking" distribution. The skin lesions are usually melanotic and keratotic and may occasionally take the form of an intradermal cancer of the squamous cell type, but without infiltrative properties. Horizontal white lines (striations) on the fingernails and toenails are commonly seen in chronic arsenical poisoning and are considered to be a diagnostic accompaniment of arsenical polyneuritis.

Inhalation of inorganic arsenic compounds is the most common cause of chronic poisoning in the industrial situation. This condition is divided into three phases based on signs and symptoms.

**First Phase:** The worker complains of weakness, loss of appetite, some nausea, occasional vomiting, a sense of heaviness in the stomach, and some diarrhea.

**Second Phase:** The worker complains of conjunctivitis, a catarrhal state of the mucous membranes of the nose, larynx, and respiratory passage. Coryza, hoarseness, and mild tracheobronchitis may occur. Perforation of the nasal septum is common, and is probably the most typical lesion of the upper respiratory tract in occupational exposure to arsenical dust. Skin lesions, eczematoid and allergic in type, are common.

**Third Phase:** The worker complains of symptoms of peripheral neuritis, initially of hands and feet, which is essentially sensory. In more severe cases, motor paralyses occur; the first muscles affected are usually the toe extensors and the peronei. In only the most severe cases will paralysis of flexor muscles of the feet or of the extensor muscles of hands occur.

Liver damage from chronic arsenical poisoning is still debated, and as yet the question is unanswered. In cases of chronic and acute arsenical poisoning, toxic effects to the myocardium have been reported based on EKG changes. These findings, however, are now largely discounted and the EKG changes are ascribed to electrolyte disturbances concom-

itant with arsenicalism. Inhalation of arsenic trioxide and other inorganic arsenical dusts does not give rise to radiological evidence of pneumoconiosis. Arsenic does have a depressant effect upon the bone marrow, with disturbances of both erythropoiesis and myelopoiesis. Evidence is now available incriminating arsenic compounds as a cause of lung cancer as well as skin cancer.

#### MEDICAL SURVEILLANCE

In preemployment physical examinations, particular attention should be given to allergic and chronic skin lesions, eye disease, psoriasis, chronic eczematous dermatitis, hyperpigmentation of skin, keratosis and warts, baseline weight, baseline blood and hemoglobin count, and baseline urinary arsenic determinations. In annual examinations, the worker's general health, weight, and skin condition should be checked, and the worker observed for any evidence of excessive exposure or absorption of arsenic.

#### SPECIAL TESTS

Chest X-rays and lung function should be evaluated; analysis of urine, hair, or nails for arsenic should be made every 60 days as long as exposure continues.

#### PERSONAL PROTECTIVE METHODS

Workers should be trained in personal hygiene and sanitation, the use of personal protective equipment, and early recognition of symptoms of absorption, skin contact irritation, and sensitivity. With the exception of arsine and arsenic trichloride, the compounds of arsenic do not have odor or warning qualities. In case of emergency or areas of high dust or spray mist, workers should wear respirators that are supplied-air or self-contained positive-pressure type with fullface mask. Where concentrations are less than 100 x standard, workers may be able to use halfmask respirators with replaceable dust or fume filters. Protective clothing, gloves and goggles, a hood for head and neck should be provided. When liquids are processed, impervious clothing should be supplied. Clean work clothes should be supplied daily and the workers should shower prior to changing to street clothes.

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## ARSINE

### DESCRIPTION

$\text{AsH}_3$ , arsine, is a colorless gas with a slight garlic-like odor which cannot be considered a suitable warning property in concentrations below 1 ppm. Arsine's solubility is 20 ml. in 100 ml. of water at 20 C.

### SYNONYMS

Hydrogen arsenide, arseniuretted hydrogen.

### POTENTIAL OCCUPATIONAL EXPOSURES

Arsine is not used in any industrial process but this gas is generated by side reactions or unexpectedly; e.g., it may be generated in metal pickling operations, metal dressing operations, or when inorganic arsenic compounds contact sources of nascent hydrogen. It has been known to occur as an impurity in acetylene. Most occupational exposure occurs in chemical, smelting, and refining industry. Cases of exposure have come from workers dealing with zinc, tin, cadmium, galvanized coated aluminum, and silicon steel metals.

A partial list of occupations in which exposure may occur includes:

Acid dippers	Jewelers
Aniline workers	Lead burners
Bronzers	Paper makers
Dye makers	Plumbers
Etchers	Solderers
Fertilizer makers	Submarine workers
Galvanizers	Tinners

### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for arsine is 0.05 ppm. NIOSH has recommended that arsine be controlled to the same concentration as other forms of inorganic arsenic (0.002 mg/m<sup>3</sup>).

### ROUTE OF ENTRY

Inhalation of gas.

### HARMFUL EFFECTS

#### *Local*—

High concentrations of arsine gas will cause damage to the eyes. Most experts agree, however, that before this occurs systemic effects can be expected.

#### *Systemic*—

Arsine is an extremely toxic gas that can be fatal if inhaled in sufficient quantities. Acute poisoning is marked by a triad of main effects caused by massive intravascular hemolysis of the circulating red cells. Early effects may occur within an hour or two and are commonly characterized by general malaise, apprehension, giddiness, headache, shivering, thirst, and abdominal pain with vomiting. In severe acute cases

the vomitus may be blood stained and diarrhea ensues as with inorganic arsenical poisoning. Pulmonary edema has occurred in severe acute poisoning.

Invariably, the first sign observed in arsine poisoning is hemoglobinuria, appearing with discoloration of the urine up to port wine hue (first of the triad). Jaundice (second of triad) sets in on the second or third day and may be intense, coloring the entire body surface a deep bronze hue. Coincident with these effects is a severe haemolytic-type anemia. Severe renal damage may occur with oliguria or complete suppression of urinary function (third of triad), leading to uremia and death. Severe hepatic damage may also occur, along with cardiac damage and EKG changes. Where death does not occur, recovery is prolonged.

In cases where the amount of inhaled arsine is insufficient to produce acute effects, or where small quantities are inhaled over prolonged periods, the hemoglobin liberated by the destruction of red cells may be degraded by the reticuloendothelial system and the iron moiety taken up by the liver, without producing permanent damage. Some hemoglobin may be excreted unchanged by the kidneys. The only symptoms noted may be general tiredness, pallor, breathlessness on exertion, and palpitations as would be expected with severe secondary anemia.

#### MEDICAL SURVEILLANCE

In preemployment physical examinations, special attention should be given to past or present kidney disease, liver disease, and anemia. Periodic physical examinations should include tests to determine arsenic levels in the blood and urine. The general condition of the blood and the renal and liver functions should also be evaluated. Since arsine gas is a by-product of certain production processes, workers should be trained to recognize the symptoms of exposure and to use appropriate personal protective equipment.

#### SPECIAL TESTS

None in common use.

#### PERSONAL PROTECTIVE METHODS

In most cases, arsine poisoning cannot be anticipated except through knowledge of the production processes. Where arsine is suspected in concentrations above the acceptable standard, the worker should be supplied with a supplied air fullface respirator or a self-contained positive pressure respirator with full facepiece.

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## ANTIMONY AND COMPOUNDS

### DESCRIPTION

Sb, antimony, is a silvery-white, soft metal insoluble in water and organic solvents. The ores most often found are stibnite, valentinite, kermesite, and senarmontite.

### SYNONYMS

None.

### POTENTIAL OCCUPATIONAL EXPOSURES

Exposure to antimony may occur during mining, smelting or refining, alloy and abrasive manufacture, and typesetting in printing. Antimony is widely used in the production of alloys, imparting increased hardness, mechanical strength, corrosion resistance, and a low coefficient of friction. Some of the important alloys are babbitt, pewter, white metal, Britannia metal and bearing metal (which are used in bearing shells), printing-type metal, storage battery plates, cable sheathing, solder, ornamental castings, and ammunition. Pure antimony compounds are used as abrasives, pigments, flameproofing compounds, plasticizers, and catalysts in organic synthesis; they are also used in the manufacture of tartar emetic, paints, lacquers, glass, pottery, enamels, glazes, pharmaceuticals, pyrotechnics, matches, explosives. In addition they are used in dyeing, for blueing steel, and in coloring aluminum, pewter, and zinc. A highly toxic gas, stibine, may be released from the metal under certain conditions.

A partial list of occupations in which exposure may occur includes:

Bronzers	Paint makers
Ceramic makers	Pewter workers
Drug makers	Rubber makers
Fireworks makers	Textile workers
Leather mordanters	Typesetters
Miners	

### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for antimony and its compounds is 0.5 mg/m<sup>3</sup>, expressed as Sb (see also Stibine).

### ROUTE OF ENTRY

Ingestion or inhalation of dust or fume; percutaneous absorption.

## HARMFUL EFFECTS

*Local—*

Antimony and its compounds are generally regarded as primary skin irritants. Lesions generally appear on exposed, moist areas of the body, but rarely on the face. The dust and fumes are also irritants to the eyes, nose, and throat, and may be associated with gingivitis, anemia, and ulceration of the nasal septum and larynx. Antimony trioxide causes a dermatitis known as "antimony spots." This form of dermatitis results in intense itching followed by skin eruptions. A diffuse erythema may occur, but usually the early lesions are small erythematous papules. They may enlarge, however, and become pustular. Lesions occur in hot weather and are due to dust accumulating on exposed areas that are moist due to sweating. No evidence of eczematous reaction is present, nor an allergic mechanism.

*Systemic—*

Systemic intoxication is uncommon from occupational exposure. However, miners of antimony may encounter dust containing free silica; cases of pneumoconiosis in miners have been termed "silico-antimoni-osis." Antimony pneumoconiosis, per se, appears to be a benign process.

Antimony metal dust and fumes are absorbed from the lungs into the blood stream. Principal organs attacked include certain enzyme systems (protein and carbohydrate metabolism), heart, lungs, and the mucous membrane of the respiratory tract. Symptoms of acute oral poisoning include violent irritation of the nose, mouth, stomach, and intestines, vomiting, bloody stools, slow shallow respiration, pulmonary congestion, coma, and sometimes death due to circulatory or respiratory failure. Chronic oral poisoning presents symptoms of dry throat, nausea, headache, sleeplessness, loss of appetite, and dizziness. Liver and kidney degenerative changes are late manifestations.

Antimony compounds are generally less toxic than antimony. Antimony trisulfide, however, has been reported to cause myocardial changes in man and experimental animals. Antimony trichloride and pentachloride are highly toxic and can irritate and corrode the skin. Antimony fluoride is extremely toxic, particularly to pulmonary tissue and skin.

## MEDICAL SURVEILLANCE

Preemployment and periodic examinations should give special attention to lung disease, skin disease, disease of the nervous system, heart and gastrointestinal tract. Lung function, EKG's, blood, and urine should be evaluated periodically.

## SPECIAL TESTS

Blood and urine antimony levels have been suggested, but are not in common use.

## PERSONAL PROTECTIVE METHODS

A combination of protective clothing, barrier creams, gloves, and personal hygiene will protect the skin. Washing and showering facilities should be available, and eating should not be permitted in exposed areas. Dust masks and supplied air respirators should be available in all areas where the Federal standard is exceeded.

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## BARIUM AND COMPOUNDS

### DESCRIPTION

Ba, barium, a silver white metal, is produced by reduction of barium oxide. The primary sources are the minerals barite ( $\text{BaSO}_4$ ) and witherite ( $\text{BaCO}_3$ ). Barium may ignite spontaneously in air in the presence of moisture, evolving hydrogen. Barium is insoluble in water but soluble in alcohol. Most of the barium compounds are soluble in water. The peroxide, nitrate, and chlorate are reactive and may present fire hazards in storage and use.

### SYNONYMS

None.

### POTENTIAL OCCUPATIONAL EXPOSURES

Metallic barium is used for removal of residual gas in vacuum tubes and in alloys with nickel, lead, calcium, magnesium, sodium, and lithium.

Barium compounds are used in the manufacture of lithopone (a white pigment in paints), chlorine, sodium hydroxide, valves, and green flares; in synthetic rubber vulcanization, X-ray diagnostic work, glass-making, papermaking, beet-sugar purification, animal, and vegetable oil refining. They are used in the brick and tile, pyrotechnics, and electronics industries. They are found in lubricants, pesticides, glazes, textile dyes and finishes, pharmaceuticals, and in cements which will be exposed to salt water; and barium is used as a rodenticide, a flux for magnesium alloys, a stabilizer and mold lubricant in the rubber and plastics industries, an extender in paints, a loader for paper, soap, rub-

ber, and linoleum, and as a fire extinguisher for uranium or plutonium fires.

A partial list of occupations in which exposure may occur includes:

Animal oil refiners	Paint makers
Brick makers	Plastic makers
Ceramic makers	Soap makers
Glass makers	Textile workers
Ink makers	Tile makers
Linoleum makers	Wax processors

#### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for soluble barium compounds is 0.5 mg/m<sup>3</sup>.

#### ROUTES OF ENTRY

Ingestion or inhalation of dust or fume.

#### HARMFUL EFFECTS

##### *Local—*

Alkaline barium compounds, such as the hydroxide and carbonate, may cause local irritation to the eyes, nose, throat, and skin.

##### *Systemic—*

Barium poisoning is virtually unknown in industry, although the potential exists when the soluble forms are used. When ingested or given orally, the soluble, ionized barium compounds exert a profound effect on all muscles and especially smooth muscle, markedly increasing their contractility. The heart rate is slowed and may stop in systole. Other effects are increased intestinal peristalsis, vascular constriction, bladder contraction, and increased voluntary muscle tension.

The inhalation of the dust of barium sulfate may lead to deposition in the lungs in sufficient quantities to produce "baritosis"—a benign pneumoconiosis. This produces a radiologic picture in the absence of symptoms and abnormal physical signs. X-rays, however, will show disseminated nodular opacities throughout the lung fields, which are discrete, but sometimes overlap.

#### MEDICAL SURVEILLANCE

Consideration should be given to the skin, eye, heart, and lung in any placement or periodic examination.

#### SPECIAL TESTS

None have been used.

#### PERSONAL PROTECTIVE METHODS

Employees should receive instruction in personal hygiene and the importance of not eating in work areas. Good housekeeping and adequate ventilation are essential. Dust masks, respirators, or goggles may be needed where amounts of significant soluble or alkaline forms are encountered, as well as protective clothing.

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*BERYLLIUM AND COMPOUNDS*

## DESCRIPTION

Be, beryllium, is a grey-metal which combines the properties of light weight and high tensile strength. Beryllium is slightly soluble in hot water and in dilute acids and alkalis. All beryllium compounds are soluble to some degree in water. Beryl ore is the primary source of beryllium, although there are numerous other sources.

## SYNONYMS

None

## POTENTIAL OCCUPATIONAL EXPOSURES

Beryllium metal is widely used in the atomic energy field as a moderator for fission reactions, as a reflector to reduce leakage of neutrons from the reactor core, and, in a mixture with uranium, as a neutron source. Beryllium foil is the window material for X-ray tubes. Beryllium may be alloyed with a number of metals to increase hardness. Beryllium-copper alloy is the most common and is used in parts subjected to abnormal wear, extreme vibration, or shock loading such as in bushings, current-carrying springs, electric contacts and switches, and radio and radar components; it is also used in non-sparking tools. Beryllium-nickel alloy has high tensile strength, increased hardness, and age-hardening characteristics which make it useful in diamond drill-bit matrices, watch-balance wheels, and certain airplane parts. Beryllium bronzes are used in non-spark tools, electrical switch parts, watch springs, diaphragms, shims, cams, and bushings. Other alloys may be formed with zinc, magnesium, iron, aluminum, gold, silver, platinum, nickel, and steel. Beryllium also has potential for use in the aircraft and aerospace industry.

Beryllium compounds are utilized in the manufacture of ceramics and refractories, as chemical reagents and gas mantle hardeners, and in atomic energy reactions. The use of phosphors produced from beryllium oxide in fluorescent lamps has been discontinued.

Hazardous exposure to beryllium is generally associated with the milling and use of beryllium and not the mining and handling of beryl ore.

A partial list of occupations in which exposure may occur includes:

Beryllium alloy workers	Gas mantle makers
Cathode ray tube makers	Missile technicians
Ceramic makers	Nuclear reactor workers
Electric equipment makers	Refractory material makers

## PERMISSIBLE EXPOSURE LIMITS

The present Federal standard for beryllium and beryllium compounds is  $2\mu\text{g}/\text{m}^3$  as an 8-hour TWA with an acceptable ceiling con-

centration of 5  $\mu\text{g}/\text{m}^3$ . The acceptable maximum peak is 25  $\mu\text{g}/\text{m}^3$  for a maximum duration of 30 minutes. The standard recommended in the NIOSH Criteria Document is 2  $\mu\text{g Be}/\text{m}^3$  as an 8-hour TWA with a peak value of 25  $\mu\text{g Be}/\text{m}^3$  as determined by a minimum sampling time of 30 minutes.

#### ROUTE OF ENTRY

Inhalation of fume or dust.

#### HARMFUL EFFECTS

##### *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 it 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 nonpro-

ductive 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.

#### MEDICAL SURVEILLANCE

Preemployment history and physical examinations for worker applicants should include chest X-rays, baseline pulmonary function tests (FVC and FEV<sub>1</sub>), and measurement of body weight. Beryllium workers should receive a periodic health evaluation that includes: spirometry (FVC and FEV<sub>1</sub>), medical history questionnaire directed toward respiratory symptoms, and a chest X-ray. General health, liver and kidney function, and possible effects on the skin should be evaluated.

#### SPECIAL TESTS

Beryllium can be determined in the urine, but shows poor correlation with quantitative exposures. Tissue biopsies for beryllium content have also been utilized in diagnostic procedures, but often show no relation to the severity of the disease and indicate only that exposure has occurred.

#### PERSONAL PROTECTIVE METHODS

Work areas should be monitored to limit and control levels of exposure. Personnel samplers are recommended. Good housekeeping, proper maintenance, and engineering control of processing equipment and technology are essential. The importance of safe work practices and personal hygiene should be stressed. When beryllium levels exceed the accepted standards, the workers should be provided with respiratory protective devices of the appropriate class, as determined on the basis of the actual or projected atmospheric concentration of airborne beryllium at the worksite. Protective clothing should be provided all workers

who are subject to exposure in excess of the standard. This should include shoes or protective shoe covers as well as other clothing. The clothing should be reissued clean on a daily basis. Workers should shower following each shift prior to change to street clothes.

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## BISMUTH AND COMPOUNDS

#### DESCRIPTION

Bi, bismuth, is a pinkish-silver, hard, brittle metal. It is found as the free metal in ores such as bismutite and bismuthinite and in lead ores. Bismuth is soluble in some mineral acids and insoluble in water. Most bismuth compounds are soluble in water.

#### SYNONYMS

None.

#### POTENTIAL OCCUPATIONAL EXPOSURES

Bismuth is used as a constituent of tempering baths for steel alloys, in lowmelting point alloys which expand on cooling, in aluminum and steel alloys to increase machinability, and in printing type metal. Bismuth compounds are found primarily in pharmaceuticals as antiseptics, antacids, antiluetics, and as a medicament in the treatment of acute angina. They are also used as a contrast medium in roentgenoscopy and in cosmetics.

A partial list of occupations in which exposure may occur includes:

Chemists	Permanent magnet makers
Cosmetic workers	Pigment makers
Disinfectant makers	Solder makers
Fuse makers	Steel alloy makers
Laboratory workers	Tin lusterers

#### PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for bismuth or its compounds.

#### ROUTE OF ENTRY

Ingestion of powder or inhalation of dust.

#### HARMFUL EFFECTS

##### *Local*—

Bismuth and bismuth compounds have little or no effect on intact skin and mucous membrane. Absorption occurs only minimally through broken skin.

##### *Systemic*—

There is no evidence connecting bismuth and bismuth compounds

with cases of industrial poisoning. All accounts of bismuth poisoning are from the soluble compounds used previously in therapeutics. Fatalities and near fatalities have been recorded chiefly as a result of intravenous or intramuscular injection of soluble salts. Principal organs affected by poisoning are the kidneys and liver. Chronic intoxication from repeated oral or parenteral doses causes "bismuth line." This is a gum condition with black spots of buccal and colonic mucosa, superficial stomatitis, foul breath, and salivation.

#### MEDICAL SURVEILLANCE

No special considerations are necessary other than following good general health practices. Liver and kidney function should be followed if large amounts of soluble salts are ingested.

