CHEMICAL HAZARDS

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Raw materials from many sources are converted by the chemical industry into the substances used by the industry itself to produce cosmetics, detergents and soaps, drugs, dyes, pigments, explosives, fertilizers, petrochemicals, inks, paints, pesticides, plastics, synthetic fibers, and many other products. Other industries use the substances in the production of durable and nondurable goods.

This section deals with the harmful health effects of some of these substances. Items discussed include the capacity of the substance to produce local and/or systemic effects, special diagnostic tests that may aid diagnosis of the illness by identification of the agent, potential occupational exposures, protection methods, and references.

HARMFUL EFFECTS. Under the heading Harmful Effects are given only the chief or dominant effects that characterize the usual responses to the toxic agent. Because of the lack of information on the mutagenic, teratogenic, or carcinogenic effects of many chemicals, consideration is given only in specific instances to these effects.

Local and systemic effects are given in an effort to categorize the effects of the toxic agent. It was arbitrarily decided to limit local effects to the skin and eyes and to the mucous membranes of the upper respiratory tract. Systemic effects include the manifestations elicited by the absorption of the toxic agent into the body and its distribution to the internal organs and particularly the effects of the agent on the tissues of the lower respiratory tract.

ROUTE OF ENTRY. The route of entry section is intended to supply information on the avenue by which the toxic agent is most likely to gain entrance into the body when encountered in the industrial environment.

SPECIAL DIAGNOSTIC TESTS. Ordinary tests such as complete blood counts, urinalyses, and chest roentgenograms are not included under the heading Special Diagnostic Tests. Similarly, liver and kidney function tests and cutaneous patch tests have not been included, even though they may be of considerable diagnostic importance. It is felt that the physician need not be reminded of the methods for determining abnormalities in the target organs which are mentioned under Systemic Effects.

It should be pointed out that many of these special diagnostic tests are difficult to carry out and should be performed only by qualified laboratories. In addition, the fact should be kept in mind that normal values may vary somewhat, even those from competent analytical laboratories.

Because of the absence of significant, interpretable information, no reference is made to behavioral patterns of response to toxic agents.

PERMISSIBLE EXPOSURE LIMITS. In the section on Permissible Exposure Limits, the standard as currently promulgated by the Secretary of the U. S. Department of Labor under the Occupational Safety and Health Act of 1970 (PL 91-596) is given. These standards contain the permissible exposure limit as found in 29 CFR 1910.1000 as of January 1, 1977. Where NIOSH has published a recommended revision to the OSHA regulation, the NIOSH recommended level is also noted. In addition in some instances the assigned Threshold Limit Value (TLV) appearing in the current (1976) list published by the American Conference of Governmental Industrial Hygienists (ACGIH) is given.

The Department of Labor promulgates standards that are mandatory in the United States; the TLVs issued by the ACGIH are guidelines and are to be considered as practical guides in the control of health hazards and should not be regarded as fine lines between safe and dangerous exposure levels. In 1971, most of the 1968 ACGIH TLV list was included in the standard for air contaminants (29 CFR 1910.93 amended to 1910.1000). United States standards are first published in the Federal Register (FR), which is available by subscription from the U. S. Government Printing Office. These regulations are then codified in the Code of Federal Regulations (CFR), generally in Title 29. This publication is also available from the Superintendent of Documents, U.S. Government Printing Office, Washington, D. C. Since these regulations are mandatory and often stipulate medical requirements as to diagnostic tests, it would be wise to check this reference if there is any question about an illness resulting from occupational exposure.

POTENTIAL OCCUPATIONAL EXPOSURES. The list of occupations appended to a particular chemical substance lists occupations in which workers are potentially exposed to the toxic agent. Whether the exposure to the toxic agent constitutes a hazard depends upon such factors as concentration of the agent, how the agent is handled and used, duration of exposure, susceptibility of the worker to the agent, and the health protection practices that may be adopted by management. Thus, all hazardous situations imply an exposure, but all exposures are not hazardous.

SYMPTOMS AND EXPOSURE. In resolving the problem of the relationship between the signs and symptoms presented by the worker and the potential toxic exposure in his occupation, it is important to understand the ways by which a toxic chemical enters the body and to secure factual information as to the physical and chemical characteristics of the work environment and the personal hygiene of the worker. At the same time, it is essential to recognize that 1) chemical formulas offer, at most, only rough guides to the prediction of toxic response and 2) the forms of acute and chronic toxicity are so often dissimilar that prediction cannot be made of the nature of chronic toxicity from the acute manifestations.

SELECTION OF CHEMICALS. Most of the known occupational disease-producing chemicals are given; certain chemicals, however, are excluded because of insufficient data.

The chemicals given are arranged by chemical groups; for example, aliphatic hydrocarbons, alcohols, glycols. Some chemicals are discussed in the section on pesticides.

REFERENCES. A number of excellent secondary references are available on chemical hazards. These include the series of NIOSH Criteria Documents, the AIHA Hygienic Guides, and the ACGIH Documentation of the Threshold Limit Values for Substances in Workroom Air. An additional source is the Medical Surveillance Guidelines from the OSHA-NIOSH Standards Completion Program. These provide detailed information on toxicology, signs and symptoms, special tests, treatments, surveillance, and prevention. Specific references cited in a section are listed at the end of that section. Supplemental references covering some chemical groups are provided in bibliographies for those groups.

General texts of interest are listed in the following bibliography.

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ALIPHATIC HYDROCARBONS

Aliphatic hydrocarbons are saturated or unsaturated, branched or unbranched open carbon chains. Within this group there are three subgroups: alkanes (saturated hydrocarbons), alkenes (unsaturated hydrocarbons with one or more double bonds), and alkynes (unsaturated hydrocarbons with one or more triple bonds). Synonyms are paraffins, olefins, and acetylenes, respectively. Compounds of lower molecular weight containing fewer than 4 carbons are usually gases at room temperature, whereas larger molecules, containing from 5 to 16 carbons, are liquids, and those having more than 16 carbons are usually solids.

Aliphatic hydrocarbons are derived from petroleum by the cracking, distillation, and fractionation of crude oil. Most of these compounds are used industrially in mixtures, such as natural gas, petroleum naphtha, gasoline, kerosene, and mineral spirits. Aliphatic hydrocarbons are used principally as fuels, refrigerants, propellants, dry cleaning agents, lubricants, solvents, and chemical intermediates.

Aliphatic hydrocarbons are asphyxiants and central nervous system depressants. Lower members of the series, methane and ethane, are pharmacologically less active than higher members of the series, their main hazards resulting from the simple displacement of oxygen and from fire and from explosion. Higher members of the series cause narcosis. At least one member (hexane) has neurotoxic properties. Another common effect is irritation of the skin and mucous membranes of the upper respiratory tract. Repeated and prolonged skin contact may result in dermatitis, due to the defatting of skin. Due to its low viscosity, aspiration of liquid may result in diffuse chemical pneumonitis, pulmonary edema, and hemorrhage. Contamination of aliphatic hydrocarbons by benzene significantly increases the hazard. Therefore, it is important that benzene content, if suspected, be determined.

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ACETYLENE

DESCRIPTION

HC≡CH, acetylene, is a colorless gas with a faint ethereal odor.

SYNONYMS

Ethine, ethyne, narcylene.

POTENTIAL OCCUPATIONAL EXPOSURES

Acetylene can be burned in air or oxygen and is used for brazing, welding, cutting, metallizing, hardening, flame scarfing, and local heating in metallurgy. The flame is also used in the glass industry. Chemically, acetylene is used in the manufacture of vinyl chloride, acrylonitrile, synthetic rubber, vinyl acetate, trichloroethylene, acrylate, butyrolactone, 1,4-butanediol, vinyl alkyl ethers, pyrrolidone, and other substances.

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

- Acetaldehyde makers
- Acetone makers
- Alcohol makers
- Braziers
- Carbon black makers
- Ceramic makers
- Copper purifiers
- Descalers
- Drug makers
- Dye makers
- Foundry workers
- Gougers
- Hardeners
- Heat treaters
- Lead burners
- Rubber makers
- Scarfers

PERMISSIBLE EXPOSURE LIMITS

No Federal standard has been established. NIOSH has recommended a ceiling limit of 2,500 ppm.

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

Acetylene is nonirritating to skin or mucous membranes.

Systemic—

At high concentrations pure acetylene may act as a mild narcotic and asphyxiant. Most accounted cases of illness or death can be attributed to acetylene containing impurities of arsine, hydrogen sulfide, phosphine, carbon disulfide, or carbon monoxide.

Initial signs and symptoms of exposure to harmful concentrations of impure acetylene are rapid respiration, air hunger, followed by im-

paired mental alertness and muscular incoordination. Other manifestations include cyanosis, weak and irregular pulse, nausea, vomiting, prostration, impairment of judgment and sensation, loss of consciousness, convulsions, and death. Low order sensitization of myocardium to epinephrine resulting in ventricular fibrillation may be possible.

MEDICAL SURVEILLANCE

No specific considerations are needed.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Acetylene poisoning can quite easily be prevented if 1) there is adequate ventilation and 2) impurities are removed when acetylene is used in poorly ventilated areas. General industrial hygiene practices for welding, brazing, and other metallurgical processes should also be observed.

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ALICYCLIC HYDROCARBONS

DESCRIPTION

Cyclopropane: C_3H_6
Cyclohexane: C_6H_{12}
Cyclohexene: C_6H_{10}
Methylcyclohexane: C_7H_{14}

Alicyclic hydrocarbons are saturated or unsaturated molecules in which three or more carbon atoms are joined to form a ring structure. The saturated compounds are called cycloalkanes, cycloparaffins, or naphthenes. The cyclic hydrocarbons with one or more double bonds are called cycloalkenes or cyclo-olefins. These compounds are colorless liquids.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Uses vary with compounds. Cyclopropane is used as an anesthetic. Cyclohexane is used as a chemical intermediate, as a solvent for fats, oils, waxes, resins, and certain synthetic rubbers, and as an extractant of essential oils in the perfume industry. Cyclohexene is used in the manufacture of adipic, maleic, and cyclohexane carboxylic acid. Methylcyclohexane is used as a solvent for cellulose ethers and in the production of organic synthetics.

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

Adipic acid makers
Benzene makers
Fat processors
Fungicide makers
Lacquerers
Nylon makers
Oil processors
Paint removers
Plastic molders
Resin makers
Rubber makers
Varnish removers
Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are:

Cyclohexane 300 ppm (1050 mg/m^3);
Cyclohexene 300 ppm (1015 mg/m^3);
Methylcyclohexane 500 ppm (2000 mg/m^3).

Presently there is no standard for cyclopropane.

(Note: The 1976 ACGIH lists a TLV of 400 ppm (1,600 mg/m^3) for methylcyclohexane.)

ROUTE OF ENTRY

Inhalation of gas or vapor.

HARMFUL EFFECTS

Local—

Repeated and prolonged contact with liquid may cause defatting of the skin and a dry, scaly, fissured dermatitis. Mild conjunctivitis may result from acute vapor exposure.

Systemic—

Alicyclic hydrocarbons are central nervous system depressants, although their acute toxicity is low. Symptoms of acute exposure are excitement, loss of equilibrium, stupor, coma, and, rarely, death as a result of respiratory failure. The concentration of cyclopropane required to produce surgical anesthesia is low, and there is a wide margin between anesthetic and toxic concentrations. The myocardium may become more sensitive to epinephrine during narcosis with cyclopropane. Severe diarrhea and vascular collapse resulting in heart, lung, liver, and brain degeneration have been reported in oral administration of alicyclic hydrocarbons to animals.

The danger of chronic poisoning is relatively slight because these compounds are almost completely eliminated from the body. Metabolism of cyclohexane, for example, results in cyclohexanone and cyclohexanol entering the bloodstream and does not include the metabolites of phenol, as with benzene. Damage to the hematopoietic system does not occur except when exposure is compounded with benzene, which may be a contaminant. Alicyclic hydrocarbons are excreted in the urine as sulfates or glucuronides, the particular content of each varying. Small quantities of these compounds are not metabolized and may be found in blood, urine, and expired breath.

MEDICAL SURVEILLANCE

Consider possible irritant effects to the skin and respiratory tract in any preplacement or periodic examination, as well as any renal or liver complications.

SPECIAL TESTS

None in common use. Some metabolites have been found in blood and urine.

PERSONAL PROTECTIVE METHODS

Skin protection with barrier creams or gloves.

Workers exposed to high concentrations of gas or vapor may need masks.

BIBLIOGRAPHY

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1,3-BUTADIENE

DESCRIPTION

$H_2C=CH-CH=CH_2$, 1,3-butadiene, is a colorless, flammable gas with a pungent, aromatic odor. Because of its low flash point, 1,3-butadiene's fire and explosion hazard may be more serious than its health hazard.

SYNONYMS

Biethylene, bivinyl, butadiene monomer, divinyl, erythrene, methylallene, pyrrolylene, vinylethylene.

POTENTIAL OCCUPATIONAL EXPOSURES

1,3-Butadiene is used chiefly as the principal monomer in the manufacture of many types of synthetic rubber. Presently, butadiene is finding increasing usage in the formation of rocket fuels, plastics, and resins.

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

- Organic chemical synthesizers
- Resin makers
- Rocket fuel handlers
- Rocket fuel makers
- Rubber makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for 1,3-butadiene is 1,000 ppm (2,200 mg/m^3).

ROUTE OF ENTRY

Inhalation of gas or vapor.

HARMFUL EFFECTS

Local—

Butadiene gas is slightly irritating to the eyes, nose, and throat. Dermatitis and frostbite may result from exposure to liquid and evaporating gas.

Systemic—

In high concentrations, 1,3-butadiene gas can act as an irritant, producing cough, and as a narcotic, producing fatigue, drowsiness, headache, vertigo, loss of consciousness, respiratory paralysis, and death. One report states that chronic exposure may result in central nervous system disorders, diseases of the liver and biliary system, and tendencies toward hypotension, leukopenia, increase in ESR, and decreased hemoglobin content in the blood. These changes have not been seen by most observers in humans.

MEDICAL SURVEILLANCE

No specific considerations are needed.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Masks are recommended in contaminated areas with high concentrations of the gas.

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GASOLINE

DESCRIPTION

Gasoline is a highly flammable, mobile liquid with a characteristic odor.

SYNONYMS

Petrol, motor spirits, benzin.

POTENTIAL OCCUPATIONAL EXPOSURES

Gasoline is used as a fuel, diluent, and solvent throughout industry. A partial list of occupations in which exposure may occur includes:

- Filling station attendants
- Garage mechanics
- Gasoline engine operators
- Motor transport drivers
- Pipeline workers
- Refinery workers
- Tank car cleaning crews

PERMISSIBLE EXPOSURE LIMITS

Presently, the composition of gasoline is so varied that a single Federal standard for all types of gasoline is not applicable. It is recommended, however, that atmospheric concentrations should be limited by the aromatic hydrocarbon content.

ROUTE OF ENTRY

Most cases of poisoning reported have resulted from inhalation of vapor and ingestion. It is not known whether gasoline poisoning may be compounded by percutaneous absorption.

HARMFUL EFFECTS

Local—

Gasoline is irritating to skin, conjunctiva, and mucous membranes. Dermatitis may result from repeated and prolonged contact with the liquid, which may defat the skin. Certain individuals may develop hypersensitivity.

Systemic—

Gasoline vapor acts as a central nervous system depressant. Exposure to low concentrations may produce flushing of the face, staggering gait, slurred speech, and mental confusion. In high concentrations, gasoline vapor may cause unconsciousness, coma, and possibly death resulting from respiratory failure.

Other signs also may develop following acute exposure. These signs are early acute hemorrhage of the pancreas, centrilobular cloudy swelling and fatty degeneration of the liver, fatty degeneration of the proximal convoluted tubules and glomeruli of the kidneys, and passive congestion of the spleen.

Ingestion and aspiration of the liquid gasoline usually occurs during siphoning.

Chemical pneumonitis, pulmonary edema, and hemorrhage may follow. Aromatic hydrocarbon content may also cause hematopoietic changes. Absorption of alkyl lead antiknock agents contained in many gasolines poses an additional problem especially where there is prolonged skin contact. The existence of chronic poisoning has not been established.

MEDICAL SURVEILLANCE

No special considerations are necessary.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Barrier creams and impervious gloves, protective clothing.
Masks in heavy exposure to vapors.

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n-HEPTANE

DESCRIPTION

$CH_3(CH_2)_5CH_3$, n-heptane, is a clear liquid which is highly flammable and volatile.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

n-Heptane is used as a solvent and as a standard in testing knock of gasoline engines.

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

Process workers (where heptane is used as the solvent)
Refinery laboratory workers
Refinery workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for n-heptane is 500 ppm (2000 mg/m^3). (Note: The 1976 ACGIH lists a TLV of 400 ppm (1,600 mg/m^3).

ROUTE OF ENTRY

Inhalation of the vapor.

HARMFUL EFFECTS

Local—

n-Heptane can cause dermatitis and mucous membrane irritation. Aspiration of the liquid may result in chemical pneumonitis, pulmonary edema, and hemorrhage.

Systemic—

Systemic effects may arise without complaints of mucous membrane irritation. Exposure to high concentrations causes narcosis producing vertigo, incoordination, intoxication characterized by hilarity, slight nausea, loss of appetite, and a persisting gasoline taste in the mouth. These effects may be first noticed on entering a contaminated area. n-Heptane may cause low order sensitization of the myocardium to epinephrine.

MEDICAL SURVEILLANCE

Preplacement examinations should evaluate the skin and general health, including respiratory, liver, and kidney function.

SPECIAL TESTS

None have been commonly used.

PERSONAL PROTECTIVE METHODS

Barrier creams and gloves. Masks where exposed to vapor.

n-HEXANE

DESCRIPTION

$CH_3(CH_2)_4CH_3$, n-hexane, is a colorless, volatile liquid and is highly flammable.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

n-Hexane is used as a solvent, particularly in the extraction of edible fats and oils, as a laboratory reagent, and as the liquid in low temperature thermometers. Technical and commercial grades consist of 45 to 85% n-hexane, as well as cyclopentanes, isohexane, and from 1 to 6% benzene.

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

Fat processors
Oil processors
Thermometer makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 500 ppm (1800 mg/m^3) for workroom exposure to n-hexane. (Note: The 1976 ACGIH lists a TLV of 100 ppm (360 mg/m^3).)

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Dermatitis and irritation of mucous membranes of the upper respiratory tract.

Systemic—

Asphyxia may be produced by high concentrations. Acute exposure may cause narcosis resulting in slight nausea, headache, and dizziness. Myocardial sensitization to epinephrine may occur but is of low order. Peripheral neuropathy has been reported resulting from exposure to n-hexane.

MEDICAL SURVEILLANCE

Consider the skin, respiratory system, central and peripheral nervous system, and general health in preplacement and periodic examinations.

SPECIAL TESTS

None in use.

PERSONAL PROTECTIVE METHODS

Barrier creams and gloves are recommended, as are masks where workers are exposed to vapors.

BIBLIOGRAPHY

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KEROSENE

DESCRIPTION

Kerosene is a pale yellow or clear, mobile liquid, composed of a mixture of petroleum distillates, having a characteristic odor. Chemically, it is composed of aliphatic hydrocarbons with 10 to 16 carbons per molecule and benzene and naphthalene derivatives.

SYNONYMS

Kerosine, coal-oil, range-oil.

POTENTIAL OCCUPATIONAL EXPOSURES

Kerosene is used as a fuel for lamps, stoves, jets, and rockets. It is also used for degreasing and cleaning metals and as a vehicle for insecticides.

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

- Farmers
- Garage workers
- Grease removers
- Heating fuel handlers
- Insecticide workers
- Jet fuel handlers
- Metal cleaners
- Petroleum refinery workers

PERMISSIBLE EXPOSURE LIMITS

Presently there is no Federal standard for kerosene vapor in workroom air.

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

The liquid may produce primary skin irritation as a result of de-

fatting. Aspiration of liquid may cause extensive pulmonary injury. Because of its low surface tension, kerosene may spread over a large area, causing pulmonary hemorrhage and chemical pneumonitis. Kerosene mist may also cause mucous membrane irritation.

Systemic—

Inhalation of high concentrations may cause headache, nausea, confusion, drowsiness, convulsions, and coma. When kerosene is ingested, it may cause nausea, vomiting, and, in severe cases, drowsiness progressing to coma, and death by hemorrhagic pulmonary edema and renal involvement.

MEDICAL SURVEILLANCE

No specific considerations are needed.

SPECIAL TESTS

None in use.

PERSONAL PROTECTIVE METHODS

Barrier creams, gloves, and protective clothing are recommended. Where workers are exposed to vapors, masks are recommended.

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NAPHTHA

DESCRIPTION

Naphthas derived from both petroleum and coal tar are included in this group. Petroleum naphthas composed principally of aliphatic hydrocarbons are termed "close-cut" fractions. "Medium-range" and "wide-range" fractions are made up of 40 to 80 per cent aliphatic hydrocarbons, 25 to 50 per cent naphthenic hydrocarbons, 0 to 10 per cent benzene, and 0 to 20 per cent other aromatic hydrocarbons.

Coal tar naphtha is a mixture of aromatic hydrocarbons, principally toluene, xylene, and cumene. Benzene, however, is present in appreciable amounts in those coal tar naphthas with low boiling points.

SYNONYMS

Petroleum naphtha: ligroin, benzine, petroleum ether, petroleum benzine.

POTENTIAL OCCUPATIONAL EXPOSURES

Naphthas are used as organic solvents for dissolving or softening rubber, oils, greases, bituminous paints, varnishes, and plastics. The less flammable fractions are used in dry cleaning, the heavy naphthas serving as bases for insecticides.

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

Chemical laboratory workers	Petroleum refinery workers
Detergent makers	Rubber coaters
Dry cleaners	Solvent workers
Fat processors	Stainers
Insecticide workers	Varnish makers
Metal degreasers	Wax makers
Oil processors	Wool processors
Painters	Xylene makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for petroleum naphtha is 500 ppm (2,000 mg/m^3); for coal tar naphtha it is 100 ppm (400 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor. Percutaneous absorption of liquid is probably not important in development of systemic effects unless benzene is present.

HARMFUL EFFECTS

Local—

The naphthas are irritating to the skin, conjunctiva, and the mucous membranes of the upper respiratory tract. Skin "chapping" and photosensitivity may develop after repeated contact with the liquid. If confined against skin by clothing, the naphthas may cause skin burn.

Systemic—

Petroleum naphtha has a lower order of toxicity than that derived from coal tar, where the major hazard is brought about by the aromatic hydrocarbon content. Sufficient quantities of both naphthas cause central nervous system depression. Symptoms include inebriation, followed by headache and nausea. In severe cases, dizziness, convulsions, and unconsciousness occasionally result. Symptoms of anorexia and nervousness have been reported to presist for several months following an acute overexposure, but this appears to be rare. One fraction, hexane, has been reported to have been associated with peripheral neuropathy. (See Hexane.) If benzene is present, coal tar naphthas may produce blood changes such as leukopenia, aplastic anemia, or leukemia. The kidneys and spleen have also been affected in animal experiments. (See Benzene.)

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should include the central nervous system. If benzene exposure is present, workers should have a periodic complete blood count (CBC) including hematocrit, hemoglobin, white blood cell count and differential count, mean corpusclar volume and platelet count, reticulocyte count, serum bilirubin determination, and urinary phenol in the preplacement examination and

at 3-month intervals. There are no specific diagnostic tests for naphtha exposure but urinary phenols may indicate exposure to aromatic hydrocarbons. It should be noted that benzene content of vapor may be higher than predicted by content in the liquid.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Workers should use barrier creams, protective clothing, gloves and masks where exposure to the vapor is likely.

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NATURAL GAS

DESCRIPTION

Natural gas consists primarily of methane (85%) with lesser amounts of ethane (9%), propane (3%), nitrogen (2%), and butane (1%). Methane is a colorless, odorless, flammable gas.

SYNONYMS

Marsh gas.

POTENTIAL OCCUPATIONAL EXPOSURES

Natural gas is used principally as a heating fuel. It is transported as a liquid under pressure. It is also used in the manufacture of various chemicals including acetaldehyde, acetylene, ammonia, carbon black, ethyl alcohol, formaldehyde, hydrocarbon fuels, hydrogenated oils, methyl alcohol, nitric acids, synthesis gas, and vinyl chloride. Helium can be extracted from certain types of natural gas.

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

- Coal miners
- Electric power plant workers
- Gas fuel users
- Helium extractors
- Hydrogen makers
- Nitric acid makers
- Organic chemical synthesizers
- Petroleum refinery workers
- Synthetic gas makers
- Vinyl chloride makers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for natural gas, methane, nitrogen, or butane. The Federal standard for propane is 1000 ppm (1,800 mg/m^3). (Note: the 1974 ACGIH lists a TLV of 600 ppm (145 mg/m^3) for butane as an intended change.)

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

Upon escape from pressurized tanks, natural gas may cause frostbite.

Systemic—

Natural gas is a simple asphyxiant. Displacement of air by the gas may lead to shortness of breath, unconsciousness, and death from hypoxemia. Incomplete combustion may produce carbon monoxide.

MEDICAL SURVEILLANCE

No specific considerations are needed.

SPECIAL TESTS

None are in use.

PERSONAL PROTECTIVE METHOD

Adequate ventilation should quite easily prevent any potential hazard.

PARAFFIN

DESCRIPTION

Paraffin is a white, somewhat translucent solid and consists of a mixture of solid aliphatic hydrocarbons. It may be obtained from petroleum.

SYNONYMS

Paraffin wax, hard paraffin.

POTENTIAL OCCUPATIONAL EXPOSURES

Paraffin is used in the manufacture of paraffin paper, candles, food package materials, varnishes, floor polishes, and cosmetics. It is also used in waterproofing and extracting of essential oils from flowers for perfume.

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

Candle makers	Polish makers
Cosmetic makers	Varnish makers
Perfume makers	Waxpaper makers

PERMISSIBLE EXPOSURE LIMITS

Paraffin wax fume has no established Federal standard; however, in 1975 the ACGIH recommended a TLV of 0.2 mg/m^3 for paraffin wax fume.

ROUTE OF ENTRY

Inhalation of fumes.

HARMFUL EFFECTS

Local—

Occasionally sensitivity reactions have been reported.

Chronic exposure can produce chronic dermatitis, wax boils, folliculitis, comedones, melanoderma, papules, and hyperkeratoses.

Systemic—

Carcinoma of the scrotum in wax pressmen exposed to crude petroleum wax has been documented. Other malignant lesions of an exposed area in employees working with finished paraffin are less well documented. Carcinoma of the scrotum, occurring in workmen exposed 10 years or more, began as a hyperkeratotic nevus-like lesion and developed into a squamous cell carcinoma. The lesions can metastasize to regional inguinal and pelvic lymph nodes. Paraffinoma has been reported from use of paraffin for cosmetic purposes.

MEDICAL SURVEILLANCE

Medical examinations should be concerned especially with the skin. Surveillance should be continued indefinitely.

SPECIAL TESTS

None appear useful.

PERSONAL PROTECTIVE METHODS

Strict personal hygienic measures and protective clothing form the basis of a protective program.

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TURPENTINE

DESCRIPTION

Turpentine is the oleoresin from species of Pinus Pinacae trees. The crude oleoresin (gum turpentine) is a yellowish, sticky, opaque mass and the distillate (oil of turpentine) is a colorless, volatile liquid. Chemically, it contains alpha pinene, beta pinene, camphene, monocyclic terpenes, and terpene alcohols.

SYNONYMS

Gum turpentine, oil of turpentine, spirit of turpentine, gum spirit, gum [derived from pine resin], wood turpentine [derived from pine stumps], sulfate wood pulp waste.

POTENTIAL OCCUPATIONAL EXPOSURES

Turpentines have found wide use as chemical feedstock for the manufacture of floor, furniture, shoe, and automobile polishes, camphor, cleaning materials, inks, putty, mastics, cutting and grinding fluids, paint thinners, resins, and degreasing solutions. Recently, alpha and beta pinenes, which can be extracted, have found use as volatile bases for various compounds.

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

- Art glass workers
- Belt dressing makers
- Camphor makers
- Drug makers
- Furniture polish makers
- Ink makers
- Insecticide makers
- Lacquer makers
- Lithographers
- Oil additive makers
- Paint workers
- Pine oil makers
- Resin makers
- Rubber workers
- Solvent workers
- Stain makers
- Varnish workers
- Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for turpentine is 100 ppm (560 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor and percutaneous absorption of liquid are the usual paths of occupational exposure. However, symptoms have been reported to develop from percutaneous absorption alone.

HARMFUL EFFECTS

Local—

High vapor concentrations are irritating to the eyes, nose, and bronchi. Aspiration of liquid may cause direct lung irritation resulting in pulmonary edema and hemorrhage. Turpentine liquid may produce contact dermatitis. Eczema from turpentine is quite common and has been attributed to the auto-oxidation products of the terpenes (formic acid, formaldehyde, and phenols). This hypersensitivity usually develops in a small portion of the working population. Liquid turpentine splashed in the eyes may cause corneal burns and demands emergency treatment.

Systemic—

Turpentine vapor in acute concentrations may cause central nervous system depression. Symptoms include headache, anorexia, anxiety, excitement, mental confusion, and tinnitus. Convulsions, coma, and death have been reported in animal experiments.

Turpentine vapor also produces kidney and bladder damage. Chronic nephritis with albuminuria and hematuria has been reported as a result of repeated exposures to high concentrations. Predisposition to pneumonia may also occur from such exposures. Recovery usually takes from a few days to a few weeks. Several animal experiments of chronic

low level exposure have produced no ill effects to the central nervous system, kidneys, bladder, or blood.

MEDICAL SURVEILLANCE

Consideration should be given to skin disease or skin allergies in any preplacement or periodic examinations. Liver, renal, and respiratory disease should also be considered.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Rubber gloves, protective clothing, masks for high concentrations.

ALCOHOLS

The alcohols are hydrocarbons in which one or more hydrogen atoms are substituted by hydroxyl (-OH) groups. Compounds with one hydroxyl group are referred to as alcohol. Glycols have two, and glycerols have three, substituted hydroxyl groups. They are widely used as industrial solvents in a variety of products.

In general, alcohols are irritating to mucous membranes. Their toxicity varies, but usually they produce some narcotic effect. They have some disinfectant action and, because of their lipid solubility, most are absorbed to some extent through the skin.

ALLYL ALCOHOL

DESCRIPTION

$H_2C=CHCH_2OH$, allyl alcohol, is a colorless liquid with a pungent odor.

SYNONYMS

Vinyl carbinol, propenyl alcohol, 2-propenol-1, propenol-3.

POTENTIAL OCCUPATIONAL EXPOSURES

Allyl alcohol is primarily used in the production of allyl esters. These compounds are used as monomers and prepolymers in the manufacture of resins and plastics. Allyl alcohol is also used in the preparation of pharmaceuticals, in organic syntheses, and as a fungicide and herbicide.

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

- Acrolein makers
- Allyl ester makers
- Drug makers
- Fungicide makers
- Glycerine makers
- Herbicide makers
- Organic chemical synthesizers
- Plasticizer makers
- Resin makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 2 ppm (5 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor; percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Liquid and vapor are highly irritating to eyes and upper respiratory tract. Skin irritation and burns have occurred from contact with liquid but are usually delayed in onset and may be prolonged.

Systemic—

Local muscle spasms occur at sites of percutaneous absorption. Pulmonary edema, liver and kidney damage, diarrhea, delirium, convulsions, and death have been observed in laboratory animals, but have not been reported in man.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include the eyes, skin, respiratory tract, and liver and kidney function.

SPECIAL TESTS

No specific test is available.

PERSONAL PROTECTIVE METHODS

Protective clothing to prevent skin contact should be made of neoprene; these must be discarded at the first sign of deterioration. The odor and irritant properties of allyl alcohol should be sufficient warning to prevent serious injury.

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AMYL ALCOHOL

DESCRIPTION

$C_5H_{11}OH$, amyl alcohol, has eight isomers. All are colorless liquids, except the isomer 2-dimethyl-1-propanol, which is a crystalline solid.

Amyl alcohols are obtained from fusel oil which forms during the fermentation of grain, potatoes, or beets for ethyl alcohol. The fusel oil is a mixture of amyl alcohol isomers, and the composition is determined somewhat by the sugar source. Amyl alcohols may be prepared by acid hydrolysis of a petroleum fraction.

SYNONYMS

Pentanols, pentyl alcohols, fusel oil, grain oil, potato spirit, potato oil.

POTENTIAL OCCUPATIONAL EXPOSURES

Amyl alcohols are used in the manufacture of lacquers, paints, varnishes, paint removers, shoe cements, perfumes, pharmaceuticals, chemicals, rubber, plastics, fruit essences, explosives, hydraulic fluids, ore-flotation agents, in the preparation of other amyl derivatives, in the extraction of fats, and in the textile and petroleum refining industries.

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

- Amyl acetate makers
- Amyl nitrite makers
- Explosive makers
- Fat processors
- Flotation workers
- Lacquer makers
- Mordanters
- Oil processors
- Painters
- Perfume makers
- Petroleum refiners
- Photographic chemical makers
- Plastic makers
- Rubber makers
- Shoe finishers
- Textile workers
- Wax processors

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for 3-methyl-1-butanol (isomyl alcohol) is 100 ppm (360 mg/m^3). There are no standards for the other isomers.

ROUTE OF ENTRY

Inhalation of vapor, percutaneous absorption.

HARMFUL EFFECTS

Local—

The liquid and vapor are mild irritants to the membranes of the eyes and upper respiratory tract and skin.

Systemic—

In low concentrations, amyl alcohol may cause irritation of nose and throat, nausea, vomiting, flushing, headache, diplopia, vertigo, and muscular weakness. In higher dosage, it is a narcotic.

MEDICAL SURVEILLANCE

Consider possible irritant effects on skin and respiratory tract in any preplacement or periodic examinations.

SPECIAL TESTS

None in common use. Amyl alcohol can be determined in blood.

PERSONAL PROTECTIVE METHODS

Barrier creams and personal protective clothing should be used to prevent skin contact.

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n-BUTYL ALCOHOL

DESCRIPTION

$CH_3(CH_2)_3OH$, n-butyl alcohol, is a colorless volatile liquid with a pungent odor.

SYNONYMS

1-Butanol, butyl hydroxide, propylcarbinol, butyric alcohol, hydroxybutane, n-butanol, n-propylcarbinol.

POTENTIAL OCCUPATIONAL EXPOSURES

n-Butyl alcohol is used as a solvent for paints, lacquers, varnishes, natural and synthetic resins, gums, vegetable oils, dyes, camphor, and alkaloids. It is also used as an intermediate in the manufacture of pharmaceuticals and chemicals and in the manufacture of artificial leather, safety glass, rubber and plastic cements, shellac, raincoats, photographic films, perfumes, and in plastic fabrication.

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

- Alkaloid makers
- Detergent makers
- Drug makers
- Dye makers
- Lacquerers
- Photographic film makers
- Plasticizer makers
- Stainers
- Urea-formaldehyde resin makers
- Varnish makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 100 ppm (300 mg/m^3). (Note: the 1976 ACGIH lists a TLV of 50 ppm (150 mg/m^3).)