#### SPECIAL TESTS

None have been proposed.

#### PERSONAL PROTECTIVE METHODS

Personal hygiene should be stressed, and eating should not be permitted in work areas. Dust masks should be worn in dusty areas to prevent inadvertent ingestion of the soluble bismuth compounds.

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## **BORON AND COMPOUNDS**

*(excluding the hydrides)*

#### DESCRIPTION

Boron, B, is a brownish-black powder and may be either crystalline or amorphous. It does not occur free in nature and is found in the minerals borax, colemanite, boronatrocalcite, and boracite. Boron is slightly soluble in water under certain conditions.

Boric acid,  $H_3BO_3$ , is a white, amorphous powder. Saturated solutions at 0 C contain 2.6% acid; at 100 C, 28% acid. Boric acid is soluble 1 gm/18 ml in cold water.

Borax,  $Na_2B_4O_7 \cdot 5H_2O$ , is a colorless, odorless crystalline solid. Borax is slightly soluble in water.

Boron trifluoride,  $BF_3$ , is a colorless gas with a pungent, suffocating odor. It decomposes in water, forming boric acid and fluoboric acid and hydrolyzes in air giving rise to dense, white fumes.

Boron oxide,  $B_2O_3$ , is a vitreous, colorless, crystalline, hygroscopic solid and slightly soluble in water.

#### SYNONYMS

B, none; boric acid, boracic acid; borax, tincal; boron trifluoride, boron fluoride; boron oxide, boric oxide.

## POTENTIAL OCCUPATIONAL EXPOSURES

Boron is used in metallurgy as a degasifying agent and is alloyed with aluminum, iron, and steel to increase hardness. It is also a neutron absorber in nuclear reactors.

Boric acid is a fireproofing agent for textiles, a weatherproofing agent for wood, a preservative, and an antiseptic. It is used in the manufacture of glass, pottery, enamels, glazes, cosmetics, cements, porcelain, borates, leather, carpets, hats, soaps, and artificial gems, and in tanning, printing, dyeing, painting, and photography. It is a constituent in powders, ointments, nickeling baths, electric condensers and is used for impregnating wicks and hardening steel.

Borax is used as a soldering flux, preservative against wood fungus, and as an antiseptic. It is used in the manufacture of enamels and glazes and in tanning, cleaning compounds, for fireproofing fabrics and wood, and in artificial aging of wood.

Boron trifluoride is used as a catalyst, a flux for soldering magnesium, a fumigant, for protecting molten magnesium and its alloys from oxidation and in ionization chambers to detect weak neutrons.

Boric acid is used in the manufacture of glass, enamels and glazes, in metallurgy, and in the analysis of silicates to determine  $\text{SiO}_2$  and alkalis.

A partial list of occupations in which exposure may occur includes:

Alloy makers	Nuclear instrument makers
Antiseptic makers	Organic chemical synthesizers
Enamel makers	Tannery workers
Fumigant workers	Textile fireproofers
Glass makers	Wood workers

## PERMISSIBLE EXPOSURE LIMITS

The applicable Federal standards are: Boron trifluoride 1 ppm ( $3 \text{ mg/m}^3$ ) as a ceiling value; and Boron oxide  $15 \text{ mg/m}^3$ .

## ROUTE OF ENTRY

Inhalation of dust, fumes, and aerosols; ingestion.

## HARMFUL EFFECTS

*Local—*

These boron compounds may produce irritation of the nasal mucous membranes, the respiratory tract, and eyes.

*Systemic—*

These effects vary greatly with the type of compound. Acute poisoning in man from boric acid or borax is usually the result of application of dressings, powders, or ointment to large areas of burned or abraded skin, or accidental ingestion. The signs are: nausea, abdominal pain, diarrhea and violent vomiting, sometimes bloody, which may be accompanied by headache and weakness. There is a characteristic erythematous rash followed by peeling. In severe cases, shock with fall in arterial pressure, tachycardia, and cyanosis occur. Marked CNS irrita-

tion, oliguria, and anuria may be present. The oral lethal dose in adults is over 30 grams. Little information is available on chronic oral poisoning, although it is reported to be characterized by mild GI irritation, loss of appetite, disturbed digestion, nausea, possibly vomiting, and erythematous rash. The rash may be "hard" with a tendency to become purpuric. Dryness of skin and mucous membranes, reddening of tongue, cracking of lips, loss of hair, conjunctivitis, palpebral edema, gastro-intestinal disturbances, and kidney injury have also been observed.

Although no occupational poisonings have been reported, it was noted that workers manufacturing boric acid had some atrophic changes in respiratory mucous membranes, weakness, joint pains, and other vague symptoms. The biochemical mechanism of boron toxicity is not clear but seems to involve action on the nervous system, enzyme activity, carbohydrate metabolism, hormone function, and oxidation processes, coupled with allergic effects. Borates are excreted principally by the kidneys.

The toxic action of the halogenated borons (boron trifluoride and trichloride) is considerably influenced by their halogenated decomposition products. They are primary irritants of the nasal passages, respiratory tract, and eyes in man. Animal experiments showed a fall in inorganic phosphorous level in blood and on autopsy, pneumonia, and degenerative changes in renal tubules. Long term exposure leads to irritation of the respiratory tract, dysproteinemia, reduction in cholinesterase activity, increased nervous system lability. High concentrations showed a reduction of acetyl carbonic acid and inorganic phosphorous in blood, and dental fluorosis.

Skin and respiratory tract irritation and central nervous system effects have been reported from animal experiments with amine and alkylboranes. The alkylboranes seem to be more toxic than the amino compounds and decaborane, but less toxic than pentaborane. No toxic effects have been attributed to elemental boron.

#### MEDICAL SURVEILLANCE

No specific considerations are needed for boric acid or borates except for general health and liver and kidney function. In the case of boron trifluoride, the skin, eyes, and respiratory tract should receive special attention. In the case of the boranes, central nervous system and lung function will also be of special concern.

#### SPECIAL TESTS

None in common use.

#### PERSONAL PROTECTIVE METHODS

Exposed workers should be educated in the proper use of protective equipment and there should be strict adherence to ventilating provisions in work areas. Workers involved with the manufacture of boric acid should be provided with masks to prevent inhalation of dust and fumes. Where exposure is to halogenated borons, or boranes, masks and supplied air respirators are necessary in areas of dust, gas, or fume

concentration. In some areas protective clothing, gloves, and goggles may be necessary.

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## BORON HYDRIDES

#### DESCRIPTION

**Diborane:**  $B_2H_6$ , boroethane, diboron hexahydride. Diborane is a colorless gas with a nauseating odor. It ignites spontaneously in moist air, and on contact with water, hydrolyzes exothermically forming hydrogen and boric acid.

**Pentaborane:**  $B_5H_9$ , pentaboron monohydride. Pentaborane is a colorless, volatile liquid with an unpleasant, sweetish odor. It ignites spontaneously in air, decomposes at 150 C and hydrolyzes in water.

**Decaborane:**  $B_{10}H_{14}$ , decaboron tetradecahydride. This is a white crystal with a bitter odor. It hydrolyzes very slowly in water.

#### SYNONYMS

Boranes, hydrogen borides.

#### POTENTIAL OCCUPATIONAL EXPOSURES

Diborane is used as a catalyst for olefin polymerization, a rubber vulcanizer, a reducing agent, a flame-speed accelerator, a chemical intermediate for other boron hydrides, and as a doping agent; and in rocket propellants and in the conversion of olefins to trialkyl boranes and primary alcohols.

Pentaborane is used in rocket propellants and in gasoline additives.

Decaborane is used as a catalyst in olefin polymerization, in rocket propellants, in gasoline additives, and as a vulcanizing agent for rubber.

A partial list of occupations in which exposure may occur includes:

Dope makers	Plastic makers
Gasoline additive makers	Rocket fuel makers
Gasoline makers	Rubber makers
Organic chemical synthesizers	

#### PERMISSIBLE EXPOSURE LIMITS

The applicable Federal standards are: Diborane 0.1 ppm (0.1 mg/m<sup>3</sup>); Pentaborane 0.005 ppm (0.01 mg/m<sup>3</sup>); Decaborane 0.05 (0.03 mg/m<sup>3</sup>) skin.

#### ROUTES OF ENTRY

Inhalation and percutaneous absorption.

#### HARMFUL EFFECTS

##### *Local—*

Vapors of boron hydrides are irritating to skin and mucous mem-

branes. Pentaborane and decaborane show marked irritation of skin and mucous membranes, necrotic changes, serious kerato-conjunctivitis with ulceration, and corneal opacification.

#### *Systemic—*

Pentaborane is the most toxic of boron hydrides. Intoxication is characterized predominantly by CNS signs and symptoms. Hyperexcitability, headaches, muscle twitching, convulsions, dizziness, disorientation, and unconsciousness may occur early or delayed for 24 hours or more following excessive exposure. Slight intoxication results in nausea and drowsiness. Moderate intoxication leads to headache, dizziness, nervous excitation, and hiccups. There may be muscular pains and cramps, spasms in face and extremities, behavioral changes, loss of mental concentration, incoordination, disorientation, cramps, convulsions, semi-coma, and persistent leukocytosis after 40-48 hours. Liver function tests and elevated nonprotein nitrogen and blood urea levels suggest liver and kidney damage.

Decaborane's toxic effects are similar to pentaborane. Symptoms of CNS damage predominate; however, they are not as marked as the pentaborane.

Diborane is the least toxic of the boron hydrides. In acute poisoning, the symptoms are similar to metal fume fever: tightness, heaviness and burning in chest, coughing, shortness of breath, chills, fever, pericardial pain, nausea, shivering, and drowsiness. Signs appear soon after exposure or after a latent period of up to 24 hours and persist for 1-3 days or more. Pneumonia may develop later. Reversible liver and kidney changes were seen in rats exposed to very high gas levels. This has not been noted in man. Subacute poisoning is characterized by pulmonary irritation symptoms, and if this is prolonged, CNS symptoms such as headaches, dizziness, vertigo, chills, fatigue, muscular weakness, and only infrequent transient tremors, appear. Convulsions do not occur. Chronic exposure leads to wheezing, dyspnea, tightness, dry cough, rales, and hyperventilation which persist for several years.

#### MEDICAL SURVEILLANCE

Preemployment and periodic physical examinations to determine the status of the workers' general health should be performed. These examinations should be concerned especially with any history of central nervous system disease, personality or behavioral changes, as well as liver, kidney, or pulmonary disease of any significant nature. Chest X-rays and blood, liver, and renal function studies may be helpful.

#### SPECIAL TESTS

None in common use.

#### PERSONAL PROTECTIVE METHODS

Constant vigilance in the storage and handling of boron hydrides is required. Continuing worker education in the use of personal protective

equipment is necessary even when maximum engineering safety measures are applied.

Adequate sanitation facilities including showers and facilities for eating away from exposure area should be provided. Workers should wash thoroughly when leaving exposure areas. Protective clothing impervious to the liquid and gas compounds are necessary. When skin is contaminated by splash or spill, immediate clothes change with thorough washing of the skin area is necessary. Showering after the shift and before changing to street clothes should be required. Masks, either dust, vapor or supplied air type depending on the compound being used in the work place, should be used by all exposed personnel and should be fullface type.

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## BRASS

#### DESCRIPTION

Brass is a term used for alloys of copper and zinc. The ratio of the two compounds is generally 2 to 1, although different types of brass may have different proportions. Brass may contain significant quantities of lead. Bronze is also a copper alloy, usually with tin; however, the term bronze is applied to many other copper alloys, some of which contain large amounts of zinc.

#### SYNONYMS

None.

#### POTENTIAL OCCUPATIONAL EXPOSURES

Brass may be cast into bearings and other wearing surfaces, steam and water valves and fittings, electrical fittings, hardware, ornamental castings, and other equipment where special corrosion-resistance properties, pressure tightness, and good machinability are required. Wrought forms of brass such as sheets, plates, bars, shapes, wire, and tubing are also widely used.

A partial list of occupations in which exposure may occur includes:

Bench molders	Junk metal refiners
Braziers	Welders
Bronzers	Zinc founders
Core makers	Zinc smelters
Galvanizers	

**PERMISSIBLE EXPOSURE LIMITS**

There is no Federal standard for brass; however, there are standards for its constituents: Lead (inorganic) ( $0.2 \text{ mg/m}^3$ ); Zinc Oxide fume ( $5 \text{ mg/m}^3$ ); Copper fume ( $0.1 \text{ mg/m}^3$ ).

**ROUTE OF ENTRY**

Inhalation of fume.

**HARMFUL EFFECTS***Local—*

Brass dust and slivers may cause dermatitis by mechanical irritation.

*Systemic—*

Since zinc boils at a lower temperature than copper, the fusing of brass is attended by liberation of considerable quantities of zinc oxide. Inhalation of zinc oxide fumes may result in production of signs and symptoms of metal fume fever (see Zinc Oxide). Brass founder's ague is the name often given to metal fume fever occurring in brass-founding industry.

Brass foundings may also release sufficient amounts of lead fume to produce lead intoxication (see Lead-Inorganic).

**MEDICAL SURVEILLANCE**

See Zinc Oxide and/or Lead-Inorganic.

**SPECIAL TESTS**

Blood lead values may be useful if lead fume or dust exposure is suspected. (See Lead.)

**PERSONAL PROTECTIVE METHODS**

See Zinc Oxide and/or Lead-Inorganic.

**CADMIUM AND COMPOUNDS****DESCRIPTION**

Cd, cadmium, is a bluish-white metal. The only cadmium mineral, greenockite, is rare; however, small amounts of cadmium are found in zinc, copper, and lead ores. It is generally produced as a by-product of these metals, particularly zinc. Cadmium is insoluble in water but is soluble in acids.

**SYNONYMS**

None.

**POTENTIAL OCCUPATIONAL EXPOSURES**

Cadmium is highly corrosion resistant and is used as a protective coating for iron, steel, and copper; it is generally applied by electro-

plating, but hot dipping and spraying are possible. Cadmium may be alloyed with copper, nickel, gold, silver, bismuth, and aluminum to form easily fusible compounds. These alloys may be used as coatings for other materials, welding electrodes, solders, etc. It is also utilized in electrodes of alkaline storage batteries, as a neutron absorber in nuclear reactors, a stabilizer for polyvinyl chloride plastics, a deoxidizer in nickel plating, an amalgam in dentistry, in the manufacture of fluorescent lamps, semiconductors, photocells, and jewelry, in process engraving, in the automobile and aircraft industries, and to charge Jones reducers.

Various cadmium compounds find use as fungicides, insecticides, nematocides, polymerization catalysts, pigments, paints, and glass; they are used in the photographic industry and in glazes. Cadmium is also a contaminant of superphosphate fertilizers.

Exposure may occur during the smelting and refining of cadmium-containing zinc, lead, and copper ores, and during spraying, welding, cutting, brazing, soldering, heat treating, melting, alloying and salvage operations which require burning of cadmium-containing materials.

A partial list of occupations in which exposure may occur includes:

Alloy makers	Pesticide workers
Battery makers	Solder workers
Dental amalgam makers	Textile printers
Engravers	Welders
Metalizers	Zinc refiners
Paint makers	

#### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for cadmium fume is 0.1 mg/m<sup>3</sup> (as Cd) as an 8-hour TWA with an acceptable ceiling of 3 mg/m<sup>3</sup>. For cadmium dust, the standard is 0.2 mg/m<sup>3</sup> (Cd) as an 8-hour TWA with an acceptable maximum ceiling of 0.6 mg/m<sup>3</sup>. NIOSH has recommended a TWA limit of 40 µg/m<sup>3</sup> with a ceiling limit of 200 µg in a 5-minute sampling period.

#### ROUTES OF ENTRY

Inhalation or ingestion of fumes or dust.

#### HARMFUL EFFECTS

##### *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 Cd has a very long 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 Cd 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 adsorption 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.

#### MEDICAL SURVEILLANCE

In preemployment physical examinations, emphasis should be given to a history of or the actual presence of significant kidney disease, smoking history, and respiratory disease. A chest X-ray and baseline pulmonary function study is recommended. Periodic examinations should emphasize the respiratory system, including pulmonary function tests, kidneys, and blood.

#### SPECIAL TESTS

A low molecular weight proteinuria may be the earliest indication of renal toxicity. The trichloroacetic acid test may pick this up, but

more specific quantitative studies would be preferable. If renal disease due to cadmium is present, there may also be increased excretion of calcium, amino acids, glucose, and phosphates.

#### PERSONAL PROTECTIVE METHODS

Most important is the requirement that each worker be adequately protected by the use of effective respiratory protection: either by dust masks, vapor canister respirators, or supplied air respirators. Clothing should be changed after each shift and clean work clothing issued each day. Food should not be eaten in contaminated work areas. Workers should shower after each shift before changing to street clothes.

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## CARBONYLS

#### DESCRIPTION

Metal carbonyls have the general formula  $Me_x(CO)_y$  in which Me is the metal and x and y are whole numbers. They are generally produced by direct reaction between carbon monoxide and the finely divided metal; however, chromium, molybdenum, and tungsten carbonyls can be produced by the Grignard method, and platinum metals, iron and rhenium carbonyls may be obtained from metal sulfides, halides, or oxides. The carbonyls react with oxidizing agents and may ignite spontaneously. Reaction with water or steam results in the liberation of carbon monoxide; and on heating, the carbonyls decompose forming carbon monoxide and the finely divided metal powder which may ignite. Some of the more important carbonyls are:

Chromium carbonyl:  $Cr(CO)_6$ . Colorless crystals.

Cobalt tricarbonyl:  $(Co(CO)_3)_4$ . Black crystal.

Cobalt tetracarbonyl:  $(Co(CO)_4)_2$ . Orange crystals or dark brown microscopic crystals.

Cobalt carbonyl hydride:  $\text{HCo}(\text{CO})_4$ . Below  $-26.2\text{ C}$ , exists as light yellow solid. At room temperature, a gas. It begins to decompose in air above  $-26\text{ C}$ .

Cobalt nitrosocarbonyl:  $\text{Co}(\text{CO})_3(\text{NO})$ . Cherry red liquid.

Iron tetracarbonyl:  $(\text{Fe}(\text{CO})_4)_3$ . Dark green lustrous crystals.

Iron pentacarbonyl:  $\text{Fe}(\text{CO})_5$ . Viscous yellow liquid.

Iron nonacarbonyl:  $\text{Fe}_2(\text{CO})_9$ . Yellow to orange crystals.

Iron carbonyl hydride:  $\text{H}_2\text{Fe}(\text{CO})_4$ . A gas. Begins to decompose at  $-10\text{ C}$ .

Iron nitrosyl carbonyl:  $\text{Fe}(\text{NO})_2(\text{CO})_2$ . Dark red crystals.

Molybdenum hexacarbonyl:  $\text{Mo}(\text{CO})_6$ . White crystals.

Nickel carbonyl:  $\text{Ni}(\text{CO})_4$ . Colorless liquid.

Osmium carbonyl chloride:  $\text{Os}(\text{CO})_2\text{Cl}_3$ . Dark brown. Deliquescent.

Ruthenium pentacarbonyl:  $\text{Ru}(\text{CO})_5$ . Colorless liquid. Very volatile.

Tungsten carbonyl:  $\text{W}(\text{CO})_6$ . Colorless crystals.

#### SYNONYMS

Chromium carbonyl: None.

Cobalt tricarbonyl: Tetracobalt dodecacarbonyl.

Cobalt tetracarbonyl: Dicobalt octacarbonyl.

Cobalt carbonyl hydride: Cobalt tetracarbonyl hydride.

Cobalt nitrosocarbonyl: None.

Iron tetracarbonyl: None.

Iron pentacarbonyl: None.

Iron nonacarbonyl: Enneacarbonyl.

Iron carbonyl hydride: None.

Iron nitrosyl carbonyl: None.

Molybdenum hexacarbonyl: Molybdenum carbonyl.

Nickel carbonyl: Nickel tetracarbonyl.

Osmium carbonyl chloride: None.

Ruthenium pentacarbonyl: None.

Tungsten carbonyl: None.

#### POTENTIAL OCCUPATIONAL EXPOSURES

Metal carbonyls are used in isolating certain metals from complex ores, in the preparation of high purity metals, for the production of carbon steel and metallizing, and as catalysts in organic synthesis. Pure metal powders from carbonyls are used in the electronics industry for radiofrequency transformers.  $\text{Fe}(\text{CO})_5$  is used as a gasoline additive in Europe and as an antidetonator.

Metal carbonyls may be formed during other processes: in the Bessemer converter in the steel industry; inadvertent introduction of carbon monoxide onto metal catalyst beds; storage of carbon monoxide in steel cylinders producing  $\text{Fe}(\text{CO})_5$ ; slowly flowing water or gas in an iron pipe generating  $\text{Fe}(\text{CO})_5$ ; and in the Fischer-Tropsch process for the liquefaction of coal.

A partial list of occupations in which exposure may occur includes:

Acetylene welders	Nickel refiners
Blast furnace workers	Organic chemical synthesizers
Metal refiners	Petroleum refinery workers
Mond process workers	

#### PERMISSIBLE EXPOSURE LIMITS

There are no specific standards for the metal carbonyls, other than nickel carbonyl. (See under Nickel Carbonyl this section.)

#### ROUTES OF ENTRY

Inhalation of vapor or dust. Percutaneous absorption of liquids may occur.

#### HARMFUL EFFECTS

##### *Local—*

Aside from skin irritation caused by the specific metal liberated when the metal carbonyl decomposes, no local effects have been reported.

##### *Systemic—*

Metal carbonyls as a group have somewhat similar toxicological effects, although there are differences in degrees of toxicity which range from moderate to extremely mild. Nickel carbonyl is the best known and is highly toxic, capable of causing pulmonary edema. Exposures during the Mond process have been associated with an increased incidence of lung and nasal sinuses cancer. Cancer has been produced in rats in the lung, liver, and kidneys.

The toxicity of carbonyls depends in part on the toxic character of the metal component and in part on the volatility and stability of the carbonyl itself.  $\text{Ni}(\text{CO})_4$  has a very high vapor pressure, plus stability at room temperature. There are no reports of human injury following exposure to cobalt carbonyls. Cobalt tetracarbonyl has an odor so offensive at low levels of concentration that it provides an effective warning against toxic exposure. Iron pentacarbonyl may cause similar pulmonary symptoms to those of nickel carbonyl. Animal studies indicate that the inhalation of fumes and dusts of carbonyls causes respiratory irritation and disturbances to the central nervous system.

#### MEDICAL SURVEILLANCE

(See Nickel Carbonyl.) Preemployment physical examinations should give particular attention to the respiratory tract and skin. Periodic examinations should include the respiratory tract and nasal sinuses, smoking history as well as general health. A baseline chest X-ray should be available and pulmonary function followed.

#### SPECIAL TESTS

Urinary nickel level determinations for a few days after an acute

exposure may be useful. Little information is available as to the value of biochemical studies in the case of the other carbonyls.

#### PERSONAL PROTECTIVE METHODS

In areas where either dust or vapors of the metal carbonyls are encountered, the worker should wear appropriate supplied air respirators. Where the danger of splash or spill of liquids exists, impervious protective clothing should be used.

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## CERIUM AND COMPOUNDS

#### DESCRIPTION

Ce, cerium, a soft, steel-gray metal, is found in the minerals monazite, cerite, and orthite. It may form either tri- or tetravalent compounds. The cerious salts are usually white and the ceric salts are yellow to orange-red. Cerium decomposes in water and is soluble in dilute mineral acids.

#### SYNONYMS

None.

#### POTENTIAL OCCUPATIONAL EXPOSURES

Cerium and its compounds are used as a catalyst in ammonia synthesis, a deoxidizer to improve the mechanical quality and refine grain size of steel, an opacifier in certain enamels, an arc-stabilizer in carbon arc lamps, an abrasive for polishing mirrors and lenses, a sedative and as a medicinal agent for vomiting during pregnancy. It is used in the manufacture of topaz yellow glass, spheroidal cast iron, incandescent gas mantles and in decolorizing glass, to prevent mildew in textiles, and to produce a vacuum in neon lamps and electronic tubes. Alloyed with aluminum, magnesium, and manganese, it increases resistance to creep and fatigue. Ferro-cerium is the pyrophoric alloy in gas cigarette lighters, and an alloy of magnesium, cerium, and zirconium is utilized for jet engine parts.

A partial list of occupations in which exposure may occur includes:

Alloy makers	Lighter flint makers
Ammonia makers	Metal refiners
Enamel makers	Phosphor makers
Glass (vitreous) makers	Rocket fuel makers
Ink makers	Textile workers

#### PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for cerium or its compounds.

## ROUTE OF ENTRY

Inhalation of dust.

## HARMFUL EFFECTS

*Local—*

No local effects have been reported due to cerium and its compounds.

*Systemic—*

There are no records of injury to human beings from either the industrial or medicinal use of cerium. The main risk to workers is from dust in mining and production areas. Recent reports in the literature describe "Cer-pneumoconiosis," a condition found in a group of graphic arts workers who use carbon arc lights in their work. Chest X-rays reveal small, miliary, homogeneously distributed infiltrates. Cer-pneumoconiosis cannot be considered a dust disease of the lung similar to silicosis. In the later stages of the reaction to the dust of carbon arc lamps, perifocal emphysema, and slight fibrosis of lungs are noted. It has been speculated that these changes may have been due to inhalation of substances containing radioactive elements of the thorium chain. To date, these views have not been confirmed by animal experimentation, autopsy, or human biopsy. Animal experimentation has demonstrated increased coagulation time from organic preparations of cerium, disturbance of lipid metabolism from cerium and its nitrates, and profound effects on metabolism and intestinal muscle causing loss of motility from cerium chloride.

## MEDICAL SURVEILLANCE

Chest X-rays should be taken as a part of preemployment and periodic physical examinations.

## SPECIAL TESTS

None in common use.

## PERSONAL PROTECTIVE METHODS

In areas of carbon arc lights, workers should wear effective dust filters or respirators. In mining and production areas, workers should wear effective dust filters or respirators suitable for the particulate size of air borne dust.

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**CHROMIUM AND ITS COMPOUNDS**

## DESCRIPTION

This group includes chromium trioxide ( $\text{CrO}_3$ ), chromium (VI) oxide, chromic acid anhydride and its aqueous solutions. Chromium may exist in one of three valence states in compounds, +2, +3, and +6. Chromic acid, along with chromates, is in the hexavalent form.

Chromium trioxide is produced from chromite ore by roasting with alkali or lime, (calcium oxide) leaching, crystallization of the soluble chromate or dichromate followed by reaction with sulfuric acid. Chromic acid anhydride mixed with water gives chromic acid and dichromic acid.

#### SYNONYMS

None.

#### POTENTIAL OCCUPATIONAL EXPOSURES

Chromium trioxide is used in chrome plating, copper stripping, aluminum anodizing, as a catalyst, in refractories, in organic synthesis, and photography.

A partial list of occupations in which exposure may occur includes:

Anodizers	Photoengravers
Copper etchers	Photographers
Electroplaters	Process engravers
Glass workers	Stainless steel workers
Lithographers	Textile workers
Metal workers	Welders
Oil purifiers	

#### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for chromic acid and chromates is 0.1 mg/m<sup>3</sup> as a ceiling concentration. The NIOSH Criteria for a Recommended Standard would set work place limits for chromic acid of 0.05 mg/m<sup>3</sup> as chromium trioxide as a TWA with a ceiling concentration of 0.1 mg/m<sup>3</sup> as chromium trioxide determined by a sampling time of 15 minutes.

#### ROUTES OF ENTRY

Percutaneous absorption, inhalation, and ingestion.

#### HARMFUL EFFECTS

##### *Local—*

In some workers, chromium compounds act as allergens which cause dermatitis to exposed skin. They may also produce pulmonary sensitization. Chromic acid has a direct corrosive effect on the skin and the mucous membranes of the upper respiratory tract; and although rare, the possibility of skin and pulmonary sensitization should be considered.

##### *Systemic—*

Chromium compounds in the +3 state are of a low order of toxicity. In the +6 state, chromium compounds are irritants and corrosive, which can enter the body by ingestion, inhalation, and through the skin. Typical industrial hazards are: inhalation of the dust and fumes released during the manufacture of dichromate from chromite ore; inhalation of chromic acid mist during the electroplating and surface treatment of metals; and skin contact in various manufacturing processes.

Acute exposures to dust or mist may cause coughing and wheezing, headache, dyspnea, pain on deep inspiration, fever, and loss of weight.

Tracheobronchial irritation and edema persist after other symptoms subside. In electroplating operations, workers may experience a variety of symptoms including lacrimation, inflammation of the conjunctiva, nasal itch and soreness, epistaxis, ulceration and perforation of the nasal septum, congested nasal mucosa and turbinates, chronic asthmatic bronchitis, dermatitis and ulceration of the skin, inflammation of laryngeal mucosa, cutaneous discoloration, and dental erosion. Hepatic injury has been reported from exposure to chromic acid used in plating baths, but appears to be rare.

Working in the chromate-producing industry increases the risk of lung cancer.

#### MEDICAL SURVEILLANCE

Preemployment physical examinations should include: a work history to determine past exposure to chromic acid and hexavalent chromium compounds, exposure to other carcinogens, smoking history, history of skin or pulmonary sensitization to chromium, history or presence of dermatitis, skin ulcers, or lesions of the nasal mucosa and/or perforation of the septum, and a chest X-ray. On periodic examinations an evaluation should be made of skin and respiratory complaints, especially in workers who demonstrate allergic reactions. Chest X-rays should be taken yearly for workers over age 40, and every five years for younger workers. Blood, liver, and kidney function should be evaluated periodically.

#### SPECIAL TESTS

Urinary chromate values have been studied in relation to exposure, but their value is questionable.

#### PERSONAL PROTECTIVE METHODS

Full body protective clothing should be worn in areas of chromic acid exposure, and impervious gloves, aprons, and footwear should be worn in areas where spills or splashes may contact the skin. Where chromic acid may contact the eyes by spills or splashes, impervious protective goggles or face shield should be worn. All clothing should be changed at the end of the shift and showering encouraged prior to change to street clothes. Clean clothes should be reissued at the start of the shift. Respirators should be used in areas where dust, fumes, or mist exposure exceeds Federal standards or where brief concentrations exceed the TWA, and for emergencies. Dust fumes and mist filter type respirators or supplied air respirators should be supplied all workers exposed, depending on concentration of exposure.

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## COBALT AND COMPOUNDS

### DESCRIPTION

Co, cobalt, is a silver-grey, hard, brittle, magnetic metal. It is relatively rare; the important mineral sources are the arsenides, sulfides, and oxidized forms. It is generally obtained as a by-product of other metals, particularly copper. Cobalt is insoluble in water, but soluble in acids.

### SYNONYMS

None.

### POTENTIAL OCCUPATIONAL EXPOSURES

Nickel-aluminum-cobalt alloys are used for permanent magnets. Alloys with nickel, aluminum, copper, beryllium, chromium, and molybdenum are used in the electrical, automobile, and aircraft industries. Cobalt is added to tool steels to improve their cutting qualities and is used as a binder in the manufacture of tungsten carbide tools.

Various cobalt compounds are used as pigments in enamels, glazes, and paints, as catalysts in afterburners, and in the glass, pottery, photographic, electroplating industries.

Radioactive cobalt ( $^{60}\text{Co}$ ) is used in the treatment of cancer.

A partial list of occupations in which exposure may occur includes:

Alloy makers	Nickel workers
Catalyst workers	Paint dryer makers
Ceramic workers	Porcelain colorers
Drug makers	Rubber colorers
Electroplaters	Synthetic ink makers
Glass colorers	

### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for cobalt, metal fume and dust, is 0.1 mg/m<sup>3</sup>.

### ROUTE OF ENTRY

Inhalation of dust or fume.

### HARMFUL EFFECTS

#### *Local—*

Cobalt dust is mildly irritating to the eyes and to a lesser extent to the skin. It is an allergen and has caused allergic sensitivity type dermatitis in some industries where only minute quantities of cobalt are used. The eruptions appear in the flexure creases of the elbow, knee, ankles, and neck. Cross sensitization occurs between cobalt and nickel, and to chromium when cobalt and chromium are combined.

*Systemic—*

Inhalation of cobalt dust may cause an asthma-like disease with cough and dyspnea. This situation may progress to interstitial pneumonia with marked fibrosis. Pneumoconiosis may develop which is believed to be reversible. Since cobalt dust is usually combined with other dusts, the role cobalt plays in causing the pneumoconiosis is not entirely clear.

Ingestion of cobalt or cobalt compounds is rare in industry. Vomiting, diarrhea, and a sensation of hotness may occur after ingestion or after the inhalation of excessive amounts of cobalt dust. Cardiomyopathy has also been reported, but the role of cobalt remains unclear in this situation.

**MEDICAL SURVEILLANCE**

In preemployment examinations, special attention should be given to a history of skin diseases, allergic dermatitis, baseline allergic respiratory diseases, and smoking history. A baseline chest X-ray should be taken. Periodic examinations should be directed toward skin and respiratory symptoms and lung function.

**SPECIAL TESTS**

None are in common use.

**PERSONAL PROTECTIVE METHODS**

Where dust levels are excessive, dust respirators should be used by all workers. Protective clothing should be issued to all workers and changed on a daily basis. Showering after each shift is encouraged prior to change to street clothes. Gloves and barrier creams may be helpful in preventing dermatitis.

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**COPPER AND COMPOUNDS****DESCRIPTION**

Cu, copper, is a reddish-brown metal which occurs free or in ores such as malachite, cuprite, and chalcopyrite. It may form both mono- and divalent compounds. Copper is insoluble in water, but soluble in nitric acid and hot sulfuric acid.

**SYNONYMS**

None.

**POTENTIAL OCCUPATIONAL EXPOSURES**

Metallic copper is an excellent conductor of electricity and is widely

used in the electrical industry in all gauges of wire for circuitry, coil, and armature windings, high conductivity tubes, commutator bars, etc. It is made into castings, sheets, rods, tubing, and wire, and is used in water and gas piping, roofing materials, cooking utensils, chemical and pharmaceutical equipment, and coinage. Copper forms many important alloys: Be-Ce alloy, brass, bronze, gun metal, bell metal, German silver, aluminum bronze, silicon bronze, phosphor bronze, and manganese bronze.

Copper compounds are used as insecticides, algicides, molluscicides, plant fungicides, mordants, pigments, catalysts, and as a copper supplement for pastures, and in the manufacture of powdered bronze paint and percussion caps. They are also utilized in analytical reagents, in paints for ships' bottoms, in electroplating, and in the solvent for cellulose in rayon manufacture.

A partial list of occupations in which exposure may occur includes:

Asphalt makers	Pigment makers
Battery makers	Rayon makers
Electroplaters	Solderers
Fungicide workers	Wallpaper makers
Gem colorers	Water treaters
Lithographers	Wood preservative workers

#### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for copper fume is  $0.1 \text{ mg/m}^3$ , and for copper dusts and mists,  $1 \text{ mg/m}^3$ .

#### ROUTE OF ENTRY

Inhalation of dust or fume.

#### HARMFUL EFFECTS

##### *Local—*

Copper salts act as irritants to the intact skin causing itching, erythema, and dermatitis. In the eyes, copper salts may cause conjunctivitis and even ulceration and turbidity of the cornea. Metallic copper may cause keratinization of the hands and soles of the feet, but it is not commonly associated with industrial dermatitis.

##### *Systemic—*

Industrial exposure to copper occurs chiefly from fumes generated in welding copper-containing metals. (See Brass.) The fumes and dust cause irritation of the upper respiratory tract, metallic taste in the mouth, nausea, metal fume fever, and in some instances discoloration of the skin and hair. Inhalation of dusts, fumes, and mists of copper salts may cause congestion of the nasal mucous membranes, sometimes of the pharynx, and on occasions, ulceration with perforation of the nasal septum. If the salts reach the gastrointestinal tract, they act as irritants, producing salivation, nausea, vomiting, gastric pain, hemorrhagic gastritis, and diarrhea. It is unlikely that poisoning by ingestion in industry would

progress to a serious point as small amounts induce vomiting and empty the stomach of copper salts.

Chronic human intoxication occurs rarely and then only in individuals with Wilson's disease (hepatolenticular degeneration). This is a genetic condition caused by the pairing of abnormal autosomal recessive genes in which there is abnormally high absorption, retention, and storage of copper by the body. The disease is progressive and fatal if untreated.

#### MEDICAL SURVEILLANCE

Consider the skin, eyes, and respiratory system in any placement or periodic examinations.

#### PERSONAL PROTECTIVE METHODS

In areas where copper dust or fume is excessive, workers should be provided with proper dust or fume filters or supplied air respirator with full facepiece.

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## GERMANIUM

#### DESCRIPTION

Ge, germanium, is a greyish-white, lustrous, brittle metalloid. It is never found free and occurs most commonly in argyrodite and germanite. It is generally produced from germanium containing minerals or as a by-product in zinc production or coal processing. Germanium is insoluble in water.

#### SYNONYMS

None.

#### POTENTIAL OCCUPATIONAL EXPOSURES

Because of its semiconductor properties, germanium is widely used in the electronic industry in rectifiers, diodes, and transistors. It is alloyed with aluminum, aluminum-magnesium, antimony, bronze, and tin to increase strength, hardness, or corrosion resistance. In the process of alloying germanium and arsenic, arsine may be released; stibine is released from the alloying of germanium and antimony. Germanium is also used in the manufacture of optical glass, lenses for infrared applications, red-fluorescing phosphors, and cathodes for electronic valves, and in electroplating, in the hydrogenation of coal, and as a catalyst, particularly at low temperatures. Certain compounds are used medically.

Industrial exposures to the dust and fumes of the metal or oxide generally occur during separation and purification of germanium, weld-

ing, multiple-zone melting operations, or cutting and grinding of crystals. Germanium tetrahydride (germanium hydride, Germane, monogermane) and other hydrides are produced by the action of a reducing acid on a germanium alloy.

A partial list of occupations in which exposure may occur includes:

Alloy makers	Rectifier makers
Dental alloy makers	Semiconductor makers
Electroplaters	Transistor makers
Glass makers	Vacuum tube makers
Phosphor makers	Residue workers

#### PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for germanium or its compounds; however, the ACGIH recently added a TLV for germanium tetrahydride of 0.2 ppm (0.6 mg/m<sup>3</sup>).

#### ROUTE OF ENTRY

Inhalation of gas, vapor, fume, or dust.

#### HARMFUL EFFECTS

##### *Local—*

The dust of germanium dioxide is irritating to the eyes. Germanium tetrachloride causes irritation of the skin.

##### *Systemic—*

Germanium tetrachloride is an upper respiratory irritant and may cause bronchitis and pneumonitis. Prolonged exposure to high level concentrations may result in damage to the liver, kidney, and other organs. Germanium tetrahydride is a toxic hemolytic gas capable of producing kidney damage.

#### MEDICAL SURVEILLANCE

Consider respiratory, liver, and kidney disease in any placement or periodic examinations.

#### SPECIAL TESTS

None commonly used, but can be determined in urine.

#### PERSONAL PROTECTIVE METHODS

In dust areas, protective clothing and gloves may be necessary to protect the skin, and goggles to protect the eyes. In areas where germanium tetrachloride is in high concentrations, dust-fume masks or supplied air respirators with full facepiece should be supplied to all workers. Personal hygiene is to be encouraged, with change of clothes following each shift and showering prior to change to street clothes.

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## IRON COMPOUNDS

### DESCRIPTION

Fe, iron, is a malleable, silver-grey metal. Ferric oxide is a dense, dark red powder or lumps. Hematite is the most important iron ore and is generally found as red hematite (red iron ore, mainly  $\text{Fe}_2\text{O}_3$ ) and brown hematite (brown iron ore, mainly limonite, a hydrated sesquioxide of iron). Magnetic iron oxide,  $\text{Fe}_3\text{O}_4$  is black. Iron is insoluble in water. Iron oxide is soluble in hydrochloric acid.

### SYNONYMS

None.

### POTENTIAL OCCUPATIONAL EXPOSURES

Iron is alloyed with carbon to produce steel. The addition of other elements (e.g., manganese, silicon, chromium, vanadium, tungsten, molybdenum, titanium, niobium, phosphorus, zirconium, aluminum, copper, cobalt, and nickel) imparts special characteristic to the steel.