ROUTE OF ENTRY

Inhalation of vapor and percutaneous absorption.

HARMFUL EFFECTS

Local—

The liquid is a primary skin irritant. The vapor is an irritant to the conjunctiva and mucous membranes of the nose and throat. A mild keratitis characterized by corneal vacuoles has been noted at vapor concentrations over 200 ppm.

Systemic—

Inhalation of high concentrations, in addition to the local effects, have produced transitory and persistent dizziness with Meniere's syndrome. Slight headache and drowsiness may also occur.

MEDICAL SURVEILLANCE

Consider irritant effects on eyes, respiratory tract, and skin in any preplacement or periodic examinations.

SPECIAL TESTS

None have been used. Blood levels can be determined.

PERSONAL PROTECTIVE METHODS

Barrier creams and protective clothing should be used where skin contact may occur.

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ETHYL ALCOHOL

DESCRIPTION

CH_3CH_2OH, ethyl alcohol, is a colorless, volatile, flammable liquid. Ethyl alcohol is produced by fermentation and distillation or by synthesis.

SYNONYMS

Ethanol, grain alcohol, spirit of wine, cologne spirit, ethyl hydroxide, ethyl hydrate.

POTENTIAL OCCUPATIONAL EXPOSURES

Ethyl alcohol is used in the chemical synthesis of a wide variety of compounds such as acetaldehyde, ethyl ether, ethyl chloride, and butadiene. It is a solvent or processing agent in the manufacture of pharmaceuticals, plastics, lacquers, polishes, plasticizers, perfumes, cosmetics, rubber accelerators, explosives, synthetic resins, nitrocellulose, adhesives, inks, and preservatives. It is also used as an antifreeze and as a fuel.

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

- Acetaldehyde makers
- Acetic anhydride makers
- Adhesive makers
- Beverage makers
- Detergent makers
- Distillers
- Dye makers
- Ethyl ether makers
- Histology technicians
- Ink makers
- Lacquer makers
- Motor fuel blenders
- Organic chemical synthesizers
- Rubber makers
- Shellac processors
- Solvent workers
- Stainers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1,000 ppm (1,900 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor and percutaneous absorption.

HARMFUL EFFECTS

Local—

Mild irritation of eye and nose occurs at very high concentrations.

The liquid can defat the skin, producing a dermatitis characterized by drying and fissuring.

Systemic—

Prolonged inhalation of high concentrations, besides the local effect on the eyes and upper respiratory tract, may produce headache, drowsiness, tremors, and fatigue. Tolerance may be a factor in individual response to a given air concentration.

Bizarre symptoms (other than typical manifestations of intoxication) may result from the denaturants often present in industrial ethyl alcohol. Ethyl alcohol may act as an adjuvant, increasing the toxicity of other inhaled, absorbed, or ingested chemical agents. An exception is methanol where ethyl alcohol counteracts methanol toxicity.

MEDICAL SURVEILLANCE

No special considerations needed.

SPECIAL TESTS

Ethyl alcohol can readily be determined in blood, urine, and expired air.

PERSONAL PROTECTIVE METHODS

Personal protective equipment is recommended where skin contact may occur.

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ETHYLENE CHLOROHYDRIN

DESCRIPTION

CH_2ClCH_2OH, ethylene chlorohydrin, is a colorless liquid with an ether-like odor.

SYNONYMS

Glycol chlorohydrin, 2-chloroethanol, β-chloroethyl alcohol.

POTENTIAL OCCUPATIONAL EXPOSURES

Ethylene chlorohydrin is used in the synthesis of ethylene glycol, ethylene oxide, amines, carbitols, indigo, malonic acid, novocaine, and in other reactions where the hydroxyethyl group ($-CH_2CH_2OH$) is introduced into organic compounds, for the separation of butadiene from hydrocarbon mixtures, in dewaxing and removing cycloalkanes from mineral oil, in the refining of rosin, in the manufacture of certain pesti-

cides, and in the extraction of pine lignin. In the lacquer industry, it is used as a solvent for cellulose acetate, cellulose esters, resins and waxes, and in the dyeing and cleaning industry, it is used to remove tar spots, as a cleaning agent for machines, and as a solvent in fabric dyeing. It has also found use in agriculture in speeding up sprouting of potatoes and in treating seeds to inhibit biological activity.

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

- Cellulose acetate makers
- Drug makers
- Dye makers
- Ethyl cellulose workers
- Indigo makers
- Lacquer makers
- Novocaine makers
- Organic chemical synthesizers
- Potato growers
- Procaine makers
- Resin workers
- Textile dyers and printers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (16 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor; percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

High vapor concentrations are irritating to the eyes, nose, throat, and skin.

Systemic—

Ethylene chlorohydrin is extremely toxic and in addition to local irritation of eyes, respiratory tract, and skin, inhalation of the vapor may produce nausea, vomiting, dizziness, headache, thirst, delirium, low blood pressure, collapse, and unconsciousness. The urine may show red cells, albumin, and casts. Death may occur in high concentrations with damage to the lung and brain. There is little margin of safety between early reversible symptoms and fatal intoxication. The toxic effects may be related to its metabolites, chloroacetaldehyde and chloroacetic acid.

MEDICAL SURVEILLANCE

Preplacement examination, including a complete history and physical should be performed. Examination of the respiratory system, liver, kidneys, and central nervous system should be stressed. The skin should be examined. A chest X-ray should be taken and pulmonary function tests performed (FVC-FEV).

The above procedures should be repeated on an annual basis, except that the X-ray is needed only when indicated by pulmonary function testing.

SPECIAL TESTS

None commonly used. Presence in blood can probably be determined by appropriate gas chromatographic methods.

PERSONAL PROTECTIVE METHODS

The liquid readily penetrates rubber. Protective clothing should be discarded at first sign of deterioration. Barrier creams may be used and scrupulous personal hygiene should be practiced.

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METHYL ALCOHOL

DESCRIPTION

CH_3OH, methyl alcohol, is a colorless, volatile liquid with a mild odor.

SYNONYMS

Methanol, carbinol, wood alcohol, wood spirit.

POTENTIAL OCCUPATIONAL EXPOSURES

Methyl alcohol is used as a starting material in organic synthesis of chemicals such as formaldehyde, methacrylates, methyl amines, methyl halides, and ethylene glycol, and as an industrial solvent for inks, resins, adhesives, and dyes for straw hats. It is an ingredient in paint and varnish removers, cleaning and dewaxing preparations, spirit duplicating fluids, embalming fluids, antifreeze mixtures, and enamels and is used in the manufacture of photographic film, plastics, celluloid, textile soaps, wood stains, coated fabrics, shatterproof glass, paper coating, waterproofing formulations, artificial leather, and synthetic indigo and other dyes. It has also found use as an extractant in many processes, an antidetonant fuel-injection fluid for aircraft, a rubber accelerator, and a denaturant for ethyl alcohol.

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

Acetic acid makers
Art glass workers
Bookbinders
Bronzers
Dyers
Enamel makers
Ester makers
Feather workers
Felt hat makers
Foundry workers
Gilders
Ink makers
Lasters
Leather workers
Millinery workers
Painters
Photoengravers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 200 ppm (260 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor; percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Contact with liquid can produce defatting and a mild dermatitis. Methyl alcohol is virtually nonirritating to the eyes or upper respiratory tract below 2,000 ppm, and it is difficult to detect by odor at less than this level.

Systemic—

Methyl alcohol may cause optic nerve damage and blindness. Its toxic effect is thought to be mediated through metabolic oxidation products, such as formaldehyde or formic acid, and may result in blurring of vision, pain in eyes, loss of central vision, or blindness. Other central nervous system effects result from narcosis and include headache, nausea, giddiness, and loss of consciousness. Formic acid, may produce acidosis. These symptoms occur principally after oral ingestion and are very rare after inhalation.

MEDICAL SURVEILLANCE

Consider eye disease and visual acuity in any periodic or placement examinations, as well as skin and liver and kidney functions.

SPECIAL TESTS

Determination of methyl alcohol in blood, and methyl alcohol and formic acid in urine. Estimation of alkali reserve which may be impaired because of acidosis following accidental ingestion.

PERSONAL PROTECTIVE METHODS

Barrier creams and protective clothing.

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PROPYL ALCOHOL

DESCRIPTION

There are two isomers of propyl alcohol, n-propyl alcohol (CH_3-CH_2CH_2OH) and isopropyl alcohol ($CH_3CHOHCH_3$). Both are colorless, volatile liquids.

SYNONYMS

n-Propyl alcohol: 1-Propanol, propylic alcohol. Isopropyl alcohol: Isopropanol, 2-propanol, secondary propyl alcohol, dimethylcarbinol.

POTENTIAL OCCUPATIONAL EXPOSURES

Isopropyl alcohol is the more widely used of the two isomers. In the pharmaceutical industry, it has replaced ethyl alcohol in liniments,

skin lotions, cosmetics, permanent wave preparations, hair tonics, mouth washes, and skin disinfectants and is widely used as a rubbing alcohol. Isopropyl alcohol is used in the manufacture of acetone, isopropyl derivatives, and safety glass, as a solvent in perfumes, resins and plastics, dye solutions, nitrocellulose lacquers, and in many extraction processes and is an ingredient of antifreezes, deicing agents, liquid soaps, and window cleaners. Further applications include use as a preservative and dehydrating agent, a coupling agent in oil emulsions, an extracting agent for sulfonic acids from petroleum oils, and for coatings in textiles. n-Propyl alcohol is used in lacquers, dopes, cosmetics, dental lotions, cleaners, polishes, and pharmaceuticals and as a surgical antiseptic. It is a solvent for vegetable oils, natural gums and resins, rosin, shellac, certain synthetic resins, ethyl cellulose, and butyral.

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

- Disinfectant makers
- Drug makers
- Gum processors
- Metal degreasers
- Nurses
- Perfume makers
- Polish makers
- Resin makers
- Soap makers
- Stainers
- Vegetable oil processors
- Wax makers
- Window cleaning fluid makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for n-propyl alcohol is 200 ppm (500 mg/m^3) and for isopropyl alcohol, 400 ppm (980 mg/m^3).

ROUTE OF ENTRY

Isopropyl alcohol: Inhalation of vapor.
n-Propyl alcohol: Inhalation of vapor, percutaneous absorption.

HARMFUL EFFECTS

Local—

The two isomers are similar in physical and in most physiological properties. The vapors are mildly irritating to the conjunctiva and mucous membranes of the upper respiratory tract.

Systemic—

No cases of poisoning from industrial exposure have been recorded for either isomer. n-Propyl alcohol can produce mild central nervous system depression; isopropyl alcohol is potentially narcotic in high concentrations.

MEDICAL SURVEILLANCE

No specific considerations are needed.

SPECIAL TESTS

Isopropyl alcohol and its metabolite, acetone, may be detected in blood, urine, and body tissues.

PERSONAL PROTECTIVE METHODS

Clothing and barrier creams are recommended.

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GLYCOLS AND DERIVATIVES

Glycols are dihydric alcohols which are colorless, viscous liquids. Because these compounds are soluble in alcohol and water and have high boiling points and low freezing points, they are used as solvents and antifreeze. Ethylene glycol, like ethyl alcohol, is often called by the class name, i.e., glycol. These compounds have relatively low toxicity, and the major hazard appears when the liquids are heated during processing.

ETHYLENE GLYCOL

DESCRIPTION

$HOCH_2CH_2OH$, ethylene glycol, is a colorless, odorless, viscous liquid with a sweetish taste.

SYNONYMS

1, 2-Ethanediol, glycol alcohol, glycol, EG.

POTENTIAL OCCUPATIONAL EXPOSURES

Because of ethylene glycol's physical properties, it is used in antifreeze, hydraulic fluids, electrolytic condensors, and heat exchangers. It is also used as a solvent and as a chemical intermediate for ethylene glycol dinitrate, glycol esters, and resins.

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

- Antifreeze makers
- Brake fluid makers
- Explosive makers
- Glue makers
- Hydraulic fluid makers
- Ink makers
- Metal cleaners
- Metal polishers
- Paint makers
- Resin makers
- Textile makers
- Tobacco workers
- Wax makers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard; however, ACGIH in 1975 recommended a TLV of 10 mg/m^3 for particulate ethylene glycol and 100 ppm (260 mg/m^3) for the vapor form.

ROUTE OF ENTRY

Inhalation of particulate or vapor. Percutaneous absorption may also contribute to intoxication.

HARMFUL EFFECTS

Local—

None.

Systemic—

Ethylene glycol's vapor pressure is such that at room temperature toxic concentrations are unlikely to occur. Poisoning resulting from vapor usually occurs only if ethylene glycol liquid is heated; therefore, occupational exposure is rare. Chronic symptoms and signs include: anorexia, oliguria, nystagmus, lymphocytosis, and loss of consciousness. Inhalation seems to primarily result in central nervous system depression and hematopoietic dysfunction, whereas, ingestion may result in depression followed by respiratory and cardiac failure, renal and brain damage.

MEDICAL SURVEILLANCE

No special considerations are needed.

SPECIAL TESTS

Urinalysis for oxalic acid, an ethylene glycol metabolite, may be useful in diagnosis of poisoning by oral ingestion.

PERSONAL PROTECTIVE METHODS

Masks should be worn in areas of vapor concentration.

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ETHYLENE GLYCOL ETHERS AND DERIVATIVES

DESCRIPTION

Ethylene glycol monoethyl ether: $CH_3CH_2OCH_2CH_2OH$.

Ethylene glycol monoethyl ether acetate: $CH_3CH_2OCH_2CH_2OOCCH_3$.

Ethylene glycol monomethyl ether: $CH_3OCH_2CH_2OH$.

Ethylene glycol monomethyl ether acetate: $CH_3OCH_2CH_2OOCCH_3$.

Ethylene glycol monobutyl ether: $CH_3CH_2CH_2CH_2OCH_2CH_2OH$.

These substances are colorless liquids with a slight odor.

SYNONYMS

Ethylene glycol monoethyl ether: cellosolve, 2-ethoxyethanol.

Ethylene glycol monoethyl ether acetate: cellosolve acetate, 2-ethoxyethyl acetate.

Ethylene glycol monomethyl ether: methyl cellosolve, 2-methoxyethanol.

Ethylene glycol monomethyl ether acetate: methyl cellosolve acetate, 2-methoxyethyl acetate.

Ethylene glycol monobutyl ether: butyl cellosolve, 2-butoxyethanol.

POTENTIAL OCCUPATIONAL EXPOSURES

Ethylene glycol ethers are used as solvents for resins, lacquers, paints, varnishes, gum, perfume, dyes and inks, and as a constituent of painting pastes, cleaning compounds, liquid soaps, cosmetics, nitrocellulose, and hydraulic fluids. Acetate derivatives are used as solvents for oils, greases and ink, in the preparation of lacquers, enamels, and adhesives, and to dissolve resins and plastics.

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

- Cellophane sealers
- Cleaning solution makers
- Dry cleaners,
- Film makers
- Hydraulic fluid makers
- Ink makers
- Lacquer makers
- Nail polish makers
- Oil processors
- Plastic makers
- Printers
- Stainers
- Textile dyers
- Wax processors

PERMISSIBLE EXPOSURE LIMITS

The Federal standards for these compounds are:

Ethylene glycol monoethyl ether	200 ppm	740 mg/m^3
Ethylene glycol monoethyl ether acetate	100 ppm	540 mg/m^3
Ethylene glycol monomethyl ether	25 ppm	80 mg/m^3
Ethylene glycol monomethyl ether acetate	25 ppm	[1]20 mg/m^3
Ethylene glycol monobutyl ether	50 ppm	240 mg/m^3

ACGIH in 1975 recommended a TLV of 25 ppm (120 mg/m^3) for ethylene glycol monomethyl ether acetate.

ROUTE OF ENTRY

Inhalation of vapor and percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Ethylene glycol ethers are only mildly irritating to the skin. Vapor may cause conjunctivitis and upper respiratory tract irritation. Temporary corneal clouding may also result and may last several hours. Acetate derivatives cause greater eye irritation than the parent compounds. The butyl and methyl ethers may penetrate skin readily.

Systemic—

Acute exposure to these compounds results in narcosis, pulmonary

edema, and severe kidney and liver damage. Symptoms from repeated overexposure to vapors are fatigue and lethargy, headache, nausea, anorexia, and tremor. Anemia and encephalopathy have been reported with ethylene glycol monomethyl ether. Rats show increased hemolysis of erythrocytes from inhalation of ethylene glycol monobutyl ether. This has not been shown in man. Acute poisoning by ingestion resembles ethylene glycol toxicity, with death from renal failure.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should evaluate blood, central nervous system, renal and liver functions, as well as the skin and respiratory tract.

SPECIAL TESTS

None currently used.

PERSONAL PROTECTIVE METHODS

Use glasses and protective clothing to prevent skin absorption. Respiratory protection may be needed if ventilation is poor or if compounds are heated or atomized.

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ETHERS AND EPOXY COMPOUNDS

Ethers are organic molecules which contain a carbon-oxygen-carbon linkage. Colorless, volatile liquids, these compounds are generally used industrially as solvents and chemical feedstock for organic synthetics. Ethyl ether, the simplest ether, has been used as a general anesthetic and has been historically known as "ether." Occupationally, exposure to chlorinated ethers is much more significant. Two compounds, bis-(chloromethyl) ether and chloromethyl methyl ether, have produced carcinoma and, therefore, have received much attention recently. Dioxane, although it is not chlorinated, has also shown potential tumorigenic hazard. Skin, eye, and mucous membrane irritation is common to all the chemicals covered. Pulmonary edema, with the added hazard of delayed appearance, may also occur as a result of particular ether exposures.

Epoxy compounds are cyclic ethers with the structure -C-O-C-. The most important industrially are the alpha-epoxy compounds in which the epoxy group is in the 1-2 position. These are the most reactive and are used as chemical intermediates in the manufacture of surface-active agents, plasticizers, synthetic resins, solvents, etc.

BIS(CHLOROMETHYL) ETHER

DESCRIPTION

$ClCH_2OCH_2Cl$, bis(chloromethyl) ether, is a colorless, volatile liquid with a suffocating odor. This substance may form spontaneously in warm moist air by the combination of formaldehyde and hydrogen chloride.

SYNONYMS

BCME, sym-dichloromethyl ether.

POTENTIAL OCCUPATIONAL EXPOSURES

Exposure to bis(chloromethyl) ether may occur in industry and in the laboratory. This compound is used as an alkylating agent in the manufacture of polymers, as a solvent for polymerization reactions, in the preparation of ion exchange resins, and as an intermediate for organic synthesis.

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

Ion exchange resin makers	Organic chemical synthesizers
Laboratory workers	Polymer makers

PERMISSIBLE EXPOSURE LIMITS

Bis(chloromethyl) ether is included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTE OF ENTRY

Inhalation of vapor and perhaps, but to a lesser extent, percutaneous absorption.

HARMFUL EFFECTS

Local—

Vapor is severely irritating to the skin and mucous membranes and may cause corneal damage which may heal slowly.

Systemic—

Bis(chloromethyl) ether has an extremely suffocating odor even in minimal concentration so that experience with acute poisoning is not available. It is not considered a respiratory irritant at concentrations of 10 ppm. Bis(chloromethyl) ether is a known human carcinogen. Animal experiments have shown increases in lung adenoma incidence; olfactory esthesioneuroepitheliomas which invaded the sinuses, cranial vault, and brain; skin papillomas and carcinomas; and subcutaneous fibrosarcomas. There have been several reports of increased incidence of human lung carcinomas (primarily small cell undifferentiated) among ether workers exposed to bis(chloromethyl) ether as an impurity. The latency period is relatively short — 10 to 15 years. Smokers as well as non-smokers may be affected.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should include an examination of the skin and respiratory tract, including chest X-ray. Sputum cytology has been suggested as helpful in detecting early malignant changes, and in this connection a smoking history is of importance. Possible effects on the fetus should be considered.

SPECIAL TESTS

None have been suggested.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and should be appropriate for protection of all skin or respiratory contact. Full body protective clothing and gloves should be used on entering areas of potential exposure. Those employed in handling operations should be provided with full face, supplied air respirators of continuous flow or pressure demand type. On exit from a regulated area, employees should remove and leave protective clothing and equipment at the point of exit, to be placed in impervious containers at the end of the work shift for decontamination or disposal. Showers should be taken before dressing in street clothes.

BIBLIOGRAPHY

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Theiss, A. M., W. Hey, and H. Zeller. 1973. Zur toxicologie von dichlormethyl-aether — verdacht auf kanzerogene wirkung auch bein menschen. Zentralbl. Arbeirsmed. Arbeitsschutz. 23:97.

CHLOROMETHYL METHYL ETHER

DESCRIPTION

$ClCH_2OCH_3$, chloromethyl methyl ether, is a volatile, corrosive liquid. Commercial chloromethyl methyl ether contains from 1 to 7 per cent bis(chloromethyl) ether, a known carcinogen.

SYNONYMS

CMME, methyl chloromethyl ether, monochloromethyl ether, chloromethoxymethane.

POTENTIAL OCCUPATIONAL EXPOSURES

Chloromethyl methyl ether is a highly reactive methylating agent and is used in the chemical industry for synthesis of organic chemicals. Most industrial operations are carried out in closed process vessels so that exposure is minimized.

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

PERMISSIBLE EXPOSURE LIMITS

Chloromethyl methyl ether is included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTE OF ENTRY

Inhalation of vapor and possibly percutaneous absorption.

HARMFUL EFFECTS

Local—

Vapor exposure results in severe irritation of the skin, eyes, and nose. Rabbit skin tests using undiluted material resulted in skin necrosis.

Systemic—

Chloromethyl methyl ether is only moderately toxic given orally. Acute exposure to chloromethyl methyl ether vapor may result in pulmonary edema and pneumonia.

Several studies of workers with CMME manufacturing exposure have shown an excess of bronchiogenic cancer predominately of the small cell-undifferentiated type with relatively short latency period (typically 10-15 years). Therefore, commercial grade chloromethyl methyl ether must be considered a carcinogen. At present it is not known whether or not chloromethyl methyl ether's carcinogenic activity is due to bis(chloromethyl) ether (BCME) contamination, but this may be a moot question inasmuch as two of the hydrolysis products of CMME can combine to form BCME. Animal experiments to determine chloromethyl methyl ether's ability to produce skin cancer indicated marginal carcinogenic activity; highly pure CMME was used. Inhalation studies, using technical grade CMME showed only one bronchiogenic cancer and one esthesioneuroepithelioma out of 79 animals exposed.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should include an examination of the skin and respiratory tract, including a chest X-ray. Sputum cytology has been suggested as helpful in detecting early malignant changes, and in this connection a detailed smoking history is of importance. Possible effects on the fetus should be considered.

SPECIAL TESTS

None have been suggested.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all skin or respiratory contact. Full body protective clothing and gloves should be used on entering areas of partial exposure. Those employed in handling operations should be provided with fullface, sup-

plied air respirators of continuous flow or pressure demand type. On exit from a regulated area, employees should be required to remove and leave protective clothing and equipment at the point of exit, to be placed in impervious containers at the end of the work-shift for decontamination or disposal. Showers should be taken prior to dressing in street clothes.

BIBLIOGRAPHY

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DICHLOROETHYL ETHER

DESCRIPTION

$ClCH_2CH_2OCH_2CH_2Cl$, dichloroethyl ether, is a clear, colorless liquid with a pungent, fruity odor.

SYNONYMS

Dichloroether, dichloroethyl oxide, sym-dichloroethyl ether, bis-(2-chloroethyl) ether, 2, 2-dichloroethyl ether.

POTENTIAL OCCUPATIONAL EXPOSURES

Dichloroethyl ether is used in the manufacture of paint, varnish, lacquer, soap, and finish remover. It is also used as a solvent for cellulose esters, naphthalenes, oils, fats, greases, pectin, tar, and gum; in drycleaning; in textile scouring; and in soil fumigation.

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

- Cellulose ester makers
- Degreasers
- Drycleaners
- Ethyl cellulose processors
- Fat processors
- Gum processors
- Lacquer makers
- Oil processors
- Paint makers
- Soap makers
- Tar processors
- Textile scourers
- Varnish workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for dichloroethyl ether is 15 ppm (90 mg/m^3); however, the ACGIH recommended TLV in 1975 was 5 ppm (30 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor, percutaneous absorption.

HARMFUL EFFECTS

Local—

Irritation of the conjunctiva of the eyes with profuse lacrimation, irritation to mucous membranes of upper respiratory tract, coughing, and nausea may result from exposure to vapor. The liquid when placed in animal eyes has produced damage. Vapors in minimal concentrations (3 ppm) are distinctly irritating and serve as a warning property.

Systemic—

In animal experiments dichloroethyl ether has caused severe irritation of the respiratory tract and pulmonary edema. Animal experiments have also shown dichloroethyl ether to be capable of causing drowsiness, dizziness, and unconsciousness at high concentrations. Except for accidental inhalation of high concentrations, the chief hazard in industrial practice is a mild bronchitis which may be caused by repeated exposure to low concentrations.

MEDICAL SURVEILLANCE

Consideration should be given to the skin, eyes, and respiratory tract, and to the central nervous system in placement or periodic examinations.

SPECIAL TESTS

None have been proposed.

PERSONAL PROTECTIVE METHODS

In cases of vapor concentrations, protective clothing with full-face respirator with air supply should be worn. Skin protection (gloves, protective clothing) is needed to prevent skin absorption. Goggles should be used to prevent eye burns.

BIBLIOGRAPHY

Schrenk, H. H. 1933. Acute response of guinea pigs to vapors of some new commercial organic compounds. VII. Dichloroethyl ether. Public Health Reports 48:1389.

DIOXANE

DESCRIPTION

$OCH_2CH_2OCH_2CH_2$, dioxane, is a volatile, colorless liquid that may form explosive peroxides during storage.

SYNONYMS

1,4-Diethylene dioxide, diethylethene ether, 1,4-dioxane, para-dioxane.

POTENTIAL OCCUPATIONAL EXPOSURES

Dioxane finds its primary use as a solvent for cellulose acetate,

dyes, fats, greases, lacquers, mineral oil, paints, polyvinyl polymers, resins, varnishes, and waxes. It finds particular usage in paint and varnish strippers, as a wetting agent and dispersing agent in textile processing, dye baths, stain and printing composition, and in the preparation of histological slides.

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

Adhesive workers
Cellulose acetate workers
Cement workers
Degreasers
Deodorant makers
Detergent workers
Emulsion makers
Fat processors
Glue makers
Histology technicians
Lacquer makers
Metal cleaners
Oil processors
Paint makers
Polish makers
Shoe cream makers
Varnish remover makers
Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 100 ppm (360 mg/m^3); however, the ACGIH 1975 recommended TLV was 50 ppm (180 mg/m^3) of technical grade.

ROUTE OF ENTRY

Inhalation of vapor as well as percutaneous absorption.

HARMFUL EFFECTS

Local—

Liquid and vapor may be irritating to eyes, nose, and throat.

Systemic—

Exposure to dioxane vapor may cause drowsiness, dizziness, loss of appetite, headache, nausea, vomiting, stomach pain, and liver and kidney damage. Prolonged skin exposure to the liquid may cause drying and cracking.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should be directed to symptoms of headache and dizziness, as well as nausea and other gastrointestinal disturbances. The condition of the skin and of renal and liver function should be considered.

SPECIAL TESTS

No specific bio-monitoring tests are available.

PERSONAL PROTECTIVE METHODS

In areas of vapor concentration, protective clothing, barrier creams, gloves, and masks should be used.

BIBLIOGRAPHY

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Johnstone, R. T. 1959. Death due to dioxane? Arch Ind. Health 20:445.

EPICHLOROHYDRIN

DESCRIPTION

CH_2OCHCH_2Cl, epichlorohydrin, is a colorless liquid with a chloroform-like odor.

SYNONYMS

Epi, chloropropylene oxide, 1-chloro-2,3-epoxypropane, chloromethyloxidrane, 2-epichlorohydrin.

POTENTIAL OCCUPATIONAL EXPOSURES

Epichlorohydrin is used in the manufacture of many glycerol and glycidol derivatives and epoxy resins, as a stabilizer in chlorine-containing materials, as an intermediate in the preparation of cellulose esters and ethers, paints, varnishes, nail enamels, and lacquers, and as a cement for celluloid.

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

- Cellulose ether workers
- Epoxy resin makers
- Glycerol derivative makers
- Glycerophosphoric acid makers
- Glycidol derivative makers
- Gum processors
- Lacquerers
- Lacquer makers
- Nail enamel makers
- Organic chemical synthesizers
- Paint makers
- Resin makers
- Solvent workers
- Varnish makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (19 mg/m^3). NIOSH has recommended a time-weighted average limit of 2 mg/m^3 with a ceiling concentration of 19 mg/m^3 based on a 15-minute sampling period.

ROUTE OF ENTRY

Inhalation of vapor, percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Epichlorohydrin is highly irritating to eyes, skin, and respiratory tract. Skin contact may result in delayed blistering and deep-seated pain. Allergic eczematous contact dermatitis occurs occasionally.

Systemic—

The earliest symptoms of intoxication may be referable to the gastrointestinal tract (nausea, vomiting, abdominal discomfort) or pain in the region of the liver. Labored breathing, cough, and cyanoses may be

evident and the onset of chemical pneumonitis may occur several hours after exposure. Animals exposed repeatedly to this chemical have developed lung, kidney, and liver injury.

MEDICAL SURVEILLANCE

Consider possible effects on the skin, eyes, lungs, liver, and kidney in preplacement or periodic examinations.

SPECIAL TESTS

None currently used.

PERSONAL PROTECTIVE METHODS

Goggles and rubber, protective clothing should be worn. Epichlorohydrin slowly penetrates rubber, so all contaminated clothing should be thoroughly washed. Respirators are required in areas of vapor concentrations.

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Shell Chemical Company. 1972. Epichlorohydrin — Industrial Hygiene Bulletin. Shell Chemical Company, New York.

ETHYLENE OXIDE

DESCRIPTION

H_2COCH_2, ethylene oxide, is a colorless gas with a sweetish odor.

SYNONYMS

1,2-Epoxyethane, oxirane, dimethylene oxide, anprolene.

POTENTIAL OCCUPATIONAL EXPOSURES

Ethylene oxide is used as an intermediate in organic synthesis for ethylene glycol, polyglycols, glycol ethers esters, ethanolamines, acrylonitrile, plastics, and surface-active agents. It is also used as a fumigant for foodstuffs and textiles, an agricultural fungicide, and for sterilization, especially for surgical instruments.

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

- Acrylonitrile makers
- Butyl cellosolve makers
- Detergent makers
- Disinfectant makers
- Ethanolamine makers
- Ethylene glycol makers
- Exterminators
- Foodstuff fumigators
- Fumigant makers
- Fungicide workers
- Gasoline sweeteners
- Grain elevator workers
- Organic chemical synthesizers
- Polyglycol makers
- Polyoxirane makers
- Rocket fuel handlers
- Surfactant makers
- Textile fumigators

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 50 ppm (90 mg/m^3).

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

Aqueous solutions of ethylene oxide or solutions formed when the anhydrous compound comes in contact with moist skin are irritating and may lead to a severe dermatitis with blisters, blebs, and burns. It is also absorbed by leather and rubber and may produce burns or irritation. Allergic eczematous dermatitis has also been reported. Exposure to the vapor in high concentrations leads to irritation of the eyes. Severe eye damage may result if the liquid is splashed in the eyes. Large amounts of ethylene oxide evaporating from the skin may cause frostbite.

Systemic—

Breathing high concentrations of ethylene oxide may cause nausea, vomiting, irritation of the nose, throat, and lungs. Pulmonary edema may occur. In addition, drowsiness and unconsciousness may occur. Ethylene oxide has been found to cause cancer in female mice exposed to it for prolonged periods.

Ethylene oxide is a well-known mutagen in commercial use in plants. No mutagenic effect has been demonstrated in man or animals.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should consider the skin and eyes, allergic history, the respiratory tract, blood, liver, and kidney function.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Eyes and skin should be protected and protective clothing changed when it is contaminated. In areas of high vapor concentration, respirators should be supplied to cover the face, including eyes. Shoes contaminated by this chemical should be discarded.

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Jacobson, K. H., E. B. Hackley, and L. Feinsilver. 1956. The toxicity of inhaled ethylene oxide and propylene oxide vapors. AMA Arch. Ind. Health 13:237.

Sexton, R. J., and E. V. Henson. 1949. Dermatological injuries by ethylene oxide. J. Ind. Hyg. Toxicol. 31:297.

Sulovska, K., D. Lindgren, G. Eriksson, and L. Ehrenberg. 1969. The mutagenic effect of low concentrations of ethylene oxide in air. Hereditas 62:264.

ETHYL ETHER

DESCRIPTION

$CH_3CH_2OCH_2CH_3$, ethyl ether, is a colorless, mobile, highly flammable, volatile liquid with a characteristic pungent odor.

SYNONYMS

Anesthetic ether, diethyl ether, diethyl oxide, ether, ethoxyethane, ethyl oxide, sulfuric ether.

POTENTIAL OCCUPATIONAL EXPOSURES

Ethyl ether is used as a solvent for waxes, fats, oils, perfumes, alkaloids, dyes, gums, resins, nitrocellulose, hydrocarbons, raw rubber, and smokeless powder. It is also used as an inhalation anesthetic, a refrigerant, in diesel fuels, in dry cleaning, as an extractant, and as a chemical reagent for various organic reactions.

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

- Acetic acid makers
- Alcohol denaturers
- Collodion makers
- Diesel fuel blenders
- Drug makers
- Explosive makers
- Fat processors
- Gasoline engine primers
- Dye makers
- Gum processors
- Motor fuel makers
- Nitrocellulose makers
- Oil processors
- Perfume makers
- Rayon makers
- Rubber workers
- Smokeless powder makers
- Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 400 ppm (1,200 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Ethyl ether vapor is mildly irritating to the eyes, nose, and throat. Contact with liquid may produce a dry, scaly, fissured dermatitis.

Systemic—

Ethyl ether has predominantly narcotic properties. Overexposed individuals may experience drowsiness, vomiting, and unconsciousness. Death may result from severe overexposure. Chronic exposure results in some persons in anorexia, exhaustion, headache, drowsiness, dizziness, excitation, and psychic disturbances. Albuminuria has been reported. Chronic exposure may cause an increased susceptibility to alcohol.

MEDICAL SURVEILLANCE

Preplacement or periodic examinations should evaluate the skin and respiratory tract, liver, and kidney function. Persons with a past history of alcoholism may be at some increased risk due to possibility of ethyl ether addiction (known as "ether habit").

SPECIAL TESTS

Tests for exposure may include expired breath for unmetabolized ethyl ether and blood for ethyl ether content by oxidation with chromate solution or by gas chromatographic methods.

PERSONAL PROTECTIVE METHODS

Barrier creams, gloves, protective clothing, and, in areas of vapor concentration, fullface respirator should be used.

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ESTERS

Esters are organic compounds with the structure R-COOR′. They are generally the result of the reaction between an organic or inorganic acid and alcohol with the elimination of water; however, other reactions, such as between an alcohol and an acid halide, will also form esters. The organic acid may be aliphatic or aromatic and may contain other substituents. Mono-, di-, or tricarboxylic esters may be formed.