Occupational exposures occur during mining, transporting, and preparing of ores and during the production and refining of the metal and alloys. In addition, certain workers may be exposed while using certain iron-containing materials: welders, grinders, polishers, silver finishers, metal workers, and boiler scalers.

A partial list of occupations in which exposure may occur includes:

Arc cutters	Metalizers
Bessemer operators	Seam welders
Electric arc welders	Stainless steel makers
Flame cutters	Steel foundry workers
Friction saw operators	

### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for iron oxide fume is  $10 \text{ mg/m}^3$ . There are no standards for other iron compounds.

### ROUTE OF ENTRY

Inhalation of dust.

### HARMFUL EFFECTS

#### *Local*—

Soluble iron salts, especially ferric chloride and ferric sulfate, are cutaneous irritants and their aerosols are irritating to the respiratory tract. Iron compounds as a class are not associated with any particular industrial risk.

#### *Systemic*—

The inhalation of iron oxide fumes or dust may cause a benign pneumoconiosis (siderosis). It is probable that the inhalation of pure iron oxide does not cause fibrotic pulmonary changes, whereas the inhalation of iron oxide plus certain other substances may cause injury.

On the basis of epidemiological evidence, exposure to hematite dust increases the risk of lung cancer for workers working underground, but not for surface workers. It may be, however, that hematite dust becomes carcinogenic only in combination with radioactive material, ferric oxide, or silica. There is no evidence that hematite dust or ferric oxide causes cancer in any part of the body other than the lungs.

Iron compounds derive their dangerous properties from the radical with which the iron is associated. Iron pentacarbonyl is one of the more dangerous metal carbonyls. It is highly flammable and toxic. Symptoms of overexposure closely resemble those caused by Ni(CO)<sub>4</sub>, and consist of giddiness and headache, occasionally accompanied by fever, cyanosis, and cough due to pulmonary edema. Death may occur within 4 to 11 days due to pneumonia, liver damage, vascular injury, and central nervous system degeneration.

#### MEDICAL SURVEILLANCE

Special consideration should be given to respiratory disease and lung function in placement and periodic examinations. Smoking history should be known. Chest X-rays and pulmonary function should be evaluated periodically especially if symptoms are present.

#### PERSONAL PROTECTIVE METHODS

Dust masks are recommended for all workers exposed to areas of elevated dust concentrations and especially those workers in underground mines. In areas where iron oxide fumes are excessive, vapor canister masks or supplied air masks are recommended. Generally speaking, protective clothing is not necessary, but attention to personal hygiene, showering, and clothes changing should be encouraged.

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## LEAD - INORGANIC

#### DESCRIPTION

Pb, inorganic lead, includes lead oxides, metallic lead, lead salts, and organic salts such as lead soaps, but excludes lead arsenate and organic lead compounds. Lead is a blue-grey metal which is very soft and malleable. Commercially important lead ores are galena, cerussite, anglesite, crocoisite, wulfenite, pyromorphite, matlockite, and vanadinite. Lead is slightly soluble in water in presence of nitrates, ammonium salts, and carbon dioxide.

#### SYNONYMS

None.

## POTENTIAL OCCUPATIONAL EXPOSURES

Metallic lead is used for lining tanks, piping, and other equipment where pliability and corrosion resistance are required such as in the chemical industry in handling corrosive gases and liquids used in the manufacture of sulfuric acid; in petroleum refining; and in halogenation, sulfonation, extraction, and condensation processes; and in the building industry. It is also used as an ingredient in solder, a filler in the automobile industry, and a shielding material for X-rays and atomic radiation; in manufacture of tetraethyl lead and organic and inorganic lead compounds, pigments for paints and varnishes, storage batteries, flint glass, vitreous enameling, ceramics as a glaze, litharge rubber, plastics, and electronic devices. Lead is utilized in metallurgy and may be added to bronze, brass, steel, and other alloys to improve their characteristics. It forms alloys with antimony, tin, copper, etc. It is also used in metallizing to provide protective coatings and as a heat treatment bath in wire drawing.

Exposures to lead dust may occur during mining, smelting, and refining, and to fume, during high temperature (above 500 C) operations such as welding or spray coating of metals with molten lead.

There are numerous applications for lead compounds, some of the more common being in the plates of electric batteries and accumulators, as compounding agents in rubber manufacture, as ingredients in paints, glazes, enamels, glass, pigments, and in the chemical industry.

A partial list of occupations in which exposure may occur includes:

Battery makers	Insecticide workers
Brass founders	Lubricant makers
Ceramic makers	Match makers
Enamel workers	Painters
Glass makers	Plumbers
Imitation pearl makers	Solderers

## PERMISSIBLE EXPOSURE LIMITS

The Federal standard for lead and its inorganic compounds is 0.2 mg/m<sup>3</sup> as a time-weighted average. The NIOSH Criteria Document recommends a time-weighted average value of 0.15 mg Pb/m<sup>3</sup>.

## ROUTES OF ENTRY

Ingestion of dust; inhalation of dust or fume.

## HARMFUL EFFECTS

*Local*—

None.

*Systemic*—

The early effects of lead poisoning are nonspecific and, except by laboratory testing, are difficult to distinguish from the symptoms of minor seasonal illnesses. The symptoms are decreased physical fitness, fatigue, sleep disturbance, headache, aching bones and muscles, diges-

tive symptoms (particularly constipation), abdominal pains, and decreased appetite. These symptoms are reversible and complete recovery is possible.

Later findings include anemia, pallor, a 'lead line' on the gums, and decreased hand-grip strength. Lead colic produces an intense periodic abdominal cramping associated with severe constipation and, occasionally, nausea and vomiting. Alcohol ingestion and physical exertion may precipitate these symptoms. The peripheral nerve affected most frequently is the radial nerve. This will occur only with exposure over an extended period of time and causes "wrist drop." Recovery is slow and not always complete. When the central nervous system is affected, it is usually due to the ingestion or inhalation of large amounts of lead. This results in severe headache, convulsions, coma, delirium, and possibly death. The kidneys can also be damaged after long periods of exposure to lead, with loss of kidney function and progressive azotemia.

Because of more efficient material handling methods and biological monitoring, serious cases of lead poisoning are rare in industry today.

#### MEDICAL SURVEILLANCE

In preemployment physical examinations, special attention is given to neurologic and renal disease and baseline blood lead levels. Periodic physical examinations should include hemoglobin determinations, tests for blood lead levels, and evaluation of any gastrointestinal or neurologic symptoms. Renal function should be evaluated.

#### SPECIAL TESTS

Periodic evaluation of blood lead levels are widely used as an indicator of increased or excessive lead absorption. Other indicators are blood and urine coproporphyrin III and delta amino low valence acid dehydrase (ALAD). Erythrocytic protoporphyrin determinations may also be helpful.

#### PERSONAL PROTECTIVE METHODS

Workers should be supplied with full body work clothing and caps (hard hats). The dust should be removed (vacuumed) before leaving after the shift. Showering after each shift prior to changing to street clothes should be encouraged. Dust and fume masks or supplied air respirators should be supplied to all employees exposed to concentrations above the TWA standard and in all emergencies. Food should not be eaten in contaminated areas.

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## LEAD - ALKYL

### DESCRIPTION

Both tetraethyl (TEL) and tetramethyl (TM) lead are colorless liquids; however, they are generally mixed with dyes to identify them. TEL is insoluble in water, but soluble in organic solvents. TML is only slightly soluble in organic solvents. Tetraethyl lead will decompose in bright sunlight yielding needle-like crystals of tri-, di-, and mono-ethyl lead compounds, which have a garlic odor.

### SYNONYMS

Tetraethyl lead: TEL. Tetramethyl lead: TML.

### POTENTIAL OCCUPATIONAL EXPOSURES

TEL and TML are used singly or together as "antiknock" ingredients in gasoline. Exposure may occur during synthesis, handling, transport, or mixing with gasoline.

A partial list of occupations in which exposure may occur includes:

Gasoline additive workers

Storage tank cleaners

### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for tetraethyl lead is 0.075 mg Pb/m<sup>3</sup> and for tetramethyl lead 0.07 mg Pb/m<sup>3</sup>.

### ROUTES OF ENTRY

Inhalation of vapor and percutaneous absorption of liquid. TML is more volatile than TEL and therefore may present more of an inhalation hazard. If the tri-, di-, and mono-ethyl lead compounds are dried, the dust may be inhaled producing the same symptomatology as TEL.

### HARMFUL EFFECTS

#### *Local—*

Liquid alkyl lead may penetrate the skin without producing appreciable local injury. However, the decomposition products of TEL (i.e., mono-, di-, tri-ethyl lead compounds) in dust form may be inhaled and result in irritation of the upper respiratory tract and possibly paroxysmal sneezing. This dust, when in contact with moist skin or ocular membranes, may cause itching, burning, and transient redness. TEL itself may be irritating to the eyes.

#### *Systemic—*

The absorption of a sufficient quantity of tetraethyl lead, whether briefly at a high rate, or for prolonged periods at a lower rate, may cause acute intoxication of the central nervous system. Mild degrees of intoxication cause headache, anxiety, insomnia, nervous excitation, and minor gastrointestinal symptoms with a metallic taste in the mouth. The most noticeable clinical sign of tetraethyl lead poisoning is encephal-

opathy which may give rise to a variety of symptoms, which include mild anxiety, toxic delirium with hallucinations, delusions, convulsions, and acute toxic psychosis. Physical signs are not prominent; but bradycardia, hypotension, increased reflexes, tremor, and slight weight loss have been reported. No peripheral neuropathy has been observed. When the interval between the termination of (either brief or prolonged) exposure and the onset of symptoms is delayed (up to 8 days) the prognosis is guardedly hopeful, but when the time interval is short (few hours), an early fatal outcome may result. Recovered patients show no residual damage to the nervous system, although recovery may be prolonged.

Diagnosis depends on developing a history of exposure to organic lead compounds, followed by the onset of encephalopathy. Biochemical measurements are helpful but not diagnostic. Blood lead is usually not elevated in proportion to the degree of intoxication. Urine amino-levulinic acid, and coproporphyrin excretion will show values close to normal with no correlation with the severity of intoxication. Erythrocyte protoporphyrin also remains within normal range.

No cases of poisoning from absorption of tetramethyl lead have been found. The compound responsible for almost all cases of organic lead poisoning is tetraethyl lead. Animal experimentation, however, indicates that a similar intoxication can be caused by tetramethyl lead.

#### MEDICAL SURVEILLANCE

In both preemployment and periodic physical examinations, the worker's general health should be evaluated, and special attention should be given to neurologic and emotional disorders.

#### SPECIAL TESTS

None seem to be useful.

#### PERSONAL PROTECTIVE METHODS

A training program should stress the importance of personal hygiene and encourage the proper use of personal protective equipment. Showers, lavatories, and locker rooms are necessary. Workers should be required to make a complete change of clothing at the beginning and end of each shift and to shower prior to changing to street clothes. Eating should not be permitted in work areas. In areas where vapor concentrations of TEL exceed the standard, dust masks, organic vapor canister masks, or supplied air respirators should be furnished and required to be worn. In areas of spills or splash, impervious clothing should be worn and goggles furnished.

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## MAGNESIUM AND COMPOUNDS

### DESCRIPTION

Magnesium is a light, silvery-white metal and is a fire hazard. It is found in dolomite, magnesite, brucite, periclase, carnallite, kieserite and as a silicate in asbestos, talc, olivine, and serpentine. It is also found in sea water, brine wells, and salt deposits. It is insoluble in water and ordinary solvents.

### SYNONYMS

None.

### POTENTIAL OCCUPATIONAL EXPOSURES

Magnesium alloyed with manganese, aluminum, thorium, zinc, cerium, and zirconium is used in aircraft, ships, automobiles, hand tools, etc. because of its lightness. Dow metal is the general name for a large group of alloys containing over 85% magnesium. Magnesium wire and ribbon are used for degassing valves in the radio industry and in various heating appliances; as a deoxidizer and desulfurizer in copper, brass, and nickel alloys; in chemical reagents; as the powder in the manufacture of flares, incendiary bombs, tracer bullets, and flashlight powders; in the nuclear energy process; and in a cement of magnesium oxide and in magnesium chloride for floors.

A partial list of occupations in which exposure may occur includes:

Alloy makers	Organic chemical synthesizers
Antiseptic makers	Pigment makers
Battery makers	Steel makers
Drug makers	Textile workers
Flare makers	Welders
Fungicide makers	

### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for magnesium oxide fume is 15 mg/m<sup>3</sup>.

### ROUTE OF ENTRY

Inhalation of fume.

## HARMFUL EFFECTS

### *Local*—

Magnesium and magnesium compounds are mild irritants to the conjunctiva and nasal mucosa, but are not specifically toxic. Magnesium in finely divided form is readily ignited by a spark or flame, and splatters and burns at above 2,300 F. On the skin, these hot particles are capable of producing second and third degree burns, but they respond to treatment as other thermal burns do. Metallic magnesium foreign bodies in the skin cause no unusual problems in man. In animal experiments, however, they have caused "gas gangrene"—massive localized gaseous tumors with extensive necrosis.

### *Systemic*—

Magnesium in the form of nascent magnesium oxide can cause metal fume fever if inhaled in sufficient quantity. Symptoms are analogous to those caused by zinc oxide: cough, oppression in the chest, fever, and leukocytosis. There is no evidence that inhalation of magnesium dust has led to lung injury. It has been noted that magnesium workers show a rise in serum magnesium — although no significant symptoms of ill health have been identified. Some investigators have reported higher incidence of digestive disorders and have related this to magnesium absorption, but the evidence is scant. In foundry casting operations, hazards exist from the use of fluoride fluxes and sulfur-containing inhibitors which produce fumes of fluorides and sulfur dioxide.

## MEDICAL SURVEILLANCE

No specific recommendations.

## SPECIAL TESTS

None.

## PERSONAL PROTECTIVE METHODS

Employees should receive training in the use of personal protective equipment, proper methods of ventilation, and fire suppression. Protective clothing should be designed to prevent burns from splatters. Masks to prevent inhalation of fumes may be necessary under certain conditions, but generally this can be controlled by proper ventilation. Dust masks may be necessary in areas of dust concentration as in transfer and storage areas, but adequate ventilation generally provides sufficient protection.

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**MANGANESE AND COMPOUNDS****DESCRIPTION**

Mn, manganese, is a reddish-grey or silvery, soft metal. The most important ore containing manganese is pyrolusite. Manganese may also be produced from ferrous scrap used in the production of electric and open-hearth steel. Manganese decomposes in water and is soluble in dilute acid.

**SYNONYMS**

None

**POTENTIAL OCCUPATIONAL EXPOSURES**

Most of the manganese produced is used in the iron and steel industry in steel alloys, e.g., ferromanganese, silicomanganese, Manganin, spiegeleisen, and as an agent to reduce oxygen and sulfur content of molten steel. Other alloys may be formed with copper, zinc, and aluminum. Manganese and its compounds are utilized in the manufacture of dry cell batteries ( $MnO_2$ ), paints, varnishes, inks, dyes, matches and fireworks, as a fertilizer, disinfectants, bleaching agent, laboratory reagent, drier for oils, an oxidizing agent in the chemical industry, particularly in the synthesis of potassium permanganate, and as a decolorizer and coloring agent in the glass and ceramics industry.

Exposure may occur during the mining, smelting and refining of manganese, in the production of various materials, and in welding operations with manganese coated rods.

A partial list of occupations in which exposure may occur includes:

Battery makers	Glass makers
Ceramic makers	Ink makers
Drug makers	Match makers
Electric arc welders	Paint makers
Feed additive makers	Varnish makers
Foundry workers	Water treaters

**PERMISSIBLE EXPOSURE LIMITS**

The Federal standard for manganese is  $5 \text{ mg/m}^3$  as a ceiling value.

**ROUTES OF ENTRY**

Inhalation of dust or fume; limited percutaneous absorption of liquids.

**HARMFUL EFFECTS**

*Local*—

Manganese dust and fumes are only minor irritants to the eyes and mucous membranes of the respiratory tract, and apparently completely innocuous to the intact skin.

*Systemic—*

Chronic manganese poisoning has long been recognized as a clinical entity. The dust or fumes (manganous compounds) enter the respiratory tract and are absorbed into the blood stream. Manganese is then deposited in major body organs and has a special predilection for the liver, spleen, and certain nerve cells of the brain and spinal cord. Among workers there is a very marked variation in individual susceptibility to manganese. Some workers have worked in heavy exposure for a lifetime and have shown no signs of the disease; others have developed manganese intoxication with as little as 49 days of exposure.

The early phase of chronic manganese poisoning is most difficult to recognize, but it is also most important to recognize since early removal from the exposure may arrest the course of the disease. The onset is insidious, with apathy, anorexia, and asthenia. Headache, hypersomnia, spasms, weakness of the legs, arthralgias, and irritability are frequently noted. Manganese psychosis follows with certain definitive features: unaccountable laughter, euphoria, impulsive acts, absentmindedness, mental confusion, aggressiveness, and hallucinations. These symptoms usually disappear with the onset of true neurological disturbances, or may resolve completely with removal from manganese exposure.

Progression of the disease presents a range of neurological manifestations that can vary widely among individuals affected. Speech disturbances are common: monotonous tone, inability to speak above a whisper, difficult articulation, incoherence, even complete muteness. The face may take on masklike quality, and handwriting may be affected by micrographia. Disturbances in gait and balance occur, and frequently propulsion, retropropulsion, and lateropropulsion are affected, with no movement for protection when falling. Tremors are frequent, particularly of the tongue, arms, and legs. These will increase with intentional movements and are more frequent at night. Absolute detachment, broken by sporadic or spasmodic laughter, ensues, and as in extrapyramidal affections, there may be excessive salivation and excessive sweating. At this point the disease is indistinguishable from classical Parkinson's disease.

Chronic manganese poisoning is not a fatal disease although it is extremely disabling.

Manganese dust is no longer believed to be a causative factor in pneumonia. If there is any relationship at all, it appears to be as an aggravating factor to a preexisting condition. Freshly formed fumes have been reported to cause fever and chills similar to metal fume fever.

**MEDICAL SURVEILLANCE**

Preemployment physical exams should be directed toward the individual's general health with special attention to neurologic and personality abnormalities. Periodic physical examinations may be required as often as every two months. Special emphasis should be given to behavioral and neurological changes: speech defects, emotional distur-

bances, hypertonia, tremor, equilibrium, difficulty in walking or squatting, adiadochokinesis, and handwriting.

#### SPECIAL TESTS

There are no laboratory tests which can be used to diagnose manganese poisoning.

#### PERSONAL PROTECTIVE METHODS

In areas where the ceiling value standards are exceeded, dust masks or respirators are necessary. Education in the use and necessity of these devices is important.

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## MERCURY - INORGANIC

#### DESCRIPTION

Hg, inorganic mercury, is here taken to include elemental mercury, inorganic mercury compounds, and organic mercury compounds, excluding alkyl mercury compounds. Metallic mercury is a silver-white liquid at room temperature. It occurs as the free metal or as cinnabar (HgS). Mercury is produced from the ore by roasting or reduction.

#### SYNONYMS

Quicksilver, hydrargyrum.

#### POTENTIAL OCCUPATIONAL EXPOSURES

Elemental and inorganic mercury compounds are used in the manufacture of scientific instruments (barometers, thermometers, etc.), electric equipment (meters, switches, batteries, rectifiers, etc.), mercury vapor lamps, incandescent electric lamps, X-ray tubes, artificial silk, radio valves, amalgams with copper, tin, silver, or gold, and solders with lead and tin. In the chemical industry, it is used as a fluid cathode for the electrolytic production of caustic soda (sodium hydroxide), chlorine, and acetic acid. It is utilized in gold, silver, bronze, and tin plating, tanning and dyeing, feltmaking, taxidermy, textile manufacture, photography and photoengraving, in extracting gold and silver from ores, in paints and pigments, in the preparation of drugs and disinfectants in the pharmaceutical industry, and as a chemical reagent.

The aryl mercury compounds such as phenylmercury are primarily used as disinfectants, fungicides for treating seeds, antiseptics, herbicides, preservatives, mildew-proofing agents, denaturants for ethyl alcohol, germicides, and bactericides.

Hazardous exposure may occur during mining and extraction of

mercury and in the use of mercury and its compounds. Elemental mercury readily volatilizes at room temperature.