Esters are an industrially important group of compounds. They are used in plastics and resins, as plasticizers, in lacquer solvents, in flavors and perfumes, in pharmaceuticals, and in industries such as automotive, aircraft, food processing, chemical, pharmaceutical, soap, cosmetic, surface coating, textiles, and leather.

There are four basic types of physiological effects of esters, and these can generally be related to structure. 1) Anesthesia and primary irritation are characteristic of most simple aliphatic esters. 2) Lacrimation, vesication, and lung irritation are due to the halogen atom in halogenated esters. 3) Cumulative organic damage to the nervous system or neuropathy can be caused by some, but not all, phosphate esters. 4) Most aliphatic and aromatic esters used as plasticizers are physiologically inert.

ACETATES

DESCRIPTION

Methyl acetate: CH_3COOCH_3.
Ethyl acetate: $CH_3COOC_2H_5$.
n-Propyl acetate: $CH_3COOC_3H_7$.
Isopropyl acetate: $CH_3COOCH(CH_3)_2$.
n-Butyl acetate: $CH_3COOC_4H_9$.
Amyl acetate: $CH_3COOC_5H_{11}$.

The acetates are colorless, volatile, flammable liquids.

SYNONYMS

Methyl acetate: none.
Ethyl acetate: acetic ether, vinegar naphtha.
n-Propyl acetate: acetic acid-propyl ester.
Isopropyl acetate: none.
n-Butyl acetate: butyl ethanoate, acetic acid butyl ester.
Amyl acetate: isoamyl acetate, pear oil, banana oil, amyl acetate ester, pentyl acetate.

POTENTIAL OCCUPATIONAL EXPOSURES

The acetates are a group of solvents for cellulose nitrate, cellulose acetate, ethyl cellulose, resins, rosin, cumar, elemi, phenolics, oils, fats, and celluloid. They are also used in the manufacture of lacquers, paints, varnishes, enamel, perfumes, dyes, dopes, plastic and synthetic finishes (e.g., artificial leather), smokeless powder, photographic film, footwear, pharmaceuticals, food preservatives, artificial glass, artificial silk, furniture polish, odorants, and other organic syntheses.

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

- Cellulose acetate makers
- Cumar makers
- Dope makers
- Dye makers
- Elemi makers
- Lacquer makers
- Nitrocellulose makers
- Paint makers
- Perfume makers
- Resin makers
- Rosin makers
- Varnish makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are:

Methyl acetate	200 ppm	610 mg/m^3
Ethyl acetate	400 ppm	1,400 mg/m^3
n-Propyl acetate	200 ppm	840 mg/m^3
Isopropyl acetate	250 ppm	950 mg/m^3
n-Butyl acetate	150 ppm	710 mg/m^3
Isoamyl acetate	100 ppm	525 mg/m^3
n-Amyl acetate	100 ppm	525 mg/m^3
sec-Amyl acetate	125 ppm	650 mg/m^3
sec-Butyl acetate	200 ppm	950 mg/m^3
tert-Butyl acetate	200 ppm	950 mg/m^3

ROUTE OF ENTRY

Inhalation and ingestion.

HARMFUL EFFECTS

Local—

In higher concentrations, acetates are irritants to the mucous membranes. All irritate eyes and nasal passages in varying degrees. Prolonged exposure can cause irritation of the intact skin. These local effects are the primary risk in industry.

Systemic—

All acetates may cause headache, drowsiness, and unconsciousness if concentrations are high enough. Those effects are relatively slow and gradual in onset and slow in recovery after exposure.

MEDICAL SURVEILLANCE

Consider initial effects on skin and respiratory tract in any preplacement or periodical examinations, as well as liver and kidney function.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Barrier creams and protective clothing with gloves should be used, as well as fullface masks in areas of vapor concentration.

BIBLIOGRAPHY

Von Oettingen, W. F. 1960. The aliphatic acids and their esters: toxicity and potential dangers. The saturated monobasic acids and their esters: aliphatic acids with three to eighteen carbons and their esters. AMA Arch. Ind. Health 21:100.

ETHYL SILICATE

DESCRIPTION

$(CH_2H_5O)_4Si$, ethyl silicate, is a colorless, flammable liquid with a sharp odor detectable at 85 ppm.

SYNONYMS

Tetraethyl orthosilicate, tetraethoxy silane.

POTENTIAL OCCUPATIONAL EXPOSURES

Ethyl silicate is used in production of cases and molds for casting of metals and as a hardener for water and weather-resistant concrete.

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

- Acidproof cement makers
- Adhesive makers
- Brick preserver makers
- Building coaters
- Cement preserver makers
- Heat resistant paint makers
- Lacquer makers
- Metal casters
- Plaster preserver makers
- Silicate paint makers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard; however, the ACGIH recommended TLV is 100 ppm (approximately 850 mg/m^3) determined as a time-weighted average. This TLV has not been confirmed in human exposure. At 3,000 ppm, ethyl silicate vapors are intolerable.

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Ethyl silicate is a primary irritant to the eyes and the nose.

Systemic—

Damage to the lungs, liver, and kidneys, and anemia have been observed in animal experiments but have not been reported for human exposure.

MEDICAL SURVEILLANCE

Placement or periodic examinations should include the skin, eyes, respiratory tract, as well as liver and kidney functions.

SPECIAL TESTS

None currently used.

PERSONAL PROTECTIVE METHODS

Fullface masks in areas of vapor concentration.

BIBLIOGRAPHY

Pozzini, U. C., and C. P. Carpenter. 1951. Response of rodents to repeated inhalation of vapors of tetraethyl orthosilicate. Arch. Ind. Hyg. Occup. Med. 4:465.

FORMATES

DESCRIPTION

Methyl formate: $HCOOCH_3$.
Ethyl formate: $HCOOC_2H_5$.
These are colorless, mobile, flammable liquids with agreeable odors.

SYNONYMS

Methyl formate: Methyl methanoate.
Ethyl formate: None.

POTENTIAL OCCUPATIONAL EXPOSURES

Formates are solvents for cellulose nitrate, oils, greases, fats, cellulose acetate, fatty acids, acetylcellulose, collodion, and celluloid. They are also used as larvicides, fumigants, flavoring agents in the production of lemonade, rum, arrack, and essences, and they are used in chemical synthesis.

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

- Cellulose acetate workers
- Flavoring makers
- Fumigant makers
- Fumigators
- Grain fumigators
- Nitrocellulose workers
- Organic chemical synthesizers
- Pesticide workers
- Tobacco fumigators

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are:

Methyl formate	100 ppm	250 mg/m^3
Ethyl formate	100 ppm	300 mg/m^3

ROUTE OF ENTRY

Inhalation, ingestion, and skin absorption.

HARMFUL EFFECTS

Local—

Methyl formate is a mild irritant to mucous membranes, especially eyes and respiratory system. Ethyl formate may be irritating to skin and mucous membranes in high concentrations.

Systemic—

Methyl formate has an irritant and narcotic effect, and in high concentrations may cause drowsiness and unconsciousness. Systemic intoxication in industry is rare.

MEDICAL SURVEILLANCE

Consider eye and respiratory disease or symptoms in any placement or follow-up examinations.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Barrier creams should be used to protect the skin, and masks should be used in areas of vapor concentration.

BIBLIOGRAPHY

Von Oettingen, W. F. 1959. The aliphatic acids and their esters — toxicity and potential dangers. The saturated monobasic aliphatic acids and their esters. AMA Arch. Ind. Health 20:517.

CARBOXYLIC ACIDS AND ANHYDRIDES

The carboxylic acids and acid anhydrides have similar properties because of their acid characteristics. Carboxylic acids, and those compounds with the COOH moiety, may be aliphatic or aromatic and may have more than one carboxyl group. The acid anhydrides are derivatives of carboxylic acids.

These compounds have a primary irritant effect, the degree determined in part by acid dissociation and water solubility. Some may cause severe tissue damage similar to that seen with strong mineral acids. Sensitization may also occur, but is more common with the anhydrides than the acids.

ACETIC ACID

DESCRIPTION

CH_3 COOH, acetic acid, is a colorless liquid with a pungent vinegar-like odor. Glacial acetic acid contains 99% acid.

SYNONYMS

Ethanoic acid, ethylic acid, methane carboxylic acid, pyroligneous acid, vinegar acid.

POTENTIAL OCCUPATIONAL EXPOSURES

Acetic acid is widely used as a chemical feedstock for the production of vinyl plastics, acetic anhydride, acetone, acetanilide, acetyl chloride, ethyl alcohol, ketene, methyl ethyl ketone, acetate esters, and cellulose acetates. It is also used alone in the dye, rubber, pharmaceutical, food preserving, textile, and laundry industries. It is utilized, too, in the manufacture of Paris green, white lead, tint rinse, photographic chemicals, stain removers, insecticides, and plastics.

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

- Acetate ester makers
- Acetate fiber makers
- Aspirin makers
- Dye makers
- Food preservers
- Insecticide makers
- Laundry workers
- Photographic chemical makers
- Plastic makers
- Resin makers
- Rubber makers
- Stain removers
- Textile printers
- Tint rinse makers
- White lead makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 10 ppm (25 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Acetic acid vapor may produce irritation of the eyes, nose, throat, and lungs. Inhalation of concentrated vapors may cause serious damage to the lining membranes of the nose, throat, and lungs. Contact with concentrated acetic acid may cause severe damage to the skin and severe eye damage, which may result in loss of sight. Repeated or prolonged exposure to acetic acid may cause darkening, irritation of the skin, erosion of the exposed front teeth, and chronic inflammation of the nose, throat, and bronchi.

Systemic—

Bronchopneumonia and pulmonary edema may develop following acute overexposure. Chronic exposure may result in pharyngitis and catarrhal bronchitis. Ingestion, though not likely to occur in industry, may result in penetration of the esophagus, bloody vomiting, diarrhea, shock, hemolysis, and hemoglobinuria which is followed by anuria.

MEDICAL SURVEILLANCE

Consideration should be given to the skin, eyes, teeth, and respiratory tract in placement or periodic examinations.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

When working with glacial acetic acid, personal protective equipment, protective clothing, gloves, and goggles should be worn. Eye fountains and showers should be available in areas of potential exposure.

BIBLIOGRAPHY

Capellini, A., and E. Sartorelli. 1967. Epidodio di intossicizione collettiva da anidride acetica ed acido acetica. Med. Lav. 58:108.

Henson, E. V. 1959. Toxicology of the fatty acids. J. Occup. Med. 1:339.

Von Oettingen, W. F. 1960. The aliphatic acids and their esters: toxicity and potential dangers. The saturated monobasic acids and their esters: aliphatic acids with three to eighteen carbons and their esters. AMA Arch. Ind. Health 21:100.

ACETIC ANHYDRIDE

DESCRIPTION

$CH_3COOCOCH_3$, acetic anhydride, is a colorless, strongly refractive liquid which has a strongly irritating odor.

SYNONYMS

Acetic oxide, acetyl oxide, ethanoic anhydride.

POTENTIAL OCCUPATIONAL EXPOSURES

Acetic anhydride is used as an acetylating agent or as a solvent in the manufacture of cellulose acetate, acetanilide, synthetic fibers, plastics, explosives, resins, pharmaceuticals, perfumes, and flavorings; and it is used in the textile dyeing industry.

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

- Acetate fiber makers
- Acetic acid makers
- Aspirin makers
- Cellulose acetate fiber makers
- Drug makers
- Dye makers
- Explosive makers
- Flavoring makers
- Perfume makers
- Photographic film makers
- Plastic makers
- Resin makers
- Textile makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (20 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

In high concentrations, vapor may cause conjunctivitis, photophobia, lacrimation, and severe irritation of the nose and throat. Liquid acetic anhydride does not cause a severe burning sensation when it comes in contact with the skin. If it is not removed, the skin may become white and wrinkled, and delayed severe burns may occur. Both liquid and vapor may cause conjunctival edema and corneal burns, which may develop into temporary or permanent interstitial keratitis with cor-

neal opacity due to progression of the infiltration. Contact and, occasionally, hypersensivitity dermatitis may develop.

Systemic—

Immediate complaints following concentrated vapor exposure include conjunctival and nasopharyngeal irritation, cough, and dyspnea. Necrotic areas of mucous membranes may be present following acute exposure.

MEDICAL SURVEILLANCE

Consideration should be given to the skin, eyes, and respiratory tract in any placement or periodic examinations.

SPECIAL TESTS

None currently used.

PERSONAL PROTECTIVE METHODS

Personal protective equipment (protective clothing, gloves, and goggles) should be used. Eye fountains and showers should be made available in areas where contact might occur.

BIBLIOGRAPHY

Grant, W. M. 1962. Toxicology of the Eye. Charles C. Thomas Publishers, Springfield, Illinois.

Takhirov, M. T. 1969. Hygienic standard for acetic acid and acetic anhydride. Hyg. Sanit. 34:122.

FORMIC ACID

DESCRIPTION

HCOOH, formic acid, is a colorless, flammable, fuming liquid, with a pungent odor.

SYNONYMS

Methanoic acid, formylic acid, hydrogen carboxylic acid.

POTENTIAL OCCUPATIONAL EXPOSURES

Formic acid is a strong reducing agent and is used as a decalcifier. It is used in dyeing color fast wool, electroplating, coagulating latex rubber, regenerating old rubber, and dehairing, plumping, and tanning leather. It is also used in the manufacture of acetic acid, airplane dope, allyl alcohol, cellulose formate, phenolic resins, and oxalate; and it is used in the laundry, textile, insecticide, refrigeration, and paper industries.

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

Airplane dope makers
Allyl alcohol makers
Dyers
Electroplaters
Insecticide makers
Lacquer makers
Laundry workers
Leather makers
Paper makers
Perfume makers
Rubber workers
Textile makers
Wine makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (9 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor and percutaneous absorption.

HARMFUL EFFECTS

Local—

The primary hazard of formic acid results from severe irritation of the skin, eyes, and mucous membranes. Lacrimation, increased nasal discharge, cough, throat discomfort, erythema, and blistering may occur depending upon solution concentrations.

Systemic—

These have not been reported from inhalation exposure and are unlikely due to its good warning properties.

Swallowing formic acid has caused a number of cases of severe poisoning and death. The symptoms found in this type of poisoning include salivation, vomiting, burning sensation in the mouth, bloody vomiting, diarrhea, and pain. In severe poisoning, shock may occur. Later, breathing difficulties may develop. Kidney damage may also be present.

MEDICAL SURVEILLANCE

Consideration should be given to possible irritant effects on the skin, eyes, and lungs in any placement or periodic examinations.

SPECIAL TESTS

None currently used.

PERSONAL PROTECTIVE METHODS

Workers should be supplied with protective clothing, gloves, and goggles. Respiratory protection will be needed in areas of high vapor exposure.

BIBLIOGRAPHY

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Malorny, G. 1969. Die akute und chronische toxizitat der ameisenaure und ihrer formiate. Ernaechrungswiss. 9:332.

Von Oettingen, W. F. 1959. The aliphatic acids and their esters — toxicity and potential dangers. The saturated monobasic aliphatic acids and their esters. AMA Arch. Ind. Health 20:517.

OXALIC ACID

DESCRIPTION

HOOCCOOH. $2H_2O$, oxalic acid in solution, is a colorless liquid. Anhydrous oxalic acid is monoclinic in form and is produced by careful drying of the crystalline dihydrate.

SYNONYMS

Dicarboxylic acid, ethane-di-acid, ethanedioic acid.

POTENTIAL OCCUPATIONAL EXPOSURES

Oxalic acid is used as an analytic reagent and in the manufacture of dyes, inks, bleaches, paint removers, varnishes, wood and metal cleansers, dextrin, cream of tartar, celluloid, oxalates, tartaric acid, purified methyl alcohol, glycerol, and stable hydrogen cyanide. It is also used in the photographic, ceramic, metallurgic, rubber, leather, engraving, pharmaceutical, paper, and lithographic industries.

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

- Bleach makers
- Celluloid makers
- Ceramic makers
- Cream of tartar makers
- Dye makers
- Glycerine makers
- Ink makers
- Laundry workers
- Paper makers
- Rubber makers
- Tannery workers
- Textile dyers
- Printers
- Wood bleachers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1 mg/m^3.

ROUTE OF ENTRY

Inhalation of mist and, occasionally, dust.

HARMFUL EFFECTS

Local—

Liquid has a corrosive action on the skin, eyes, and mucous membranes, which may result in ulceration. Local prolonged contact with extremities may result in localized pain, cyanosis, and even gangrenous changes probably resulting from localized vascular damage.

Systemic—

Chronic exposure to mist or dust has been reported to cause chronic inflammation of the upper respiratory tract. Ingestion is of lesser importance occupationally. Symptoms appear rapidly and include shock, collapse, and convulsive seizures. Such cases may also have marked kidney damage with deposition of calcium oxalate in the lumen of the renal tubules.

MEDICAL SURVEILLANCE

Evaluate skin, respiratory tract, and renal functions in placement or periodic examinations.

SPECIAL TESTS

The presence of increased urinary oxalate crystals may be helpful in evaluating oral poisoning. Determination of blood calcium and oxalate levels may also be used for this purpose.

PERSONAL PROTECTIVE METHODS

Protective clothing and goggles should be worn when working in areas where direct contact is possible. Respiratory protection from mist or dust may be needed.

BIBLIOGRAPHY

Klauder, J. V., L. Shelanski, and K. Gabriel. 1955. Industrial uses of fluorine and oxalic acid. AMA Arch. Ind. Health 12:412.

PHTHALIC ANHYDRIDE

DESCRIPTION

$C_8H_4O_3$, phthalic anhydride, is moderately flammable, white, lustrous, solid, with needle-like crystals.

SYNONYMS

Phthalic acid anhydride, benzene-o-dicarboxylic acid anhydride, phthalandione.

POTENTIAL OCCUPATIONAL EXPOSURES

Phthalic anhydride is used in the manufacture of phthaleins, benzoic acid, alkyd and polyester resins, synthetic indigo, and phthalic acid, which is used as a plasticizer for vinyl resins. To a lesser extent, it is used in the production of alizarin dye, anthranilic acid, anthraquinone, diethyl phthalate, dimethyl phthalate, erythrosin, isophthalic acid, methyl aniline, phenolphthalein, phthalamide, sulfathalidine, and terephthalic acid. It has also found use in pesticides and herbicides, as well as perfumes.

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

- Alizarin dye makers
- Alkyd resin makers
- Automobile finish makers
- Cellulose acetate plasticizer makers
- Dacron fiber makers
- Erythrosin makers
- Insecticide makers
- Mylar plastic makers
- Organic chemical synthesizers
- Phthalein makers
- Resin makers
- Vat dye makers
- Vinyl plasticizer makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 2 ppm (12 mg/m^3).

ROUTE OF ENTRY

Inhalation of dust, fume, or vapor.

HARMFUL EFFECTS

Local—

Phthalic anhydride, in the form of a dust, fume, or vapor, is a potent irritant of the eyes, skin, and respiratory tract. The irritant effects are worse on moist surfaces. Conjunctivitis and skin erythema, burning, and contact dermatitis may occur. If the chemical is held in contact with the skin, as under clothes or shoes, skin burns may develop. Hypersensitivity may develop in some individuals. Inhalation of the dust or vapors may cause coughing, sneezing, and a bloody nasal discharge. Impurities, naphthoquinone, as well as maleic anhydride, may also contribute to eye, skin, and pulmonary irritation.

Systemic—

Repeated exposure may result in bronchitis, emphysema, allergic asthma, urticaria, and chronic eye irritation.

MEDICAL SURVEILLANCE

Emphasis should be given to a history of skin or pulmonary allergy, and preplacement and periodic examinations should evaluate the skin, eye, and lungs, as well as liver and kidney functions. The hydrolysis product, phthalic acid, is rapidly excreted in the urine, although this has not been used in biological monitoring. Diagnostic patch testing may be helpful in evaluating skin allergy.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Proper ventilation, rubber gloves, protective clothing, head coverings, and goggles are recommended when repeated or prolonged contact is possible. Respiratory protection may be needed in dusty areas or where fumes or vapors are present.

BIBLIOGRAPHY

Ghezzi, I., and P. Scott. 1965. Clinical contribution on the pathology induced by phthalic and maleic anhydride. Med. Lav. 56:746.

Merlevede, E., and J. Elskens. 1957. The toxicity of phthalic anhydride, maleic anhydride, and the phthalates. Arch. Belg. Med. Soc. 15:445.

ALDEHYDES AND KETONES

Aldehydes and ketones are aliphatic or aromatic organic compounds which contain the carbonyl group, C=O.

The aldehydes, R−CH=O, are used primarily as chemical feedstock because of their relatively high reactivity. They are volatile, colorless liquids, with the exception of formaldehyde, which is a gas, and can exhibit additional hazard due to its flammability. Typically, these compounds are strongly irritating to the skin, eyes, and respiratory tract. Acute exposure may result in pulmonary injuries such as edema, bronchitis, and bronchopneumonia. Skin and pulmonary sensitization may develop in some individuals and result in contact dermatitis and, more rarely, asthmatic attacks. After hypersensitivity develops, individuals may develop symptoms due to other aldehydes. For this reason, medical surveillance and industrial hygiene practices are of importance.

Ketones are characterized by the structure R-O-R. They are similar in their chemical and toxicological properties, and all are flammable, colorless liquids with a pungent odor similar to acetone. They are used as industrial solvents and raw materials or as intermediates in chemical synthesis. Prolonged exposure is usually precluded by the intense irritation of the eyes and respiratory tract.

ACETALDEHYDE

DESCRIPTION

CH_3CHO, acetaldehyde, is a flammable, volatile, colorless liquid with a characteristic odor. It is produced by oxidation of alcohol with a metallic catalyst, by hydration of acetylene, or, usually, by direct oxidation of ethylene.

SYNONYMS

Acetic aldehyde, aldehyde, ethanol, ethyl aldehyde.

POTENTIAL OCCUPATIONAL EXPOSURES

Acetaldehyde can be reduced or oxidized to form acetic acid, acetic anhydride, acrolein, aldol, buranol, chloral, 2-methyl-5-ethyl pyridine, paraldehyde, and pentaerythritol. It is also used in the manufacture of disinfectants, drugs, dyes, explosives, flavorings, lacquers, mirrors (silvering), perfume, photographic chemicals, phenolic and urea resins, rubber accelerators and antioxidants, varnishes, vinegar, and yeast.

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

- Acetic acid makers
- Antioxidant makers
- Disinfectant makers
- Explosives workers
- Flavoring makers
- Mirror silverers
- Paraldehyde makers
- Urea resin makers
- Rubber makers
- Vinegar makers
- Yeast makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 200 ppm (360 mg/m^3); however, the ACGIH 1976 recommended TLV is 100 ppm (180 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

The liquid and the fairly low levels of the vapor are irritating to the eyes, skin, upper respiratory passages, and bronchi. Repeated exposure may result in dermatitis, rarely, and skin sensitization.

Systemic—

Acute involuntary exposure to high levels of acetaldehyde vapors may result in pulmonary edema, preceded by excitement, followed by narcosis. It has been postulated that these symptoms may have been similar to those of alcohol, which is converted to acetaldehyde and acetic acid. Chronic effects have not been documented, and seem unlikely, since voluntary inhalation of toxicologically significant levels of acetaldehyde are precluded by its irritant properties at levels as low as 200 ppm (360 mg/m^3 of air).

MEDICAL SURVEILLANCE

Consideration should be given to skin, eyes, and respiratory tract in any preplacement or periodic examinations.

SPECIAL TESTS

None appear needed, but effects of exposure can be determined from blood results by gas chromatographic methods, if desired.

PERSONAL PROTECTIVE METHODS

Protective clothing, gloves, goggles, and respiratory protective equipment where high concentrations of the gas or vapor are expected.

BIBLIOGRAPHY

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Egle, J. L. 1972. Effects of inhaled acetaldehyde and propionaldehyde on blood pressure and heart rate. Toxicol. Appl. Pharmacol. 23:131.

Fairhall, L. T. 1969. Industrial Toxicology, 2nd ed. Hafner Publishing Company, New York. p. 138.

James, T. N., and E. S. Bear. 1967. Effects of ethanol and acetaldehyde on the heart. Am. Heart J. 74:243.

ACROLEIN

DESCRIPTION

$H_2C=CHCHO$, acrolein, is a clear, yellowish liquid which is a petroleum byproduct. It is commercially produced by the oxidation of propylene.

SYNONYMS

Acraldehyde, acrylic aldehyde, allyl aldehyde, propenal.

POTENTIAL OCCUPATIONAL EXPOSURES

Acrolein is the feedstock for several types of plastics, plasticizers, acrylates, textile finishes, synthetic fibers, and methionines. It is also used in the manufacture of colloidal forms of metals, in perfumes, and, due to its pungent odor, as a warning agent in methyl chloride refrigerants. Other potential exposures may arise when acrolein vapor is given off when oils and fats containing glycerol are heated.

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

- Acrylate makers
- Fat processors
- Methionine makers
- Perfume makers
- Plastic makers
- Refrigerant makers
- Renderers
- Rubber makers
- Textile resin makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for exposure to acrolein is 0.1 ppm (0.25 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor and percutaneous absorption.

HARMFUL EFFECTS

Local—

In the liquid or pungent vapor form, acrolein produces intense irritation to the eye and mucous membranes of the respiratory tract. Skin burns and dermatitis may result from prolonged or repeated exposure. Sensitization in a few individuals may also occur.

Systemic—

Because of acrolein's pungent, offensive odor and the intense irritation of the conjunctiva and upper respiratory tract, severe toxic effects from acute exposure are rare, as workmen will not tolerate the vapor even in minimal concentrations. Acute exposure to acrolein may cause bronchial inflammation, resulting in bronchitis or pulmonary edema.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should consider respiratory, skin, and eye disease. Pulmonary function tests may be helpful.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Protection in handling and transporting is advocated. Suitable ventilation and protective clothing should be provided for employees working in areas of possible exposure. Protective respiratory equipment in areas of vapor concentration.

BIBLIOGRAPHY

Champeix, J., L. Courtial, E. Perche, and P. Catalina. 1966. Bronchopneumopathe aigue par vapeurs d'acroleine. Arch. Mal. Prof. 17:794.

Gusev, M. I., A. I. Schechnikova, I. S. Dronov, M. D. Grebenskova, and A. I. Golovina. 1966. Determination of the daily average maximum permissible concentration of acrolein in the atmosphere. Hyg. Sanit. 31(1):8.

FORMALDEHYDE

DESCRIPTION

HCHO, formaldehyde, is a colorless, pungent gas. It is produced commercially by the catalytic oxidation of methyl alcohol and sold in aqueous solution containing 30-50% formaldehyde and from 0-15% methanol, which is added to prevent polymerization. Formaldehyde solution is called formalin, formol, or morbicid.

SYNONYMS

Oxomethane, oxymethylene, methylene oxide, formic aldehyde, methyl aldehyde.

POTENTIAL OCCUPATIONAL EXPOSURES

Formaldehyde has found wide industrial usage as a fungicide, germicide, and in disinfectants and embalming fluids. It is also used in the manufacture of artificial silk and textiles, latex, phenol, urea, thiourea and melamine resins, dyes, and inks, cellulose esters and other organic molecules, mirrors, and explosives. It is also used in the paper, photographic, and furniture industries.

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

- Anatomists
- Biologists
- Deodorant makers
- Disinfectant makers
- Embalming fluid makers
- Formaldehyde resin makers
- Hide preservers
- Ink makers
- Latex makers
- Photographic film makers
- Textile printers
- Wood preservers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 3 ppm determined as a TWA. The acceptable ceiling concentration is 5 ppm with an acceptable maximum peak above this value of 10 ppm for a maximum duration of 30 minutes. ACGIH in 1975 recommended a TLV of 2 ppm (3 mg/m^3) as a ceiling value. NIOSH has recommended a ceiling of 1 ppm (1.2 mg/m^3) for any 30-minute sampling period.

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

Formaldehyde gas may cause severe irritation to the mucous membranes of the respiratory tract and eyes. The aqueous solution splashed in the eyes may cause eye burns. Urticaria has been reported following inhalation of gas. Repeated exposure to formaldehyde may cause dermatitis either from irritation or allergy.

Systemic—

Systemic intoxication is unlikely to occur since intense irritation of upper respiratory passages compels workers to leave areas of exposure. If workers do inhale high concentrations of formaldehyde, coughing, difficulty in breathing, and pulmonary edema may occur. Ingestion, though usually not occurring in industrial experience, may cause severe irritation of the mouth, throat, and stomach.

MEDICAL SURVEILLANCE

Consider the skin, eyes, and respiratory tract in any preplacement or periodic examination, especially if the patient has a history of allergies.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Prevention of intoxication may be easily accomplished by supplying adequate ventilation and protective clothing. Barrier creams may also be helpful. In areas of high vapor concentrations, full protective face masks with air supply is needed, as well as protective clothing.

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FURFURAL

DESCRIPTION

$C_5H_4O_2$, furfural, is an aromatic heterocyclic aldehyde with an amber color and aromatic odor. This liquid is obtained from cereal straws and brans containing pentosans by hydrolysis and dehydration with sulfuric acid.

SYNONYMS

Furfurol (a misnomer), furfuraldahyde, artificial ant oil, pyromucic aldehyde, furol, 2-furaldehyde.

POTENTIAL OCCUPATIONAL EXPOSURES

Furfural is used as a solvent for wood resin, nitrated cotton, cellulose acetate, and gums. It is used in the production of phenolic plastics, thermosetting resins, refined petroleum oils, dyes, and varnishes. It is also utilized in the manufacture of pyromucic acid, vulcanized rubber, insecticides, fungicides, herbicides, germicides, furan derivatives, polymers, and other organic chemicals.

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

- Adipic acid makers
- Butadiene refiners
- Cellulose acetate makers
- Disinfectant workers
- Dye makers
- Herbicide makers
- Lysine makers
- Metal refiners
- Nylon makers
- Organic chemical synthesizers
- Pyromucic acid makers
- Road builders

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (20 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapor, percutaneous absorption.

HARMFUL EFFECTS

Local—

Liquid and concentrated vapor are irritating to the eyes, skin, and mucous membranes of the upper respiratory tract. Eczematous dermatitis as well as skin sensitization, resulting in allergic contact dermatitis and photosensitivity, may develop following repeated exposure.

Systemic—

Workers chronically exposed to the vapor have had complaints of headache, fatigue, itching of the throat, lacrimation, loss of the sense of taste, numbness of the tongue, and tremor. Occupational overexposure is relatively rare due to the liquid's low vapor pressure, and symptoms usually disappear rapidly after removal from exposure.

MEDICAL SURVEILLANCE

Consider skin irritation and skin allergies (especially to aldehydes) in preplacement or periodic examinations. Also consider possible respiratory irritant effects.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

Protective clothing and adequate ventilation should be provided in areas where toxic exposure may occur. In areas of vapor concentrations, fullface masks may be required.

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KETONES

DESCRIPTION

The ketone family includes:

Acetone: CH_3COCH_3
Diacetone: $(CH_3)_2COHCH_2COCH_3$
Methyl ethyl ketone: $CH_3COCH_2CH_3$
Methyl n-propyl ketone: $CH_3CH_2CH_2COCH_3$
Methyl n-butyl ketone: $CH_3COCH_2CH_2CH_2CH_3$
Methyl isobutyl ketone: $(CH_3)_2CHCH_2COCH_3$

SYNONYMS

Acetone: 2-Propanone, dimethyl ketone, beta-ketopropane, pyroacetic ether.

Diacetone: Diacetone alcohol, diacetonyl alcohol, dimethylacetonyl

carbinol, 4-hydroxy-4 methyl-2-pentanone, 2-methyl-2-pentanol-4-one.

Methyl ethyl ketone: Butanone, 2-butanone, MEK, ethyl methyl ketone,

Methyl n-propyl ketone: Ethyl acetone, 2-pentanone, MPK.

Methyl n-butyl ketone: n-Butyl methyl ketone, propyl acetone, 2-hexanone, MBK.

Methyl isobutyl ketone: Hexone, isopropylacetone, 4-methyl-2-pentanone, MIBK.

POTENTIAL OCCUPATIONAL EXPOSURES

This group of ketone solvents has many uses in common. They are low-cost solvents for resins, lacquers, oils, fats, collodion, cotton, cellulose acetate, nitrocellulose, cellulose esters, epoxy resins, gums, pigments, dyes, vinyl polymers, and copolymers. They are used as chemical intermediates, in the manufacture of smokeless powder and explosives, and in the paint, lacquer, varnish, plastics, dyeing, celluloid, photographic, cement, rubber, artificial silk and leather, synthetic rubber, and lubricating oil industries. They are also used in hydraulic fluids, metal cleaning compounds, quick drying inks, airplane dopes, compositions for paper and textiles, pharmaceuticals, cosmetics, and as paint removers and dewaxers.

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

- Adhesive makers
- Celluloid makers
- Dope workers
- Dye makers
- Explosive makers
- Garage mechanics
- Lacquer and oil processors
- Shoe makers
- Solvent workers
- Varnish and stain makers
- Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are:

Acetone	1,000 ppm	2,400 mg/m^3
Diacetone	50 ppm	240 mg/m^3
Methyl ethyl ketone	200 ppm	590 mg/m^3
Methyl n-propyl ketone	200 ppm	700 mg/m^3
Methyl n-butyl ketone	100 ppm	410 mg/m^3
Methyl isobutyl ketone	100 ppm	410 mg/m^3

ROUTES OF ENTRY

Inhalation of vapor, percutaneous absorption.

HARMFUL EFFECTS

Local—

These solvents may produce a dry, scaly, and fissured dermatitis after repeated exposure. High vapor concentrations may irritate the conjunctiva and mucous membranes of the nose and throat, producing eye and throat symptoms.

Systemic—

In high concentrations, narcosis is produced, with symptoms of headache, nausea, light headedness, vomiting, dizziness, incoordination, and unconsciousness.

Recent reports indicate that exposure of workers to methyl n-butyl ketone has been associated with the development of peripheral neuropathy. Rat experiments have also shown nerve changes characteristic of peripheral neuropathy after exposure to 1,300 ppm.

MEDICAL SURVEILLANCE

Preplacement examinations should evaluate skin and respiratory conditions. In the case of methyl n-butyl ketone, special attention should be given to the central and peripheral nervous systems.

SPECIAL TESTS

Acetone can be determined in the blood, urine, and expired air, and has been used as an index of exposure. A neurotoxic metabolite of methyl n-butyl ketone has been found in the rat. This is not present with methyl isobutyl ketone.

PERSONAL PROTECTIVE METHODS

Contact with skin and eyes should be avoided by the use of protective clothing. In areas of high vapor concentration, masks should be used.

BIBLIOGRAPHY

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ALIPHATIC HALOGENATED HYDROCARBONS

Halogenated hydrocarbons typically are colorless, volatile liquids with excellent organic solvent properties. Chemically, they consist of saturated or unsaturated carbon chains in which one hydrogen atom or more have been replaced by one or more halogens (fluorine, chlorine, bromine, or iodine). Hydrocarbons having only one or two halogens are usually flammable and less toxic than similar hydrocarbons with complete halogen substitution. Hydrocarbons containing fluorine tend to be less toxic, while hydrocarbons containing bromine or iodine are generally more toxic than hydrocarbons containing chlorine. The latter, chlorinated hydrocarbons, are widely used because of their low cost.

Halogenated hydrocarbons have found wide use as solvents in degreasing, dewaxing, drycleaning, and extracting processes. Other uses are as aerosol propellants, fumigants, insecticides, refrigerants, and

chemical intermediates for drugs, plastics, and synthetic rubber. Some of these compounds have been used medically as anesthetics and anthelmintics. The toxicologic effects of halogenated hydrocarbons vary from one compound to another but, generally, most cause central nervous system depression. Also common to most is the defatting of the skin which may lead to dermatitis. Upon inhalation of high concentrations of vapor, liver or kidney injury may occur, but it should be noted that while some compounds may have no effect, others may affect only one of these two organs, and still others may affect both. Pulmonary irritation and damage to the hematopoietic system may also occur after exposure to certain compounds.

Medical surveillance of workers exposed to halogenated hydrocarbons should include periodic examinations, including urinalysis to check for renal damage and appropriate tests for liver dysfunction. Evidence of acute exposure to chlorinated compounds may be obtained in some cases by analysis of expired air obtained soon after exposure. If acute poisoning is suspected, epinephrine should not be used in treatment as it has been noted that epinephrine injected during narcosis caused by exposure to certain halogenated solvents may lead to cardiac arrhythmia.

CARBON TETRACHLORIDE

DESCRIPTION

CCl_4, carbon tetrachloride, is a colorless, nonflammable liquid with a characteristic odor. Oxidative decomposition by flame causes phosgene and hydrogen chloride to form.

SYNONYMS

Tetrachloromethane, perchloromethane.

POTENTIAL OCCUPATIONAL EXPOSURES

Carbon tetrachloride is used as a solvent for oils, fats, lacquers, varnishes, rubber, waxes, and resins. Fluorocarbons are chemically synthesized from it. It is also used as an azeotropic drying agent for spark plugs, a dry cleaning agent, a fire extinguishing agent, a fumigant, and an anthelmintic agent. The use of this solvent is widespread, and substitution of less toxic solvents when technically possible is recommended.

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

- Chemists
- Degreasers
- Fat processors
- Firemen
- Fluorocarbon makers
- Grain fumigators
- Ink makers
- Insecticide makers
- Lacquer makers
- Metal cleaners
- Propellant makers
- Refrigerant makers
- Rubber makers
- Solvent workers
- Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 10 ppm (65 mg/m^3) as an 8-hour TWA

with an acceptable ceiling concentration of 25 ppm; acceptable maximum peaks above the ceiling of 200 ppm are allowed for one 5-minute duration in any 4-hour period. NIOSH has recommended a ceiling limit of 2 ppm based on a 1-hour sampling period at a rate of 750 ml/min.

ROUTES OF ENTRY

Inhalation of vapor. Percutaneous absorption has been demonstrated in animals.

HARMFUL EFFECTS

Local—

Carbon tetrachloride solvent removes the natural lipid cover of the skin. Repeated contact may lead to a dry, scaly, fissured dermatitis. Eye contact is slightly irritating, but this condition is transient.

Systemic—

Excessive exposure may result in central nervous system depression, and gastrointestinal symptoms may also occur. Following acute exposure, signs and symptoms of liver and kidney damage may develop. Nausea, vomiting, abdominal pain, diarrhea, enlarged and tender liver, and jaundice result from toxic hepatitis. Diminished urinary volume, red and white blood cells in the urine, albuminuria, coma, and death may be consequences of acute renal failure. The hazard of systemic effects is increased when carbon tetrachloride is used in conjunction with ingested alcohol.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include an evaluation of alcohol intake and appropriate tests for liver and kidney functions. Special attention should be given to the central and peripheral nervous system, the skin, and blood.

SPECIAL TESTS

Expired air and blood levels may be useful as indicators of exposure.

PERSONAL PROTECTIVE METHODS

Barrier creams, gloves, protective clothing, and masks should be used as appropriate where exposure occurs.

BIBLIOGRAPHY

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Luse, S. A., and W. G. Wood. 1967. The brain in fatal carbon tetrachloride poisoning. Arch. Neurol. 17:304.

Nielson, V. K., and J. Larsen. 1965. Acute renal failure due to carbon tetrachloride poisoning. Acta. Med. Scand. p. 178.

CHLOROFORM

DESCRIPTION

$CHCl_3$, chloroform, is a clear, colorless liquid with a characteristic odor. Though nonflammable, chloroform decomposes to form hydrochloric acid, phosgene, and chlorine upon contact with a flame.

SYNONYMS

Trichloromethane, methenyl chloride.

POTENTIAL OCCUPATIONAL EXPOSURES

Chloroform was one of the earliest general anesthetics, but its use for this purpose has been abandoned because of toxic effects. Chloroform is widely used as a solvent (especially in the lacquer industry); in the extraction and purification of penicillin and other pharmaceuticals; in the manufacture of artificial silk, plastics, floor polishes, and fluorocarbons; and in sterilization of catgut.

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

- Chemists
- Drug makers
- Fluorocarbon makers
- Lacquer workers
- Polish makers
- Silk synthesizers
- Solvent workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 50 ppm (240 mg/m^3). The ACGIH recommended 1976 TLV is 25 ppm. NIOSH's recommended limit is a ceiling of 2 ppm based on a 1-hour sample collected at 750 l/min.

ROUTE OF ENTRY

Inhalation of vapors.

HARMFUL EFFECTS

Local—

Chloroform may produce burns if left in contact with the skin.

Systemic—

Chloroform is a relatively potent anesthetic at high concentrations. Death from its use as an anesthetic has resulted from liver damage and from cardiac arrest. Exposure may cause lassitude, digestive disturbance, dizziness, mental dullness, and coma. Chronic overexposure has been shown to cause enlargement of the liver and kidney damage. Alcoholics seem to be affected sooner and more severely from chloroform exposure. Disturbance of the liver is more characteristic of exposure than central nervous system depression or renal injury. There is some animal experimental evidence that suggests chloroform may be a carcinogen.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include appropriate

tests for liver and kidney functions, and special attention should be given to the nervous system, the skin, and to the history of alcoholism.

SPECIAL TESTS

Expired air and blood levels may be useful in estimating levels of acute exposure.

PERSONAL PROTECTIVE METHODS

Protective clothing and gloves should be worn to protect the skin, and masks should be worn in areas of high vapor concentration.

BIBLIOGRAPHY

Challen, P. J. R., D. E. Hickish, and J. R. Bedford. 1958. Chronic chloroform intoxication. Br. J. Ind. Med. 15:243.

CHLOROPRENE

DESCRIPTION

$H_2C=CC_1-CH=CH_2$, chloroprene, is a colorless, flammable liquid posessing a pungent odor.

SYNONYMS

2-chloro-1,3-butadiene.

POTENTIAL OCCUPATIONAL EXPOSURES

The only major use of chloroprene is in the production of artificial rubber (neoprene, duprene).

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

Duprene makers
Neoprene makers
Rubber makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 25 ppm (90 mg/m^3). Because of suspected carcinogenic and/or mutagenic potential, the limit may be lowered.

ROUTES OF ENTRY

Inhalation of vapor and skin absorption.

HARMFUL EFFECTS

Local—

Chloroprene acts as a primary irritant on contact with skin, conjunctiva, and mucous membranes and may result in dermatitis, conjunctivitis, and circumscribed necrosis of the cornea. Temporary hair loss has been reported during the manufacture of polymers.

Systemic—

Inhalation of high concentrations may result in anesthesia and respiratory paralysis. Chronic exposure may produce damage to the lungs,

nervous system, liver, kidneys, spleen, and myocardium. Russian studies suggest that chloroprene exposure is associated with an increased incidence of cancer of the skin and lungs, but this has not been confirmed. Fetal effects have been noted in rodents.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include an evaluation of the skin, eyes, respiratory tract, and central nervous system. Liver and kidney function should be evaluated.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

Protective clothing, chemical safety goggles, air-supplied or self-contained respirators, and safety harnesses should be worn where there is exposure to the liquid or high concentrations of the vapor.

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Lloyd, J. W., P. Decouflé, and R. W. Moore, Jr. 1975. Background information on chloroprene. J. Occup. Med. 17:263.

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1,2-DIBROMOETHANE

DESCRIPTION

$BrCH_2CH_2Br$, 1,2-dibromoethane, is a colorless nonflammable liquid with a chloroformlike odor.

SYNONYMS

Ethylene dibromide, ethylene bromide, sym-dibromoethane.

POTENTIAL OCCUPATIONAL EXPOSURES

1,2-Dibromoethane is used principally as a fumigant for ground pest control and as a constituent of ethyl gasoline. It is also used in fire extinguishers, gauge fluids, and waterproofing preparations; and it is used as a solvent for celluloid, fats, oils, and waxes.

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

- Antiknock compound makers
- Cabbage growers
- Drug makers
- Farmers
- Fat processors
- Fire extinguisher makers
- Fumigant workers
- Gum processors
- Lead scavenger makers
- Resin makers
- Termite controllers
- Tetraethyl lead makers
- Wool reclaimers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 20 ppm (145 mg/m^3) as an 8-hour TWA with an acceptable ceiling concentration of 300 ppm; acceptable maximum peaks above the ceiling of 50 ppm are allowed for 5 minutes duration.

ROUTES OF ENTRY

Inhalation of the vapor and absorption through the skin.

HARMFUL EFFECTS

Local—

Prolonged contact of the liquid with the skin may cause erythema, blistering, and skin ulcers. These reactions may be delayed 24-48 hours. Dermal sensitization to the liquid may develop. The vapor is irritating to the eyes and to the mucous membranes of the respiratory tract.

Systemic—

Inhalation of the vapor may result in severe acute respiratory injury, central nervous system depression, and severe vomiting. Animal experiments have produced injury to the liver and kidneys.

MEDICAL SURVEILLANCE

Preemployment and periodic examinations should evaluate the skin and eyes, respiratory tract, and liver and kidney functions.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

1,2-Dibromoethane can penetrate most types of rubber and leather protective clothing, and, therefore, protective clothing made from other materials should be provided. Masks must be worn in areas with excessive vapor concentrations.

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1,2-DICHLOROETHANE

DESCRIPTION

$Cl_1CH_2CH_2Cl$, 1,2-dichloroethane, is a colorless, flammable liquid which has a pleasant odor, sweetish taste.

SYNONYMS

Ethylene dichloride, sym-dichloroethane, ethylene chloride, glycol dichloride, β-dichloroethane.

POTENTIAL OCCUPATIONAL EXPOSURES

In recent years, 1,2-dichloroethane has found wide use in the manufacture of ethyl glycol, diaminoethylene, chloroecholine chloride, polyvinyl chloride, nylon, viscose rayon, styrene-butadiene rubber, and various plastics. It is a solvent for resins, asphalt, bitumen, rubber, cellulose acetate, cellulose ester, and paint; a degreaser in the engineering, textile, and petroleum industries; and an extracting agent for soya bean oil and caffeine. It is also used as an antiknock agent in gasoline, a pickling agent, a fumigant, and a drycleaning agent. It has found use in photography, xerography, water softening, and in the production of adhesives, cosmetics, pharmaceuticals, and varnishes.

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

- Adhesive makers
- Bakelite processors
- Camphor workers
- Drycleaners
- Exterminators
- Gasoline blenders
- Insecticide makers
- Metal degreasers
- Ore upgraders
- Solvent workers
- Textile cleaners
- Vinyl chloride makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 50 ppm (200 mg/m^3) as an 8-hour TWA with an acceptable ceiling concentration of 100 ppm. Acceptable maximum peaks above the ceiling of 200 ppm are allowed for 5 minutes duration in any 3-hour period.

ROUTES OF ENTRY

Inhalation of vapor and skin absorption of liquid.

HARMFUL EFFECTS

Local—

Repeated contact with liquid can produce a dry, scaly, fissured dermatitis. Liquid and vapor may also cause eye damage.

Systemic—

Inhalation of high concentrations may cause nausea, vomiting, mental confusion, dizziness, and pulmonary edema. Chronic exposure has been associated with liver and kidney damage.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include an evaluation of the skin and liver and kidney functions.

SPECIAL TESTS

None commonly used; can be determined in expired air.

PERSONAL PROTECTIVE METHODS

Protective clothing, goggles, and gloves are to be worn. In high vapor concentrations,use fullface masks and supplied air respirators.

BIBLIOGRAPHY

Heppel, L. A., P. A. Neal, T. L. Perrin, K. M. Endicott, and V. T. Porterfield. 1945. The toxicity of 1, 2-dichloroethane (ethylene) (sic III). Its acute toxicity and the effect of protective agents. J. Pharmacol. Exp. Ther. 84:53.

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Yodiken, R. E., and J. R. Babcock, Jr., 1973. 1,2-Dichloroethane poisoning. Arch. Environ. Health 26:281.

1,2-DICHLOROETHYLENE

DESCRIPTION

ClCH=CHCl, 1,2-dichloroethylene, exists in two isomers, cis 60% and trans 40%. There are variations in toxicity between these two forms. At room temperature, it is a liquid with a slight acrid, ethereal odor. Gradual decomposition results in hydrochloric acid formation in the presence of ultraviolet light or upon contact wth hot metal.

SYNONYMS

Acetylene dichloride, sym-dichloroethylene, 1,2-dichloroethene.

POTENTIAL OCCUPATIONAL EXPOSURES

1,2-Dichloroethylene is used as a solvent for waxes, resins, and acetyl cellulose. It is also used in the extraction of rubber, as a refrigerant, in the manufacture of pharmaceuticals and artificial pearls, and in the extraction of oils and fats from fish and meat.

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

Carbolic acid processors
Drug makers
Drycleaners
Solvent workers
Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 200 ppm (790 mg/m^3).

ROUTE OF ENTRY

Inhalation of the vapor.

HARMFUL EFFECTS

Local—

This liquid can act as a primary irritant producing dermatitis and irritation of mucous membranes.

Systemic—

1,2-Dichloroethylene acts principally as a narcotic, causing central nervous system depression. Symptoms of acute exposure include dizzi-

ness, nausea and frequent vomiting, and central nervous system intoxication similar to that caused by alcohol. Renal effects, when they do occur, are transient.

MEDICAL SURVEILLANCE

Consider possible irritant effects on skin or respiratory tract as well as liver and renal function in preplacement or periodic examinations.

SPECIAL TESTS

None commonly used; expired air analyses may be useful in detecting exposure.

PERSONAL PROTECTIVE METHODS

Barrier creams and gloves are needed to protect the skin. In high vapor concentrations, masks and protective clothing are required.

BIBLIOGRAPHY

McBirney, R. S. 1954. Trichloroethylene and dichloroethylene poisoning. AMA Arch. Ind. Hyg. Occup. Med. 10:130.

ETHYL CHLORIDE

DESCRIPTION

CH_3CH_2Cl, ethyl chloride, is a flammable gas with an ethereal odor, burning taste. It is flammable, and the products of combustion include phosgene and hydrogen chloride.

SYNONYMS

Monochloroethane, hydrochloric ether, chloroethane, chlorethyl.

POTENTIAL OCCUPATIONAL EXPOSURES

Ethyl chloride is used as an ethylating agent in the manufacture of tetraethyl lead, dyes, drugs, and ethyl cellulose. It can be used as a refrigerant and as a local anesthetic (freezing).

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

- Anesthetists
- Dentists
- Drug makers
- Ethylation workers
- Fat and oil processors
- Nurses
- Perfume makers
- Phosphorus and sulfur processors
- Physicians
- Refrigeration workers
- Resin makers
- Sulfur processors
- Tetraethyl lead makers
- Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1000 ppm (2600 mg/m^3).

ROUTES OF ENTRY

Inhalation of gas and slight percutaneous absorption.

HARMFUL EFFECTS

Local—

The liquid form of ethyl chloride is mildly irritating to skin and eyes. Frostbite can occur due to rapid liquid evaporation.

Systemic—

Ethyl chloride exposure may produce headache, dizziness, incoordination, stomach cramps, and eventual loss of consciousness. In high concentrations, it is a respiratory tract irritant, and death due to cardiac arrest has been recorded. Renal damage has been reported in animals. Effects from chronic exposure have not been reported.

MEDICAL SURVEILLANCE

Consider possible acute cardiac effects in any preplacement or periodic examination.

SPECIAL TESTS

None commonly used. Ethyl chloride is excreted in expired air.

PERSONAL PROTECTIVE METHODS

In high vapor concentrations, use mask with full face protection, gloves, and protective clothing.

BIBLIOGRAPHY

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FLUOROCARBONS

DESCRIPTION

Chemically, fluorocarbons are hydrocarbons containing fluorine and include compounds that may contain other halogens in addition to fluorine. Generally, these compounds are colorless, nonflammable gases, though a few are liquids at room temperature. Decomposition of chlorine-containing fluoromethanes caused by contact with an open flame or hot metal produces hydrogen chloride, hydrogen fluoride, phosgene, carbon dioxide, and chlorine.

SYNONYMS

See listing of economically important fluorocarbons under "Permissible Exposure Limits." Also, Freon® is a trademark for a number of fluorocarbons used particularly in refrigeration products and equipment.

POTENTIAL OCCUPATIONAL EXPOSURES

The fluorocarbons are used primarily as refrigerants and polymer intermediates. They are also used as aerosol propellants, anesthetics, fire extinguishers, foam blowing agents, in drycleaning, and in degreasing of electronic equipment. The fluorocarbons have found wide use due to their relatively low toxicity.

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

- Aerosol bomb workers
- Ceramic mold makers
- Drycleaners
- Drug makers
- Fire extinguisher workers
- Heat transfer workers
- Metal conditioners
- Plastic makers
- Pressurized food makers
- Refrigeration makers
- Rocket fuel makers
- Solvent workers

PERMISSIBLE EXPOSURE LIMITS

Federal standards for selected fluorocarbons of economic importance are listed as follows:

Compound	Federal Standards	
	ppm	mg/m³
Bromotrifluoromethane Fluorocarbon 13B1	1,000	6,100
Dibromodifluoromethane Fluorocarbon 12B2	100	860
Dichlorodifluoromethane Fluorocarbon 12	1,000	4,950
Dichloromonofluoromethane Fluorocarbon 21	1,000	4,200
Dichlorotetrafluoroethane Fluorocarbon 114	1,000	7,000
Fluorotrichloromethane Fluorocarbon 11	1,000	5,600
1,1,1,2-Tetrachloro-2,2-difluoroethane Fluorocarbon 112	500	4,170
1,1,2,2-Tetrachloro-1,2-difluoroethane Fluorocarbon 112	500	4,170
1,1,2-Trichloro-1,2,2-trifluoroethane Fluorocarbon 113	1,000	7,600
Bromochlorotrifluoroethane Fluorocarbon 123B1	No Standard	
Chlorodifluoromethane Fluorocarbon 22	No Standard (ACGIH TLV of 1,000 ppm)	
Chloropentafluoroethane Fluorocarbon 115	No Standard	
Chlorotrifluoroethylene Fluorocarbon 1113	No Standard	
Chlorotrifluoromethane Fluorocarbon 13	No Standard	
Difluoroethylene Fluorocarbon 1131	No Standard	
Fluoroethylene Fluorocarbon 1141	No Standard	
Hexafluoropropylene Fluorocarbon 1216	No Standard	
Octafluorocyclobutane Fluorocarbon C-318	No Standard	
Tetrafluoroethylene Fluorocarbon 1114	No Standard	

ROUTE OF ENTRY

Inhalation of vapor or gas.

HARMFUL EFFECTS

Local—

These compounds may produce mild irritation to the upper respiratory tract. Dermatitis occurs only rarely. Decomposition products may also be the cause of these effects.

Systemic—

Mild central nervous system depression may occur in cases of exposure to very high concentrations of fluorocarbons. Symptoms from acute exposure may manifest themselves in occasional tremor and incoordination. It has been reported that dizziness has resulted from an exposure to 5% dichlorodifluoromethane and unconsciousness from exposure to 15%. Cardiac arrhythmias, with sudden death, have occurred from breathing some of these chemicals. Typically, fluorocarbons have very low levels of toxicity, and their predominant hazard is from simple asphyxia.

Fluoroalkenes are more toxic than fluoroalkanes. Liver and kidney damage has been reported to occur from chronic exposure to fluoroalkenes, whereas no chronic effects have been reported from fluoroalkanes.

MEDICAL SURVEILLANCE

Though these compounds are of a low level of toxicity, they should not be considered inert. There are no specific diagnostic tests for the toxic effects occurring at very high concentrations. Preplacement and periodic examinations should consider possible cardiac effects from acute exposure.

SPECIAL TESTS

None commonly used. Compounds are usually excreted rapidly in expired air.

PERSONAL PROTECTIVE METHODS

Simple ventilation can avert acute poisoning. Masks are rarely needed.

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METHYL AND ETHYL BROMIDE

DESCRIPTION

CH_3Br, methyl bromide, is a colorless, nearly odorless gas. It is synthesized from sodium bromide, methyl alcohol, and sulfuric acid.

C_2H_5Br, ethyl bromide, is a colorless, volatile, flammable liquid possessing an etherlike odor, burning taste. It becomes yellowish on exposure to air. It is produced from potassium bromide, ethyl alcohol, and sulfuric acid.

SYNONYMS

Methyl bromide: bromomethane.

Ethyl bromide: bromoethane, monobromoethane, monobromomethane.

POTENTIAL OCCUPATIONAL EXPOSURES

Methyl Bromide: The primary use of methyl bromide is as an insect fumigant for soil, grain, warehouses, mills, ships, etc. It is also used as a chemical intermediate and a methylating agent, a refrigerant, a herbicide, a fire extinguishing agent, a low-boiling solvent in aniline dye manufacture; for degreasing wool; for extracting oils from nuts, seeds, and flowers; and in ionization chambers.

Ethyl bromide: This chemical is used as an ethylating agent in organic synthesis and gasoline, as a refrigerant, and as an extraction solvent. It has limited use as a local anesthetic.

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

Anesthetists	Grain fumigators
Color makers	Refrigerant makers
Dye makers	Soil fumigators
Fire extinguisher workers	Solvent workers
Fruit fumigators	Wool degreasers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for methyl bromide is 20 ppm (80 mg/m^3) as a ceiling value. The Federal standard for ethyl bromide is 200 ppm (890 mg/m^3) as a TWA. The ACGIH recommended TLV for methyl bromide is 15 ppm (60 mg/m^3) as a TWA.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

Methyl bromide is irritating to the eyes, skin, and mucous membranes of the upper respiratory tract. In cases of moderate skin exposure, there may be an itching dermatitis, and in severe cases, vesicles and second-degree burns. Methyl bromide may be absorbed by leather, resulting in prolonged skin contact. Repeated or prolonged skin contact with ethyl bromide may cause irritation.

Systemic—

High concentrations of either methyl or ethyl bromide may cause lung irritation which may result in pulmonary edema and death. Acute exposure to methyl bromide may produce delayed effects. Onset of symptoms is usually delayed from 30 minutes to 6 hours; the first to appear are malaise, visual disturbances, headaches, nausea, vomiting, somnolence, vertigo, and tremor in the hands. The tremor may become more severe and widespread, developing into epileptiform type convul-

sions followed by coma and death due to pulmonary or circulatory failure or both. A period of delirium and mania may precede convulsions, but convulsions have been reported without any other warning symptoms. Kidney damage may occur; permanent brain damage may result.

In chronic poisoning, the effects of methyl bromide are usually limited to the central nervous system: Lethargy; muscular pains; visual, speech, and sensory disturbances; and mental confusion being the most prominent complaints. Ethyl bromide exposure has not been associated with chronic effects other than skin irritation.

MEDICAL SURVEILLANCE

Methyl bromide: Evaluate the central nervous system, respiratory tract, and skin in preplacement and periodic examinations.

Ethyl bromide: No specific considerations needed.

SPECIAL TESTS

None in common use. Blood bromide levels have been measured in cases of methyl bromide intoxication, but their value in routine monitoring of exposure has not been established.

PERSONAL PROTECTIVE METHODS

Rubber, not leather, protective clothing should be utilized. When masks are worn, they should provide full face protection.

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METHYL CHLORIDE

DESCRIPTION

CH_3Cl, methyl chloride, is a colorless gas possessing a faint, sweet odor.

SYNONYMS

Monochloromethane, chloromethane.

POTENTIAL OCCUPATIONAL EXPOSURES

Methyl chloride is used as a methylating and chlorinating agent in organic chemistry. In petroleum refineries it is used as an extractant for greases, oils, and resins. Methyl chloride is also used as a solvent in the synthetic rubber industry, as a refrigerant, and as a propellant in polystyrene foam production. In the past it has been used as a local anesthetic (freezing).

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

Aerosol packagers	Methyl cellulose makers
Drug makers	Polystyrene foam makers
Flavor extractors	Refrigeration workers
Low temperature solvent workers	Rubber makers
Methylation workers	Vapor pressure thermometer makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 100 ppm (210 mg/m^3) as an 8-hour TWA, with an acceptable ceiling concentration of 200 ppm; acceptable maximum peaks above the ceiling of 300 ppm are allowed for 5 minutes duration in a 3-hour period.

ROUTE OF ENTRY

Percutaneous absorption.

HARMFUL EFFECTS

Local—

Skin contact with the discharge from the pressurized gas may cause frostbite. The liquid may damage eyes.

Systemic—

Signs and symptoms of chronic exposure include staggering gait, difficulty in speech, nausea, headache, dizziness, and blurred vision. Vomiting has also occurred in some cases. These effects may be observed following a latency period of several hours.

Acute exposure is much like chronic except that the latency period is shorter and the effects more severe. Coma or convulsive seizures may occur. Acute poisoning predominantly depresses the central nervous system, but renal and hepatic damage may also occur. Recently noted in these cases is the depression of bone marrow activity. Recovery from severe exposure may take as long as 2 weeks.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should give careful consideration to a previous history of the central nervous system, and to renal or hepatic disorders.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Masks should be used in areas of high vapor concentrations.

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METHYLENE CHLORIDE

DESCRIPTION

$CHCl_2$, methylene chloride, is a nonflammable, colorless liquid with a pleasant aromatic odor noticeable at 300 ppm (this, however, should not be relied upon as an adequate warning of unsafe concentrations).

SYNONYMS

Dichloromethane, methylene dichloride, methylene bichloride.

POTENTIAL OCCUPATIONAL EXPOSURES

Methylene chloride is used mainly as a low temperature extractant of substances which are adversely affected by high temperature. It can be used as a solvent for oil, fats, waxes, bitumen, cellulose acetate, and esters. It is also used as a paint remover and as a degreaser.

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

- Aerosol packagers
- Anesthetic makers
- Bitumen makers
- Degreasers
- Fat extractors
- Flavoring makers
- Leather finish workers
- Oil processors
- Paint remover makers
- Resin makers
- Solvent workers
- Stain removers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 500 ppm (1,740 mg/m^3) as an 8-hour TWA with an acceptable ceiling concentration of 1,000 ppm; acceptable maximum peaks above the ceiling of 2,000 ppm are allowed for 5 minutes duration in a 2-hour period.

Note: The 1976 ACGIH TLV is 200 ppm (720 mg/m^3.)

NIOSH has recommended a time-weighted average limit of 75 ppm in the absence of occupational exposure to carbon monoxide greater than 9 ppm (TWA). For carbon monoxide exposures greater than 9 ppm, a formula is recommended. NIOSH also recommended a ceiling limit of 500 ppm as determined by a 15-minute sampling period.

ROUTES OF ENTRY

Inhalation of vapors and percutaneous absorption of liqiud.

HARMFUL EFFECTS

Local—

Repeated contact with methylene chloride may cause a dry, scaly, and fissured dermatitis. The liquid and vapor are irritating to the eyes and upper respiratory tract at higher concentrations. If the liquid is held in contact with the skin, it may cause skin burns.

Systemic—

Methylene chloride is a mild narcotic. Effects from intoxication include headache, giddiness, stupor, irritability, numbness, and tingling

in the limbs. Irritation to the eyes and upper respiratory passages occurs at higher dosages. In severe cases, observers have noted toxic encephalopathy with hallucinations, pulmonary edema, coma, and death. Cardiac arrythmias have been produced in animals but have not been common in human experiences. Exposure to this agent may cause elevated carboxyhemoglobin levels which may be significant in smokers, or workers with anemia or heart disease, and those exposed to CO.

MEDICAL SURVEILLANCE

Changes in liver, respiratory tract, and central nervous system should be considered during preplacement or periodic medical examinations. Smoking history should be known; anemias or cardiovascular disease may increase the hazard.

SPECIAL TESTS

The metabolism and excretion of methylene chloride has been thoroughly studied. Blood and expired air analyses are useful indicators of exposure. Carboxyhemoglobin levels may be useful indicators of excessive exposure, especially in nonsmokers.

PERSONAL PROTECTIVE METHODS

Protective clothing, gloves to prevent skin contact, and, in areas of high concentration, fullface masks.

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PROPYLENE DICHLORIDE

DESCRIPTION

$CH_3CHClCH_2Cl$, propylene dichloride, is a colorless liquid with a characteristic unpleasant odor.

SYNONYMS

1,2-Dichloropropane, propylene chloride.

POTENTIAL OCCUPATIONAL EXPOSURES

Propylene dichloride is widely used for degreasing and drycleaning; it is also used as a soil fumigant and in the manufacture of cellulose

plastics, rubber, waxes, scouring compounds and other manufacture of organic synthetics.

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

Cellulose plastic makers
Drycleaners
Fat processors
Fumigant workers
Gum processors
Metal degreasers
Oil processors
Organic chemical synthesizers
Rubber makers
Scouring compound makers
Solvent workers
Stain removers
Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 75 ppm (350 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Propylene dichloride may cause dermatitis by defatting the skin. More severe irritation may occur if it is confined against the skin by clothing. Undiluted, it is moderately irritating to the eyes, but does not cause permanent injury.

Systemic—

In animal experiments, acute exposure to propylene dichloride produced central nervous system narcosis, fatty degeneration of the liver and kidneys.

MEDICAL SURVEILLANCE

Evaluate the skin and liver and renal function on a periodic basis, as well as cardiac and respiratory status and general health.

SPECIAL TESTS

None in common use. Propylene dichloride can be determined in expired air.

PERSONAL PROTECTIVE METHODS

Barrier creams or gloves and protective clothing. Masks in areas of vapor concentration.

BIBLIOGRAPHY

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TETRACHLOROETHANE

DESCRIPTION

$CHCl_2CHCl_2$, tetrachloroethane, is a heavy, volatile liquid which is nonflammable and has a sweetish, chloroform-like odor. Oxidative

decomposition of tetrachloroethane by ultraviolet radiation or by contact with hot metal results in the formation of small quantities of phosgene, hydrochloric acid, carbon monoxide, carbon dioxide, or dichloroacetyl chloride.

SYNONYMS

1,1,2,2-Tetrachloroethane, sym-tetrachloroethane, acetylene tetrachloride, ethanetetrachloride.

POTENTIAL OCCUPATIONAL EXPOSURES

Tetrachloroethane is used as a drycleaning agent, as a fumigant, in cement, and in lacquers. It is used in the manufacture of tetrachloroethylene, artificial silk, artificial leather, and artificial pearls. Recently, its use as a solvent has declined due to replacement by less toxic compounds. It is also used in the estimation of water content in tobacco and many drugs, and as a solvent for chromium chloride impregnation of furs.

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

- Biologists
- Drycleaners
- Fat processors
- Fumigators
- Gasket makers
- Herbicide workers
- Insecticide workers
- Lacquer workers
- Metal cleaners
- Mineralogists
- Oil processors
- Paint makers
- Phosphorus processors
- Resin makers
- Soil treaters
- Solvent workers
- Varnish workers
- Waxers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (35 mg/m^3). NIOSH has recommended a time-weighted average limit of 1 ppm.

ROUTES OF ENTRY

Inhalation of vapor and absorption of liquid through the skin. There is some evidence that tetrachloroethane absorbed through the skin affects the central nervous system only.

HARMFUL EFFECTS

Local—

Repeated or prolonged contact with this chemical can produce a scaly and fissured dermatitis.

Systemic—

Early effects brought on by tetrachloroethane narcotic action include tremors, headache, a prickling sensation and numbness of limbs, loss of kneejerk, and excessive sweating. Paralysis of the interossei muscles of the hands and feet and disappearance of ocular and pharyngeal reflexes have also occurred due to peripheral neuritis which may de-

velop later. Blood changes include increases in mononuclear leukocytes, progressive anemia, and a slight thrombocytosis.

Clinical symptoms following these changes are fatigue, headache, constipation, insomnia, irritability, anorexia, and nausea. Later on, liver dysfunction may result in complaints of general malaise, drowsiness, loss of appetite, nausea, an unpleasant taste in the mouth, and abdominal discomfort. This may be followed by jaundice, mental confusion, stupor or delirium, hematemesis, convulsions, and purpuric rashes.

Pulmonary edema ascribed to capillary injury has been noted in severe cases, along with renal damage, though it is not known to what extent this contributes to the total toxic picture. Nephritis may develop, and the urine may contain albumin and casts. Fatty degeneration of the myocardium has been reported only in animal experiments.

MEDICAL SURVEILLANCE

Preplacement and periodic examination should be comprehensive because of the possible involvement of many systems. Special attention should be given to liver, kidney, and bone marrow function, as well as to the central and peripheral nervous systems. Alcoholism may be a predisposing factor.

SPECIAL TESTS

None commonly used. Blood or breath analyses may be useful.

PERSONAL PROTECTIVE METHODS

Gloves and protective clothing should be worn, and appropriate respirators or masks should be used in areas of elevated vapor concentration.

BIBLIOGRAPHY

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TETRACHLOROETHYLENE

DESCRIPTION

$Cl_2C=CCl_2$, tetrachloroethylene, is a clear, colorless, nonflammable liquid with a characteristic odor. The odor is noticeable at 50 ppm, though after a short period it may become inconspicuous, thereby becoming an unreliable warning signal.

SYNONYMS

Perchloroethylene, carbon dichloride, ethylene tetrachloride.

POTENTIAL OCCUPATIONAL EXPOSURES

Tetrachloroethylene is a widely used solvent with particular use as a drycleaning agent, a degreaser, a chemical intermediate, a fumigant, and medically as an anthelmintic.

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

Cellulose ester processors	Metal degreasers
Degreasers	Printers
Dope processors	Rubber workers
Drug makers (anthelmintics)	Soap workers
Drycleaners	Solvent workers
Electroplaters	Tar processors
Ether processors	Vacuum tube makers
Fumigant workers	Wax makers
Gum processors	Wool scourers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 100 ppm (670 mg/m^3), as an 8-hour TWA with an acceptable ceiling concentration of 200 ppm; acceptable maximum peaks above the ceiling of 300 ppm are allowed for 5 minutes duration in a 3-hour period. NIOSH has recommended a time-weighted average limit of 50 ppm and a ceiling limit of 100 ppm determined by 15-minute samples, twice daily.

ROUTES OF ENTRY

Inhalation of vapor and percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Repeated contact may cause a dry, scaly, and fissured dermatitis. High concentrations may produce eye and nose irritation.