A partial list of occupations in which exposure may occur includes:

Amalgam makers	Gold extractors
Bactericide makers	Jewelers
Battery makers	Paper makers
Caustic soda makers	Photographers
Dental amalgam makers	Taxidermists
Fungicide makers	

#### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for mercury is 1 mg/10m<sup>3</sup> as a ceiling value. The recommended standard is 0.05 mg Hg/m<sup>3</sup> as a TWA.

#### ROUTES OF ENTRY

Inhalation of dust or vapor; percutaneous absorption of elemental mercury.

#### HARMFUL EFFECTS

##### *Local—*

Mercury is a primary irritant of skin and mucous membranes. It may occasionally be a skin sensitizer.

##### *Systemic—*

Acute poisoning due to mercury vapors affects the lungs primarily, in the form of acute interstitial pneumonitis, bronchitis, and bronchiolitis.

Exposure to lower levels over prolonged periods produces symptom complexes that can vary widely from individual to individual. These may include weakness, fatigability, loss of appetite, loss of weight, insomnia, indigestion, diarrhea, metallic taste in the mouth, increased salivation, soreness of mouth or throat, inflammation of gums, black line on the gums, loosening of teeth, irritability, loss of memory, and tremors of fingers, eyelids, lips, or tongue. More extensive exposures, either by daily exposures or one-time, can produce extreme irritability, excitability, anxiety, delirium with hallucinations, melancholia, or manic depressive psychosis. In general, chronic exposure produces four classical signs: gingivitis, sialorrhea, increased irritability, and muscular tremors. Rarely are all four seen together in an individual case.

Either acute or chronic exposure may produce permanent changes to affected organs and organ systems.

#### MEDICAL SURVEILLANCE

Preemployment and periodic examinations should be concerned especially with the skin, respiratory tract, central nervous system, and kidneys. The urine should be examined and urinary mercury levels determined periodically. Signs of weight loss, gingivitis, tremors, personality changes, and insomnia would be suggestions of possible mercury intoxication.

## SPECIAL TESTS

Urine mercury determination may be helpful as an index of amount of absorption. Opinions vary as to the significance of a given level. Generally, 0.1 to 0.5 mg Hg/liter of urine is considered significant.

## PERSONAL PROTECTIVE METHODS

In areas where the exposures are excessive, respiratory protection shall be provided either by full face canister type mask or supplied air respirator, depending on the concentration of mercury fumes. Above 50 mg Hg/cu m requires supplied air positive pressure fullface respirators. Full body work clothes including shoes or shoe covers and hats should be supplied, and clean work clothes should be supplied daily. Showers should be available and all employees encouraged to shower prior to change to street clothes. Work clothes should not be stored with street clothes in the same locker. Food should not be eaten in the work area:

*MERCURY - ALKYL*

## DESCRIPTION

Methyl mercury compounds: methyl mercury dicyandiamide -  $\text{CH}_3\text{HgNHC}(:\text{NH})\text{NHCN}$ . Soluble in water.

Ethyl mercury compounds: ethylmercuric chloride:  $\text{C}_2\text{H}_5\text{HgCl}$ . Insoluble in water. Ethylmercuric phosphate:  $(\text{C}_2\text{H}_5\text{Hg})\text{PO}$ . Soluble in water. N-(Ethylmercuric)-p-toluenesulphonanilide:  $\text{C}_6\text{H}_5\text{N}(\text{HgC}_2\text{H}_5)\text{-SO}_2\text{C}_6\text{H}_4\text{CH}_3$ . Practically insoluble in water.

## SYNONYMS

Methyl mercury compounds: Methyl mercury dicyandiamide: none. Cyano (methyl mercury) guanidine: panogen.

Ethyl mercury compounds: Ethylmercuric chloride: ceresan. Ethylmercuric phosphate: new ceresan. N-(Ethylmercuric)-p-toluenesulphonanilide: ceresan m.

## POTENTIAL OCCUPATIONAL EXPOSURES

These compounds are used in treating seeds for fungi and seed-borne diseases, as timber preservatives, and disinfectants.

A partial list of occupations in which exposure may occur includes:

Disinfectant makers	Seed handlers
Fungicide makers	Wood preservers

## PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 0.01 mg/m<sup>3</sup> as an 8-hour TWA with an acceptable ceiling of 0.04 mg/m<sup>3</sup>.

## ROUTES OF ENTRY

Inhalation of dust, percutaneous absorption.

## HARMFUL EFFECTS

*Local—*

Alkyl mercury compounds are primary skin irritants and may cause dermatitis. When deposited on the skin, they give no warning, and if contact is maintained, can cause second-degree burns. Sensitization may occur.

*Systemic—*

The central nervous system, including the brain, is the principal target tissue for this group of toxic compounds. Severe poisoning may produce irreversible brain damage resulting in loss of higher functions.

The effects of chronic poisoning with alkyl mercury compounds are progressive. In the early stages, there are fine tremors of the hands, and in some cases, of the face and arms. With continued exposure, tremors may become coarse and convulsive; scanning speech with moderate slurring and difficulty in pronunciation may also occur. The worker may then develop an unsteady gait of a spastic nature which can progress to severe ataxia of the arms and legs. Sensory disturbances including tunnel vision, blindness, and deafness are also common.

A later symptom, constriction of the visual fields, is rarely reversible and may be associated with loss of understanding and reason which makes the victim completely out of touch with his environment. Severe cerebral effects have been seen in infants born to mothers who had eaten large amounts of methyl mercury contaminated fish.

## MEDICAL SURVEILLANCE

Preplacement and periodic physical examinations should be concerned particularly with the skin, vision, central nervous system, and kidneys. Consideration should be given to the possible effects on the fetus of alkyl mercury exposure in the mother. Constriction of visual fields may be a useful diagnostic sign. (See Mercury-Inorganic.)

## SPECIAL TESTS

Blood and urine levels of mercury have been studied, especially in the case of methyl mercury. A precise correlation has not been found between exposure levels and concentrations. They may be of some value in indicating that exposure has occurred, however.

## PERSONAL PROTECTIVE METHODS

(See Mercury-Inorganic.)

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**MOLYBDENUM AND COMPOUNDS****DESCRIPTION**

Mo, molybdenum, is a silver-white metal or a greyish-black powder. Molybdenite is the only important commercial source. This ore is often associated with copper ore. Molybdenum is insoluble in water and soluble in hot concentrated nitric and sulfuric acid.

**SYNONYMS**

None

**POTENTIAL OCCUPATIONAL EXPOSURES**

Most of the molybdenum produced is used in alloys: steel, stainless steel, tool steel, cast iron, steel mill rolls, manganese, nickel, chromium, and tungsten. The metal is used in electronic parts (contacts, spark plugs, X-ray tubes, filaments, screens, and grids for radios), induction heating elements, electrodes for glass melting, and metal spraying applications. Molybdenum compounds are utilized as lubricants; as pigments for printing inks, lacquers, paints, for coloring rubber, animal fibers, and leather, and as a mordant; as catalysts for hydrogenation cracking, alkylation, and reforming in the petroleum industry, in Fischer-Tropsch synthesis, in ammonia production, and in various oxidation-reduction and organic cracking reactions; as a coating for quartz glass; in vitreous enamels to increase adherence to steel; in fertilizers, particularly for legumes; in electroplating to form protective coatings; and in the production of tungsten.

Hazardous exposures may occur during high-temperature treatment in the fabrication and production of molybdenum products, spraying applications, or through loss of catalyst.  $\text{MoO}_3$  sublimates above 800 C.

A partial list of occupations in which exposure may occur includes:

Ceramic makers	Metal platers
Drug makers	Petroleum refinery workers
Electroplaters	Steel alloy makers
Fertilizer makers	Tannery workers
Glass makers	Vacuum tube makers

**PERMISSIBLE EXPOSURE LIMITS**

The Federal standards are: Molybdenum: soluble compounds, 5 mg/m<sup>3</sup>. Molybdenum: insoluble compounds, 15 mg/m<sup>3</sup>.

**ROUTE OF ENTRY**

Inhalation of dust or fume.

**HARMFUL EFFECTS****Local—**

Molybdenum trioxide may produce irritation of the eyes and mucous membranes of the nose and throat. Dermatitis from contact with molybdenum is unknown.

### *Systemic—*

No reports of toxic effects of molybdenum in the industrial setting have appeared. It is considered to be an essential trace element in many species, including man. Animal studies indicate that insoluble molybdenum compounds are of a low order of toxicity (e.g., disulfide, oxides, and halides). Soluble compounds (e.g., sodium molybdate) and freshly generated molybdenum fumes, however, are considerably more toxic. Inhalation of high concentrations of molybdenum trioxide dust is very irritating to animals and has caused weight loss, diarrhea, loss of muscular coordination, and a high mortality rate. Molybdenum trioxide dust is more toxic than the fumes. Large oral doses of ammonium molybdate in rabbits caused some fetal deformities. Excessive intake of molybdenum may produce signs of a copper deficiency.

### MEDICAL SURVEILLANCE

Preemployment and periodic physical examinations should evaluate any irritant effects to the eyes or respiratory tract and the general health of the worker. Although molybdenum compounds are of low order of toxicity, animal experimentation indicates protective measures should be employed against the more soluble compounds and molybdenum trioxide dust and fumes. The normal intake of copper in the diet appears to be sufficient to prevent systemic toxic effects due to molybdenum poisoning.

### SPECIAL TESTS

None in common use.

### PERSONAL PROTECTIVE METHODS

Where dust and fumes exceed the standard, molybdenum workers should be supplied with dust masks or supplied air respirators. Full body work clothes are advisable with daily change of clothes and showering before changing to street clothes.

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## *NICKEL AND COMPOUNDS*

### DESCRIPTION

Ni, nickel, is a hard, ductile, magnetic metal with a silver-white color. It is insoluble in water and soluble in acids. It occurs free in meteorites and in ores combined with sulfur, antimony, or arsenic. Processing and refining of nickel is accomplished by either the Orford (sodium sulfide and electrolysis) or the Mond (nickel carbonyl) processes. In the latter, impure nickel powder is reacted with carbon monoxide to form gaseous nickel carbonyl which is then treated to deposit high purity metallic nickel.

## SYNONYMS

None.

## POTENTIAL OCCUPATIONAL EXPOSURES

Nickel forms alloys with copper, manganese, zinc, chromium, iron, molybdenum, etc. Stainless steel is the most widely used nickel alloy. An important nickel-copper alloy is Monel metal, which contains 66% nickel and 32% copper and has excellent corrosion resistance properties. Permanent magnets are alloys chiefly of nickel, cobalt, aluminum, and iron.

Elemental nickel is used in electroplating, anodizing aluminum, casting operations for machine parts, and in coinage; in the manufacture of acid-resisting and magnetic alloys, magnetic tapes, surgical and dental instruments, nickel-cadmium batteries, nickel soaps in crankcase oils, and ground-coat enamels, colored ceramics, and glass. It is used as a catalyst in the hydrogenation of fats, oils, and other chemicals, in synthetic coal oil production, and as an intermediate in the synthesis of acrylic esters for plastics.

Exposure to nickel may also occur during mining, smelting, and refining operations.

A partial list of occupations in which exposure may occur includes:

Battery makers	Oil hydrogenators
Ceramic makers	Paint makers
Chemists	Pen point makers
Dyers	Spark plug makers
Enamelers	Textile dyers
Ink makers	Varnish makers
Magnet makers	

## PERMISSIBLE EXPOSURE LIMITS

The Federal standard for nickel metal and its soluble compounds is 1 mg/m<sup>3</sup> expressed as Ni.

## ROUTE OF ENTRY

Inhalation of dust or fume.

## HARMFUL EFFECTS

*Local—*

Skin sensitization is the most commonly seen toxic reaction to nickel and nickel compounds and is seen frequently in the general population. This often results in chronic eczema "Nickel itch," with lichenification resembling atopic or neurodermatitis. Nickel and its compounds are also irritants to the conjunctiva of the eye and the mucous membrane of the upper respiratory tract.

*Systemic—*

Elemental nickel (as deposited from inhalation of nickel carbonyl) and nickel salts are probably carcinogenic, producing an increased incidence of cancer of the lung and nasal passages. Effects on the heart

muscle, brain, liver, and kidney have been seen in animal studies. Pulmonary eosinophilia (Loeffler's syndrome) has been reported in one study to be caused by the sensitizing property of nickel. Finely divided nickel has also shown some carcinogenic effects in rats by injection, and in guinea pigs by inhalation.

#### MEDICAL SURVEILLANCE

Preemployment physical examinations should evaluate any history of skin allergies or asthma, other exposures to nickel or other carcinogens, smoking history, and the respiratory tract. Lung function should be studied and chest X-rays periodically evaluated. Special attention should be given to the nasal sinuses and skin.

#### SPECIAL TESTS

Serum and urinary nickel can be determined, although opinions vary as to their value in monitoring exposures.

#### PERSONAL PROTECTIVE METHODS

Full body protective clothing is advisable, as is the use of barrier creams to prevent skin sensitization and dermatitis. In areas of dust or fumes, masks or supplied air respirators are mandatory where concentrations exceed the standard limits. Clean work clothing should be provided daily; and showering should be required before changing to street clothes. No food should be eaten in work areas.

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## NICKEL CARBONYL

#### DESCRIPTION

$\text{Ni}(\text{CO})_4$ , nickel carbonyl, is a colorless, highly volatile, flammable liquid with a musty odor. It decomposes above room temperature producing carbon monoxide and finely divided nickel. It is soluble in organic solvents.

#### SYNONYMS

Nickel tetracarbonyl.

#### POTENTIAL OCCUPATIONAL EXPOSURES

The primary use of nickel carbonyl is in the production of nickel by the Mond process. Impure nickel powder is reacted with carbon monoxide to form gaseous nickel carbonyl which is then treated to deposit high purity metallic nickel and release carbon monoxide. Other uses include gas plating, the production of nickel products; in chemical

synthesis as a catalyst, particularly for oxo reactions (addition reaction of hydrogen and carbon monoxide with unsaturated hydrocarbons to form oxygen-function compounds), e.g., synthesis of acrylic esters, and as a reactant.

A partial list of occupations in which exposure may occur includes:

Foundry workers	Organic chemical synthesizers
Gas platers	Petroleum refinery workers
Mond process workers	

#### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for nickel carbonyl is 0.001 ppm (0.007 mg/m<sup>3</sup>).

#### ROUTES OF ENTRY

Inhalation of vapor. It may be possible for appreciable amounts of the liquid to be absorbed through the skin.

#### HARMFUL EFFECTS

##### *Local—*

Nickel dermatitis may develop. (See Nickel and Compounds.)

##### *Systemic—*

Symptoms of exposure to the toxic vapors of nickel carbonyl are of two distinct types. Immediately after exposure, symptoms consist of frontal headache, giddiness, tightness of the chest, nausea, weakness of limbs, perspiring, cough, vomiting, cold and clammy skin, and shortness of breath. Even in exposures sufficiently severe to cause death, the initial symptoms disappear quickly upon removal of the subject to fresh air. Symptoms may be so mild during this initial phase that they go unrecognized.

Severe symptoms may then develop insidiously hours or even days after exposure. The delayed syndrome usually consists of retrosternal pain, tightness in the chest, dry cough, shortness of breath, rapid respiration, cyanosis, and extreme weakness. The weakness may be so great that respiration can be sustained only by oxygen support. Fatal cases are usually preceded by convulsion and mental confusion, with death occurring from 4 - 11 days following exposure. The syndrome represents a chemical pneumonitis with adrenal cortical suppression.

Nickel carbonyl is carcinogenic to the same degree as elemental nickel. (See Nickel and Compounds.)

#### MEDICAL SURVEILLANCE

(See Nickel and Compounds.)

#### SPECIAL TESTS

Urinary nickel levels for several days after acute exposures may be helpful.

#### PERSONAL PROTECTIVE METHODS

(See Nickel and Compounds.)

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## OSMIUM AND COMPOUNDS

## DESCRIPTION

Os, osmium, is a blue-white metal. It is found in platinum ores and in the naturally occurring alloy osmiridium. Osmium when heated in air or when the finely divided form is exposed to air at room temperature oxidizes to form tetroxide ( $\text{OsO}_4$ , osmic acid). It has a nauseating odor.

## SYNONYMS

None.

## POTENTIAL OCCUPATIONAL EXPOSURES

Osmium may be alloyed with platinum metals, iron, cobalt, and nickel, and it forms compounds with tin and zinc. The alloy with iridium is used in the manufacture of fountain pen points, engraving tools, record player needles, electrical contacts, compass needles, fine machine bearings, and parts for watch and lock mechanisms. The metal is a catalyst in the synthesis of ammonia, and in the dehydrogenation of organic compounds. It is also used as a stain for histological examination of tissues. Osmium tetroxide is used as an oxidizing agent and as a fixative for tissues in electron microscopy. Other osmium compounds find use in photography. Osmium is no longer used in incandescent lights and in fingerprinting.

A partial list of occupations in which exposure may occur includes:

Alloy makers	Synthetic ammonia makers
Histology technicians	Platinum hardeners
Organic chemical synthesizers	

## PERMISSIBLE EXPOSURE LIMITS

There is presently no Federal standard for osmium itself; the standard for osmium tetroxide is  $0.002 \text{ mg/m}^3$ .

## ROUTE OF ENTRY

Inhalation of vapor or fume.

## HARMFUL EFFECTS

*Local—*

Osmium metal is innocuous, but persons engaged in the production of the metal may be exposed to acids and chlorine vapors. Osmium

tetroxide vapors are poisonous and extremely irritating to the eyes; even in low concentrations, they may cause weeping and persistent conjunctivitis. Longer exposure can result in damage to the cornea and blindness. Contact with skin may cause discoloration (green or black) dermatitis and ulceration.

*Systemic—*

Inhalation of osmium tetroxide fumes is extremely irritating to the respiratory system, causing tracheitis, bronchitis, bronchial spasm, and difficulty in breathing which may last several hours. Longer exposures can cause serious inflammatory lesions of the lungs (bronchopneumonia with suppuration and gangrene). Slight kidney damage was seen in rabbits inhaling lethal concentrations of vapor for 30 minutes. Some fatty degeneration of renal tubules was seen in one fatal human case along with bronchio pneumonia following an accidental overexposure.

**MEDICAL SURVEILLANCE**

Consider the skin, eyes, respiratory tract, and renal function in placement or periodic examinations.

**SPECIAL TESTS**

None in common use.

**PERSONAL PROTECTIVE METHODS**

In areas where the concentration of osmium tetroxide fumes or vapors are excessive, fullface masks or supplied air respirators are necessary. Even low concentrations can cause severe irritation of the eyes. This can usually be prevented by proper ventilation (exhaust hoods, etc.) and the use of goggles. Gloves should be used to prevent burns of the skin and hands. Precautions should be taken to provide protection against acids and chlorine vapors in areas where the metal is produced. (See chlorine.)

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**PHOSPHINE**

**DESCRIPTION**

PH<sub>3</sub>, phosphine, is a colorless gas with an odor of decaying fish. Phosphine presents an additional hazard in that it ignites at very low temperature. Phosphine is soluble in water 26 ml/100 ml at 17 C and in organic solvents.

**SYNONYMS**

Hydrogen phosphide, phosphoretted hydrogen, phosphorus trihydride.

## POTENTIAL OCCUPATIONAL EXPOSURES

Phosphine is only occasionally used in industry, and exposure usually results accidentally as a by-product of various processes. Exposures may occur when acid or water comes in contact with metallic phosphides (aluminum phosphide, calcium phosphide). These two phosphides are used as insecticides or rodenticides for grain, and phosphine is generated during grain fumigation. Phosphine may also evolve during the generation of acetylene from impure calcium carbide, as well as during metal shaving, sulfuric acid tank cleaning, rust proofing, and ferrosilicon, phosphoric acid and yellow phosphorus explosive handling.

A partial list of occupations in which exposure may occur includes:

Acetylene workers	Metal slag workers
Cement workers	Metallic phosphate workers
Firemen	Organic chemical synthesizers
Grain fumigators	Rustproofers
Metal refiners	Welders

## PERMISSIBLE EXPOSURE LIMITS

0.3 ppm (0.4 mg/m<sup>3</sup>) is the Federal standard for occupational exposure to phosphine determined as TWA.

## ROUTE OF ENTRY

Inhalation of vapor.

## HARMFUL EFFECTS

*Local—*

Phosphine's strong odor may be nauseating. However, irritation to the eyes or skin is undocumented, and some authors indicate that lachrymation, if it occurs, results as a systemic effect rather than from local irritation.