Systemic—

Acute exposure to tetrachloroethylene may cause central nervous system depression, hepatic injury, and anesthetic death. Cardiac arrhythmias and renal injury have been produced in animal experiments. Signs and symptoms of overexposure include malaise, dizziness, headache, increased perspiration, fatigue, staggering gait, and slowing of mental ability. These usually subside quickly upon removal into the open air.

MEDICAL SURVEILLANCE

Evaluate skin, and liver and kidney function, as well as central nervous system. Alcoholism may be a predisposing factor.

SPECIAL TESTS

Breath analyses may be helpful in evaluating exposures.

Workers with preemployment histories of liver, kidney, or nervous disorders should be advised as to possible increased risk.

PERSONAL PROTECTIVE METHODS

Skin protection in the form of barrier creams, gloves, and protective clothing should be used. In areas of vapor concentration, fullface masks should be worn.

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1,1,1-TRICHLOROETHANE

DESCRIPTION

CH_3CCl_8, 1,1,1-trichloroethane, is a colorless, nonflammable liquid with an odor similar to chloroform. Upon contact with hot metal or exposure to ultraviolet radiation, it will decompose to form the irritant gases hydrochloric acid, phosgene, and dichloroacetylene.

SYNONYMS

Methyl chloroform.

POTENTIAL OCCUPATIONAL EXPOSURES

In recent years, 1,1,1-trichloroethane has found wide use as a substitute for carbon tetrachloride. In liquid form it is used as a degreaser and for cold cleaning, dip-cleaning, and bucket cleaning of metals. Other industrial applications of 1,1,1-trichloroethane's solvent properties include its use as a drycleaning agent, a vapor degreasing agent, and a propellant.

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

- Degreasers
- Drycleaners
- Machinery cleaners
- Metal degreasers
- Propellant makers
- Stain removers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 350 ppm (1,900 mg/m^3). NIOSH has recommended a 350 ppm ceiling as determined by a 15-minute sampling period.

ROUTES OF ENTRY

Inhalation of vapor and moderate skin absorption.

HARMFUL EFFECTS

Local—

Liquid and vapor are irritating to eyes on contact. This effect is usually noted first in acute exposure cases. Mild conjunctivitis may develop but recovery is usually rapid. Repeated skin contact may produce a dry, scaly, and fissured dermatitis, due to the solvent's defatting properties.

Systemic—

1,1,1-trichloroethane acts as a narcotic and depresses the central nervous system. Acute exposure symptoms include dizziness, incoordination, drowsiness, increased reaction time, unconsciousness, and death.

MEDICAL SURVEILLANCE

Consider the skin, liver function, cardiac status, especially arrythmias, in preplacement or periodic examinations.

SPECIAL TESTS

Expired air analyses may be useful in monitoring exposure.

PERSONAL PROTECTIVE METHODS

1,1,1-Trichloroethane attacks natural rubber; therefore, protective clothing of leather, polyvinyl alcohol, or neoprene is recommended. In areas of high concentrations, fullface masks should be worn.

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1,1,2-TRICHLOROETHANE

DESCRIPTION

$CH_2ClCHCl_2$, 1,1,2-trichloroethane, is a colorless, nonflammable liquid. It is an isomer of 1,1,1-trichloroethane but should not be confused with it toxicologically. 1,1,2-Trichloroethane is comparable to carbon tetrachloride and tetrachloroethane in toxicity.

SYNONYMS

Vinyl trichloride.

POTENTIAL OCCUPATIONAL EXPOSURES

1,1,2-Trichloroethane is used as a chemical intermediate and as a solvent, but is not as widely used as its isomer 1,1,1-trichloroethane.

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

Organic chemical synthesizers
Solvent makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 10 ppm (45 mg/m^3) as a TWA.

ROUTES OF ENTRY

Inhalation of vapor and absorption through the skin.

HARMFUL EFFECTS

Local—

Irritation to eyes and nose, and infection of the conjunctiva have been shown in animals.

Systemic—

Little is known of the toxicity of 1,1,2-trichloroethane since no human toxic effects have been reported. Animal experiments show 1,1,2-trichloroethane to be a potent central nervous system depressant. The injection of anesthetic doses in animals was associated with both liver and renal neurosis.

MEDICAL SURVEILLANCE

Consider the skin, central nervous system, and liver and kidney function. Alcoholism may be a synergistic factor.

SPECIAL TESTS

None commonly used, but expired air analyses may be useful in monitoring exposure.

PERSONAL PROTECTIVE METHODS

Protective clothing and gloves should be worn. Respirators should be used in areas of high vapor concentration.

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TRICHLOROETHYLENE

DESCRIPTION

$ClCH=CCl_2$, trichloroethylene, a colorless, nonflammable, noncorrosive liquid has the "sweet" odor characteristic of some chlorinated hydrocarbons. Decomposition of trichloroethylene, due to contact with hot metal or ultraviolet radiation, forms products including chlorine gas, hydrogen chloride, and phosgene. Dichloroacetylene may be formed from the reaction of alkali with trichloroethylene.

SYNONYMS

Ethylene trichloride, ethinyl trichloride, trichloroethene.

POTENTIAL OCCUPATIONAL EXPOSURES

Trichloroethylene is primarily used as a solvent in vapor degreasing. It is also used for extracting caffeine from coffee, as a drycleaning agent,

and as a chemical intermediate in the production of pesticides, waxes, gums, resins, tars, paints, varnishes, and specific chemicals such as chloroacetic acid.

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

Anesthetic makers	Metal cleaners
Caffeine processors	Oil processors
Cleaners	Perfume makers
Disinfectant makers	Printers
Degreasers	Resin workers
Drug makers	Rubber cementers
Drycleaners	Shoe makers
Dye makers	Soap makers
Electronic equipment cleaners	Solvent workers
Fat processors	Textile cleaners
Glass cleaners	Tobacco denicotinizers
Mechanics	Varnish workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 100 ppm (535 mg/m^3) as an 8-hour TWA with an acceptable ceiling concentration of 200 ppm; acceptable maximum peaks above the ceiling of 300 ppm are allowed for 5 minutes duration in a 2-hour period. The NIOSH Criteria for a Recommended Standard recommends limits of 100 ppm as a TWA and a peak of 150 ppm determined by a sampling time of 10 minutes.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

Exposure to trichloroethylene vapor may cause irritation of the eyes, nose, and throat. The liquid, if splashed in the eyes, may cause burning irritation and damage. Repeated or prolonged skin contact with the liquid may cause dermatitis.

Systemic—

Acute exposure to trichloroethylene depresses the central nervous system exhibiting such symptoms as headache, dizziness, vertigo, tremors, nausea and vomiting, irregular heart beat, sleepiness, fatigue, blurred vision, and intoxication similar to that of alcohol. Unconsciousness and death have been reported. Alcohol may make the symptoms of trichloroethylene overexposure worse. If alcohol has been consumed, the overexposed worker may become flushed. Trichloroethylene addiction and peripheral neuropathy have been reported. Recent reports indicate that exposure to trichloroethylene may induce liver tumors in mice.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include the skin,

respiratory, cardiac, central, and peripheral nervous systems, as well as liver and kidney function. Alcohol intake should be evaluated.

SPECIAL TESTS

Expired air analysis and urinary metabolites have been used to monitor exposure.

PERSONAL PROTECTIVE METHODS

Gloves and protective clothing should be worn, and fullface mask should be used in areas of excessive vapor concentrations.

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Pardys, S., and M. Brotman. 1974. Trichloroethylene and alcohol: a straight flush. J. Am. Med. Assoc. 229:521.

VINYL CHLORIDE

DESCRIPTION

$CH_2=CHCl$, vinyl chloride, is a flammable gas at room temperature and is usually encountered as a cooled liquid. The colorless liquid forms a vapor which has a pleasant ethereal odor.

SYNONYMS

Chloroethylene, chloroethene, monochloroethylene.

POTENTIAL OCCUPATIONAL EXPOSURES

Vinyl chloride is used as a vinyl monomer in the manufacture of polyvinyl chloride and other resins. It is also used as a chemical intermediate and as a solvent.

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

Polyvinyl resin makers
Organic chemical synthesizers
Rubber makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for exposure to vinyl chloride sets a limit of 1 ppm over an 8-hour period, and a ceiling of 5 ppm averaged over any period not exceeding 15 minutes.

ROUTES OF ENTRY

Vinyl chloride gas is absorbed by inhalation. Skin absorption has been suggested but experimental evidence is presently lacking.

HARMFUL EFFECTS

Local—

Vinyl chloride is a skin irritant, and contact with the liquid may cause frostbite upon evaporation. The eyes may be immediately and severely irritated.

Systemic—

Vinyl chloride depresses the central nervous system causing symptoms which resemble mild alcohol intoxication. Lightheadedness, some nausea, and dulling of visual and auditory responses may develop in acute exposures. Death from severe vinyl chloride exposure has been reported.

Chronic exposure of workers involved in reactor vessel entry and hand cleaning may result in the triad of acro-osteolysis, Raynaud's phenomenon, and sclerodermatus skin changes. Chronic exposure may also cause hepatic damage.

Vinyl chloride is regarded as a human carcinogen, and a causal agent of angiosarcoma of the liver. Excess cancer of the lung and the lymphatic and nervous systems has also been reported. Experimental evidence of tumor induction in a variety of organs, including liver, lung, brain, and kidney, as well as nonmalignant alterations, such as fibrosis and connective tissue deterioration, indicate the multisystem oncogenic and toxicologic effects of vinyl chloride.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should emphasize liver function and palpation. Liver scans and grey-scale ultrasonography have been useful in detecting liver tumors. Medical histories should include alcoholic intake; past hepatitis; exposure to hepatotoxic agents, drugs and chemicals; past blood transfusions; past hospitalizations. Radiographic examinations of the hands may be helpful if acroosteolysis is suspected. Long term followup of exposed persons is essential as in the case of other carcinogens.

SPECIAL TESTS

None in common use. Metabolism is being studied.

PERSONAL PROTECTIVE METHODS

Where vinyl chloride levels cannot meet the standard, workers should be required to wear respiratory protection, either air supplied respirator or, if the level does not exceed 25 ppm, a chemical cartridge or cannister type gas mask. In hazard areas, proper protective clothing to prevent skin contact with the vinyl chloride or polyvinyl chloride residue should be worn.

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ALIPHATIC AMINES

The aliphatic amines are derivatives of ammonia (NH_3) in which one or more hydrogen atoms are replaced by alkyl or alkanol radicals. They tend to have a characteristic fishlike ammonia odor in the free base form.

These compounds are generally prepared by alkylation of ammonia or hydrogenation of the appropriate nitrite. They are widely used in industry, particularly as chemical intermediates.

The amines are basic compounds and may form strongly alkaline solutions which can be highly irritating and cause damage on contact with eyes and skin. Skin absorption may be significant as many are capable of cutaneous sensitization. Some members of this series may have physiologic or pharmacologic effects—e.g., histamine liberation and vasodilation, but, in general, local effects predominate in industrial exposures.

Because of the strong irritant properties of aliphatic amines, eyes, skin, and respiratory tract should be protected from exposure to them.

N-BUTYLAMINE

DESCRIPTION

CH_3-CH_2-CH_2-CH_2-NH_2, n-butylamine, is a flammable colorless liquid with an ammoniacal odor.

SYNONYMS

1-aminobutane.

POTENTIAL OCCUPATIONAL EXPOSURES

n-Butylamine is used in pharmaceuticals, dyestuffs, rubber, chemicals, emulsifying agents, photography, desizing agents for textiles, pesticides, and synthetic agents.

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

- Butylaminophenol makers
- Chemists
- Drug makers
- Dye makers
- Emulsifier makers
- Insecticide makers
- Petroleum dewaxers
- Rubber makers
- Tanning chemical makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (15 mg/m^3) as a ceiling value.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

Butylamine vapor is irritating to the nose, throat, and eyes. Contact with the liquid may produce severe eye damage and skin burns.

Systemic—

Inhalation of concentrations at or above the threshold limit may produce mild headaches and flushing of the skin and face.

Butylamine vapor has produced pulmonary edema in animal experiments.

MEDICAL SURVEILLANCE

Evaluate risks of eye or skin injury and respiratory irritation in periodic or placement examinations.

SPECIAL TESTS

None have been developed.

PERSONAL PROTECTIVE METHODS

Protective clothing and goggles should be worn where possibility of skin or eye contact with the liquid exists. In areas of elevated vapor concentration, fullface masks with organic vapor canister or supplied air respirators and protective clothing should be worn. The odor and irritation of the mucous membranes cannot be relied upon for exposure control.

ETHANOLAMINES

DESCRIPTION

Monoethanolamine: $H_2NCH_2CH_2OH$, Diethanolamine: $HN(CH_2CH_2OH)$, Triethanolamine: $N(CH_2CH_2,(OH)_3$. All three compounds are water soluble liquids. Monoethanolamine has a low vapor pressure while the vapor pressure of the other ethanolamines is very low. Monoethanolamine and diethanolamine have ammonia odors while triethanolamine has only a faint non-ammonia odor. The acid salts have less odor and are of low volatility. Ethanolamines can be detected by odor as low as 2-3 ppm.

SYNONYMS

Monoethanolamine: Ethanolamine, 2-aminoethanol, colamine
Diethanolamine: 2,2'-Iminodiethanol
Triethanolamine: 2,2',2"-Nitrilotriethanol

POTENTIAL OCCUPATIONAL EXPOSURES

Monoethanolamine is widely used in industry to remove carbon dioxide and hydrogen from natural gas, to remove hydrogen sulfide and carbonyl sulfide, as an alkaline conditioning agent, and as an intermediate for soaps, detergents, dyes, and textile agents.

Diethanolamine is an absorbent for gases, a solubilizer for 2,4-dichlorophenoxyacetic acid (2,4-D), and a softener and emulsifier intermediate for detergents. It also finds use in the dye and textile industry.

Triethanolamine is used as a plasticizer, neutralizer for alkaline dispersions, lubricant additive, corrosion inhibitor, and in the manufacture of soaps, detergents, shampoos, shaving preparations, face and hand creams, cements, cutting oils, insecticides, surface active agents, waxes, polishes, and herbicides.

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

- Cement makers
- Detergent makers
- Dye makers
- Emulsifier makers
- Herbicide makers
- Insecticide makers
- Natural gas workers
- Plastic workers
- Polish makers
- Soap makers
- Surfactant makers
- Textile workers
- 2,4-D makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for monoethanolamine is 3 ppm (6 mg/m^3). There are no standards for the other compounds.

ROUTES OF ENTRY

Inhalation of vapor and percutaneous absorption.

HARMFUL EFFECTS

Local—

Ethanolamine has had wide use in industry, yet reports of injury

in man are lacking. Ethanolamine in animal experiments was highly irritating to the skin, eyes, and respiratory tract. Diethanolamine and triethanolamine produced much less irritation. In human experiments, ethanolamine produced only redness of the skin.

Systemic—

No specific published data on human exposure are available. Animal experiments indicate that it is a central nervous system depressant. Acute high level exposures produced pulmonary damage and non-specific hepatic and renal lesions in animals.

MEDICAL SURVEILLANCE

Evaluate possible irritant effects on skin and eyes.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Protective clothing should be worn, and in areas of elevated vapor concentrations, fullface masks should be supplied.

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ETHYLENEDIAMINE

DESCRIPTION

$H_2N\text{-}CH_2\text{-}CH_2\text{-}NH_2$, ethylenediamine, is a strongly alkaline, colorless, clear, thick, liquid with an ammonia odor.

SYNONYMS

Ethanediamine, 1,2-diaminoethane.

POTENTIAL OCCUPATIONAL EXPOSURES

Ethylenediamine is used as a solvent, an emulsifier for casein and shellac solutions, a stabilizer in rubber latex, a chemical intermediate in the manufacture of dyes, corrosion inhibitors, synthetic waxes, fungicides, resins, insecticides, asphalt wetting agents, and pharmaceuticals, and also in controlling acidity or alkalinity.

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

- Albumin processors
- Casein processors
- Drug makers
- Dye makers
- Emulsion workers
- Ethylenediamine tetraacetic acid (EDTA) makers
- Fungicide makers
- Insecticide makers
- Oil neutralizers
- Resin makers
- Rubber makers
- Shellac processors
- Surfactant makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 10 ppm (25 mg/m³).

ROUTES OF ENTRY

Inhalation of vapor and percutaneous absorption.

HARMFUL EFFECTS

Local—

Ethylenediamine vapor may cause irritation of the nose and tingling of the face. Cutaneous sensitivity has been reported.

In animal experiments, the liquid has produced severe irritation of the eyes and corneal damage. It has also produced severe irritation and necrosis.

Systemic—

In animal experiments, high concentrations of ethylenediamine vapor have produced damage to liver, lungs, and kidneys.

MEDICAL SURVEILLANCE

Consider possible irritant effects on skin, eyes and respiratory system. History of allergic redness of skin or asthmatic symptoms may be important in placement and periodic examinations.

SPECIAL TESTS

None have been developed.

PERSONAL PROTECTIVE METHODS

Protective clothing, gloves, and goggles should be worn to protect the skin and eyes. Fullface masks with organic vapor canisters must be used in areas of high vapor concentrations. Recent reports indicate that a non-occupational allergic contact dermatitis may develop after use of pharmaceuticals containing ethylenediamine.

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CYANIDES AND NITRILES

This class of compounds contains the —CN group and includes, in addition to cyanides and nitriles, related chemicals such as cyanogens, isocyanates, and cyanamides.

Hydrogen cyanide and its soluble salts are rapidly acting poisons. The cyanide ion when released in the body is capable of inhibiting many enzymes, the most sensitive being cytochrome oxidase. Deaths from acute exposure are due to chemical asphyxia at the cellular level.

In high concentrations, nitriles (R-CN) can cause similar symptoms, but the onset is slower. It is believed that the CN ion is released from

the nitriles, the rate of release determining the toxicity. In addition, nitriles may be primary irritants.

Other —CN derivatives differ in their toxic properties and may not have the same mechanism of action.

There seems to be a detoxification mechanism for cyanide ion; the enzyme rhodanese (transulfurase) is the catalyst for the reaction

$$S_2O_3^{=} + CN^{-} \rightarrow SO_3^{=} + SCN^{-}$$

in which cyanide ion is converted to thiocyanate ion. Theoretically, this endogenously produced thiocyanate may have lesser toxic effects, similar to those seen in thiocyanate therapy, at high doses. It is unlikely that this is of practical significance, however, because of the minute quantities of CN^- and SCN^- ions involved in poisoning cases.

There are several tests of biological tissues available which are suitable for diagnostic purposes of acute intoxication but not for routine medical surveillance. Thiocyanate is excreted in the urine and may be present in the serum; smokers have a higher thiocyanate level than nonsmokers. Cyanide ion may also be found in blood and tissues.

ACETONITRILE

DESCRIPTION

CH_3-CN, acetonitrile, is a colorless liquid with an ether-like odor.

SYNONYMS

Methyl cyanide, ethanenitrile, cyanomethane.

POTENTIAL OCCUPATIONAL EXPOSURES

Acetonitrile is used as an extractant for animal and vegetable oils, as a solvent, particularly in the pharmaceutical industry, and as a chemical intermediate.

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

Animal oil processors
Organic chemical synthesizers
Vegetable oil processors

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 40 ppm (70 mg/m^3).

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

At high concentrations, nose and throat irritation have been reported. Splashes of the liquid in the eyes may cause irritation. Acetonitrile may cause slight flushing of the face and a feeling of chest tightness.

Systemic—

Acetonitrile has a relatively low acute toxicity, but there have been reports of severe and fatal poisonings in man after inhalation of high concentrations. Signs and symptoms may include nausea, vomiting, respiratory depression, weakness, chest or abdominal pain, hematemesis, convulsions, shock, unconsciousness, and death. In most cases there is a latent period of several hours between exposure and onset of symptoms. It has been thought that acetonitrile itself has relatively little toxic effect and that the delayed response is due to the slow release of cyanide. No chronic disease has been reported.

MEDICAL SURVEILLANCE

Consider the skin, respiratory tract, heart, central nervous system, renal and liver function in placement and periodic examinations. A history of fainting spells or convulsive disorders might present an added risk to persons working with toxic nitriles.

SPECIAL TESTS

None commonly used. Blood CN can be determined but may be of little help in evaluating low level exposures.

PERSONAL PROTECTIVE METHODS

Protective clothing should be worn, and in areas of high concentration, air supplied respirators and complete skin protection are necessary. Workers in these areas must be educated to the nature of acetonitrile hazard. They should also be trained in artificial respiration and in the use of amyl nitrite antidote in emergency situations.

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ACRYLONITRILE

DESCRIPTION

$CH_2=CH-CN$, acrylonitrile, is a colorless liquid with a faint acrid odor. It is both flammable and explosive.

SYNONYMS

Vinyl cyanide, cyanoethylene, propene nitrile.

POTENTIAL OCCUPATIONAL EXPOSURES

Acrylonitrile is used in the manufacture of synthetic fibers, acrylostyrene plastics, acrylonitrile-butadiene styrene plastics, nitrile rubbers, chemicals, and adhesives. It is also used as a pesticide.

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

- Acrylic resin makers
- Organic chemical synthesizers
- Pesticide workers
- Rubber makers
- Synthetic fiber makers
- Textile finish makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 20 ppm (45 mg/m^3).

ROUTES OF ENTRY

Inhalation and percutaneous absorption. It may be absorbed from contaminated rubber or leather.

HARMFUL EFFECTS

Local—

Acrylonitrile may cause irritation of the eyes. Repeated and prolonged exposure may produce skin irritation. When acrylonitrile is held in contact with the skin (e.g., after being absorbed into shoe leather or clothing), it may produce blistering after several hours of no apparent effect. Unless the contaminated clothing is removed promptly and the area washed off, blistering will occur.

Systemic—

Acrylonitrile exposure may produce nausea, vomiting, headache, sneezing, weakness, and light-headedness. Exposure to high concentrations may produce profound weakness, asphyxia, and death.

MEDICAL SURVEILLANCE

Consider the skin, respiratory tract, heart, central nervous system, renal and liver function in placement and periodic examinations. A history of fainting spells or convulsive disorders might present an added risk to persons working with toxic nitriles.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

Leather should not be used in protective clothing since it is readily penetrated by acrylonitrile. Rubber clothing should be frequently washed and inspected because it will soften and swell. Acrylonitrile should be handled with all of the same precautions as taken for hydrogen cyanide, and workers' education should be identical. Liquid splashed on skin should be immediately washed off. Eyes should be protected from splash (goggles), and, in areas of vapor concentration, special cyanide masks or air supplied masks should be provided. Workers should be trained in artificial respiration and in the use of amyl nitrite antidote in emergency situations.

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CALCIUM CYANAMIDE

DESCRIPTION

NCN=Ca, calcium cyanamide, is a blackish-grey, shiny powder.

SYNONYMS

Nitrolim, calcium carbimide, cyanamide.

POTENTIAL OCCUPATIONAL EXPOSURES

Calcium cyanamide is used in agriculture as a fertilizer, herbicide, defoliant for cotton plants, and pesticide. It is also used in the manufacture of dicyanidamide and calcium cyanide, as a desulfurizer in the iron and steel industry, and in steel hardening.

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

- Ammonia makers
- Cotton defoliant workers
- Cyanamide makers
- Fertilizer workers
- Herbicide workers
- Nitrogen compound makers
- Organic chemical synthesizers
- Steel workers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for calcium cyanamide. (Note: The 1976 ACGIH TLV was 0.5 mg/m^3.)

ROUTE OF ENTRY

Inhalation of dust.

HARMFUL EFFECTS

Local—

Calcium cyanamide is a primary irritant of the mucous membranes of the respiratory tract, eyes, and skin. Inhalation may result in rhinitis, pharyngitis, laryngitis, and bronchitis. Conjunctivitis, keratitis, and corneal ulceration may occur. An itchy erythematous dermatitis has been reported and continued skin contact leads to the formation of slowly healing ulcerations on the palms and between the fingers. Sensitization occasionally develops. Chronic rhinitis and perforation of the nasal septum have been reported after long exposures. All local effects appear to be due to the caustic nature of cyanamide.

Systemic—

Calcium cyanamide causes a characteristic vasomotor reaction. There is erythema of the upper portions of the body, face, and arms,

accompanied by nausea, fatigue, headache, dyspnea, vomiting, oppression in the chest, and shivering. Circulatory collapse may follow in the more serious cases. The vasomotor response may be triggered or intensified by alcohol ingestion. Pneumonia or lung edema may develop. Cyanide ion is not released in the body, and the mechanism of toxic action is unknown.

MEDICAL SURVEILLANCE

Evaluate skin, respiratory tract, and history of alcohol intake in placement or periodic examinations.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

In addition to personal protective equipment, waterproof barrier creams may be used to provide additional face and skin protection. Personal hygiene measures are to be encouraged, such as showering after work and a complete change of clothing. In areas of heavy dust concentrations, fullface dust masks are recommended.

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o-CHLOROBENZYLIDENE MALONITRILE

DESCRIPTION

$ClC_6H_4CH=C(CN)_2$ o-chlorobenzylidene malonitrile (OCBM), is a white crystalline solid.

SYNONYMS

OCBM, CS.

POTENTIAL OCCUPATIONAL EXPOSURES

OCBM is used as a riot control agent.

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

Riot controllers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 0.05 ppm (0.4 mg/m^3).

ROUTE OF ENTRY

Inhalation.

HARMFUL EFFECTS

Local—

OCBM is extremely irritating and acts on exposed sensory nerve endings (primarily in the eyes and upper respiratory tract). The signs

and symptoms from exposure to the vapor are conjunctivitis and pain in the eyes, lacrimation, erythema of the eyelids, blepharospasms, irritation and running of the nose, burning in the throat, coughing and constricted feeling in the chest, and excessive salivation. Vomiting may occur if saliva is swallowed. Most of the symptoms subside after exposure ceases. Burning on the exposed skin is increased by moisture. With heavy exposure, vesiculation and erythema occur. Photophobia has been reported.

Systemic—

Animal experiments indicate that OCBM has a relatively low toxicity. The systemic changes observed in human experiments are nonspecific reactions to stress. OCBM is capable of sensitizing guinea pigs; there also appears to be a cross-reaction in guinea pigs previously sensitized to 1-chloroacetophenone (CN)

MEDICAL SURVEILLANCE

Consideration should be given to the eyes, skin, and respiratory tract in any placement or periodic evaluations.

SPECIAL TESTS

None have been proposed.

PERSONAL PROTECTIVE METHODS

Because of its extremely irritant properties, those using OCBM in high concentrations should wear respirators and eye protection.

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Punte, C. L., E. J. Owens, and P. J. Gutentag. 1963. Exposures to orthochlorobenzylidene malonitrile. Arch. Environ. Health 6:366.

HYDROGEN CYANIDE

DESCRIPTION

Hydrogen cyanide, a colorless gas or liquid, intensely poisonous, with the odor of bitter almonds, is highly flammable and explosive and is a very weak acid. Hydrogen cyanide, HCN (together with its soluble salts), owes its toxicity to the $-CN$ moiety and not to its acid properties. HCN vapor is released when cyanide salts come in contact with any acid.

SYNONYMS

Hydrocyanic acid, prussic acid.

POTENTIAL OCCUPATIONAL EXPOSURES

Hydrogen cyanide is used as a fumigant, in electroplating, and in chemical synthesis of acrylates and nitriles, particularly acrylonitrile. It may be generated in blast furnaces, gas works, and coke ovens. Cyanide salts have a wide variety of uses, including electroplating, steel hardening, fumigating, gold and silver extraction from ores, and chemical synthesis.

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

- Acid dippers
- Acrylate makers
- Ammonium salt makers
- Blast furnace workers
- Cellulose product treaters
- Coke oven workers
- Cyanogen makers
- Electroplaters
- Fumigant workers
- Gas workers
- Gold extractors
- Jewelers
- Organic chemical synthesizers
- Polish makers
- Silver extractors
- Steel workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for hydrogen cyanide is 10 ppm (11 mg/m^3). NIOSH has recommended 5 mg/m^3 expressed as cyanide and determined as a ceiling concentration based on a 10-minute sampling period.

ROUTES OF ENTRY

Inhalation of vapor and percutaneous absorption of liquid and concentrated vapor.

HARMFUL EFFECTS

Local—

Hydrogen cyanide is a mild upper respiratory irritant and may cause slight irritation of the nose and throat. There may also be irritation from skin and eye contact with the liquid. Hydrogen cyanide liquid may cause eye irritation.

Systemic—

Hydrogen cyanide is an asphyxiant. It inactivates certain enzyme systems, the most important being cytochrome oxidase, which occupies a fundamental position in the respiratory process and is involved in the ultimate electron transfer to molecular oxygen. Inhalation, ingestion, or skin absorption of hydrogen cyanide may be rapidly fatal. Larger doses may cause loss of consciousness, cessation of respiration, and death. Lower levels of exposure may cause weakness, headache, confusion, nausea, and vomiting. These symptoms may be followed by unconsciousness and death.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include the cardiovascular and central nervous systems, liver and kidney function, blood, history of fainting or dizzy spells.

SPECIAL TESTS

Blood CN levels may be useful during acute intoxication. Urinary thiocyanate levels have been used but are nonspecific and are elevated in smokers.

PERSONAL PROTECTIVE METHODS

If personal protective equipment is necessary, air supplied or self-contained gas masks specific for hydrogen cyanide, and clothing impervious to HCN vapor should be worn. Eye protection can be provided by fullface respirators or goggles. All personnel working with processes involving cyanides should be specially trained so that they fully understand the hazard, and so they will faithfully follow all rules laid down for safe handling.

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ISOCYANATES

DESCRIPTION

Both toluene diisocyanate (TDI) and methylene bisphenyl isocyanate (MDI) are liquids and may exist in different isomers: 2,4-toluene diisocyanate and methylene bisphenyl 4,4'-diisocyanate. Other less commonly used isocyanates are hexamethylene diisocyanate (HDI) and 1,5-naphthalene diisocyanate (NDI).

SYNONYMS

Toluene diisocyanate: TDI, tolylene diisocyanate, diisocyanatotoluene. Methylene bisphenyl isocyanate: MDI, diphenylmethane diisocyanate, methane diisocyanate.

POTENTIAL OCCUPATIONAL EXPOSURES

TDI is more widely used than MDI. Polyurethanes are formed by the reaction of isocyanates with polyhydroxy compounds. Since the reaction proceeds rapidly at room temperature, the reactants must be mixed in pots or spray guns just before use. These resins can be produced with various physical properties, e.g., hard, flexible, semirigid foams, and have found many uses, e.g., upholstery padding, thermal insulation, molds, surface coatings, shoe innersoles, and in rubbers, adhesives, paints, and textile finishes. Because of TDI's high volatility, exposure can occur in all phases of its manufacture and use. MDI has a much lower volatility, and problems generally arise only in spray applications.

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

- Adhesive workers
- Insulation workers
- Isocyanate resin workers
- Lacquer workers
- Organic chemical synthesizers
- Paint sprayers
- Polyurethane makers
- Rubber workers
- Ship burners
- Textile processors
- Wire coating workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for MDI is 0.02 ppm (0.2 mg/m^3) as a ceiling value. The Federal standard for the 2,4 isomer of TDI is also 0.02 ppm (0.14 mg/mg^3) as a ceiling value. However, the standard recommended in the NIOSH Criteria Document for TDI is 0.005 ppm (0.036 mg/m^3) as a TWA and 0.02 for any 20-minute period.

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

TDI and MDI may cause irritation of the eyes, respiratory tract, and skin. The irritation may be severe enough to produce bronchitis and pulmonary edema. Nausea, vomiting, and abdominal pain may occur. If liquid TDI is allowed to remain in contact with the skin, it may produce redness, swelling, and blistering. Contact of liquid TDI with the eyes may cause severe irritation, which may result in permanent damage if untreated. Swallowing TDI may cause burns of the mouth and stomach.

Systemic—

Sensitization to TDI and MDI may occur, which may cause an asthmatic reaction with wheezing, dyspnea, and cough. These symptoms may first occur during the night following exposure to these chemicals. Some decrease in lung function in the absence of symptoms has been observed in some workers exposed to TDI for long periods of time.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should include chest roentgenograph, pulmonary function tests, and an evaluation of any respiratory disease or history of allergy. Periodic pulmonary function tests may be useful in detecting the onset of pulmonary sensitization.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Protective clothing and goggles should be worn if there is a possibility of contact with the liquids. In areas of vapor concentration, full-

face masks with organic vapor canisters or respirators with supplied air and fullface pieces should be worn.

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AROMATIC HYDROCARBONS

Aromatic hydrocarbons are characterized by the presence of the aromatic nucleus. The basic aromatic nucleus is benzene, C_6H_6. In benzene, the carbon atoms are arranged as a regular hexagon, with a hydrogen atom attached to each of the carbon atoms. The bond between each of the carbon atoms is neither a single bond nor a double bond, but an intermediate form of higher stability. (The electronic character of the benzene nucleus is usually referred to as "resonance.") The fact that the bonds are intermediate between single and double results in all of the carbon atoms being equivalent. The hydrogen atoms on the aromatic nucleus may be replaced by other univalent elements or groups. Aromatic hydrocarbons encompass compounds that include only carbon and hydrogen.

Aromatic hydrocarbons have enjoyed wide usage as solvents and as chemical intermediates. Benzene, the typical aromatic hydrocarbon, has been replaced as a commercial solvent by toluene and other less toxic compounds. These chemicals are also used as feedstock for many organic compounds and are used in the manufacture of fuels, dyes, pharmaceuticals, plastics, resins, and polyesters.

Typically, the vapor of aromatic hydrocarbons causes central nervous system depression or other effects, and, depending on the compound, hepatic, renal, or bone marrow disorders. Vapor is absorped through the lungs, and the liquid may be absorbed through the skin. Repeated and prolonged skin contact may cause defatting of the skin, which leads to dermatitis.

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BENZENE

DESCRIPTION

C_6H_6, benzene, is a clear, volatile, colorless, highly flammable liquid with a charactersitic odor. The most common commercial grade

contains 50-100% benzene, the remainder consisting of toluene, xylene, and other constituents which distill below 120 C.

SYNONYMS

Benzol, phenyl hydride, coal naphtha, phene, benxole, cyclohexatriene.

POTENTIAL OCCUPATIONAL EXPOSURES

Benzene is used as a constituent in motor fuels, as a solvent for fats, inks, oils, paints, plastics, and rubber, in the extraction of oils from seeds and nuts, and in photogravure printing. It is also used as a chemical intermediate. By alkylation, chlorination, nitration, and sulfonation, chemicals such as styrene, phenols, and maleic anhydride are produced. Benzene is also used in the manufacture of detergents, explosives, pharmaceuticals, and dyestuffs.

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

- Adhesive makers
- Asbestos product impregnators
- Dry-battery makers
- Chemists
- Benzene hexachloride makers
- Burnishers
- Carbolic acid makers
- Chlorinated benzene workers
- Detergent makers
- Dye makers
- Furniture finishers
- Glue makers
- Linoleum makers
- Maleic acid makers
- Nitrobenzene makers
- Petrochemical workers
- Putty makers
- Rubber makers
- Styrene makers
- Welders

PERMISSIBLE EXPOSURE LIMITS

The Federal emergency standard for benzene effective May 21, 1977, is 1 ppm for an 8-hour TWA, with 5 ppm as a maximum peak above the acceptable ceiling for a maximum duration of 15 minutes.

ROUTES OF ENTRY

Inhalation of vapor which may be supplemented by percutaneous absorption although benzene is poorly absorbed through intact skin.

HARMFUL EFFECTS

Local—

Exposure to liquid and vapor may produce primary irritation to skin, eyes, and upper respiratory tract. If the liquid is aspirated into the lung, it may cause pulmonary edema and hemorrhage. Erythema, vesiculation, and dry, scaly dermatitis may also develop from defatting of the skin.

Systemic—

Acute exposure to benzene results in central nervous system depression. Headache, dizziness, nausea, convulsions, coma, and death

may result. Death has occurred from large acute exposure as a result of ventricular fibrillation, probably caused by myocardial sensitization to endogenous epinephrine. Early reported autopsies revealed hemorrhages (non-pathognomonic) in the brain, pericardium, urinary tract, mucous membranes, and skin.

Chronic exposure to benzene is well documented to cause blood changes. Benzene is basically a myelotoxic agent. Erythrocyte, leukocyte, and thrombocyte counts may first increase, and then aplastic anemia may develop with anemia, leukopenia, and thrombocytopenia. The bone marrow may become hypo- or hyper-active and may not always correlate with peripheral blood.

Recent epidemiologic studies along with case reports of benzene related blood dyscrasias and chromosomal aberrations have led NIOSH to conclude that benzene is leukemogenic. The evidence is most convincing for acute myelogenous leukemia and for acute erythroleukemia, but a connection with chronic leukemia has been noted by a few investigators.

Recent work has shown increases in the rate of chromosomal aberations associated with benzene myelotoxicity. These changes in the bone marrow are stable or unstable and may occur several years after exposure has ceased. "Stable" changes may give rise to leukemic clones and seem to involve chromosomes of the G group.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should be concerned especially with effects on the blood and bone marrow and with a history of exposure to other myelotoxic agents or drugs or of other diseases of the blood. Preplacement laboratory exams should include: (a) complete blood count (hematocrit, hemoglobin, mean corpuscular volume, white blood count, differential count, and platelet estimation), (b) reticulocyte count, (c) serum bilirubin, and (d) urinary phenol.

The type and frequency of periodic hematologic studies should be related to the data obtained from biologic monitoring and industrial hygiene studies, as well as any symptoms or signs of hematologic effects. Recommendations for proposed examinations have been made in the criteria for a recommended standard. Examinations should also be concerned with other possible effects such as those on the skin, central nervous system, and liver and kidney functions.

SPECIAL TESTS

Biologic monitoring should be provided to all workers subject to benzene exposure. It consists of sampling and analysis of urine for total phenol content. The objective of such monitoring is to be certain that no worker absorbs an unacceptable amount of benzene. Unacceptable absorption of benzene, posing a risk of benzene poisoning, is considered to occur at levels of 75 mg phenol per liter of urine (with urine specific gravity corrected to 1.024), when determined by methods specified in the NIOSH "Criteria for Recommended Standard - Benzene." Alter-

native methods shown to be equivalent in accuracy and precision may also be useful. Biological monitoring should be done at quarterly intervals. If environmental sampling and analysis are equal to or exceed accepted safe limits, the urinary phenol analysis should be conducted every two weeks. This increased monitoring frequency should continue for at least 2 months after the high environmental level has been demonstrated.

Two follow-up urines should be obtained within one week after receipt of the original results, one at the beginning and the other at the end of the work week. If original elevated findings are confirmed, immediate steps should be taken to reduce the worker's absorption of benzene by improvement in environmental control, personal protection, personal hygiene, and administrative control.

PERSONAL PROTECTIVE METHODS

Protective clothing should be worn at all times; benzene-wetted clothing should be changed at once. Impervious clothing and gloves to cover exposed areas of body should be worn where exposure is continuous. In areas where there is likelihood of spill or splash, face shields or goggles should be provided. In areas of elevated vapor concentration, organic vapor cartridge masks or supplied air or self-contained breathing apparatus may be required.

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DIPHENYL

DESCRIPTION

$C_6H_5C_6H_5$, diphenyl, is a colorless to light yellow, leaflet solid with a potent characteristic odor.

SYNONYMS

Biphenyl, phenylbenzene.

POTENTIAL OCCUPATIONAL EXPOSURES

Diphenyl is a fungistat for oranges which is applied to the inside of shipping containers and wrappers. It is also used as a heat transfer agent and as an intermediate in organic synthesis. Diphenyl is produced by thermal dehydration of benzene.

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

Orange packers
Fungicide workers
Organic chemical synthesizers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 0.2 ppm (1 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapor or dust; percutaneous absorption.

HARMFUL EFFECTS

Local—

Repeated exposure to dust may result in irritation of skin and respiratory tract. The vapor may cause moderate eye irritation. Repeated skin contact may produce a sensitization dermatitis.

Systemic—

In acute exposure, diphenyl exerts a toxic action on the central nervous system, on the peripheral nervous system, and on the liver. Symptoms of poisoning are headache, diffuse gastrointestinal pain, nausea, indigestion, numbness and aching of limbs, and general fatigue. Liver function tests may show abnormalities. Chronic exposure is characterized mostly by central nervous system symptoms, fatigue, headache, tremor, insomnia, sensory impairment, and mood changes. Such symptoms are rare, however.

MEDICAL SURVEILLANCE

Consider skin, eye, liver function and respiratory tract irritation in any preplacement or periodic examination.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Because of its low vapor pressure and low order of toxicity, it does not usually present a major problem in industry. Protective creams, gloves, and masks with organic vapor canisters for use in areas of elevated vapor concentrations should suffice. Elevated temperature may increase the requirement for protective methods or ventilation.

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NAPHTHALENE

DESCRIPTION

$C_{10}H_8$, naphthalene, is a white crystalline solid with a characteristic "moth ball" odor.

SYNONYMS

Naphthalin, moth flake, tar camphor, white tar.

POTENTIAL OCCUPATIONAL EXPOSURES

Naphthalene is used as a chemical intermediate or feedstock for synthesis of phthalic, anthranilic, hydroxyl (naphthols), amino (naphthylamines), and sulfonic compounds which are used in the manufacture of various dyes. Naphthalene is also used in the manufacture of hydronaphthalenes, synthetic resins, lampblack, smokeless powder, and celluloid. Naphthalene has been used as a moth repellent.

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

- Beta naphthol makers
- Celluloid makers
- Coal tar workers
- Dye chemical makers
- Fungicide makers
- Hydronaphthalene makers
- Lampblack makers
- Moth repellent workers
- Phthalic anhydride makers
- Smokeless powder makers
- Tannery workers
- Textile chemical makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 10 ppm (50 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor or dust.

HARMFUL EFFECTS

Local—

Naphthalene is a primary irritant and causes erythema and dermatitis upon repeated contact. It is also an allergen and may produce dermatitis in hypersensitive individuals. Direct eye contact with the dust has produced irritation and cataracts.

Systemic—

Inhaling high concentrations of naphthalene vapor or ingesting may cause intravascular hemolysis and its consequences. Initial symptoms include eye irritation, headache, confusion, excitement, malaise, profuse sweating, nausea, vomiting, abdominal pain, and irritation of the bladder. There may be progressive jaundice, hematuria, hemoglobinuria, renal tubular blockade, and acute renal shutdown. Hematologic features include red cell fragmentation, icterus, severe anemia with nucleated red cells, leukocytosis, and dramatic decreases in hemoglobin, hematocrit, and red cell count. Individuals with a deficiency of glucose-6-phosphate dehydrogenase in erythrocytes are more susceptible to hemolysis by naphthalene.

MEDICAL SURVEILLANCE

Consider eyes, skin, blood, liver, and renal function in placement and follow-up examinations. Low erythrocyte glucose 6-phosphate dehydrogenase increases risk.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

As used in industry, they are rarely necessary. In dusty areas and areas of high vapor concentration, dust type or organic vapor canister masks should be supplied. Skin protection with gloves, barrier creams, or protective clothing may be useful.

STYRENE/ETHYL BENZENE

DESCRIPTION

$C_6H_5CH=CH_2$, styrene, is a colorless to yellowish, very refractive, oily liquid with a penetrating odor.

$C_6H_5C_2H_5$, ethyl benzene, is a colorless flammable liquid with a pungent odor.

SYNONYMS

Styrene: Cinnamene, cinnemenol, cinnamol, phenethylene, phenylethylene, styrene monomer, styrol, styrolene, vinyl benzene.

Ethyl benzene: Ethylbenzol, phenylethane, EB.

POTENTIAL OCCUPATIONAL EXPOSURES

Upon heating to 200 C, styrene polymerizes to form polystyrene, a plastic. It is also used in combination with 1,3-butadiene or acrylonitrile to form copolymer elastomers, butadiene-styrene rubber, and acrylonitrile-butadienestyrene (ABS). It is also used in the manufacture of resins, polyesters, and insulators.

Ethyl benzene is used in the manufacture of cellulose acetate, styrene, and synthetic rubber. It is also used as a solvent or diluent and as a component of automotive and aviation gasoline.

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

- Adhesive makers
- Aviation fuel blenders
- Emulsifier agent makers
- Fibrous glass moulders
- Insulator makers
- Lacquer workers
- Organic chemical synthesizers
- Petroleum refinery workers
- Polyester resin laminators
- Polystyrene makers
- Potting compound workers
- Protective coating workers
- Resin makers
- Rubber makers
- Solvent workers
- Varnish makers.

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for styrene for an 8-hour TWA is 100 ppm (420 mg/m^3). The acceptable ceiling concentration is 200 ppm with an acceptable maximum peak of 600 ppm for a maximum duration of 5 minutes in any 3 hours. The Federal standard for ethyl benzene is 100 ppm (435 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapor; percutaneous absorption.

HARMFUL EFFECTS

Local—

Liquid and vapor are irritating to the eyes, nose, throat, and skin. The liquids are low-grade cutaneous irritants, and repeated contact may produce a dry, scaly, and fissured dermatitis.

Systemic—

Acute exposure to high concentrations may produce irritation of the mucous membranes of the upper respiratory tract, nose, and mouth, followed by symptoms of narcosis, cramps, and death due to respiratory center paralysis. Effects of short-term exposure to styrene under laboratory conditions include prolonged reaction time and decreased manual dexterity.

MEDICAL SURVEILLANCE

Consider possible irritant effects on the skin, eyes, and respiratory tract in any preplacement or periodic examinations, as well as blood, liver, and kidney function.

SPECIAL TESTS

None in common use. Mandelic acid in urine has been used as a measure of the intensity of styrene exposure.

PERSONAL PROTECTIVE METHODS

Barrier creams or gloves and protective clothing may be all that are needed where the vapor concentrations do not exceed existing standards. Where vapor concentration exists above allowable standards, masks with organic vapor canisters and face plates or respirators with air supply are recommended. Clothing saturated with styrene or ethylbenzene should be changed at once. Personal hygiene is encouraged with frequent changes of work clothes.

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TOLUENE

DESCRIPTION

$C_6H_5CH_3$, toluene, is a clear, colorless, noncorrosive liquid with a sweet, pungent, benzene-like odor.

SYNONYMS

Toluol, methylbenzene, phenylmethane, methylbenzol.

POTENTIAL OCCUPATIONAL EXPOSURES

Toluene may be encountered in the manufacture of benzene. It is

also used as a chemical feed for toluene diisocyanate, phenol, benzyl and benzyl derivatives, benzoic acid, toluene sulfonates, nitrotoluenes, vinyl toluene, and saccharin; as a solvent for paints and coatings; or as a component of automobile and aviation fuels.

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

- Aviation fuel blenders,
- Benzene makers
- Chemical laboratory workers
- Coke oven workers
- Gasoline blenders
- Lacquer workers
- Paint thinner makers
- Perfume makers
- Petrochemical workers
- Rubber cement makers
- Saccharin makers
- Solvent workers
- Toluene diisocyanate makers
- Vinyl toluene makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 200 ppm as an 8-hour TWA with an acceptable ceiling concentration of 300 ppm; acceptable maximum peaks above the ceiling of 500 ppm are allowed for 10 minutes duration. NIOSH has recommended a limit of 100 ppm (TWA) with a ceiling of 200 ppm for a ten minute sampling period.

ROUTES OF ENTRY

Inhalation of vapor and percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Toluene may cause irritation of the eyes, respiratory tract, and skin. Repeated or prolonged contact with liquid may cause removal of natural lipids from the skin, resulting in dry, fissured dermatitis. The liquid splashed in the eyes may cause irritation and reversible damage.

Systemic—

Acute exposure to toluene predominantly results in central nervous system depression. Symptoms and signs include headache, dizziness, fatigue, muscular weakness, drowsiness, incoordination with staggering gait, skin paresthesias, collapse, and coma.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should evaluate possible effect on skin, central nervous system, as well as liver and kidney function. Hematologic studies should also be done if there is significant contamination of the solvent with benzene.

SPECIAL TESTS

Hippuric acid levels above 5 g/liter of urine may result from exposure greater than 200 ppm determined as a TWA. Blood levels can also be determined for toluene.

PERSONAL PROTECTIVE METHODS

Where vapor concentration exists above allowable standards, em-

ployees should be provided with respirators (air supplied) or gas masks with organic vapor canister and fullface plate. Impervious clothing, gloves, or other coverings to protect potentially exposed areas of the body should be supplied to employees in operations requiring continued exposure to liquid toluene. Toluene-wet clothing should be immediately removed unless impervious, and work clothing changed at least twice a week. Safety glasses or goggles should be worn in areas where splash or spill is likely.

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XYLENE

DESCRIPTION

$C_6H_4(CH_3)_2$, xylene, exists in three isomeric forms, ortho-, meta- and para-xylene. Commercial xylene is a mixture of these three isomers and may also contain ethyl benzene as well as small amounts of toluene, trimethyl benzene, phenol, thiophene, pyridine, and other non-aromatic hydrocarbons. Metaxylene is predominant in commercial xylene and shares physical properties with ortho-xylene in that both are mobile, colorless, flammable liquids. Para-xylene, at low temperature (13-14 C), forms colorless plates or prisms.

SYNONYMS

Xylol, dimethylbenzene.

POTENTIAL OCCUPATIONAL EXPOSURES

Xylene is used as a solvent; as a constituent of paint, lacquers, varnishes, inks, dyes, adhesives, cements, cleaning fluids and aviation fuels; and as a chemical feedstock for xylidines, benzoic acid, phthalic anhydride, isophthalic, and terephthalic acids, as well as their esters (which are specifically used in the manufacture of plastic materials and synthetic textile fabrics). Xylene is also used in the manufacture of quartz crystal oscillators, hydrogen peroxide, perfumes, insect repellants, epoxy resins, pharmaceuticals, and in the leather industry.

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

- Adhesive workers
- Aviation gasoline workers
- Benzoic acid makers
- Cleaning fluid makers
- Histology technicians
- Lacquer workers
- Leather workers
- Paint workers
- Phthalic anhydride makers
- Polyethylene terephthalate film makers
- Quartz crystal oscillator makers
- Solvent workers
- Synthetic textile makers
- Terephthalic acid makers
- Varnish makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 100 ppm (435 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapor and, to a small extent, percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Xylene vapor may cause irritation of the eyes, nose, and throat. Repeated or prolonged skin contact with xylene may cause drying and defatting of the skin which may lead to dermatitis. Liquid xylene is irritating to the eyes and mucous membranes, and aspiration of few milliliters may cause chemical pneumonitis, pulmonary edema, and hemorrhage. Repeated exposure of the eyes to high concentrations of xylene vapor may cause reversible eye damage.

Systemic—

Acute exposure to xylene vapor may cause central nervous system depression and minor reversible effects upon liver and kidneys. At high concentrations xylene vapor may cause dizziness, staggering, drowsiness, and unconsciousness. Also at very high concentrations, breathing xylene vapors may cause pulmonary edema, anorexia, nausea, vomiting, and abdominal pain.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should evaluate possible effects on the skin and central nervous system, as well as liver and kidney functions. Hematalogic studies should be done if there is any significant contamination of the solvent with benzene.

SPECIAL TESTS

Although metabolites are known, biologic monitoring has not been widely used. Hippuric acid or the ether glucuronide of ortho-toluic acid may be useful in diagnosis of meta-, para- and ortho-xylene exposure, respectively.

PERSONAL PROTECTIVE METHODS

When vapor concentrations exceed allowable standards, fullface masks with organic vapor canisters or air supplied respirators should be furnished. Impervious protective clothing and gloves should be worn to cover exposed portions of the body of employees exposed to liquid xylene. Xylene-wet clothing should be changed quickly. Personal hygiene, as well as appropriate changes of work clothes, is necessary. Goggles or safety glasses in areas of spill or splash, or in areas where vapors concentrate, are advised. Barrier creams may be useful.

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PHENOLS AND PHENOLIC COMPOUNDS

This group of compounds is characterized by the substitution of one or more hydrogens in a benzene ring by hydroxyl (−OH) groups. Phenol (C_6H_5OH) is the simplest of the compounds. Additional substitutions are possible. Quinone ($C_6H_4O_2$) is included in this group because it is derived from hydroquinone although its physical, chemical, and toxic properties are quite different. These substances are widely distributed in industry and some (e.g., phenolcresol) find use in pharmaceuticals because of their disinfectant action.

These materials generally enter the body by inhalation and percutaneous absorption. Their toxicity varies, but some are highly irritating to the skin, mucous membranes of the upper respiratory tract, and eyes. Some are corrosive for all tissue; cresote, a complex mixture of phenolic and aromatic compounds, may cause skin cancer. Systemic effects usually involve the central nervous or cardio-vascular systems or both; this may be accompanied by renal and hepatic damage.

Appropriate engineering controls and personal protective devices should be used to prevent absorption by either the respiratory or percutaneous route, and eye protection should be utilized where necessary.

CRESOL

DESCRIPTION

$CH_3C_6H_4OH$, cresol, is a mixture of the three isomeric cresols, ortho-,meta-, and para-cresol, and is a colorless, yellowish, brownish-yellow, or pinkish liquid with a phenolic odor. Creosols are soluble in alcohol, glycol, and dilute alkalis. Also they may be combustible.

SYNONYMS

Cresylic acid, cresylol, hydroxytoluene, methyl phenol, oxytoluene, tricresol.

POTENTIAL OCCUPATIONAL EXPOSURES

Cresol is used as a disinfectant, as an ore flotation agent, and as an intermediate in the manufacture of chemicals, dyes, plastics, and antioxidants. A mixture of isomers is generally used; the concentrations of the components are determined by the source of the cresol.

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

Antioxidant makers	Paint remover workers
Chemical disinfectant workers	Pitch workers
Dye makers	Plastic makers
Flotation agent makers	Resin makers
Foundry workers	Stain workers
Insulation enamel workers	Wool scourers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (22 mg/m^3).

ROUTES OF ENTRY

Inhalation or percutaneous absorption of liquid or vapor.

HARMFUL EFFECTS

Local—

Cresol is very corrosive to all tissues. It may cause burns if it is not removed promptly and completely and in case of extensive exposure, if it is not removed completely from contaminated areas of the body very quickly, death may result. When it contacts the skin, it may not produce any sensation immediately. After a few moments, prickling and intense burning occur. This is followed by loss of feeling. The affected skin shows wrinkling, white discoloration, and softening. Later gangrene may occur. If the chemical contacts the eyes, it may cause extensive damage and blindness. A skin rash may result from repeated or prolonged exposure of the skin to low concentrations of cresol. Discoloration of the skin may also occur from this type of exposure.

Systemic—

When cresol is absorbed into the body either through the lungs, through the skin, or mucous membranes, or by swallowing, it may cause systemic poisoning. The signs and symptoms of systemic poisoning may develop in 20 or 30 minutes. These toxic effects include: weakness of the muscles, headache, dizziness, dimness of vision, ringing of the ears, rapid breathing, mental confusion, loss of consciousness, and sometimes death.

Prolonged or repeated absorption of low concentrations of cresol through the skin, mucous membranes, or respiratory tract may cause chronic systemic poisoning. Symptoms and signs of chronic poisoning include vomiting, difficulty in swallowing, salivation, diarrhea, loss of appetite, headache, fainting, dizziness, mental disturbances, and skin rash. Death may result if there has been severe damage to the liver and kidneys.

MEDICAL SURVEILLANCE

Consider the skin, eyes, respiratory system, and liver and kidney function in placement or periodic examinations.

SPECIAL TESTS

Can be determined in urine, but because large amounts are normally present, a urine test is of little value as a procedure for evaluating exposure.

PERSONAL PROTECTIVE METHODS

Protective goggles and clothing should be worn to prevent direct contact with cresol. Masks with organic vapor canisters are advisable in areas of vapor concentration.

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CREOSOTE

DESCRIPTION

Creosote is a flammable, heavy, oily liquid with a characteristic sharp, smoky smell, and caustic burning taste. In pure form it is colorless, but the industrial product is usually brownish. It is produced by the destructive distillation of wood or coal tar at temperatures above 200 C. The chemical composition is determined by the source and may contain guaiacol, creosols, phenol, cresols, pyridine, and numerous other aromatic compounds.

SYNONYMS

Creosotum, cresote oil, brick oil.

POTENTIAL OCCUPATIONAL EXPOSURES

Creosote is used primarily as a wood preservative, and those working with the treated wood may be exposed. It is also used as a waterproofing agent, an animal dip, a constituent in fuel oil, a lubricant for die molds, as pitch for roofing, and in the manufacture of chemicals and lampblack. In the pharmaceutical industry, it is used as an antiseptic, disinfectant, antipyretic, astringent, styptic, germicide, and expectorant.

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

- Coal tar workers
- Fuel oil blenders
- Lampblack makers
- Organic chemical synthesizers
- Pitch workers
- Water proofers
- Wood preservers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for creosote.

ROUTE OF ENTRY

Skin absorption.

HARMFUL EFFECTS

Local—

The liquid and vapors are strong irritants producing local erythema,

burning, itching, pigmentation (grayish yellow to bronze), vesiculation, ulceration, and gangrene. Eye injuries include keratitis, conjunctivitis, and permanent corneal scars. Contact dermatitis is reported in industry. Photosensitization has been reported. Skin cancer may occur.

Systemic—

Symptoms of systemic illness include salivation, vomiting, vertigo, headache, loss of pupillary reflexes, hypothermia, cyanosis, convulsions, thready pulse, respiratory difficulties, and death.

MEDICAL SURVEILLANCE

Consider the skin, eyes, respiratory tract, and central nervous system in placement and periodic examination.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

Protective clothing should be worn where employees are exposed to the liquid or high vapor concentration. Masks with fullface protection and organic vapor canisters should be worn. Gloves and goggles are advisable in any area where spill or splash might occur.

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HYDROQUINONE

DESCRIPTION

$C_6H_4(OH)_2$, hydroquinone, exists as colorless, hexagonal prisms.

SYNONYMS

Quinol, hydroquinol, p-diphenol, hydrochinone, dihydroxybenzene, p-dihydroxybenzene, p-hydroxyphenol, 1,4-benzenediol.

POTENTIAL OCCUPATIONAL EXPOSURES

Hydroquinone is a reducing agent and is used as a photographic developer and as an antioxidant or stabilizer for certain materials which polymerize in the presence of oxidizing agents. Many of its derivatives are used as bacteriostatic agents, and others, particularly 2,5-bis(ethyleneimino) hydroquinone, have been reported to be good antimitotic and tumor-inhibiting agents.

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

- Antioxidant makers
- Bacteriostatic agent makers
- Drug makers
- Fur processors
- Motor fuel blenders
- Paint makers
- Organic chemical synthesizers
- Photographic developer makers
- Plastic stabilizer workers
- Stone coating workers
- Styrene monomer workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 2 mg/m³.

ROUTE OF ENTRY

Inhalation of dust.

HARMFUL EFFECTS

Local—

The dust is a mild primary irritant. Skin sensitization to the dry solid is very rare but does occur on occasion from contact with its alkaline solutions. The skin may be depigmented by repeated applications of ointments of hydroquinone, but this virtually never occurs from contact with dust or dilute water solutions. Following prolonged exposure to elevated dust levels, brownish conjunctiva stains may appear. These may be followed by corneal opacities and structural changes in the cornea which may lead to loss of visual acuity. The early pigmentary stains are reversible, while the corneal changes tend to be progressive.

Systemic—

Oral ingestion of large quantities of hydroquinone may produce blurred speech, tinnitus, tremors, sense of suffocation, vomiting, muscular twitching, headache, convulsions, dyspnea and cyanosis from methemoglobinemia, and coma and collapse from respiratory failure. The urine is usually green or brownish green. No systemic symptoms have been found following inhalation of hydroquinone dust.

MEDICAL SURVEILLANCE

Careful examination of the eyes, including visual acuity and slit lamp examinations, should be carried out in preplacement and periodic examinations. Also examine skin.

SPECIAL TESTS

Hydroquinone is excreted in the urine as a sulfate ester. This has not been helpful in following worker exposure to dust.

PERSONAL PROTECTIVE METHODS

The eyes should be protected by goggles or dust masks with full-face shield. Protective clothing is recommended along with good hygiene practice, clothes changing after each shift, and showering prior to dressing in street clothes. Oxidation of hydroquinone may produce quinone vapor which is highly irritating.

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PHENOL

DESCRIPTION

C_6H_5OH, phenol, is a white crystalline substance with a distinct aromatic, acrid odor.

SYNONYMS

Carbolic acid, phenic acid, phenylic acid, phenyl hydrate, hydroxybenzene, monohydroxybenzene.

POTENTIAL OCCUPATIONAL EXPOSURES

Phenol is used in the production or manufacture of explosives, fertilizer, coke, illuminating gas, lampblack, paints, paint removers, rubber, asbestos goods, wood preservatives, synthetic resins, textiles, drugs, pharmaceutical preparations, perfumes, bakelite, and other plastics (phenol-formaldehyde resins). Phenol also finds wide use as a disinfectant in the petroleum, leather, paper, soap, toy, tanning, dye, and agricultural industries.

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

- Coal tar workers
- Disinfectant makers
- Dye workers
- Explosive workers
- Fertilizer makers
- Illuminating gas workers
- Lampblack makers
- Organic chemical synthesizers
- Paint and paint remover workers
- Paper makers
- Rubber reclaimers
- Soap workers
- Tannery workers
- Weed killer users
- Wood preservers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (19 mg/m³).

ROUTES OF ENTRY

Inhalation of mist or vapor; percutaneous absorption of mist, vapor, or liquid.

HARMFUL EFFECTS

Local—

Phenol has a marked corrosive effect on any tissue. When it comes in contact with the eyes it may cause severe damage and blindness. On contact with the skin, it does not cause pain but causes a whitening of the exposed area. If the chemical is not removed promptly, it may cause a severe burn or systemic poisoning.

Systemic—

Systemic effects may occur from any route of exposure. These include paleness, weakness, sweating, headache, ringing of the ears, shock, cyanosis, excitement, frothing of the nose and mouth, dark colored urine, and death. If death does not occur, kidney damage may appear.

Repeated or prolonged exposure to phenol may cause chronic phenol poisoning. This condition is very rarely reported. The symptoms of chronic poisoning include vomiting, difficulty in swallowing, diarrhea, lack of appetite, headache, fainting, dizziness, dark urine, mental disturbances, and possibly, skin rash. Liver and kidney damage and discoloration of the skin may occur.

MEDICAL SURVEILLANCE

Consider the skin, eye, liver, and renal function as part of any preplacement or periodic examination.

SPECIAL TESTS

Phenol can be determined in blood or urine.

PERSONAL PROTECTIVE METHODS

In areas where there is likelihood of a liquid spill or splash, impervious protective clothing and goggles should be worn. In areas of heavy vapor concentrations, fullface mask with forced air supply should be used, as well as protective clothing, gloves, rubber boots, and apron.

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QUINONE

DESCRIPTION

$C_6H_4O_2$, quinone, exists as large yellow, monoclinic prisms; the vapors have a pungent, irritating odor.

SYNONYMS

Benzoquinone, chinone, p-benzoquinone, 1,4-benzoquinone

POTENTIAL OCCUPATIONAL EXPOSURES

Because of its ability to react with certain nitrogen compounds to form colored substances, quinone is widely used in the dye, textile, chemical, tanning, and cosmetic industries. It is used as an intermediate in chemical synthesis for hydroquinone and other chemicals.

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

- Chemical laboratory workers
- Cosmetic makers
- Dye makers
- Gelatin makers
- Hydrogen peroxide makers
- Organic chemical synthesizers
- Photographic film developers
- Protein fiber makers
- Tannery workers
- Textile workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 0.1 ppm (0.4 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Solid quinone in contact with skin or the lining of the nose and throat may produce discoloration, severe irritation, swelling, and the formation of papules and vesicles. Prolonged contact with the skin may cause ulceration. Quinone vapor is highly irritating to the eyes. Following prolonged exposure to vapor, brownish conjunctival stains may appear. These may be followed by corneal opacities and structural changes in the cornea and loss of visual acuity. The early pigmentary stains are reversible, while the corneal dystrophy tends to be progressive.

Systemic—

No systemic effects have been found in workers exposed to quinone vapor over many years.

MEDICAL SURVEILLANCE

Careful examination of the eyes, including visual acuity and slit lamp examinations, should be done during placement and periodic examinations. Also evaluate skin.

SPECIAL TESTS

No useful laboratory tests for monitoring exposure have been developed.

PERSONAL PROTECTIVE METHODS

In areas of high vapor concentrations, protection must be aimed at the eyes and respiratory tract. Fullface mask with organic vapor canisters or respirators with forced air afford protection. The skin can be damaged by contact with solid quinone, solutions, or vapor condensing on the skin, so protective clothing, gloves, and boots are indicated. Personal hygiene is encouraged, with clothes being changed after each shift or after becoming damp from contact with the liquid. Workers should shower before changing to street clothes.

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AROMATIC HALOGENATED HYDROCARBONS

Aromatic compounds having a halogen bearing side chain are extensively used in the manufacture of basic and acid colors, pharmaceuticals, pesticides, resins, and as chemical intermediates. The vapor and

liquid of some of these compounds are highly irritating to all mucous membranes and skin, and some are powerful lacrimators.

The chlorinated naphthalenes and diphenyls produce a severe and disfiguring acne on skin contact. Percutaneous absorption and inhalation of vapor may lead to severe liver damage in certain instances.

With exception of the chlorinated benzenes, the more highly chlorinated the compound, the greater the toxicity.

BENZYL CHLORIDE

DESCRIPTION

$C_6H_5CH_2Cl$, benzyl chloride is a colorless liquid with an unpleasant, irritating odor.

SYNONYMS

Alpha-chlorotoluene.

POTENTIAL OCCUPATIONAL EXPOSURES

Benzyl chloride is used in production of benzal chloride, benzyl alcohol, and benzaldehyde. Industrial usage includes the manufacture of plastics, dyes, synthetic tannins, perfumes, resins, and pharmaceuticals.

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

- Drug makers
- Dye makers
- Gasoline additive makers
- Germicide makers
- Perfume makers
- Photographic developer makers
- Plastic makers
- Resin makers
- Rubber makers
- Tannin makers
- Wetting agent makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1 ppm (5 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Benzyl chloride is a severe irritant to the eyes and respiratory tract. At 160 mg/m^3 it is unbearably irritating to the eyes and nose. Liquid contact with the eyes produces severe irritation and may cause corneal injury. Skin contact may cause dermatitis.

Systemic—

Benzyl chloride is regarded as a potential cause of pulmonary edema. One author has reported disturbances of liver functions and mild leakopenia in some workers, but this has not been confirmed. Sarcomas have been produced in rats which were injected with benzyl chloride.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include the skin, eyes, and an evaluation of the liver, kidney, respiratory tract, and blood.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Personal protective equipment should include industrial filter respirators with goggles, and protective clothing for face, hands, and arms.

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CHLORODIPHENYLS AND DERIVATIVES

DESCRIPTION

$C_{12}H_{10-x}Cl_x$, Chlorodiphenyls, are diphenyl rings in which one or more hydrogen atoms are replaced by a chlorine atom. Most widely used are chlorodiphenyl (42% chlorine), containing 3 chlorine atoms in unassigned positions, and chlorodiphenyl (54% chlorine) containing 5 chlorine atoms in unassigned positions. These compounds are light, straw-colored liquids with typical chlorinated aromatic odors; 42% chlorodiphenyl is a mobile liquid and 54% chlorodiphenyl is a viscous liquid.

Chlorinated diphenyl oxides are ethers of chlorodiphenyls and are included in this group. They range from clear, oily liquids to white to yellowish waxy solids, depending on the degree of chlorination.

SYNONYMS

Chlorobiphenyls, polychlorinated diphenyl, PCB.

POTENTIAL OCCUPATIONAL EXPOSURES

Chlorinated diphenyls are used alone and in combination with chlorinated naphthalenes. They are stable, thermoplastic, and non-flammable, and find chief use in insulation for electric cables and wires in the production of electric condensers, as additives for extreme pressure lubricants, and as a coating in foundry use.

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

- Cable coaters
- Dye makers
- Electric equipment makers
- Herbicide workers
- Lacquer makers
- Paper treaters
- Plasticizer makers
- Resin makers
- Rubber workers
- Textile flameproofers
- Transformer workers
- Wood preservers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards for dichlorophenyl (42%) and dichloro-

diphenyl (54%) are 1 mg/m³ and 0.5 mg/m³ respectively.

ROUTES OF ENTRY

Inhalation of fume or vapor and percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Prolonged skin contact with its fumes or cold wax may cause the formation of comedones, sebaceous cysts, and pustules, known as chloracne. Irritation to eyes, nose, and throat may also occur. The above standards are considered low enough to prevent systemic effects, but it is not known whether or not these levels will prevent local effects.

Systemic—

Generally, toxic effects are dependent upon the degree of chlorination; the higher the degree of substitution, the stronger the effects. Acute and chronic exposure can cause liver damage. Signs and symptoms include edema, jaundice, vomiting, anorexia, nausea, abdominal pains, and fatigue.

Studies of accidental oral intake indicate that chlorinated diphenyls are embryotoxic, causing stillbirth, a characteristic grey-brown skin, and increased eye discharge in infants born to women exposed during pregnancy.

MEDICAL SURVEILLANCE

Placement and periodic examinations should include an evaluation of the skin, lung, and liver function. Possible effects on the fetus should be considered.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Protection of exposed skin should be encouraged, since the above standards may not be low enough to prevent chloracne. Barrier creams, protective clothing, and good personal hygiene are good protective measures. Respirators should be used in areas of vapor concentration.

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Peakall, D. B. 1972. Polychlorinated diphenyls: occurrence and biological effects. Residue Rev. 44:1.

CHLORINATED BENZENES

DESCRIPTION

Chlorinated benzenes are aromatic rings with one or more chlorines

substituted for a hydrogen. Included in this group are:

Chlorobenzene: phenyl chloride, monochlorobenzene, chlorobenzol.
o-dichlorobenzene: 1,2-dichlorobenzene
m-dichlorobenzene: 1,3-dichlorobenzene.
p-dichlorobenzene: 1,4-dichlorobenzene.
1,2,3-trichlorobenzene: None.
1,2,4-trichlorobenzene: None.
1,3,5-trichlorobenzene: None.
1,2,4,5-tetrachlorobenzene: None.
Hexachlorobenzene: perchlorobenzene.