*Systemic—*

Acute effects are secondary to central nervous system depression, irritation of lungs, and damage to the liver and other organs. Most common effects include weakness, fatigue, headache, vertigo, anorexia, nausea, vomiting, abdominal pain, diarrhea, tenesmus, thirst, dryness of the throat, difficulty in swallowing, and sensation of chest pressure. In severe cases staggering gait, convulsions, and coma follow. Death may occur from cardiac arrest and, more typically, pulmonary edema, which may be latent in a manner similar to nitrogen oxide intoxication.

Chronic poisoning has been suggested by some authors and symptoms have been attributed to chronic phosphorus poisoning. However, there is evidence that phosphine may be metabolized to form nontoxic phosphates, and chronic exposure of animals has failed to produce toxic effects. Compounded with the lack of human experience and of extensive commercial usage, evidence indicates that chronic poisoning per se does not occur.

## MEDICAL SURVEILLANCE

No special considerations are necessary in placement or periodic examinations, other than evaluation of the respiratory system. If poisoning is suspected, workers should be observed for 48 hours due to the delayed onset of pulmonary edema.

## SPECIAL TESTS

None have been used.

## PERSONAL PROTECTIVE METHODS

In areas where vapors are excessive, workers should be supplied with fullface gas masks with proper cannisters or supplied air respirators.

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## PHOSPHORUS AND COMPOUNDS (EXCLUDING PHOSPHINE)

## DESCRIPTION

Phosphorus (white or yellow): P. Almost insoluble in water, but soluble in organic solvents.

Phosphoric acid:  $H_3PO_4$ . Soluble in water and alcohol.

Phosphorus trichloride:  $PCl_3$ . Decomposes in cold water.

Tetraphosphorus trisulfide:  $P_4S_3$ . Insoluble.

Red phosphorus is excluded in that it is a nontoxic allotrope, although it is frequently contaminated with a small amount of the yellow. White or yellow phosphorus is either a yellow or colorless, volatile, crystalline solid which darkens when exposed to light and ignites in air to form white fumes and greenish light. Phosphoric acid is also a crystal; however, it is typically encountered in a liquid form. Phosphorus pentachloride and phosphorus pentasulfide are white to pale yellow, fuming crystals, while tetraphosphorus trisulfide is a greenish-yellow crystal. Phosphorus trichloride and phosphorous oxychloride are also colorless, fuming liquids.

Elemental phosphorus does not occur free in nature, but is found in the form of phosphates. Phosphorus and phosphoric acid are prepared commercially from "phosphate rock" deposits of the Southern United

States and, at one time, from bone in Europe. Phosphorus, once formed, is immediately converted to less toxic substances, such as phosphoric acid. The other compounds are prepared directly from red phosphorus and chloride or sulfur respectively. Decomposition products of phosphorus compounds are also toxic and include hydrogen sulfide and phosphoric acid for sulfur-containing compounds.

#### SYNONYMS

Phosphorus: none.

Phosphoric acid:  $H_3PO_4$ , orthophosphoric acid.

Phosphorus trichloride:  $PCl_3$ , phosphorous chloride.

Phosphorus pentachloride:  $PCl_5$ , phosphoric chloride, phosphorus perchloride.

Tetraphosphorus trisulfide:  $P_4S_3$ , phosphorus sesquisulfide, trisulfurated phosphorus.

Phosphorus pentasulfide:  $P_2S_5$ , phosphoric sulfide, thiophosphoric anhydride, phosphorus persulfide.

Phosphorus oxychloride:  $POCl_3$ , phosphorylchloride.

#### POTENTIAL OCCUPATIONAL EXPOSURES

Yellow phosphorus is handled away from air so that exposure is usually limited. Phosphorus was at one time used for the production of matches or lucifers but has long since been replaced due to its chronic toxicity. Phosphorus is used in the manufacture of munitions, pyrotechnics, explosives, smoke bombs, and other incendiaries, in artificial fertilizers, rodenticides, phosphorbronze alloy, semiconductors, electroluminescent coating, and chemicals, such as, phosphoric acid and metallic phosphides. Phosphoric acid is used in the manufacture of fertilizers, phosphate salts, polyphosphates, detergents, activated carbon, animal feed, ceramics, dental cement, pharmaceuticals, soft drinks, gelatin, rust inhibitors, wax, and rubber latex. Exposure may also occur during electropolishing, engraving, photoengraving, lithograving, metal cleaning, sugar refining, and water treating. Phosphorus trichloride and phosphorus pentachloride are used in the manufacture of agricultural chemicals, chlorinated compounds, dyes, gasoline additives, acetylcellulose, phosphorus oxychloride, plasticizers, saccharin, and surfactants. Phosphorus pentasulfide and tetraphosphorus trisulfide are used in the manufacture of flotation agents, insecticides, lubricating oil, additives, ignition compounds, and matches. They are also used to introduce sulfur into agricultural, rubber, and organic chemicals.

A partial list of occupations in which exposure may occur includes:

Acetylcellulose makers	Metal refiners
Bronze alloy makers	Metallic phosphide makers
Chlorinated compound makers	Munitions workers
Electroluminescent coating makers	Pesticide workers
Fertilizer makers	Rat poison workers
Fireworks makers	Semiconductor makers
Hydraulic fluid makers	Smoke bomb makers
Incendiary makers	

## PERMISSIBLE EXPOSURE LIMITS

Federal standards are: Phosphorus (yellow) 0.1 mg/m<sup>3</sup>, phosphoric acid 1.0 mg/m<sup>3</sup>, phosphorus trichloride 3.0 mg/m<sup>3</sup>, phosphorus pentachloride 1.0 mg/m<sup>3</sup>, phosphorus pentasulfide 1.0 mg/m<sup>3</sup>.

## ROUTE OF ENTRY

Inhalation of vapor or fumes or mist.

## HARMFUL EFFECTS

*Local—*

Phosphorus, upon contact with skin, may result in severe burns, which are necrotic, yellowish, fluorescent under ultraviolet light, and have a garlic-like odor. Other phosphorus compounds are potent irritants of the skin, eyes, and mucous membranes of nose, throat, and respiratory tract. At 1 ppm, the Federal standard, phosphoric acid mist is irritating to unacclimated workers but is easily tolerated by acclimated workers. Localized contact dermatitis, particularly of the thighs and eczema of the face and hands, have been observed in workers manufacturing the "strike anywhere" matches containing tetraphosphorus trisulfide.

*Systemic—*

Acute phosphorus poisoning usually occurs as a result of accidental or suicidal ingestion. However, animal experiments indicate that acute systemic poisoning may follow skin burns. In acute cases, shock may ensue rapidly and the victim may succumb immediately. If acute attack is survived, an asymptomatic latency period of a few hours to a few days may follow. Death often occurs upon relapse from liver, kidney, cardiac, or vascular dysfunction or failure. Abnormal electrocardiograms, particularly of the QT, ST, or T wave phases, abnormal urinary and serum calcium and phosphate levels, proteinuria and aminoaciduria, and elevated serum SGPT are indicative signs. Vomitus, urine, and stools may be fluorescent in ultraviolet light, and a garlic odor of breath and eructations may be noted.

Inhalation of fumes produced by phosphorus compounds listed above may cause irritation of pulmonary tissues with resultant acute pulmonary edema. Chronic exposure may lead to cough, bronchitis, and pneumonia. The hazards of phosphorous pentasulfide are the same as for hydrogen sulfide to which it rapidly hydrolyzes in the presence of moisture.

Chronic phosphorus poisoning is a result of continued absorption of small amounts of yellow phosphorus for periods typically of ten years; however, exposures of as short as 10 months may cause phosphorus necrosis of the jaw ("phossy jaw"). Chronic intoxication is characterized by periostitis with suppuration, ulceration, necrosis, and severe deformity of the mandible and, less often, maxilla. Sequestration of bone may occur. Polymorphic leukopenia, susceptibility to bone fracture, and failure of the alveolar bone to resorb following extractions are secondary clinical signs. Carious teeth and poor dental hygiene increase susceptibility.

**MEDICAL SURVEILLANCE**

Special consideration should be given to the skin, eyes, jaws, teeth, respiratory tract, and liver. Preplacement medical and dental examination with X-ray of teeth is highly recommended in the case of yellow phosphorus exposure. Poor dental hygiene may increase the risk in yellow phosphorus exposures, and any required dental work should be completed before workers are assigned to areas of possible exposure. Workers experiencing any jaw injury, tooth extraction, or any abnormal dental conditions should be removed from areas of exposure and observed. Roentgenographic examinations may show necrosis; however, in order to prevent full development of sequestrae, the disease should be diagnosed in earlier stages. Liver function should be evaluated periodically. Pulmonary function tests may be useful when exposures are to the acid, chlorides, and sulfide compounds.

**SPECIAL TESTS**

None commonly used.

**PERSONAL PROTECTIVE METHODS**

Full body protective clothing including hat and face shield should be supplied to workers who may be exposed to spill, splashes, or spotters of phosphorus or phosphorus compounds. Inhalation of vapors or fumes can be prevented by proper ventilation in many cases, but in areas of higher concentration fullface mask respirators with proper canisters or supplied air respirators may be required. Continuing worker education of exposure risks for those in exposed areas is essential.

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**PLATINUM AND COMPOUNDS****DESCRIPTION**

Pt, platinum, is a soft, ductile, malleable, silver-white metal, insoluble in water and organic solvents. It is found in the metallic form

and as the arsenide, sperrylite. It forms complex soluble salts such as  $\text{Na}_2\text{PtCl}_6$ .

#### SYNONYMS

None.

#### POTENTIAL OCCUPATIONAL EXPOSURES

Platinum and its alloys are utilized because of their resistance to corrosion and oxidation, particularly at high temperatures, their high electrical conductivity, and their excellent catalytic properties. They are used in relays, contacts and tubes in electronic equipment, in spark plug electrodes for aircraft, and windings in high temperature electrical furnaces. Platinum alloys are used for standards for weight, length, and temperature measurement. Platinum and platinum catalysts (e.g., hexachloroplatinic acid,  $\text{H}_2\text{PtCl}_6$ ) are widely used in the chemical industry in persulfuric, nitric, and sulfuric acid production, in the synthesis of organic compounds and vitamins, and for producing higher octane gasoline. They are coming into use in catalyst systems for control of exhaust pollutants from automobiles. They are used in the equipment for handling molten glass and manufacturing fibrous glass; in laboratory, medical, and dental apparatus; in electroplating; in photography; in jewelry; and in X-ray fluorescent screens.

A partial list of occupations in which exposure may occur includes:

Alloy makers	Gasoline additive makers
Catalyst workers	Indelible ink makers
Ceramic workers	Jewelry makers
Dental alloy makers	Laboratory ware makers
Drug makers	Mirror makers
Electronic equipment makers	Spark plug makers
Electroplaters	Zinc etchers

#### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for soluble salts of platinum is  $0.002 \text{ mg/m}^3$  expressed as Pt.

#### ROUTE OF ENTRY

Inhalation of dust or mist.

#### HARMFUL EFFECTS

##### *Local—*

Hazards arise from the dust, droplets, spray, or mist of complex salts of platinum, but not from the metal itself. These salts are sensitizers of the skin, nasal mucosa, and bronchi, and cause allergic phenomena. One case of contact dermatitis from wearing a ring made of platinum alloy is recorded.

##### *Systemic—*

Characteristic symptoms of poisoning occur after 2 to 6 months' exposure and include pronounced irritation of the throat and nasal pas-

sages, which result in violent sneezing and coughing; bronchial irritation, which causes respiratory distress; and irritation of the skin, which produces cracking, bleeding, and pain. Respiratory symptoms can be so severe that exposed individuals may develop status asthmaticus. After recovery, most individuals develop allergic symptoms and experience further asthma attacks when exposed to even minimal amounts of platinum dust or mists. Mild cases of dermatitis involve only erythema and urticaria of the hands and forearms. More severe cases affect the face and neck. All pathology is limited to allergic manifestations.

#### MEDICAL SURVEILLANCE

In preemployment and periodic physical examinations, the skin, eyes, and respiratory tract are most important. Any history of skin or pulmonary allergy should be noted, as well as exposure to other irritants or allergens, and smoking history. Periodic assessment of pulmonary function may be useful.

#### SPECIAL TESTS

None commonly used.

#### PERSONAL PROTECTIVE METHODS

In areas where dust or mist are excessive, masks or air supplied respirators should be supplied. Where droplets, mist, or spray are encountered, impervious protective clothing, gloves, and goggles should be supplied.

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## SELENIUM AND COMPOUNDS

#### DESCRIPTION

Se, selenium, exists in three forms: a red amorphous powder, a grey form, and red crystals. Selenium, along with tellurium, is found in the sludges and sediments from electrolytic copper refining. It may also be recovered in flue dust from burning pyrites in sulfuric acid manufacture.

#### SYNONYMS

None.

#### POTENTIAL OCCUPATIONAL EXPOSURES

Most of the selenium produced is used in the manufacture of selenium rectifiers. It is utilized as a pigment for ruby glass, paints, and dyes, as a vulcanizing agent for rubber, a decolorized agent for green

glass, a chemical catalyst in the Kjeldahl test, and an insecticide; in the manufacture of electrodes, selenium photocells, selenium cells, and semiconductor fusion mixtures; in photographic toning baths; and for dehydrogenation of organic compounds. Se is used in radioactive scanning of the pancreas and for photostatic and X-ray xerography. It may be alloyed with stainless steel, copper, and cast steel.

Hydrogen selenide (selenium hydride,  $H_2Se$ ) is a colorless gas with a very disagreeable odor which is soluble in water. It is not used commercially. However, it may be produced by the reaction of acids or water and metal selenides or hydrogen and soluble selenium compounds. Selenium hexafluoride ( $SeF_6$ ) is a gas and is utilized as a gaseous electric insulator. Other selenium compounds are used as solvents, plasticizers, reagents for alkaloids, and flameproofing agents for textiles and wire-cable coverings.

Selenium is a contaminant in most sulfide ores of copper, gold, nickel, and silver, and exposure may occur while removing selenium from these ores.

A partial list of occupations in which exposure may occur includes:

Arc light electrode makers	Pigment makers
Copper smelters	Plastic workers
Electric rectifier makers	Pyrite roasters
Glass makers	Rubber makers
Organic chemical synthesizers	Semiconductor makers
Pesticide makers	Sulfuric acid makers
Photographic chemical makers	Textile workers

#### PERMISSIBLE EXPOSURE LIMITS

The Federal standards are: Selenium compounds (as Se): 0.2 mg/m<sup>3</sup>. Selenium hexafluoride: 0.05 ppm, 0.4 mg/m<sup>3</sup>. Hydrogen selenide: 0.05 ppm, 0.2 mg/m<sup>3</sup>.

#### ROUTES OF ENTRY

Inhalation of dust or vapor; percutaneous absorption of liquid; ingestion.

#### HARMFUL EFFECTS

##### *Local—*

Elemental selenium is considered to be relatively nonirritating and is poorly absorbed. Some selenium compounds (particularly selenium dioxide and selenium oxychloride) are strong vesicants and can cause destruction of the skin. They are strong irritants to the upper respiratory tract and eyes, and may cause irritation of the mucous membrane of the stomach. Selenium compounds also may cause dermatitis of exposed areas. Allergy to selenium dioxide has been reported in the form of an urticarial generalized rash, and may cause a pink discoloration of the eyelids and palpebral conjunctivitis ("rose-eye"). Selenium oxide also may penetrate under the free edge of the nail, causing excruciatingly painful nail beds and painful paronychia. Selenium compounds may be

absorbed through intact skin to produce systemic effects (Se sulfide in shampoo).

Selenium is considered to be an essential trace element for rats and chickens, and there is strong evidence of its essentiality in man. It is capable of antagonizing the toxic effects of certain other metals, e.g., As and Cd.

#### *Systemic—*

The effects of hydrogen selenide intoxication are similar to those caused by other irritating gases in industry: irritation of the mucous membranes of the nose, eyes, and upper respiratory tract, followed by slight tightness in the chest. These symptoms clear when the worker is removed from the exposed area. In some cases, however, pulmonary edema may develop suddenly after a latent period of six to eight hours following exposure. Selenium dioxide inhaled in large quantities may also produce pulmonary edema.

The first and most characteristic sign of selenium absorption is a garlic odor of the breath. This may be related to the excretion in the breath of small amounts of dimethyl selenide. This odor dissipates completely in seven to ten days after the worker is removed from the exposure. It cannot be relied upon as a certain guide to selenium absorption. A more subtle and earlier sign is a metallic taste in the mouth, but many workers accept this without complaint. Other systemic effects are less specific: pallor, lassitude, irritability, vague gastrointestinal symptoms (indigestion), and giddiness. Vital organs appear to escape harm from selenium absorption, but, based on the results of animal experimentation, liver and kidney damage should be regarded as possible. Liver damage and other effects have been long recognized in livestock grazing on high selenium soils. Selenium has been mentioned for its carcinogenic, anticarcinogenic, and teratogenic effects, but, to date, these effects have not been seen in man.

#### MEDICAL SURVEILLANCE

Preemployment and periodic examinations should consider especially the skin and eyes as well as liver, respiratory and kidney disease and function. The fingernails should be examined.

#### SPECIAL TESTS

Urinary selenium excretion has been used to indicate exposure in the environment and also occupational exposure. It varies with the Se content of the diet and geographic location. Dimethyl selenide can be determined in breath.

#### PERSONAL PROTECTIVE METHODS

Protective clothing with special emphasis on personal hygiene (showering and care of fingernails) should help prevent skin exposure and sensitization. Masks and supplied air respirators are needed in areas where concentrations of dust and vapors exceed the allowable standards. These should be equipped with fullface plates. Work clothing

should be changed daily and showering encouraged prior to change to street clothing.

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## STIBINE

#### DESCRIPTION

$SbH_3$ , stibine, is a colorless gas with a characteristic disagreeable odor. It is produced by dissolving zinc-antimony or magnesium-antimony in hydrochloric acid.

#### SYNONYMS

Antimony hydride.

#### POTENTIAL OCCUPATIONAL EXPOSURES

Stibine is used as a fumigating agent. Exposure to stibine usually occurs when stibine is released from antimony-containing alloys during the charging of storage batteries, when certain antimonial drosses are treated with water or acid, or when antimony-containing metals come in contact with acid. Operations generally involved are metallurgy, welding or cutting with blow torches, soldering, filling of hydrogen balloons, etching of zinc, and chemical processes.

A partial list of occupations in which exposure may occur includes:

Etchers	Storage battery workers
Solderers	Welders

#### PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 0.1 ppm (0.5 mg/m<sup>3</sup>).

#### ROUTE OF ENTRY

Inhalation of gas.

#### HARMFUL EFFECTS

##### *Local*—

No local effects have been noted.

##### *Systemic*—

Stibine is a powerful hemolytic and central nervous system poison. In acute poisoning, the symptoms are severe headache, nausea, weakness, abdominal and lumbar pain, slow breathing, and weak, irregular

pulse. One of the earliest signs of overexposure may be hemoglobinuria. Laboratory studies may show a profound hemolytic anemia. Death is preceded by jaundice and anuria. Chronic stibine poisoning in man has not been reported.

#### MEDICAL SURVEILLANCE

In preemployment and periodic examinations special attention should be given to significant blood, kidney, and liver diseases. The general health of exposed workmen should be evaluated periodically. Blood hemoglobin and urine tests for hemoglobin on persons suspected of stibine overexposure are indicated. Workers should also be advised to immediately report any red or dark urinary discoloration to the medical department. This frequently is the initial sign of stibine poisoning. (See Arsine).

#### SPECIAL TESTS

None in common use.

#### PERSONAL PROTECTIVE METHODS

In areas where stibine gas is suspected, all persons entering or working in the area should be provided with fullface gas masks or supplied air respirators.

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## SILVER AND COMPOUNDS

#### DESCRIPTION

Ag, silver, is a white metal and is extremely ductile and malleable, insoluble in water but soluble in hot sulfuric and nitric acids.

#### SYNONYMS

None.

#### POTENTIAL OCCUPATIONAL EXPOSURES

Silver may be alloyed with copper, aluminum, cadmium, lead, or antimony; the alloys are used in the manufacture of silverware, jewelry, coins, ornaments, plates, commutators, scientific instruments, automobile bearings, and grids in storage batteries. Silver is used in chrome-nickel steels, in solders and brazing alloys, in the application of metallic films on glass and ceramics, to increase corrosion resistance to sulfuric acid, in photographic films, plates and paper, as an electroplated undercoating for nickel and chrome, as a bactericide for sterilizing water, fruit juices, vinegar, etc., in busbars and windings in electrical plants, in dental amal-

gams, and as a chemical catalyst in the synthesis of aldehydes. Because of its resistance to acetic and other food acids, it is utilized in the manufacture of pipes, valves, vats, pasteurizing coils and nozzles for the milk, vinegar, cider, brewing, and acetate rayon silk industries.

Silver compounds are used in photography, silver plating, inks, dyes, coloring glass and porcelain, etching ivory, in the manufacture of mirrors, and as analytical chemical reagents and catalysts. Some of the compounds are also of medical importance as antiseptics or astringents, and in the treatment of certain diseases, particularly in veterinary medicine.

A partial list of occupations in which exposure may occur includes:

Alloy makers	Glass makers
Bactericide makers	Hair dye makers
Ceramic makers	Hard solder workers
Coin makers	Ivory etchers
Chemical laboratory workers	Mirror makers
Dental alloy makers	Organic chemical makers
Drug makers	Photographic workers
Electric equipment makers	Water treaters
Food product equipment makers	

#### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for silver metal and soluble compounds is 0.01 mg/m<sup>3</sup>.

#### ROUTES OF ENTRY

Inhalation of fumes or dust; ingestion of solutions or dust.

#### HARMFUL EFFECTS

##### *Local—*

The only local effect from metallic silver derives from the implant of small particles in the skin of the workmen (usually hands and fingers) which causes a permanent discoloration equivalent to the process of tattooing (local argyria). Silver nitrate dust and solutions are highly corrosive to the skin, eyes, and intestinal tract. The dust of silver nitrate may cause local irritation of the skin, burns of the conjunctiva, and blindness. Localized pigmentation of the skin and eyes may occur. The eye lesions are seen first in the caruncle, and then in the conjunctiva and cornea. The nasal septum and tonsillar pillars also are pigmented.

##### *Systemic—*

All forms of silver are extremely cumulative once they enter body tissues, and very little is excreted. Studies on the occurrence of argyria following injection of silver arsphenamine indicate that the onset of visible argyria begins at a total dose of about 0.9 grams of silver. Generalized argyria develops when silver oxide or salts are inhaled or possibly ingested by workmen who handle compounds of silver (nitrate, fulminate, or cyanide). The condition produces no constitutional symp-

toms, but it may lead to permanent pigmentation of the skin and eyes. The workman's face, forehead, neck, hands, and forearms develop a dark, slate-grey color, uniform in distribution and varying in depth depending on the degree of exposure. Fingernails, buccal mucosa, toenails, and covered parts of the body to a lesser degree, can also be affected by this discoloration process. The dust is also deposited in the lungs and may be regarded as a form of pneumoconiosis, although it carries no hazard of fibrosis. The existence of kidney lesions of consequence to renal function is improbable from occupational exposure.

#### MEDICAL SURVEILLANCE

Special attention should be given to other sources of silver exposure, e.g., medications or previous occupational exposure. Inspection of the nasal septum, eyes, and throat will generally give incidence of pigmentation before generalized argyria occurs. This will usually be seen first in the ear lobes, face, and hands.

#### SPECIAL TESTS

Silver is excreted principally in the feces. Urine and blood levels have not been found useful in monitoring.

#### PERSONAL PROTECTIVE METHODS

Workers involved with silver nitrate solution should be protected from spills and splashes by impervious protective clothing and chemical goggles. In areas of excessive dust levels, masks with fullface plates should be worn. Clean clothing should be provided daily and meals eaten in noncontaminated areas. Showers should be taken after each shift before change to street clothes.

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## TELLURIUM AND COMPOUNDS

#### DESCRIPTION

Te, tellurium, is a semimetallic element with a bright lustre which is insoluble in water and organic solvents. It may exist in a hexagonal crystalline form or an amorphous powder. It is found in sulfide ores and is produced as a by-product of copper or bismuth refining.

#### SYNONYMS

Aurum paradoxum, metallum problematum.

#### POTENTIAL OCCUPATIONAL EXPOSURES

The primary use of tellurium is in the vulcanization of rubber. It is also used as a carbide stabilizer in cast iron, a chemical catalyst, a coloring agent in glazes and glass, a thermocoupling material in refrig-

erating equipment, and as an additive to selenium rectifiers; in alloys of lead, copper, steel, and tin for increased resistance to corrosion and stress, workability, machinability, and creep strength, and in certain culture media in bacteriology. Since tellurium is present in silver, copper, lead, and bismuth ores, exposure may occur during purification of these ores.

A partial list of occupations in which exposure may occur includes:

Alloy makers	Lead refinery workers
Ceramic makers	Porcelain makers
Copper refinery workers	Rubber workers
Electronic workers	Semiconductor makers
Enamel makers	Silverware makers
Foundry workers	Stainless steel makers
Glass makers	Thermoelectric device makers

#### PERMISSIBLE EXPOSURE LIMITS

The applicable Federal standards are: Tellurium: 0.1 mg/m<sup>3</sup>.  
Tellurium hexafluoride: 0.02 ppm (mg/m<sup>3</sup>).

#### ROUTES OF ENTRY

Inhalation of dust or fume; percutaneous absorption from dust.

#### HARMFUL EFFECTS

##### *Local—*

The literature contains no indication of any local effect from tellurium.

##### *Systemic—*

The toxicity of tellurium and its compounds is of a low order. There is no indication that either tellurium dust or fume is damaging to the skin or lungs. Inhalation of fumes may cause symptoms, however, some of which are particularly annoying socially to the worker. The most common sign of exposure are foul (garliclike) breath and perspiration, metallic taste in the mouth, and dryness. This is probably due to the presence of dimethyl telluride. These symptoms may appear after relatively short exposures at high concentrations, or longer exposures at lower concentrations, and may persist for long periods of time after the exposure has ended. Workers also complain of afternoon somnolence and loss of appetite.

Exposure to hydrogen telluride produces symptoms of headache, malaise, weakness, dizziness, and respiratory and cardiac symptoms similar to those caused by hydrogen selenide. Pulmonary irritation and the destruction of red blood cells have been reported in studies of laboratory animals exposed to hydrogen telluride.

In other animal studies, tellurium hexafluoride was found to be a respiratory irritant which caused pulmonary edema, and metallic tellurium was shown to have a teratogenic effect on the fetus of rats.

**MEDICAL SURVEILLANCE**

Oral hygiene and the respiratory tract should receive special attention in replacement or periodic examinations.

**SPECIAL TESTS**

Urinary tellurium excretion has been studied in relation to exposure, but is of uncertain value.

**PERSONAL PROTECTIVE METHODS**

Clean change of work clothes is necessary for hygienic purposes, and showering after each shift before change to street clothes should be encouraged. Respiratory protection is indicated in areas where exposure to hydrogen telluride and tellurium hexafluoride fumes and dust are above the allowable limits.

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**THALLIUM AND COMPOUNDS****DESCRIPTION**

Tl, thallium, is a soft, heavy metal insoluble in water and organic solvents. It is usually obtained as a by-product from the flue dust generated during the roasting of pyrite ores in the smelting and refining of lead and zinc.

**SYNONYMS**

None.

**POTENTIAL OCCUPATIONAL EXPOSURES**

Thallium and its compounds are used as rodenticides, fungicides, insecticides, catalysts in certain organic reactions, and phosphor activators, in bromiodide crystals for lenses, plates, and prisms in infrared optical instruments, in photoelectric cells, in mineralogical analysis, alloyed with mercury in low temperature thermometers, switches and closures, in high-density liquids, in dyes and pigments, and in the manufacture of optical lenses, fireworks, and imitation precious jewelry. It forms a stainless alloy with silver and a corrosion-resistant alloy with lead. Its medicinal use for epilation has been almost discontinued.

A partial list of occupations in which exposure may occur includes:

Alloy makers	Glass makers
Artificial diamond makers	High refractive index makers
Chlorinated compound makers	Infrared instrument makers
Dye makers	Optical glass makers
Fireworks makers	Photoelectric cell makers
Gem makers	Rodenticide workers

#### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for thallium (soluble compounds) is 0.1 mg Tl/m<sup>3</sup>.

#### ROUTES OF ENTRY

Inhalation of dust and fume. Ingestion and percutaneous absorption of dust.

#### HARMFUL EFFECTS

##### *Local*—

Thallium salts may be skin irritants and sensitizers, but these effects occur rarely in industry.

##### *Systemic*—

Thallium is an extremely toxic and cumulative poison. In nonfatal occupational cases of moderate or long term exposure, early symptoms usually include fatigue, limb pain, metallic taste in the mouth and loss of hair, although loss of hair is not always present as an early symptom. Later, peripheral neuritis, proteinuria, and joint pains occur.

Occasionally, neurological signs are the presenting factor, especially in more severe poisonings. Long term exposure may produce optic atrophy, paraesthesias, and changes in pupillary and superficial tendon reflexes (slowed responses). Acute poisoning rarely occurs in industry, and is usually due to ingestion of thallium. When it occurs, gastrointestinal symptoms, abdominal colic, loss of kidney function, peripheral neuritis, strabismus, disorientation, convulsions, joint pain, and alopecia develop rapidly (within 3 days). Death is due to damage to the central nervous system.

#### MEDICAL SURVEILLANCE

Preplacement and periodic examinations should give special consideration to the central nervous system, gastrointestinal symptoms, and liver and kidney function. Hair loss may be a significant sign. Urine examinations may be helpful.

#### SPECIAL TESTS

Thallium has been determined in the urine, but the levels do not relate to degree, exposure, or to symptoms.

#### PERSONAL PROTECTIVE METHODS

Eating, gum chewing, and smoking should not be allowed in pro-

duction areas. Strict enforcement of high standards of personal hygiene is recommended. Appropriate respiratory protection should be used. Protective clothing, hats, goggles, and gloves may be needed to prevent dust absorption through the skin. Daily change of work clothes and showers at the end of the shift will reduce the chances of significant absorption.

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## THORIUM AND COMPOUNDS

#### DESCRIPTION

Th, thorium, is a natural radioactive element insoluble in water and organic solvents. It occurs in the minerals monazite, thorite, and thorianite, usually mixed with its distintegration products.

#### SYNONYMS

None.

#### POTENTIAL OCCUPATIONAL EXPOSURES

Metallic thorium is used in nuclear reactors to produce nuclear fuel, in the manufacture of incandescent mantles, as an alloying material, especially with some of the lighter metals, e.g., magnesium, as a reducing agent in metallurgy, for filament coatings in incandescent lamps and vacuum tubes, as a catalyst in organic synthesis, in ceramics, and in welding electrodes.

Exposures may occur during production and use of thorium-containing materials, in the casting and machining of alloy parts, and from the fume produced during welding with thorium electrodes.

A partial list of occupations in which exposure may occur includes:

Ceramic makers	Metal refiners
Gas mantle makers	Nuclear reactor workers
Incandescent lamp makers	Organic chemical synthesizers
Magnesium alloy makers	Vacuum tube makers

#### PERMISSIBLE EXPOSURE LIMITS

Maximum permissible concentration for thorium under the Federal standard (see 20 CFR Part 20-Table 1) is  $1 \times 10^{-6}$   $\mu\text{Ci}/\text{ml}$  (air).

#### ROUTES OF ENTRY

Ingestion of liquid, inhalation of dust or gas, and percutaneous absorption.

#### HARMFUL EFFECTS

##### *Local*—

Thorium and thorium compounds are relatively inert, but some

irritant effect may occur depending on the anion present. Gas and aerosols can penetrate the body by way of the respiratory system, the digestive system, and the skin.

#### *Systemic—*

Thorium and its compounds are toxicologically inert on the basis of its chemical toxicity. Only 0.001% of an ingested dose is retained in the body. Thorium, once deposited in the body, remains for long periods of time. It has a predilection for bones, lungs, lymphatic glands, and parenchymatous tissues. Characteristic effects of the activity of thorium and its disintegration products are changes in blood forming, nervous, and reticuloendothelial systems, and functional and morphological damage to lung and bone tissue. Only much later do illness and symptoms characteristic of chronic radiation disease appear. After a considerable time, neoplasms may occur and the immunological activity of the body may be reduced. External radiation with gamma rays can occur from contact with material containing mesothorium, with thorium in large quantities, and with by-products that contain disintegration products of thorium. Thorium dioxide (thorotrast) is known to cause severe radiation damage and cancer of bone, blood vessels, liver, and other organs when administered to patients for diagnostic purposes. Its use is now forbidden for introduction into body tissues. Workers in plants where thorium dioxide is produced have not experienced either chemical or radiation injury.

#### MEDICAL SURVEILLANCE

Monitoring of personnel for early symptoms and changes such as abnormal leukocytes in the blood smear may be of value.

#### SPECIAL TESTS

In cases of chronic or acute exposure, the determination of thorium in the urine or the use of whole body radiation counts and breath radon are useful methods of monitoring the exposure dose and excretion rates.

#### PERSONAL PROTECTIVE METHODS

Protection of the worker is afforded by respiratory protection with either dust masks, special canister gas masks, or supplied air respirators. Protective clothing and gloves to prevent dust settling on the skin, with daily change of work clothes, and showering after each shift before change to street clothes should be routine.

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## TIN AND COMPOUNDS

### DESCRIPTION

Sn, tin, is a soft, silvery-white metal insoluble in water. The primary commercial source of tin is cassiterite ( $\text{SnO}_2$  tinstone).

### SYNONYMS

Stannum.

### POTENTIAL OCCUPATIONAL EXPOSURES

The most important use of tin is as a protective coating for other metals such as in the food and beverage canning industry, in roofing tiles, silverwares, coated wire, household utensils, electronic components, and pistons. Common tin alloys are phosphor bronze, light brass, gun metal, high tensile brass, manganese bronze, die-casting alloys, bearing metals, type metal, and pewter. These are used as soft solders, fillers in automobile bodies, and as coatings for hydraulic brake parts, aircraft landing gear and engine parts. Metallic tin is used in the manufacture of collapsible tubes and foil for packaging.

Organic and inorganic tin compounds are important industrially in the production of drill-glass, ceramics, porcelain, enamel, glass, and inks; as a mordant it is important in the production of fungicides, anthelmintics, insecticides; as a stabilizer it is used in polyvinyl plastics and chlorinated rubber paints; and it is used in plating baths.

Exposures to tin may occur in mining, smelting, and refining, and in the production and use of tin alloys and solders.

A partial list of occupations in which exposure may occur includes:

- Babbitt metal (tin, copper, antimony) makers
- Brass (essentially copper and zinc) founders
- Britannia metal (tin, copper, antimony) makers
- Bronze (tin, copper) founders
- Dye workers
- Fungicide workers
- Pewter makers
- Pigment workers
- Plastic makers
- Solder makers
- Textile workers
- Type metal (lead, antimony, tin) makers

### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for organic tin compounds is  $0.1 \text{ mg/m}^3$  and for inorganic compounds excluding the oxides it is  $2.0 \text{ mg/m}^3$ .

### ROUTES OF ENTRY

Inhalation of dust. Ingestion, inhalation, or percutaneous absorption of organo-tins.

## HARMFUL EFFECTS

*Local—*

Certain inorganic tin salts are mild irritants to the skin and mucous membranes. They may be strongly acid or basic depending on the cation or anion present. Organic tin compounds, especially tributyl and dibutyl compounds, may cause acute burns to the skin. The burns produce little pain but may itch. They heal without scarring. Clothing contaminated by vapors or liquids may cause subacute lesions and diffuse erythematoid dermatitis on the lower abdomen, thighs, and groin of workmen who handle these compounds. The lesions heal rapidly on removal from contact. The eyes are rarely involved, but accidental splashing with tributyl tin has caused lacrimation and conjunctival edema which lasted several days; there was no permanent injury.

*Systemic—*

Exposure to dust or fumes of inorganic tin is known to cause a benign pneumoconiosis (stannosis). This form of pneumoconiosis produces distinctive progressive X-ray changes of the lungs as long as exposure persists, but there is no distinctive fibrosis, no evidence of disability, and no special complicating factors. Because tin is so radio-opaque, early diagnosis is possible.

Certain organic tin compounds, especially alkyltin compounds, are highly toxic when ingested. The trialkyl and tetraalkyl compounds cause damage to the central nervous system with symptoms of headaches, dizziness, photophobia, vomiting, and urinary retention, some weakness and flacid paralysis of the limbs in the most severe cases. Percutaneous absorption of these compounds has been postulated, but to date, deaths and serious injury have resulted only from ill-advised attempts at therapeutic use by mouth. The mechanism of action of the organo-tins is not clearly understood, although triethyltin is an extremely potent inhibitor of oxidative phosphorylation. Occasionally, mild organo-tin intoxication is seen in chemical laboratories with headache, nausea, and EEG changes.

## MEDICAL SURVEILLANCE

In the case of inorganic tin compounds, the skin and eyes are of particular interest. Chest X-rays may reveal that exposures have occurred. For organo-tins, preplacement and periodic examinations should include the skin, eyes, blood, central nervous system, and liver and kidney function.

## SPECIAL TESTS

None in use.

## PERSONAL PROTECTIVE METHODS

It is important that employees be trained in the correct use of personal protective equipment. Skin contact should be prevented by protective clothing, and, especially in the case of organic tin compounds, clean work clothes should be supplied daily and the worker required to

shower following the shift and prior to change to street clothes. In all areas of dust concentration, dust masks should be provided, and in the case of fumes, masks with proper canisters or supplied air respirators should be used.

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## TITANIUM AND COMPOUNDS

#### DESCRIPTION

Ti, titanium, is a dark-grey, lustrous metal insoluble in water. It is brittle when cold and malleable when hot. The most important minerals containing titanium are ilmenite, rutile, perovskite, and titanite or sphene.

#### SYNONYMS

None.

#### POTENTIAL OCCUPATIONAL EXPOSURES

Titanium metal, because of its low weight, high strength, and heat resistance, is used in the aerospace and aircraft industry as tubing, fittings, fire walls, cowlings, skin sections, and jet compressors, and it is also used in surgical appliances. It is used, too, as control-wire casings in nuclear reactors, as a protective coating for mixers in the pulp-paper industry and in other situations in which protection against chlorides or acids is required, in vacuum lamp bulbs and X-ray tubes, as an addition to carbon and tungsten in electrodes and lamp filaments, and to the powder in the pyrotechnics industry. It forms alloys with iron, aluminum, tin, and vanadium of which ferrotitanium is especially important in the steel industry.

Titanium dioxide (TiO<sub>2</sub>, rutile, anatase, titania) is a white pigment in the rubber, plastics, ceramics, paint, and varnish industries, in dermatological preparations, and is used as a starting material for other titanium compounds, as a gem, in curing concrete, and in coatings for welding rods.

Other titanium compounds are utilized in smoke screens, as mordants in dyeing, in the manufacture of cemented metal carbides, as thermal insulators, and in heat resistant surface coatings in paints and plastics.

A partial list of occupations in which exposure may occur includes:

Ceramic makers	Paper makers
Glass makers	Plastic makers
Incandescent lamp makers	Rayon makers
Ink makers	Smoke screen makers
Lacquer makers	Steel workers
Nuclear steel makers	Vacuum tube makers
Paint makers	Welding rod makers

#### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for titanium dioxide is 15 mg/m<sup>3</sup>. There is no standard for titanium itself or other titanium compounds.

#### ROUTE OF ENTRY

Inhalation of dust or fume.

#### HARMFUL EFFECTS

##### *Local—*

Titanium and titanium compounds are, for the most part, virtually inert and not highly toxic to man. Titanium tetrachloride, which is released into the air during maintenance of chlorinating and rectifying operations, is an exception. Titanium tetrachloride and its hydrolysis products are highly toxic and irritating. Skin exposure may cause irritation and burns, and even brief contact with the eyes may cause suppurating conjunctivitis and keratitis, followed by clouding of the cornea.

##### *Systemic—*

During the production of titanium metal, it is possible that the air may be contaminated with chlorine, hydrogen chloride, titanium tetrachloride, and similar harmful constituents. Reports of severe lung injury caused by such exposures have been recorded; in some cases the condition resembles silicotic lungs. Reports of pulmonary fibrosis due to titanium carbide are now mostly discounted, but precautions are still recommended. Titanium tetrachloride may cause injury to the upper respiratory tract and acute bronchitis.