Compounds with only a few chlorines are usually colorless liquids at room temperature and have an aromatic odor. The more highly substituted compounds are crystals (typically monoclinic).

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Chlorobenzene is used as a solvent and as an intermediate in dyestuffs. o-Dichlorobenzene is used as a solvent, fumigant, insecticide, and chemical intermediate. p-Dichlorobenzene finds use as an insecticide, chemical intermediate, disinfectant and moth preventative. Other chlorinated benzenes are not as widely used in industry but find use as chemical intermediates, and to an even lesser extent, as insecticides and solvents.

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

Cellulose acetate workers	Insecticide makers and workers
Deodorant makers	Lacquer workers
Disinfectant workers	Organic chemical synthesizers
Dyers	Paint workers
Dye makers	Resin makers
Fumigant workers	Seed disinfectors

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are:

chlorobenzene	75 ppm	350 mg/m^3
o-dichlorobenzene	50 ppm	300 mg/m^3
p-dichlorobenzene	75 ppm	450 mg/m^3

Threshold limit values for the other compounds have not as yet been established.

ROUTES OF ENTRY

Inhalation of vapor, percutaneous absorption of the liquid.

HARMFUL EFFECTS

Local—

Chlorinated benzenes are irritating to the skin, conjunctiva, and

mucous membranes of the upper respiratory tract. Prolonged or repeated contact with liquid chlorinated benzenes may cause skin burns.

Systemic—

In contrast to aliphatic halogenated hydrocarbons, the toxicity of chlorinated benzenes generally decreases as the number of substituted chlorine atoms increases. Basically, acute exposure to these compounds may cause drowsiness, incoordination, and unconsciousness. Animal exposures have produced liver damage.

Chronic exposure may result in liver, kidney, and lung damage as indicated by animal experiments.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should consider skin, liver, lung, and kidney.

SPECIAL TESTS

None commonly used. Urinary excretion of 2,5-dichlorophenol may be useful as an index of exposure.

PERSONAL PROTECTIVE METHODS

Barrier creams, protective clothing, and good personal hygiene are good preventive measures. Respirators in areas of vapor concentrations are advised.

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CHLORINATED NAPHTHALENES

DESCRIPTION

$C_{10}H_{8-x}Cl$, the chlorinated naphthalenes, are naphthalenes in which one or more hydrogen atoms have been replaced by chlorine to form wax-like substances, beginning with monochloronaphthalene and going on to the octochlor derivatives. Their physical states vary from mobile liquids to waxy-solids depending on the degree of chlorination.

SYNONYMS

Chloronaphthalenes

POTENTIAL OCCUPATIONAL EXPOSURES

Industrial exposure from individual chlorinated naphthalenes is rarely encountered; rather it usually occurs from mixtures of two or more chlorinated naphthalenes. Due to their stability, thermoplasticity, and nonflammability, these compounds enjoy wide industrial application. These compounds are used in the production of electric condensers, in the insulation of electric cables and wires, as additives to extreme pressure lubricants, as supports for storage batteries, and as a coating in foundry use.

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

Cable coaters	Rubber workers
Condenser impregnators	Solvent workers
Electric equipment makers	Transformer workers
Insecticide workers	Wire coaters
Petroleum refinery workers	Wood preservers
Plasticizer makers	

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are:

Trichloronaphthalene	5.0 mg/m^3	Set I
Tetrachloronaphthalene	2 mg/m^3	Set I
Pentachloronaphthalene	0.5 mg/m^3	Set G
Hexachloronaphthalene	0.2 mg/m^3	Set H

ROUTES OF ENTRY

Inhalation of fumes and percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Chronic exposure to chlorinated naphthalenes can cause chloracne, which consists of simple erythematous eruptions with pustules, papules, and comedones. Cysts may develop due to plugging of the sebaceous gland orifices.

Systemic—

Cases of systemic poisoning are few in number and they may occur without the development of chloracne.

It is believed that chloracne develops from skin contact and inhalation of fumes, while systemic effects result primarily from inhalation of fumes. Symptoms of poisoning may include headaches, fatigue, vertigo, and anorexia. Jaundice may occur from liver damage. Highly chlorinated naphthalenes seem to be more toxic than those chlorinated naphthalenes with a lower degree of substitution.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should be concerned particularly with skin lesions such as chloracne and with liver function.

SPECIAL TESTS

None are in common use.

PERSONAL PROTECTIVE METHODS

Skin contact should be avoided whenever possible. Barrier creams, protective clothing, and good personal hygiene are all good preventive measures. Use of respirators in areas of vapor concentration is advised.

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AROMATIC AMINES

The aromatic amines are aromatic hydrocarbons in which at least one hydrogen atom has been replaced by an amino ($-NH_2$) group. The hydrogen atoms in the amino group may be replaced by aryl or alkyl groups, giving rise to secondary and tertiary amino compounds. The aromatic amines are infrequently formed in nature although they do occur, e.g., anthranilic acid esters in grapes. They are generally synthesized by nitration of the aromatic hydrocarbon with subsequent reduction to the amine; an alternate method is by reaction of ammonia and a chloro- or hydroxy-hydrocarbon. Their most important uses are as intermediates in the manufacture of dyestuffs and pigments; however, they are also used in the chemical, textile, rubber, dyeing, paper, and other industries.

Most of the aromatic amines in the free base form are readily absorbed through the skin in addition to the respiratory route. The amino salts have a lower lipid solubility and, therefore, a lower amount of skin absorption. The two major toxic effects of these compounds are methemoglobinemia and cancer of the urinary tract. Other effects may be hematuria, cystitis, anemia, and skin sensitization.

Several of the aromatic amines have been shown to be carcinogenic in humans or animals or both. Occupational tumors of the bladder were recognized in the dyestuff industry as early as 1895. The most common site of cancer is the bladder, but cancer of the pelvis, ureter, kidney, and urethra do occur. It is thought that bladder cancer results from the presence of an active metabolite(s) of the amino compound in the urine, which acts on the bladder epithelium. Several of these metabolites have been identified and have been shown to have carcinogenic properties by implantation in mouse bladders. Man and the dog seem to be more sus-

ceptible to bladder tumors, suggesting a similarity in metabolism of the aromatic amino compounds.

The minimum exposure which produces cancer is not known. There are documented cases of tumors with exposures of less than one year; however, the latent period from first exposure to the development of tumors is usually long and ranges from 4 to over 40 years, with a mean of about 20 years. Bladder tumors are also relatively common in unexposed populations, and the incidence is considerably increased in heavy smokers. They are more common in older age males. It is unknown whether smoking plus exposure to a bladder carcinogen would be synergistic, but this seems possible, e.g., asbestos and smoking.

Clinically, occupationally induced bladder tumors are indistinguishable from those found in the general population; however, they generally occur at an earlier age than usual. These tumors may range from the extremes of benign papillomas to infiltrating carcinomas. Severe or fatal complications which may arise from papillomas are local spreading tumors, severe hemorrhage, and infection of the bladder and kidney.

Hematuria often does not appear until the tumor(s) is inoperable. Micro-examination of the urine is not specific, but routine cystoscopy is a reliable indicator of tumors at an early stage. Exfoliative cytology of urinary sediment using the stained smear method of Papanicolau permits early differentiation of malignant neoplasms and benign papillomas from normal tissue. Those individuals who give a positive test should be examined by cystoscopy and followed indefinitely. Renal pelvis, ureteric, and urethral tumors can also be detected by cytodiagnosis.

Because there may be significant skin absorption of the aromatic amines, protective clothing and polyvinyl chloride or rubber gloves should be worn, and there should be adequate wash and change facilities. Workers exposed to carcinogens should have a complete change of work clothes in addition to protective clothing. The recommended means of control of carcinogenic compounds is by engineering methods aimed at zero exposure levels and a program of periodic medical surveillance.

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2-ACETYLAMINOFLUORENE

DESCRIPTION

$CCH_2C_6H_4C_6H_3NHCOCH_3$, 2-Acetylaminofluorene, is a tan crystalline solid.

SYNONYMS

2-acetaminofluorene, N-acetylaminophenathrene, N-2-fluorenylacetamide.

POTENTIAL OCCUPATIONAL EXPOSURES

Very little 2-acetylaminofluorene is produced. It is used primarily for cancer research purposes. It was patented as a pesticide, but was never used for this purpose. Thus, occupations in which exposure may occur are those in areas of research.

PERMISSIBLE EXPOSURE LIMITS

2-Acetylaminofluorene is included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Probably by inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

Unknown.

Systemic—

2-Acetylaminofluorene's carcinogenic activity was first discovered in rats in which it produced nodular hyperplasia and cancer consistently in the bladder, kidney, pelvis, liver, and pancreas by ingestion. Later feeding experiments in dogs demonstrated bladder and liver tumors. Guinea pigs appear resistant to its carcinogenic effects. No human effects have been reported.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include history of other exposure to carcinogens, smoking history, family history, alcohol, and medications. The skin, respiratory tract, kidney, bladder, and liver should be evaluated for possible effects. Sputum and bladder cytology should be performed. Fetal effects may occur.

The scope and frequency of medical surveillance examinations can be related to the hazard, which probably is greater among research chemists or those involved in animal inhalation studies.

SPECIAL TESTS

None in common use, although urinary metabolites are known.

PERSONAL PROTECTIVE METHODS

Personal protective methods are designed to supplement engineering controls and to prevent all skin or inhalation exposure.

Full body protective clothing and gloves may be required. Those employed in handling operations should be provided with fullface, supplied air respirators of continuous flow or pressure demand type. On exit from a regulated area, employees should shower and change into

street clothes; leaving their protective clothing and equipment at the point of exit to be placed in impervious containers at the end of the work shift for decontamination or disposal. Effective methods should be used for decontamination and changing of clothes and gloves.

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AMINODIPHENYL

DESCRIPTION

$C_6H_5H_6H_4NH_2NH_2$, 4-aminodiphenyl, is a yellowish brown crystal.

SYNONYMS

Biphenyline, p-phenylaniline, xenylamine, 4-aminobiphenyl, 4-biphenylamine, p-aminobiphenyl, p-aminodiphenyl, p-biphenylamine.

POTENTIAL OCCUPATIONAL EXPOSURES

It is no longer manufactured commercially and is only used for research purposes. 4-Aminodiphenyl was formerly used as a rubber antioxidant and as a dye intermediate.

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

Diphenylamine workers
Research workers

PERMISSIBLE EXPOSURE LIMITS

4-Aminodiphenyl is included in the Federal standards for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

None reported.

Systemic—

4-Aminodiphenyl is a known human bladder carcinogen. An exposure of only 133 days has been reported to have ultimately resulted in a bladder tumor. The latent period is generally from 15 to 35 years. Acute exposure produces headaches, lethargy, cyanosis, urinary burning, and hematuria. Cystoscopy reveals diffuse hyperemia, edema, and frank slough.

MEDICAL SURVEILLANCE

Placement and periodic examinations should include an evaluation of exposure to other carcinogens; use of alcohol, smoking, and medications; and family history. Special attention should be given on a regular basis to urine sediment and cytology. If red cells or positive smears are seen, cystoscopy should be done at once. The general health of exposed persons should also be evaluated in periodic examinations.

SPECIAL TESTS

None commonly used. One urinary metabolite is 3-amino-4-hydroxydiphenyl.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all skin or respiratory contact. Full body protective clothing and gloves should be used by those employed in handling operations. Full-face, supplied air respirators of continuous flow or pressure demand type should also be used. On exit from a regulated area, employees should shower and change into street clothes, leaving their clothing and equipment at the point of exit to be placed in impervious containers at the end of the work shift for decontamination or disposal. Effective methods should be used to clean and decontaminate gloves and clothing.

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ANILINE

DESCRIPTION

$C_6H_5NH_2$, aniline, is a clear, colorless, oily liquid with a characteristic odor.

SYNONYMS

Aminobenzene, phenylamine, aniline oil, aminophen, arylamine.

POTENTIAL OCCUPATIONAL EXPOSURES

Aniline is widely used as an intermediate in the synthesis of dyestuffs. It is also used in the manufacture of rubber accelerators and antioxidants, pharmaceuticals, marking inks, tetryl, optical whitening agents, photographic developers, resins, varnishes, perfumes, shoe polishes, and many organic chemicals.

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

Acetanilide workers
Bromide makers
Coal tar workers
Disinfectant makers
Dye workers
Ink makers
Leather workers
Lithographers
Nitraniline workers
Perfume makers
Photographic chemical makers
Plastic workers
Printers
Rocket fuel makers
Rubber workers
Tetryl makers
Varnish workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (19 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapors; percutaneous absorption of liquid and vapor.

HARMFUL EFFECTS

Local—

Liquid aniline is mildly irritating to the eyes and may cause corneal damage.

Systemic—

Absorption of aniline, whether from inhalation of the vapor or from skin absorption of the liquid, causes anoxia due to the formation of methemoglobin. Moderate exposure may cause only cyanosis. As oxygen deficiency increases, the cyanosis may be associated with headache, weakness, irritability, drowsiness, dyspnea, and unconsciousness. If treatment is not given promptly, death can occur. The development of intravascular hemolysis and anemia due to aniline-induced methemoglobinemia has been postulated, but neither is observed often in industrial practice, despite careful study of numerous cases.

MEDICAL SURVEILLANCE

Preplacement and periodic physical examinations should be performed on all employees working in aniline exposure areas. These should include a work history to elicit information on all past exposures to aniline, other aromatic amines, and nitro compounds known to cause chemical cyanosis, and the clinical history of any occurrence of chemical cyanosis; a personal history to elicit alcohol drinking habits; and general physical examination with particular reference to the cardiovascular system. Persons with impaired cardiovascular status may be at greater risk from the consequences of chemical cyanosis. A preplacement complete blood count and methemoglobin estimation should be performed as baseline levels, also follow-up studies including periodic blood counts and hematocrits.

SPECIAL TESTS

Methemoglobin levels, and other abnormal hemoglobins, and/or

urine para-aminophenols, and other aniline metabolites,have been used for biologic monitoring for occupational aniline exposure.

PERSONAL PROTECTIVE METHODS

In areas of vapor concentration, the use of respirators alone is not sufficient; skin protection by protective clothing should be provided even though there is no skin contact with liquid aniline. Butyl rubber protective clothing is reportedly superior to other materials. In severe exposure situations, complete body protection has been employed, consisting of air-conditioned suit with air supplied helmet and cape. Personal hygiene practices including prompt removal of clothing which has absorbed aniline, thorough showering after work and before changing to street clothes, and clean working clothes daily are essential.

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BENZIDINE AND ITS SALTS

DESCRIPTION

$NH_2C_6H_4C_6H_4NH_2$, benzidine, is a crystalline solid with a significant vapor pressure. The salts are less volatile, but tend to be dusty.

SYNONYMS

4,4'-Biphenyldiamine, para-diaminodiphenyl, 4,4'-diaminobiphenyl, 4,4'-diphenylenediamine, benzidine base.

POTENTIAL OCCUPATIONAL EXPOSURES

Benzidine is used primarily in the manufacture of azo dyestuffs; there are over 250 of these produced. Other uses, including some which may have been discontinued, are in the rubber industry as a hardener, in the manufacture of plastic films, for detection of occult blood in feces, urine, and body fluids, in the detection of H_2O_2 in milk, in the production of security paper, and as a laboratory reagent in determining HCN, sulfate, nicotine, and certain sugars. No substitute has been found for its use in dyes.

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

- Biochemists
- Dye workers
- Medical laboratory workers
- Organic chemical synthesizers
- Plastic workers
- Rubber workers
- Wood chemists

PERMISSIBLE EXPOSURE LIMITS

Benzidine and its salts are included in a Federal standard for carcinogens; all contact with them should be avoided.

ROUTES OF ENTRY

Inhalation, percutaneous absorption, and ingestion of dust.

HARMFUL EFFECTS

Local—

Contact dermatitis due to primary irritation or sensitization has been reported.

Systemic—

Benzidine is a known human urinary tract carcinogen with an average latent period of 16 years. The first symptoms of bladder cancer usually are hematuria, frequency of urination, or pain.

MEDICAL SURVEILLANCE

Placement and periodic examinations should include an evaluation of exposure to other carcinogens; use of alcohol, smoking, and medications; and family history. Special attention should be given on a regular basis to urine sediment and cytology. If red cells or positive smears are seen, cystoscopy should be done at once. The general health of exposed persons should also be evaluated in periodic examinations.

SPECIAL TESTS

None in common use although several metabolites are known.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all skin or respiratory contact. Full body protective clothing and gloves should also be used. On exit from a regulated area employees should shower and change into street clothes, leaving their protective clothing and equipment at the point of exit to be placed in impervious containers at the end of the work shift for decontamination or disposal. Effective methods should be used to clean and decontaminate gloves and clothing.

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3,3'-DICHLOROBENZIDINE AND ITS SALTS

DESCRIPTION

$C_6H_3ClNH_2C_6H_3ClNH_2$, 3,3'-dichlorobenzidine, is a gray or purple crystalline solid.

SYNONYMS

4,4'-Diamino-3,3'-dichlorobiphenyl, 3,3'-dichlorobiphenyl-4,4'-diamine, 3,3'-dichloro-4,4-biphenyldiamine.

POTENTIAL OCCUPATIONAL EXPOSURES

The major uses of dichlorobenzidine are in the manufacture of pigments for printing ink, textiles, plastics, and crayons and as a curing agent for solid urethane plastics. There are no substitutes for many of its uses.

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

Pigment makers
Polyurethane workers

PERMISSIBLE EXPOSURE LIMITS

3,3'-Dichlorobenzidine and its salts are included in a Federal standard for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Inhalation and probably percutaneous absorption.

HARMFUL EFFECTS

Local—

May cause allergic skin reactions.

Systemic—

3,3'-Dichlorobenzidine was shown to be a potent carcinogen in rats and mice in feeding and injection experiments, but no bladder tumors were produced. However, no cases of human tumors have been observed in epidemiologic studies of exposure to the pure compound.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include history of exposure to other carcinogens, smoking, alcohol, medication, and family history. The skin, lung, kidney, bladder, and liver should be evaluated; sputum or urinary cytology may be helpful.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all skin or respiratory contact. Full body protective clothing and gloves should be used by those employed in handling operations. Fullface supplied air respirators of continuous flow or pressure demand type should also be used. On exit from a regulated area, employees should shower and change into street clothes, leaving their protective clothing and equipment at the point of exit to be placed in impervious containers at the end of the work shift for decontamination or disposal. Effective methods should be used to clean and decontaminate gloves and clothing.

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4-DIMETHYLAMINOAZOBENZENE

DESCRIPTION

$C_6H_5NNC_6H_4N(CH_3)_2$, 4-dimethylaminoazobenzene, is a flaky yellow crystal.

SYNONYMS

Aniline-N,N-dimethyl-p(phenylazo), benzeneazo dimethylaniline, fat yellow, oil yellow, butter yellow, methyl yellow.

POTENTIAL OCCUPATIONAL EXPOSURES

4-Dimethylaminoazobenzene is only used for research purposes. It was formerly used as a dye,but has been substituted by diethylaminoazobenzene. It was also formerly used for coloring margarine and butter.

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

Research workers

PERMISSIBLE EXPOSURE LIMITS

4-Dimethylaminoazobenzene is included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Probably inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

Unknown.

Systemic—

Cancer of the liver has been produced in rats and mice in feeding experiments. No human effects have been reported.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include a history of exposure to other carcinogens; use of alcohol, smoking, and medications; and family history. Special attention should be given to liver size and liver function tests.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all contact with skin and the respiratory tract. Protective clothing and gloves should be provided, and also appropriate type dust or supplied air respirators. On exit from a regulated area, employees should shower and change into street clothes, leaving their clothes at the point of exit, to be placed in impervious containers at the end of the work shift for decontamination or disposal.

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4,4'-METHYLENEBIS(2-CHLOROANILINE)

DESCRIPTION

$CH_2(C_6H_4ClNH_2)_2$, 4,4'-methylenebis (2-chloroaniline) or moca, is a yellow to light gray-tan pellet and is also available in liquid form.

SYNONYMS

Moca, 4,4'-diamino-3,3'-dichlorodiphenylmethane, 4,4'-methylene-2,2-dichloroaniline.

POTENTIAL OCCUPATIONAL EXPOSURES

Moca is primarily used in the production of solid elastomeric parts. Other uses are as a curing agent for epoxy resins and in the manufacture of cross-linked urethane foams used in automobile seats and safety padded dashboards; it is also used in the manufacture of gun mounts, jet engine turbine blades, radar systems, and components in home appliances.

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

Elastomer makers
Epoxy resin workers
Polyurethane foam workers

PERMISSIBLE EXPOSURE LIMITS

Moca is included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Inhalation; percutaneous absorption.

HARMFUL EFFECTS

Local—

None reported.

Systemic—

Feeding experiments with rats produced liver and lung cancer. No tumors were found in experiments with dogs. No tumors or other ill-

ness have been reported from chronic exposure in man except a mild cystitis which subsided within a week.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include a history of exposure to other carcinogens, alcohol and smoking habits, use of medications, and family history. Special attention should be given to liver size and function and to any changes in lung symptoms or X-rays.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all contact with skin and the respiratory tract. Protective clothing and gloves should be provided, and also appropriate type dust or supplied air respirators. On exit from a regulated area, employees should shower and change into street clothes, leaving the protective clothing and equipment at the point of exit, to be placed in impervious containers at the end of the work shift for decontamination or disposal.

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alpha-NAPHTHYLAMINE

DESCRIPTION

$C_{10}H_7NH_2$, alpha-naphthylamine, exists as white needlelike crystals which turn red on exposure to air.

SYNONYMS

1-Aminonaphthalene, naphthalidam, naphthalidine.

POTENTIAL OCCUPATIONAL EXPOSURES

alpha-Naphthylamine is used in the manufacture of dyes, condensation colors, and rubber, and in the synthesis of many chemicals such as alpha-naphthol, sodium naphthionate o-naphthionic acid, Nevile's acid, Winther's acid, sulfonated naphthylamines, alpha-naphthylthiouria (a rodenticide), and N-phenyl-alpha-naphthylamine.

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

Dye makers
Chemical synthesizers
Rubber workers

PERMISSIBLE EXPOSURE LIMITS

alpha-Naphthylamine is included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

None reported.

Systemic—

It has not been established whether alpha-naphthylamine is a human carcinogen per se or is associated with an excess of bladder cancer due to its beta-naphthylamine content. Workers exposed to alpha-naphthylamine developed bladder tumors. The mean latent period was 22 years compared to 16 years for beta-naphthylamine. One animal experiment demonstrated papillomata, but these results have never been confirmed.

MEDICAL SURVEILLANCE

Placement and periodic examinations should include an evaluation of exposure to other carcinogens; use of alcohol, smoking, and medications; and family history. Special attention should be given on a regular basis to urine sediment and cytology. If red cells or positive smears are seen, cystoscopy should be done at once. The general health of exposed persons should also be evaluated in periodic examinations.

SPECIAL TESTS

None commonly used. Some metabolites are known.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all skin or respiratory contact. Full body protective clothing and gloves should be used by those employed in handling operations. Full-face, supplied air respirators of continuous flow or pressure demand type should also be used. On exit from a regulated area, employees should shower and change into street clothes, leaving their protective clothing and equipment at the point of exit to be placed in impervious containers at the end of the work shift for decontamination or disposal. Effective methods should be used to clean and decontaminate gloves and clothing. Showers should be taken prior to dressing in street clothes.

beta-NAPHTHYLAMINE

DESCRIPTION

$C_{10}H_7NH_2$, beta-Naphthylamine, is a white to reddish crystal.

SYNONYMS

2-Naphthylamine, 2-aminonaphthalene.

POTENTIAL OCCUPATIONAL EXPOSURES

beta-Naphthylamine is presently used only for research purposes. It is present as an impurity in alpha-naphthylamine. It was widely used in the manufacture of dyestuffs, as an antioxidant for rubber, and in rubber coated cables.

A partial list of occupations in which exposure may occur includes:
beta-Naphthylamine workers
Research workers

PERMISSIBLE EXPOSURE LIMITS

beta-Naphthylamine is included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

beta-Naphthylamine is mildly irritating to the skin and has produced contact dermatitis.

Systemic—

beta-Naphthylamine is a known human bladder carcinogen with a latent period of about 16 years. The symptoms are frequent urination, dysuria, and hematuria. Acute poisoning leads to methemoglobinemia or acute hemorrhagic cystitis.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include an evaluation of exposure to other carcinogens; use of alcohol, smoking, and medications; and family history. Special attention should be given on a regular basis to urine sediment and cytology. If red cells or positive smears are seen, cystoscopy should be done at once. The general health of exposed persons should also be evaluated in periodic examinations.

SPECIAL TESTS

None in common use; some metabolites are known.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all skin or respiratory contact. Full body protective clothing and gloves should be used by those employed in handling operations. Full-face, supplied air respirators of continuous flow or pressure demand type should also be used. On exit from a regulated area, employees should shower and change into street clothes, leaving their clothing and equipment at the point of exit to be placed in impervious containers at the end of the work shift for decontamination or disposal. Effective methods should be used to clean and decontaminate gloves and clothing. Showers should be taken prior to dressing in street clothes.

NITRO COMPOUNDS

The aliphatic nitro compounds are characterized by the $-C-NO_2$ structure. Closely related chemicals are the alkyl nitrites ($-C-O-NO$), alkyl nitrates ($-C-O-NO_2$), and chloronitroparaffins (e.g., CCl_2NO_2). All differ significantly in their chemical and toxicological characteristics.

The aromatic nitro compounds, in which a nitro group is substituted directly on a benzene ring, are a more homogeneous group. Most of them can be produced by nitration of the aromatic. They are widely used, especially in explosive and dyestuff manufacture. Aromatic nitro compounds rapidly penetrate the skin, and this may be the major route of absorption. In acute exposures, they produce cyanosis and in chronic exposures, anemia. Local irritation and liver damage are also common. A portion of the absorbed dose is excreted in the urine unchanged; however the major portion is first metabolized to aminophenol derivatives before excretion. Many colorimetric tests are available for detecting the parent compounds or metabolites in the urine.

Other clinical tests which may be of value are urinalysis, blood chemistry, and blood analysis for anemia, methemoglobin, and Heinz bodies. Physical examinations are an important aspect of prevention. Individuals with cardiovascular, renal, hepatic, or respiratory diseases, blood dyscrasia, allergies, or chronic alcoholism may be at increased risk from exposure to aromatic nitro compounds.

Work practices should include protective clothing made of butyl rubber and emphasis on personal hygiene.

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DINITROBENZENE

DESCRIPTION

$C_6H_4(NO_2)_2$, dinitrobenzene, may exist in three isomers; the meta-form is the most widely used.

SYNONYMS

Dinitrobenzol.

POTENTIAL OCCUPATIONAL EXPOSURES

Dinitrobenzene is used in the synthesis of dyestuffs, dyestuff intermediates, and explosives and in celluloid production.

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

- Celluloid makers
- Dye makers
- Explosive workers
- Organic chemical synthesizers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for all isomers of dinitrobenzene is 1 mg/m³.

ROUTES OF ENTRY

Inhalation and percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Exposure to dinitrobenzene may produce yellowish coloration of the skin, eyes, and hair.

Systemic—

Exposure to any isomer of dinitrobenzene may produce methemoglobinemia, symptoms of which are headache, irritability, dizziness, weakness, nausea, vomiting, dyspnea, drowsiness, and unconsciousness. If treatment is not given promptly, death may occur. Consuming alcohol, exposure to sunlight, or hot baths may make symptoms worse. Dinitrobenzene may also cause a bitter almond taste or burning sensation in the mouth, dry throat, and thirst. Reduced vision may occur. In addition liver damage, hearing loss, and ringing of the ears may be produced. Repeated or prolonged exposure may cause anemia.

MEDICAL SURVEILLANCE

Preemployment and periodic examinations should be concerned particularly with a history of blood dyscrasias, reactions to medications, alcohol intake, eye disease, and skin and cardiovascular status. Liver and renal functions should be evaluated periodically as well as blood and general health.

SPECIAL TESTS

Methemoglobin levels should be followed until normal in all cases of suspected cyanosis. Dinitrobenzene can be determined in the urine; levels greater than 25 mg/liter may indicate significant absorption.

PERSONAL PROTECTIVE METHODS

Dinitrobenzene is readily absorbed through intact skin and its vapors are highly toxic. Protective clothing impervious to the liquid should be worn in areas where the likelihood of splash or spill exists. When splash or spill occurs on ordinary work clothes, they should be removed immediately and the area washed thoroughly. In areas of elevated vapor concentrations fullface masks with organic vapor canisters or air supplied respirators with fullface piece should be used. Daily changes of work clothing and mandatory showering at the end of each shift before changing to street clothes should be enforced.

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DINITRO-O-CRESOL

DESCRIPTION

$CH_3C_6H_2(NO_2)_2OH$, dinitro-o-cresol, exists in 9 isomeric forms of which 3,5-dinitro-o-cresol is the most important commercially. It is a yellow crystalline solid.

SYNONYMS

DNOC; 4,6-Dinitro-o-cresol is also known as 3,5-dinitro-o-cresol, 2-methyl-4,6-dinitrophenol, 3,5-dinitro-2-hydroxytoluene.

POTENTIAL OCCUPATIONAL EXPOSURES

DNOC is widely used in agriculture as a herbicide and pesticide; it is also used in the dyestuff industry.

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

Dye makers
Herbicide workers
Pesticide workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for all isomers of DNOC is 0.2 mg/m³.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

None reported except for staining of skin and hair.

Systemic—

DNOC blocks the formation of high energy phosphate compounds, and the energy from oxidative metabolism is liberated as heat. Early symptoms of intoxication by inhalation or skin absorption are elevation of the basal metabolic rate and rise in temperature accompanied by fatigue, excessive sweating, unusual thirst, and loss of weight. The clinical picture resembles in part a thyroid crisis. Weakness, fatigue, increased respiratory rate, tachycardia, and fever may lead to rapid deterioration and death. Bilateral cataracts have been seen following oral ingestion for therapeutic purposes.

These have not been seen during industrial or agricultural use.

MEDICAL SURVEILLANCE

Consider eyes, thyroid, and cardiovascular system, as well as general health.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

Since dinitro-o-cresol is used extensively in agriculture as well as

industry, worker education to the toxic properties of the chemical are necessary. Where there is a possibility of skin contamination or vapor inhalation, full protection should be provided. Impervious protective clothing and fullface masks with organic vapor canisters or air supplied respirators are advised. A clean set of work clothes daily, and showers following each shift before change to street clothes are essential.

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DINITROPHENOL

DESCRIPTION

There are six isomers of dinitrophenol of which 2,4-dinitrophenol is the most important industrially. It is an explosive, yellow crystalline solid.

SYNONYMS

DNP.

POTENTIAL OCCUPATIONAL EXPOSURES

2,4-DNP is used in the manufacturing of dyestuff intermediates, wood preservatives, pesticides, herbicides, explosives, chemical indicators, photograph developers, and also in chemical synthesis.

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

- Chemical indicator makers
- Dye makers
- Explosive workers
- Herbicide workers
- Organic chemical synthesizers
- Photographic developer makers
- Wood preservative workers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for DNP. A useful guideline of 0.2 mg/m^3 is based on data for dinitro-o-cresol.

ROUTES OF ENTRY

Percutaneous absorption and inhalation of dust and vapors.

HARMFUL EFFECTS

Local—

DNP causes yellow staining of exposed skin. Dermatitis may be due to either primary irritation or allergic sensitivity.

Systemic—

The isomers differ in their toxic effects. In general, DNP disrupts oxidative phosphorylation (as in the case of DNOC) which results in increased metabolism, oxygen consumption, and heat production. Acute intoxication is characterized by sudden onset of fatigue, thirst, sweating, and oppression of the chest. There is rapid respiration, tachycardia, and a rise in body temperature. In less severe poisoning, the symptoms are nausea, vomiting, anorexia, weakness, dizziness, vertigo, headache, and sweating. The liver may be sensitive to pressure, and there may also be jaundice. DNP poisoning is more severe in warm environments. If not fatal, the effects are rapidly and completely reversible. Chronic exposure results in kidney and liver damage and cataract formation. Occasional hypersensitivity reactions, e.g., neutropenia, skin rashes, peripheral neuritis, have been seen after oral use.

MEDICAL SURVEILLANCE

Consider skin, eyes, thyroid, blood, central nervous system, liver and kidney function, as well as general health in preplacement and periodic examinations.

SPECIAL TESTS

Can be measured in urine as such or as an aminophenol derivative.

PERSONAL PROTECTIVE METHODS

Because of its wide use in agriculture, lumbering, photography, as well as in the petrochemical industry, worker education to the toxic properties of dinitrophenol are important. Impervious protective clothing, fullface masks with organic vapor canisters or air supplied respirators are necessary in areas of high concentration of dust or vapor. Spills and splashes that contaminate clothing require the worker to immediately change clothes and wash the area thoroughly. Workers should have clean work clothes on every shift and should be required to shower prior to changing to street clothing.

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DINITROTOLUENE

DESCRIPTION

Six isomers of DNT exist, the most important being 2,4-dinitro-1-toluene.

SYNONYMS

Dinitrotoluol, DNT.

POTENTIAL OCCUPATIONAL EXPOSURES

DNT is used in the manufacture of explosives and dyes in organic synthesis, e.g., trinitrotoluene.

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

Dye makers
Explosive workers
Organic chemical synthesizers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1.5 mg/m^3.

ROUTES OF ENTRY

Inhalation of vapor and percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

None.

Systemic—

The effects from exposure to dinitrotoluene are caused by its capacity to produce anoxia due to the formation of methemoglobin. Cyanosis may occur with headache, irritability, dizziness, weakness, nausea, vomiting, dyspnea, drowsiness, and unconsciousness. If treatment is not given promptly, death may occur. The onset of symptoms may be delayed. The ingestion of alcohol may cause increased susceptibility. Repeated or prolonged exposure may cause anemia.

MEDICAL SURVEILLANCE

Preemployment and periodic examinations should be concerned particularly with a history of blood dyscrasias, reactions to medications, alcohol intake, eye disease, skin, and cardiovascular status. Liver and renal functions should be evaluated periodically as well as blood and general health.

SPECIAL TESTS

None commonly used. Forms a blue color with alcoholic NaOH.

PERSONAL PROTECTIVE METHODS

Liquid soaked clothing should be immediately removed and the skin area washed thoroughly. Impervious protective clothing should be provided if skin exposure to liquid is anticipated. In areas of elevated vapor concentration, fullface masks with organic vapor canisters or air-supplied respirators should be required.

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NITROBENZENE

DESCRIPTION

$C_6H_5NO_2$, nitrobenzene, is a pale yellow liquid whose odor resembles bitter almonds.

SYNONYMS

Nitrobenzol, oil of mirbane, oil of bitter almonds.