#### MEDICAL SURVEILLANCE

Preemployment and periodic physical examinations should give special attention to lung disease, especially if irritant compounds are involved. Chest X-rays should be included in both examinations and pulmonary function evaluated periodically. Smoking history should be taken. Careful attention should be given to the eyes and the skin.

#### PERSONAL PROTECTIVE METHODS

Employees exposed to titanium tetrachloride should wear protective clothing and respirators. In areas of dust or fumes of titanium tetrachloride, all workers should be provided with goggles and dust masks, fullface gas masks, or supplied air respirators. Clothing should be

changed daily to avoid dust inhalation from clothing, and employees should be encouraged to shower before changing to street clothes.

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## URANIUM AND COMPOUNDS

#### DESCRIPTION

U, uranium, is a hard, silvery-white amphoteric metal and is a radioactive element. In the natural state, it consists of three isotopes:  $U^{238}$  (99.28%),  $U^{234}$  (0.006%), and  $U^{235}$  (0.714%). There are over one hundred uranium minerals; those of commercial importance are the oxides and oxygenous salts. The processing of uranium ore generally involves extraction then leaching either by an acid or a carbonate method. The metal may be obtained from its halides by fused salt electrolysis.

#### SYNONYMS

None.

#### POTENTIAL OCCUPATIONAL EXPOSURES

The primary use of natural uranium is in nuclear energy as a fuel for nuclear reactors, in plutonium production, and as feeds for gaseous diffusion plants. It is also a source of radium salts. Uranium compounds are used in staining glass, glazing ceramics, and enamelling, in photographic processes, for alloying steels, and as a catalyst for chemical reactions, radiation shielding, and aircraft counterweights.

Uranium presents both chemical and radiation hazards, and exposures may occur during mining, processing of the ore, and production of uranium metal.

A partial list of occupations in which exposure may occur includes:

Atomic bomb workers	Hydrogen bomb workers
Ceramic makers	Nuclear reactor workers
Glass makers	Photographic chemical makers

#### PERMISSIBLE EXPOSURE LIMITS

The Federal standards are: uranium, soluble compounds, 0.05 mg/m<sup>3</sup>; and uranium, insoluble compounds, 0.25 mg/m<sup>3</sup>.

#### ROUTES OF ENTRY

Inhalation of fume, dust, or gas. The following uranium salts are reported to be capable of penetrating intact skin:

Uranyl nitrate,  $UO_2(NO_3)_2 \cdot 6H_2O$ .

Uranyl fluoride,  $UO_2F_2$ .

Uranium pentachloride,  $UCl_5$ .

Uranium trioxide (uranyl oxide),  $UO_3$ .

Sodium diuranate (sodium uranate (VI),  $Na_2U_2O_7 \cdot H_2O$ ).

Ammonium diuranate (ammonium uranate (VI) ( $NH_4$ )<sub>2</sub> $U_2O_7$ ).

Uranium hexafluoride,  $UF_6$ .

#### HARMFUL EFFECTS

##### *Local*—

No toxic effects have been reported, but prolonged contact with skin should be avoided to prevent radiation injury.

##### *Systemic*—

Uranium and its compounds are highly toxic substances. The compounds which are soluble in body fluids possess the highest toxicity. Poisoning has generally occurred as a result of accidents. Acute chemical toxicity produces damage primarily to the kidneys. Kidney changes precede in time and degree the effects on the liver. Chronic poisoning with prolonged exposure gives chest findings of pneumoconiosis, pronounced blood changes, and generalized injury.

It is difficult to separate the toxic chemical effects of uranium and its compounds from their radiation effects. The chronic radiation effects are similar to those produced by ionizing radiation. Reports now confirm that carcinogenicity is related to dose and exposure time. Cancer of the lung, osteosarcoma, and lymphoma have all been reported.

#### MEDICAL SURVEILLANCE

Special attention should be given to the blood, lung, kidney, and liver in preemployment physical examinations. In periodic examinations, tests for blood changes, changes in chest X-rays, or for renal injury and liver damage are advisable.

#### SPECIAL TESTS

Uranium excretion in the urine has been used as an index of exposure. Whole body counting may also be useful.

#### PERSONAL PROTECTIVE METHODS

It is important that a formal monitoring system be established to measure each employee's exposure to uranium. This industry has an excellent record of safety to this hazardous material because of good industrial hygiene practices and monitoring of work practices. Protective clothing, gloves, and respirators are necessary in cases of spills and accidents, and must be worn when dealing with soluble compounds in open systems. Closed systems are essential because of the carcinogenic effects.

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## VANADIUM AND COMPOUNDS

### DESCRIPTION

V, vanadium, is a light grey or white, lustrous powder or fused hard lump insoluble in water. It is produced by roasting the ores, thermal decomposition of the iodide, or from petroleum residues, slags from ferrovanadium production, or soot from oil burning.

### SYNONYMS

None.

### POTENTIAL OCCUPATIONAL EXPOSURES

Most of the vanadium produced is used in ferrovanadium and of this, the majority is used in high speed and other alloy steels with only small amounts in tool or structural steels. It is usually combined with chromium, nickel, manganese, boron, and tungsten in steel alloys.

Vanadium pentoxide ( $V_2O_5$ ) is an industrial catalyst in oxidation reactions, is used in glass and ceramic glazes, is a steel additive, and is used in welding electrode coatings. Ammonium metavanadate ( $NH_4VO_3$ ) is used as an industrial catalyst, a chemical reagent, a photographic developer, and in dyeing and printing. Other vanadium compounds are utilized as mordants in dyeing, in insecticides, as catalysts, and in metallurgy.

Since vanadium itself is considered nontoxic, there is little hazard associated with mining; however, exposure to the more toxic compounds, especially the oxides, can occur during smelting and refining. Exposure may also occur in conjunction with oil-fired furnace flues.

A partial list of occupations in which exposure may occur includes:

Alloy makers	Glass makers
Ceramic makers	Organic chemical synthesizers
Dye makers	Photographic chemical makers
Ferrovanadium workers	Textile dye workers

### PERMISSIBLE EXPOSURE LIMITS

The Federal standards are:  $V_2O_5$  dust, 0.5 mg/m<sup>3</sup>, and  $V_2O_5$  fume, 0.1 mg/m<sup>3</sup>, both as ceiling values.

### ROUTE OF ENTRY

Inhalation of dust or fume.

### HARMFUL EFFECTS

#### *Local—*

Vanadium compounds, especially vanadium pentoxide, are irritants to the eyes and skin. The initial eye symptoms are profuse lacrimation and a burning sensation of the conjunctiva. Skin lesions are of the eczematous type which itch intensely. In some cases there may be generalized urticaria. Workers may also exhibit greenish discoloration of the tongue. This same discoloration may be detectable on the butts of cigarettes smoked by vanadium workers.

*Systemic—*

Vanadium compounds are irritants to the respiratory tract. Entrance to the body is through inhalation of dusts or fumes. Serous or hemorrhagic rhinitis, sore throat, cough, tracheitis, bronchitis, expectoration, and chest pain, may result after even a brief exposure. More serious exposure may result in pulmonary edema and pneumonia which may be fatal. Individuals who recover may experience persistent bronchitis resembling asthma, and bouts of dyspnea; however, no chronic lung lesions have been described.

The results of experimental biochemical studies show that vanadium compounds inhibit cholesterol synthesis and the activity of the enzyme cholinesterase. A variety of other biochemical effects have been noted experimentally, but these have not been reported in relation to occupational exposures. Slightly lower cholesterol levels in blood were noted in one report, but this seems of doubtful significance.

## MEDICAL SURVEILLANCE

Preemployment and periodic physical examinations should emphasize effects on the eyes, skin, and lungs.

## SPECIAL TESTS

Urinary vanadium excretion may be useful as an index of exposure.

## PERSONAL PROTECTIVE METHODS

Employees should receive training in personal hygiene and in the use of personal protective equipment. In certain areas, masks or respirators may be necessary to prevent inhalation of dust and fumes. Protective clothing and gloves will be helpful in preventing dermatitis. Showering after each shift before changing to street clothes is very important. Clean work clothes should be supplied daily.

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**ZINC CHLORIDE**

## DESCRIPTION

ZnCl<sub>2</sub>, zinc chloride, consists of white hexagonal, deliquescent crystals, soluble in water (1 gm/0.5 ml) and in organic solvents. It may be produced from zinc sulfide ore, zinc oxide, or zinc metal.

## SYNONYMS

Butter of zinc.

### POTENTIAL OCCUPATIONAL EXPOSURES

Zinc chloride is used as a wood preservative, for dry battery cells, as a soldering flux, and in textile finishing, in vulcanized fiber, reclaiming rubber, oil and gas well operations, oil refining, manufacture of parchment paper, dyes, activated carbon, chemical synthesis, dentists' cement, deodorants, disinfecting and embalming solutions, and taxidermy. It is also produced by military screening-smoke devices.

A partial list of occupations in which exposure may occur includes:

Activated carbon makers,	Military personnel
Dental cement makers	Paper makers
Deodorant makers	Petroleum refinery workers
Disinfectant makers	Rubber workers
Dry cell battery makers	Solderers
Dye makers	Taxidermists
Embalmers	Textile finishers

### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for zinc chloride fume is 1 mg/m<sup>3</sup>.

### ROUTES OF ENTRY

Inhalation of dust and fumes; ingestion.

### HARMFUL EFFECTS

#### *Local—*

Solid zinc chloride is corrosive to the skin and mucous membranes. Aqueous solutions of 10% or more are also corrosive and cause primary dermatitis and chemical burns, especially at sites of minor trauma. Aqueous solutions are also extremely dangerous to the eyes, causing extreme pain, inflammation, and swelling, which may be followed by corneal ulceration. Zinc chloride may produce true sensitization of the skin in the form of eczematoid dermatitis. Ingestion of zinc chloride may cause serious corrosive effects in the esophagus and stomach, often complicated by pyloric stenosis.

#### *Systemic—*

There are no reports of inhalation of zinc chloride from industrial exposure. All reported experience with inhaled zinc chloride is based on exposures caused by military accidents. In all of those cases, there was severe irritation of the respiratory tract. In the more severe cases, acute pulmonary edema developed within two to four days following exposure. The fatalities reported were due to severe lung injury with hemorrhagic alveolitis and bronchopneumonia. In human experimentation with concentrations of 120 mg/m<sup>3</sup>, there were complaints of irritation of the nose, throat, and chest after 2 minutes. With exposure to 80 mg/m<sup>3</sup> for 2 minutes, the majority of subjects experienced slight nausea, all noticed the smell, and one or two coughed.

### MEDICAL SURVEILLANCE

In preemployment and periodic physical examinations, special at-

tention should be given to the skin and to the history of allergic dermatitis, as well as to exposed mucous membranes, the eyes, and the respiratory system. Chest X-rays and periodic pulmonary function studies may be helpful. Smoking history should be known.

#### SPECIAL TESTS

Urinary zinc excretion may be useful.

#### PERSONAL PROTECTIVE METHODS

Employees exposed to zinc chloride should be given instruction in personal hygiene, and in the use of personal protective equipment. Goggles should be provided in areas where splash or spill of liquid is possible. In areas with excessive dust or fume levels, respiratory protection by use of filter type dust masks or air supplied respirators with fullface pieces should be required. In areas where danger of spills or splashes exists, skin protection should be provided with rubber gloves, face shields, rubber aprons, gauntlets, suits, and rubber shoes.

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## ZINC OXIDE

#### DESCRIPTION

ZnO, zinc oxide, is an amorphous, odorless, white or yellowish-white powder, practically insoluble in water. It is produced by oxidation of zinc or by roasting of zinc oxide ore.

#### SYNONYMS

Zinc white, flowers of zinc.

#### POTENTIAL OCCUPATIONAL EXPOSURES

Zinc oxide is primarily used as a white pigment in rubber formulations and as a vulcanizing aid. It is also used in photocopying, paints, chemicals, ceramics, lacquers, and varnishes, as a filler for plastics, in cosmetics, pharmaceuticals, and calamine lotion. Exposure may occur in the manufacture and use of zinc oxide and products, or through its formation as a fume when zinc or its alloys are heated.

A partial list of occupations in which exposure may occur includes:

Alloy makers	Lacquer makers
Brass foundry workers	Paint makers
Ceramic makers	Pigment makers
Chemical synthesizers	Plastic makers
Cosmetic makers	Rubber workers
Electroplaters	Welders
Galvanizers	

#### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for zinc oxide fume is 5 mg/m<sup>3</sup>.

**ROUTE OF ENTRY**

Inhalation of dust or fumes.

**HARMFUL EFFECTS***Local—*

When handled under poor hygienic conditions, zinc oxide powder may produce a dermatitis called "oxide pox." This condition is due primarily to clogging of the sebaceous glands with zinc oxide and produces a red papule with a central plug. The area rapidly becomes inflamed and the central plug develops into a pustule which itches intensely. Lesions occur in areas of the skin that are exposed or subject to heavy perspiration. These usually clear, however, in a week to ten days with good hygiene and proper care of secondary infections.

*Systemic—*

The syndrome of metal fume fever is the only important effect of exposure to freshly formed zinc oxide fumes and zinc oxide dusts of respirable particle size. The fumes are formed by subjecting either zinc or alloys containing zinc to high temperatures. Typically, the syndrome begins four to twelve hours after sufficient exposure to freshly formed fumes of zinc oxide. The worker first notices the presence of a sweet or metallic taste in the mouth, accompanied by dryness and irritation of the throat. Cough and shortness of breath may occur, along with general malaise, a feeling of weakness, fatigue, and pains in the muscles and joints. Fever and shaking chills then develop. Fever can range from 102 to 104 F. Profuse sweats develop and the fever subsides. The entire episode runs its course in 24 - 48 hours. During the acute period, there is an elevation of the leukocyte count (rarely above 20,000/ml), and the serum LDH may be elevated. Chest X-rays are not diagnostic.

Metal fume fever produces rapid development of tolerance or short-lived relative immunity. This may be lost, however, over a weekend or holiday, and the worker may again develop the complete syndrome when he returns to work if fume levels are sufficiently high. There are no sequelae to the attacks.

Other possible systemic effects of zinc oxide are in doubt. Cases of gastrointestinal disturbance have been reported, but most authorities agree there is no evidence of chronic industrial zinc poisoning.

**MEDICAL SURVEILLANCE**

Preemployment and periodic physical examinations should be made to assess the status of the general health of the worker. Examinations are also recommended following episodes of metal fume fever or intercurrent illnesses.

**SPECIAL TESTS**

Zinc excretion in urine can be used as an index of exposure.

**PERSONAL PROTECTIVE METHODS**

Employees should receive instruction in personal hygiene and in

the causes and effects of metal fume fever. Workers exposed to zinc oxide powder should be supplied with daily clean work clothes and should be required to shower before changing to street clothes. In cases of accident or where excessive fume concentrations are present, gas masks with proper canister or supplied air respirators should be provided.

## ZIRCONIUM AND COMPOUNDS

### DESCRIPTION

Zr, zirconium, is a greyish-white, lustrous metal in the form of platelets, flakes, or a bluish-black, amorphous powder. It is never found in the free state; the most common sources are the ores zircon and baddeleyite. It is generally produced by reduction of the chloride or iodide. The metal is very reactive, and the process is carried out under an atmosphere of inert gas. The powdered metal is a fire and explosive hazard.

### SYNONYMS

None.

### POTENTIAL OCCUPATIONAL EXPOSURES

Zirconium metal is used as a "getter" in vacuum tubes, a deoxidizer in metallurgy, and a substitute for platinum; it is used in priming of explosive mixtures, flashlight powders, lamp filaments, flash bulbs, and in construction rayon spinnerets. Zirconium or its alloys (nickel cobalt, niobium, tantalum) are used as lining materials for pumps and pipes, for chemical processes, and for reaction vessels. Pure zirconium is a structural material for atomic reactors, and alloyed, particularly with aluminum, it is a cladding material for fuel rods in water-moderated nuclear reactors. A zirconium-columbian alloy is an excellent superconductor.

Zircon ( $ZrSiO_4$ ) is utilized as a foundry sand, an abrasive, a refractory in combination with zirconia, a coating for casting molds, a catalyst in alkyl and alkenyl hydrocarbon manufacture, a stabilizer in silicone rubbers, and as a gem stone; in ceramics it is used as an opacifier for glazes and enamels and in frittered glass filters. Both zircon and zirconia (zirconium oxide,  $ZrO_2$ ) bricks are used as linings for glass furnaces. Zirconia itself is used in die extrusion of metals and in spout linings for pouring metals as a substitute for lime in oxyhydrogen light, as a pigment, and an abrasive; it is used, too, in incandescent lights, as well as in the manufacture of enamels, white glass, and refractory crucibles.

Other zirconium compounds are used in metal cutting tools, thermocouple jackets, waterproofing textiles, ceramics, and in treating dermatitis and poison ivy.

A partial list of occupations in which exposure may occur includes:

Abrasive makers	Incandescent lamp makers
Ceramic workers	Metallurgists
Crucible makers	Pigment makers
Deodorant makers	Rayon spinneret makers
Enamel makers	Refractory material makers
Explosive workers	Textile waterproofers
Foundry workers	Vacuum tube makers
Glass makers	

#### PERMISSIBLE EXPOSURE LIMITS

The Federal standard for zirconium compounds is 5 mg/m<sup>3</sup> as Zr.

#### ROUTE OF ENTRY

Inhalation of dust or fume.

#### HARMFUL EFFECTS

##### *Local—*

No ill effects from industrial exposure to zirconium have been proven. A recent study from the U.S.S.R., however, reports that some workers exposed to plumbous titanate zirconate developed a mild occupational dermatitis associated with hyperhydrosis of the hands. This condition was accompanied by subjective complaints of vertigo, sweet taste in the mouth, and general indisposition. These workers were also said to have elevated thermal and pain sensitivity, and electric permeability of the horny layer, along with increased sweating, and reduced capillary resistance.

Zircon granuloma were reported in the U.S. as early as 1956. This condition arose from the use of deodorant sticks in the axillae, but it was resolved when use was stopped. Zircon is no longer used as a deodorant. Because of a possible allergic sensitivity reaction, individuals who have experienced granuloma from zirconium should avoid dust and mist.

##### *Systemic—*

Inhalation of zirconium dust and fumes has caused no respiratory or other pathological problems. Animal experiments, however, have produced interstitial pneumonitis, peribronchial abscesses, peribronchiolar granuloma, and lobular pneumonia.

#### MEDICAL SURVEILLANCE

No special considerations are needed.

#### SPECIAL TESTS

None in common use.

#### PERSONAL PROTECTIVE METHODS

Employees should be trained in the correct use of personal protective equipment. In areas of dust accumulation or high fume concentrations, respiratory protection is advised either by dust mask or supplied

air respirators. Skin protection is not generally necessary, but where there is a history of zircon granuloma from deodorants, it is probably advisable.

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## MISCELLANEOUS INORGANIC COMPOUNDS

### AMMONIA

#### DESCRIPTION

$\text{NH}_3$ , ammonia, is a colorless, strongly alkaline, and extremely soluble gas with a characteristic pungent odor.

#### SYNONYMS

None.

#### POTENTIAL OCCUPATIONAL EXPOSURES

Ammonia is used as a nitrogen source for many nitrogen-containing compounds. It is used in the production of ammonium sulfate and ammonium nitrate for fertilizers and in the manufacture of nitric acid, soda, synthetic urea, synthetic fibers, dyes, and plastics. It is also utilized as a refrigerant and in the petroleum refining, chemical, and pharmaceutical industries.

Other sources of occupational exposure include the silvering of mirrors, gluemaking, tanning of leather, and around nitriding furnaces. Ammonia is produced as a by-product in coal distillation and by the action of steam on calcium cyanamide, and from the decomposition of nitrogenous materials.

A partial list of occupations in which exposure may occur includes:

Aluminum workers	Metal powder processors
Annealers	Mirror silverers
Chemical laboratory workers	Paper makers
Chemical workers	Paper pulp makers
Dye makers	Pesticide makers
Electroplate workers	Rayon makers
Fertilizer workers	Refrigeration workers
Galvanizers	Sulfuric acid workers
Glue makers	Tannery workers
Metal extractors	Water treaters