POTENTIAL OCCUPATIONAL EXPOSURES

Nitrobenzene is used in the manufacture of explosives and aniline dyes and as a solvent and intermediate. It is also used in shoe and floor polishes, leather dressings, and paint solvents, and to mask other unpleasant odors. Substitution reactions with nitrobenzene are used to form meta-derivatives.

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

- Aniline dye makers
- Explosive makers
- Organic chemical synthesizers
- Paint makers
- Polish makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1 ppm (5 mg/m^3).

ROUTES OF ENTRY

Inhalation and percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Nitrobenzene may cause irritation of the eyes.

Systemic—

There is a latent period of 1-4 hours before signs and symptoms appear. Nitrobenzene affects the central nervous system producing fatigue, headache, vertigo, vomiting, general weakness, and in some cases severe depression, unconsciousness, and coma. Nitrobenzene is a powerful methemoglobin former; cyanosis appears when methemoglobin reaches 15%. Sulfhemoglobin formation may also contribute to nitrobenzene toxicity. Chronic exposure may lead to spleen and liver damage, jaundice, liver impairments, and hemolytic icterus. Anemia and Heinz bodies in the red blood cells have also been observed. Alcohol ingestion may increase the toxic effects.

MEDICAL SURVEILLANCE

Preemployment and periodic examinations should be concerned particularly with a history of dyscrasias, reactions to medications, alcohol intake, eye disease, skin, and cardiovascular status. Liver and renal functions should be evaluated periodically, as well as blood and general health.

SPECIAL TESTS

Follow methemoglobin levels until normal in all cases of suspected cyanosis. The metabolites in urine, p-nitro and p-amino phenol, can be used as an evidence of exposure.

PERSONAL PROTECTIVE METHODS

Impervious protective clothing should be worn in areas where risk of splash or spill exists. When splashed or spilled on ordinary work clothes, the clothes should be removed at once and the skin area washed thoroughly. In areas of vapor concentration fullface masks with organic vapor canisters or air supplied respirators should be used. Clean work clothing should be supplied daily, and showering made mandatory after each shift before workers change to street clothes.

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Salmowa, J., J. Piotrowski, and U. Neuhorn. 1963. Evaluation of exposure to nitrobenzene. Absorption of nitrobenzene vapor through lungs and excretion of p-nitrophenol in urine. Brit. J. Ind. Med. 20:41.

4-NITROBIPHENYL

DESCRIPTION

$C_6H_5C_6H_4NO_2$, 4-nitrobiphenyl, exists as yellow plates or needles.

SYNONYMS

4-Nitrodiphenyl, p-nitrobiphenyl, p-nitrodiphenyl, PNB.

POTENTIAL OCCUPATIONAL EXPOSURES

4-Nitrobiphenyl was formerly used in the synthesis of 4-aminodiphenyl. It is presently used only for research purposes; there are no commercial uses.

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

PERMISSIBLE EXPOSURE LIMITS

4-Nitrobiphenyl was included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

None reported.

Systemic—

4-Nitrobiphenyl is considered to be a human carcinogen. This is based on the evidence that it will induce bladder tumors in dogs and that human cases of bladder cancer were reported from a mixed exposure to 4-aminodiphenyl and 4-nitrobiphenyl. These human cases were attributed to 4-aminodiphenyl because the information available at the time showed that it produced bladder tumors in dogs. 4-Amino biphenyl may be a metabolite.

MEDICAL SURVEILLANCE

Placement and periodic examinations should include an evaluation of exposure to other carcinogens, as well as an evaluation of smoking, of use of alcohol and medications, and of family history. Special attention should be given on a regular basis to urine sediment and cytology. If red cells or positive smears are seen, cystoscopy should be done at once. The general health of exposed persons should also be evaluated in periodic examinations.

SPECIAL TESTS

None commonly used. Can probably be determined in the urine as a metabolite.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all skin or respiratory contact. Full body protective clothing and gloves should be used by those employed in handling operations. Full-face, supplied air respirators of continuous flow or pressure demand type should also be used. On exit from a regulated area, employees should shower and change into street clothes, leaving their protective clothing and equipment at the point of exit to be placed in impervious containers at the end of the work-shift for decontamination or disposal. Effective methods should be used to clean and decontaminate gloves and clothing.

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NITROGLYCERIN and ETHYLENE GLYCOL DINITRATE

DESCRIPTION

$C_3H_5(ONO_2)_3$, nitroglycerin.

$O_2NOCH_2OCH_2ONO_2$, ethylene glycol dinitrate.

Both are oily, yellow liquids and are highly explosive. They may be detonated by mechanical shock, heat, or spontaneous chemical reaction.

SYNONYMS

Nitroglycerin: nitroglycerol, glyceryl trinitrate, trinitroglycerol, glonoin, trinitrin.

Ethylene glycol dinitrate: nitroglycol, glycol dinitrate, ethylene dinitrate, EGDN.

POTENTIAL OCCUPATIONAL EXPOSURES

Although ethylene glycol dinitrate is an explosive in itself, it is primarily used to lower the freezing point of nitroglycerin; together these compounds are the major constituents of commercial dynamite, cordite, and blasting gelatin. Occupational exposure generally involves a mixture of the two compounds. Ethylene glycol dinitrate is 160 times more volatile than nitroglycerin. Nitroglycerin is also used as a pharmaceutical.

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

Drug makers
Explosive makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for nitroglycerin is 0.2 ppm (2 mg/m^3). The standard for ethylene glycol dinitrate and/or nitroglycerin is 0.2 ppm (1 mg/m^3) as a ceiling value, and, at concentrations greater than 0.02 ppm, personal protection may be necessary to avoid headache. These levels should be reduced when the substance is also absorbed percutaneously.

ROUTES OF ENTRY

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

HARMFUL EFFECTS

Local—

None reported.

Systemic—

Exposure to small amounts of ethylene glycol dinitrate and/or nitroglycerin by skin exposure, inhalation, or swallowing may cause severe throbbing headaches. With larger exposure, nausea, vomiting, cyanosis, palpitations of the heart, coma, cessation of breathing, and death may occur. A temporary tolerance to the headache may develop, but this is lost after a few days without exposure. On some occasions a worker may have anginal pains a few days after discontinuing repeated daily exposure.

MEDICAL SURVEILLANCE

Placement and periodic examinations should be concerned with central nervous system, blood, glaucoma, and especially history of alcoholism.

SPECIAL TESTS

None commonly used, but urinary and blood ethylene glycol dinitrate may be determined by gas chromatography.

PERSONAL PROTECTIVE METHODS

Both compounds are readily absorbed through the skin, lungs, and mucous membranes. It is, therefore, essential that adequate skin protection be provided for each worker: impervious clothing where liquids are likely to contaminate and full body clothing where dust creates the problem. All clothing should be discarded at the end of the shift and clean work clothing provided each day. Showers should be taken at the end of each shift and prior to changing to street clothing. In case of spill or splash that contaminates work clothing, the clothes should be changed at once and the skin area washed thoroughly. Masks of the dust type or organic vapor canister type may be necessary in areas of concentration of dust or vapors.

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NITROPARAFFINS

DESCRIPTION

Nitroparaffiins are characterized by a $-C-NO_2$ group and may be either mono- or poly-substituted. Only certain mononitroparaffins are included in this section: nitromethane (CH_3NO_2), nitroethane ($C_2H_5-NO_2$), 1-nitropropane ($C_3H_7-NO_2$), and 2-nitropropane ($CH_3-CH(NO_2)-CH_3$. All of these are colorless liquids. Other mononitroparaffins are not commonly used, and use of the polynitroparaffins is limited almost entirely to fuels and fuel additives.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Nitroparaffins are used as solvents for cellulose esters, vinyl copolymer, and other resins, oils, fats, waxes, and dyes. They are also used in various coating materials such as shellac, synthetic and processed rubber, paint and varnish removers, alkyl resins, and other high polymer coatings, and also in organic synthesis.

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

- Cellulose workers
- Dye makers
- Fat processors
- Organic chemical synthesizers
- Plastic makers
- Resin makers
- Rubber makers
- Stainers
- Wax makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards for these substances are: nitromethane 100 ppm (250 mg/m^3), nitroethane 100 ppm (310 mg/m^3), 1-nitropropane 25 ppm (90 mg/m^3), and 2-nitropropane 25 ppm (90 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

The nitroparaffins are irritants to the eyes and upper respiratory tract. There may be slight skin irritation due to solvent drying of skin.

Systemic—

Only one report of occupational illness from nitroparaffins has been reported. The workers were exposed to 20-45 ppm of 2-nitropropane and complained of anorexia, nausea, vomiting diarrhea, and occipital headache. Animal experiments indicate that high concentrations of nitroparaffins may produce light narcosis and central nervous system irritation. The lethal dose is generally lower than that producing significant narcosis. Liver and kidney damage have been observed in animals at lethal concentrations. Nitroparaffins release nitrate in vivo; however, methemoglobinemia and Heinz bodies have only been observed with 2-nitropropane. Experimental evidence also indicates that the toxicity of nitroparaffins increases with the size of the molecule.

MEDICAL SURVEILLANCE

Based on animal data, preplacement and periodic examination should consider respiratory and central nervous system effects as well as liver and kidney function.

SPECIAL TESTS

None commonly used. In the case of 2-nitropropane, Heinz bodies and methemoglobin levels would be of interest.

PERSONAL PROTECTIVE METHODS

Barrier creams or gloves to protect exposed skin and, where vapor concentrations are excessive, fullface mask with organic vapor canister or air supplied respirators are advised.

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NITROPHENOL

DESCRIPTION

There are three isomers of nitrophenol $NO_2C_6H_4OH$. The meta-

form is produced from m-nitroaniline, and the ortho-and para-isomers are produced by nitration of phenol. They are colorless to slightly yellowish crystals with an aromatic to sweetish odor.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Nitrophenols are used in the synthesis of dyestuffs and other intermediates and as a chemical indicator.

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

Chemical indicator makers
Organic chemical synthesizers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for nitrophenol.

ROUTES OF ENTRY

Inhalation and percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Unknown.

Systemic—

There is very little information available on the toxicity for humans of nitrophenols. Animal experiments have shown central and peripheral vagus stimulation, CNS depression, methemoglobinemia, and dyspnea. The p-isomer is the most toxic.

MEDICAL SURVEILLANCE

Based on animal studies, individuals with cardiovascular, renal, or pulmonary disease and those with anemia are probably more subject to poisoning by nitrophenol. Liver and renal function and blood should be evaluated in placement or periodic examinations.

SPECIAL TESTS

None commonly used. Nitrophenol is excreted rapidly in the urine as a conjugate. It may also be present as a metabolite of parathion.

PERSONAL PROTECTIVE METHODS

Nitrophenols are readily absorbed through intact skin and by inhalation; full body protective clothing and appropriate type organic vapor canisters in areas of concentrations of dust or vapors should be provided. Spills on work clothing necessitate immediate clothing change and thorough washing of the skin area. Clean work clothes should be supplied daily; showers should be taken at the end of each shift prior to changing to street clothes.

PICRIC ACID

DESCRIPTION

$C_6H_2(NO_2)_3OH$, picric acid, is a pale yellow, odorless, intensely bitter crystal which is explosive upon rapid heating or mechanical shock.

SYNONYMS

Picronitric acid, trinitrophenol, nitroxanthic acid, carbazotic acid, phenol trinitrate.

POTENTIAL OCCUPATIONAL EXPOSURES

Picric acid is used in the manufacture of explosives, rocket fuels, fireworks, colored glass, matches, electric batteries, and disinfectants. It is also used in the pharmaceutical and leather industries, and in dyes, copper and steel etching, forensic chemistry, histology, textile printing, and photographic emulsions.

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

- Battery makers
- Colored glass makers
- Copper etchers
- Disinfectant makers
- Drug makers
- Dye makers
- Explosive makers
- Forsenic chemists
- Histology technicians
- Matchmakers
- Photographic chemical workers
- Tannery workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for picric acid is 0.1 mg/m³.

ROUTES OF ENTRY

Inhalation and ingestion of dust; percutaneous absorption.

HARMFUL EFFECTS

Local—

Picric acid dust or solutions are potent skin sensitizers. In solid form, picric acid is a skin irritant, but in aqueous solution it irritates only hypersensitive skin. The cutaneous lesions which appear usually on exposed areas of the upper extremities consist of dermatitis with erythema, papular, and vesicular eruptions. Desquamation may occur following repeated or prolonged contact. Skin usually turns yellow upon contact, and areas around nose and mouth as well as the hair are most often affected. Dust or fume may cause eye irritation which may be aggravated by sensitization. Corneal injury may occur from exposure to picric acid dust and solutions.

Systemic—

Inhalation of high concentrations of dust by one worker caused temporary coma followed by weakness, myalgia, anuria, and later polyuria. Following ingestion of picric acid, there may be headache, vertigo, nausea, vomiting, diarrhea, yellow coloration of the skin, hema-

turia, and albuminuria. High doses may cause destruction of erythrocytes, hemorrhagic nephritis, and hepatitis. High doses which cause systemic intoxication will color all tissues yellow, including the conjunctiva and aqueous humor, and cause yellow vision.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should focus on skin disorders such as hypersensitivity atopic dermatitis, and liver and kidney function.

SPECIAL TESTS

None commonly used. It is probably excreted as picric and picramic acid in the urine.

PERSONAL PROTECTIVE METHODS

Skin protection by clothing and barrier creams can avoid the irritant and sensitizing action of picric acid. Masks of the dust type will prevent absorption by inhalation. Fullface masks are advisable or combination of chemical goggles with halfmask. Daily change of clean work clothes and showering after each shift before changing to street clothes are mandatory.

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Williams, R. T. 1959. Detoxication Mechanism, 2nd ed. J. Wiley and Sons, New York.

TETRYL

DESCRIPTION

Tetryl is a yellow solid.

SYNONYMS

Trinitrophenylmethylnitramine, nitramine, tetranitromethylaniline, pyrenite, picrylmethylnitramine, picrylnitromethylamine, N-methyl-N-2,4, 6-tetranitroaniline, tetralite.

POTENTIAL OCCUPATIONAL EXPOSURES

Tetryl is used in explosives as an intermediary detonating agent and as a booster charge; it is also used as a chemical indicator.

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

Chemical indicator makers
Explosive makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1.5 mg/m^3.

ROUTES OF ENTRY

Inhalation and skin absorption.

HARMFUL EFFECTS

Local—

Tetryl is a potent sensitizer, and allergic dermatitis is common. Dermatitis first appears on exposed skin areas, but can spread to other parts of the body in fair skinned individuals or those with poor personal hygiene. The severest forms show massive generalized edema with partial obstruction of the trachea due to swelling of the tongue, and these cases require hospitalization. Contact may stain skin and hair yellow or orange. Tetryl is acutely irritating to the mucous membranes of the respiratory tract and the eyes, causing coughing, sneezing, epistaxis, conjunctivitis, and palpebral and periorbital edema.

Systemic—

Tetryl exposure may cause irritability, easy fatigability, malaise, headaches, lassitude, insomnia, nausea, and vomiting. Anemia either of the marrow depression or deficiency type has been observed among tetryl workers. Tetryl exposure has produced liver and kidney damage in animals.

MEDICAL SURVEILLANCE

Preplacement physical examination should give special attention to those individuals with a history of allergy, blood dyscrasias, or skin, liver, or kidney disease. Periodic examinations should be directed primarily to the control of dermatitis and allergic reactions, plus any effects on the respiratory tract, eyes, central nervous system, blood, liver, or kidneys.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Skin protection is necessary by means of protective clothing and gloves. Where significant air concentration of dusts or vapors exist, masks to prevent inhalation are necessary. Daily change to clean work clothes is strongly advised, with showers after each shift mandatory, before dressing in street clothes.

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Norwood, W. D. 1943. Trinitrotoluene (TNT); its effective removal from the skin by a special liquid soap. Ind. Med. 12:206.

TRINITROTOLUENE

DESCRIPTION

TNT exists in 5 isomers; 2,4,6-trinitrotoluene is the most commonly

used. All are crystalline solids in pure form. TNT is a relatively stable high explosive.

SYNONYMS

TNT, sym-trinitrotoluol, methyltrinitrobenzene.

POTENTIAL OCCUPATIONAL EXPOSURES

TNT is used as an explosive, i.e., as a bursting charge in shells, bombs, and mines.

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

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1.5 mg/m^3.

ROUTES OF ENTRY

Inhalation of dust, fume, or vapor; ingestion of dust; percutaneous absorption from dust.

HARMFUL EFFECTS

Local—

Exposure to trinitrotoluene may cause irritation of the eyes, nose, and throat with sneezing, cough, and sore throat. It may cause dermatitis and may stain the skin, hair, and nails a yellowish color.

Systemic—

Numerous fatalities have occurred in workers exposed to TNT from toxic hepatitis or aplastic anemia. TNT exposure may also cause methemoglobinemia with cyanosis, weakness, drowsiness, dyspnea, and unconsciousness. In addition it may cause muscular pains, heart irregularities, renal irritation, cataracts, menstrual irregularities, and peripheral neuritis.

MEDICAL SURVEILLANCE

Placement or periodic examinations should give special considerations to history of allergic reactions, blood dyscrasias, reactions to medications, and alcohol intake. The skin, eye, blood, and liver and kidney function should be followed.

SPECIAL TESTS

Urine may be examined for TNT by the Webster test or for the urinary metabolite 2,6-dinitro-4-aminotoluene; however, both may be negative if there is liver injury.

PERSONAL PROTECTIVE METHODS

Protective clothing should be worn. The Webster skin test (colorimetric test with alcoholic sodium hydroxide) or indicator soap should be used to make sure workers have washed all TNT off their skins. Daily change of clean work clothes should be provided, and showers

made compulsory at the end of each shift prior to changing to street clothes.

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MISCELLANEOUS ORGANIC NITROGEN COMPOUNDS

This group of organic nitrogen compounds includes examples of heterocyclic compounds, hydrazines, substituted amides, an imine, and a nitrosoamine.

Heterocyclic nitrogen compounds contain one or more nitrogen atoms in the ring structure and are widely distributed in nature as well as in industrial use. The ring may be three, five, or six membered, and there may be other hetero atoms in addition to nitrogen.

The hydrazine compounds are characteristized by their structure. Amides are derivatives of acids, and some have wide usage as solvents. Imines are highly reactive substances of the general structure, e.g., $R_2C=NH$. Many of them appear to be biological alkylating agents and to have radiomimetic properties. They are somewhat similar in these respects to epoxy compounds, with the nitrogen group in place of an oxygen in a ring structure.

The nitroso group, e.g., $-N=O$, forms another reactive class of nitrogen compounds widely used in synthetic chemical reactions. When combined with a carbon atom, e.g., $C-N=O$, they often show skin irritant or sensitizing properties, and some are methemoglobin formers. When attached to the nitrogen of certain aliphatic amines, however, e.g., $(CH_3)_2-N-N=O$ (N-nitroso dimethyl amine), they sometimes become potent experimental animal carcinogens.

ACRIDINE

DESCRIPTION

$C_{13}H_9N$, acridine, is a colorless or light yellow crystal, very soluble in boiling water.

SYNONYMS

Dibenzopyridine, 10-azaanthracene.

POTENTIAL OCCUPATIONAL EXPOSURES

Acridine and its derivatives are widely used in the production of dyestuffs such as acriflavine, benzoflavine, and chrysaniline, and in the synthesis of pharmaceuticals such as aurinacrine, proflavine, and rivanol.

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

Chemical laboratory workers	Drug makers
Coal tar workers	Dye makers
Disinfectant makers	Organic chemical synthesizers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for acridine.

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Acridine is a severe irritant to the conjunctiva of the eyes, the mucous membranes of the respiratory tract, and the skin. It is a powerful photosensitizer of the skin. Acridine causes sneezing on inhalation.

Systemic—

Yellowish discoloration of sclera and conjunctiva may occur. Mutational properties have been ascribed to acridine, but its effect on humans is not known.

MEDICAL SURVEILLANCE

Evaluate the skin, eyes, and respiratory tract in the course of any placement or periodic examinations.

SPECIAL TESTS

None commonly used. Can be detected in blood or urine.

PERSONAL PROTECTIVE METHODS

Prevent skin, eye, or respiratory contact with protective clothing, gloves, goggles, and appropriate dust respirators. In case of spills or splashes, the skin area should be thoroughly washed and the contaminated clothing changed. Clean work clothing should be supplied on a daily basis, and the worker should shower prior to changing to street clothes.

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N,N-DIMETHYLFORMAMIDE

DESCRIPTION

$HCON(CH_3)_2$, dimethylformamide, is a colorless liquid which at

25 C is soluble in water and organic solvents. It has a fishy, unpleasant odor at relatively low concentrations, but the odor has no warning property.

SYNONYMS

DMF, the "universal organic solvent," DMFA.

POTENTIAL OCCUPATIONAL EXPOSURES

Dimethylformamide has powerful solvent properties for a wide range of organic compounds. Because of dimethyl formamide's physical properties, it has been used when solvents with a slow rate of evaporation are required.

It finds particular usage in the manufacture of polyacrylic fibers, butadiene, purified acetylene, pharmaceuticals, dyes, petroleum products, and other organic chemicals.

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

- Acetylene purifiers
- Butadiene makers
- Drug makers
- Dye makers
- Organic chemical synthesizers
- Petroleum refinery workers
- Resin makers
- Solvent workers
- Synthetic fiber makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 10 ppm (30 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapor, and it is readily absorbed through intact skin.

HARMFUL EFFECTS

Local—

Dimethylformamide exposure may cause dermatitis.

Systemic—

Inhalation of dimethylformamide or skin contact with this chemical may cause colicky abdominal pain, anorexia, nausea, vomiting, constipation, diarrhea, facial flushing (especially after drinking alcohol), elevated blood pressures, hepatomegaly, and other signs of liver damage. This chemical has produced kidney damage in animals.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should be concerned particularly with liver and kidney function and with possible effects on the skin.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Organic vapor masks or air supplied respirators may be required in elevated vapor concentrations. Percutaneous absorption should be pre-

vented by gloves and other protective clothing. Goggles should be used to prevent eye splashes. In cases of spills or splashes, the wet clothing should be immediately removed and the skin area thoroughly cleaned. Clean clothing should be issued to workers on a daily basis and showers taken before changing to street clothes.

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ETHYLENEIMINE

DESCRIPTION

H_2CNHCH_2, ethyleneimine, is a colorless volatile liquid with an ammoniacal odor.

SYNONYMS

Azacyclopropane, aziridine, dimethyleneimine, ethylenimine, vinylamine, azirane, dihydroazirine, EI.

POTENTIAL OCCUPATIONAL EXPOSURES

Ethyleneimine is a highly reactive compound and is used in many organic synthesesis. The polymerization products, polyethyleneimines, are used as auxiliaries in the paper industry and as flocculation aids in the clarification of effluents. It is also used in the textile industry for increasing wet strength, flameproofing, shrinkproofing, stiffening, and waterproofing.

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

- Effluent treaters
- Paper makers
- Polyethyleneimine makers
- Organic chemical synthesizers
- Textile makers

PERMISSIBLE EXPOSURE LIMITS

Ethyleneimine was included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

The vapor is strongly irritating to the conjunctiva and cornea, the mucous membranes of the nose, throat, and upper respiratory tract, and the skin. The liquid is a severe irritant and vesicant in humans, and

severe eye burns have followed contact with the cornea. Skin sensitization has occurred.

Systemic—

Acute exposures in humans have caused nausea, vomiting, headaches, dizziness, and pulmonary edema. In mice acute lethal exposures to vapor produced pulmonary edema, renal damage, and hematuria. Compounds with the aziridine structure have some of the properties of alkylating agents. Ethyleneimine has been reported to induce mutagenic effects in *in vitro* cultures, microorganisms, plants, and animals.

In repeated exposures rodents have developed pancytopenia and gonadal effects. Rats given twice weekly subcutaneous injections of ethyleneimine in oil for about 33 weeks developed sarcoma at the injection site and one case of transitional cell carcinoma in the kidney was observed. Feeding experiments with mice at 13 ppm in the diet for 74 weeks produced hepatomas and pulmonary tumors. These effects have not been reported in humans.

MEDICAL SURVEILLANCE

Based partly on animal experimental data, examinations should include history of exposure to other carcinogens, smoking, alcohol, medications, and family history. The skin, eye, lung, liver, and kidney should be evaluated. Sputum or urine cytology may be helpful.

SPECIAL TESTS

None in common use. Chromosomal studies have been made, but are probably not useful for routine surveillance.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering control and prevent all skin or respiratory exposure. Full body protective clothing and gloves should be used. Fullface supplied air respirators with continuous flow or pressure demand type should also be used. Eyes should be protected at all times. On exit from a regulated area employees should shower and change to street clothes, leaving their protective clothing and equipment at the point of exit, to be placed in impervious containers at the end of the work shift for decontamination or disposal.

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HEXAMETHYLENETETRAMINE

DESCRIPTION

$(CH_2)_6N_4$, hexamethylenetetramine, is an odorless, crystalline solid.

SYNONYMS

Methenamine, hexamine, formamine, ammonioformaldehyde.

POTENTIAL OCCUPATIONAL EXPOSURES

Hexamethylenetetramine is used as an accelerator in the rubber industry, as a curing agent in thermosetting plastics, as a fuel pellet for camp stoves, and in the manufacture of resins, pharmaceuticals, and explosives.

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

- Drug makers
- Explosive makers
- Fuel tablet makers
- Phenol-formaldehyde resin workers
- Resin makers
- Rubber makers
- Textile makers
- Urea-formaldehyde resin workers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for hexamethylenetetramine.

ROUTES OF ENTRY

Ingestion and skin contact.

HARMFUL EFFECTS

Local—

Very mild skin irritant.

Systemic—

None. Side effects from ingestion are urinary tract irritation, skin rash, and digestive disturbances. Large oral doses can cause severe nephritis which may be fatal.

MEDICAL SURVEILLANCE

No specific considerations are necessary.

SPECIAL TESTS

None.

PERSONAL PROTECTIVE METHODS

If repeated or prolonged skin exposure is likely, gloves or protective clothing may be needed.

HYDRAZINE and DERIVATIVES

DESCRIPTION

Hydrazine (H_2N-NH_2) is a colorless, oily liquid with an ammoniacal odor. Phenylhydrazine ($C_6H_5NHNH_2$) is an oily, colorless liquid

or a crystalline solid. Dimethylhydrazine, UDMH, ($(CH_3)_2-N-NH_2$) is a hygroscopic mobile liquid. Hydrazine and UDMH are soluble in water and alcohol. Phenylhydrazine is slightly soluble in water.

SYNONYMS

Hydrazine: Hydrazine base, diamine.

Phenylhydrazine: Hydrazinobenzene.

Dimethylhydrazine: UDMH, 1,1-dimethylhydrazine, asymmetrical dimethylhydrazine.

POTENTIAL OCCUPATIONAL EXPOSURES

Both UDMH and hydrazine are used in liquid rocket fuels. Because of its strong reducing capabilities, hydrazine is used as an intermediate in chemical synthesis and in photography and metallurgy. It is also used in the preparation of anticorrosives, textile agents, and pesticides, and as a scavenging agent for oxygen in boiler water. Hydrazine salts find use as fluxes in soft soldering and aluminum soldering. Phenylhydrazine is very reactive with carbonyl compounds and is a widely used reagent in conjunction with sugars, aldehydes, and ketones, in addition to its use in the synthesis of dyes, pharmaceuticals such as antipyrin, cryogenin, and pyramidone, and other organic chemicals. The hydrochloride salt is used in the treatment of polycythemia vera.

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

Acrylic and vinyl textile dyers
Agricultural chemical makers
Anticorrosion additive makers
Antioxidant workers
Boiler operators
Chemists
Drug makers
Insecticide makers
Jet fuel workers
Oxygen scavenger makers
Photographic developer makers
Rocket fuel workers
Solder flux makers
Water treaters

PERMISSIBLE EXPOSURE LIMITS

The Federal standard compounds are:

Hydrazine 1 ppm (1.3 mg/m³)
Phenylhydrazine 5 ppm (22 mg/m³)
Dimethylhydrazine 0.5 ppm (1 mg/m³)

ROUTES OF ENTRY

Inhalation and percutaneous absorption.

HARMFUL EFFECTS

Local—

All three compounds have similar toxic local effects due to their irritant properties. The vapor is highly irritating to the eyes, upper respiratory tract, and skin, and causes delayed eye irritation. Severe exposure may produce temporary blindness. The liquid is corrosive, producing penetrating burns and severe dermatitis. Permanent corneal lesions may occur if the liquid is splashed in the eyes. A sensitization dermatitis may be produced.

Systemic—

Inhalation of hydrazine may cause dizziness and nausea. In animals hydrazine has caused liver and kidney damage and pulmonary edema. It has also been reported to cause adenocarcinoma of the lung and liver in animals.

MEDICAL SURVEILLANCE

Based partly on experimental animal data, placement should include a history of exposure to other carcinogens, smoking, alcohol, medications, and family history. The skin, eye, lungs, liver, kidney, blood, and central nervous system should be evaluated. Sputum or urine cytology may give useful information.

SPECIAL TESTS

Hydrazine may be detected in the blood; UDMH has been measured in blood and urine. Some phenylhydrazine metabolites are known. None of these are in common use, however.

PERSONAL PROTECTIVE METHODS

Protective clothing, gloves, and goggles should be worn to reduce any skin or eye contact. Fullface supplied air masks and full protective clothing may be required if vapor concentrations are significant. Clean work clothes should be supplied on a daily basis, and workers should shower prior to change to street clothes.

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N-NITROSODIMETHYLAMINE

DESCRIPTION

$(CH_3)_2NN=O$, n-nitrosodimethylamine (DMN), is a yellow liquid of low viscosity, soluble in water, alcohol, and ether.

SYNONYMS

Dimethylnitrosamine, DMN.

POTENTIAL OCCUPATIONAL EXPOSURES

DMN is used in the manufacture of dimethylhydrazine. It has also been used as an industrial solvent and a nematocide. There are patents

for its use as a solvent in the fiber and plastics industry, as an antioxidant, as a softener for copolymers, as an additive for lubricants, and in condensers to increase the dielectric constant.

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

Dimethylhydrazine makers
Nematocide makers
Solvent workers

PERMISSIBLE EXPOSURE LIMITS

DMN is included in the Federal standard for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Inhalation of vapor and possibly percutaneous absorption.

HARMFUL EFFECTS

Local—

The liquid and vapor are not especially irritating to the skin or eyes, and warning properties are poor.

Systemic—

DMN is a highly toxic substance in most species, including man. Systemic effects are characterized by onset in a few hours of nausea and vomiting, abdominal cramps, and diarrhea. Also headache, fever, weakness, enlargement of the liver, and jaundice may occur. Chronic exposures may lead to liver damage (central necrosis), with jaundice and ascites. There have been a number of reported cases, including severe liver injury in man and one death. Autopsy revealed an acute diffuse centrolobular necrosis. Recovery occurred in other cases.

In rats, guinea pigs, and other experimental animals, DMN is a highly potent carcinogen, producing malignant tumors, primarily of the liver and kidney, but also in the lung. Both ingestion and inhalation routes have produced tumors. These have not been reported in man, but in view of its potency in various other species, the material has been presumed to be carcinogenic in man also.

MEDICAL SURVEILLANCE

Based on human experience and on animal studies, preplacement and periodic examinations should include a history of exposure to other carcinogens, alcohol and smoking habits, medications, and family history. Special attention should be given to liver size and function, and to any changes in lung symptoms or X-rays. Renal function should be followed. Sputum and urine cytology may be useful.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to prevent all contact with the skin, eyes, or respiratory tract. Full body pro-

tective clothing and gloves should be provided and also appropriate type fullface supplied air respirators of continuous flow or pressure demand type. On exit from a regulated area, employees should be required to shower before changing into street clothes, leaving their protective clothing and equipment at the point of exit, to be placed in impervious containers at the end of the work shift for decontamination or disposal.

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PYRIDINE

DESCRIPTION

C_5H_5N, pyridine, is a colorless liquid with an unpleasant odor. It is both flammable and explosive when exposed to a flame and decomposes on heating to release cyanide fumes. Pyridine is soluble in water, alcohol, and ether. The odor can be detected well below 1 ppm.

SYNONYMS

Azine.

POTENTIAL OCCUPATIONAL EXPOSURES

Pyridine is used as a solvent in the chemical industry and as a denaturant for ethyl alcohol. It is used in the manufacture of paints, explosives, dyestuffs, rubber, vitamins, sulfa drugs, and disinfectants.

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

- Alcohol denaturant makers
- Alcohol denaturers
- Drug makers
- Dye makers
- Explosive workers
- Organic chemical synthesizers
- Paint makers
- Rubber workers
- Resin workers
- Solvent workers
- Vitamin makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (15 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapor and percutaneous absorption of liquids.

HARMFUL EFFECTS

Local—

Irritation of the conjunctiva of the eye and cornea and mucous membranes of the upper respiratory tract and skin may occur. It oc-

casionally causes skin sensitization, and photosensitization has been reported.

Systemic—

Very high concentrations may cause narcosis. Repeated, intermittent, or continuous low level exposure may lead to transient effects on the central nervous system and gastrointestinal tract. The symptoms include headache, dizziness, insomnia, nervousness, anorexia, nausea, vomiting, and diarrhea. Low back pain and urinary frequency with no changes in urine sediment or liver or renal function and complete recovery have been reported to follow exposures to about 100 ppm. Liver and kidney injury have been reported from its use as an oral medication.

MEDICAL SURVEILLANCE

Placement and periodic examinations should consider possible effects on skin, central nervous system, and liver and kidney function.

SPECIAL TESTS

None in common use. Metabolites are known and can be determined in blood and urine.

PERSONAL PROTECTIVE METHODS

Rubber and plastic gloves should not be relied upon to prevent contact with pyridine as its salts penetrate the material. The odor is detectable at less than 1 ppm but cannot be relied upon as a preventive. In areas of elevated vapor concentration, workers should be supplied with fullface supplied air masks and protective clothing. Clothing that is contaminated by spills or splashes should be immediately changed and discarded and the area of involved skin thoroughly washed. Clean work clothes should be supplied daily with the worker showering after his shift before changing to street clothes.

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N,N-DIMETHYLACETAMIDE

DESCRIPTION

$CH_3CON(CH_3)_2$, dimethylacetamide, is a colorless, nonvolatile liquid.

SYNONYMS

Acetic acid dimethylamide, DMA, DMAC, acetyl dimethylamide.

POTENTIAL OCCUPATIONAL EXPOSURES

Dimethylacetamide is used commercially as a solvent in various industries.

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

Solvent workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 10 ppm (35 mg/m³), skin.

ROUTES OF ENTRY

Inhalation of vapor and absorption through intact skin.

HARMFUL EFFECTS

Local—

None known.

Systemic—

Jaundice has been noted in workers exposed chronically to dimethylacetamide vapor although skin absorption may also have occurred. Liver injury consists of cord-cell degeneration, but recovery is usually rapid. Other symptoms from large oral doses as an anticancer drug include depression, lethargy, and visual and auditory hallucinations.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should give special attention to skin, central nervous system, and liver function or disease.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

Organic vapor masks or air supplied respirators may be required in elevated vapor concentrations. Percutaneous absorption should be prevented by gloves and other protective clothing. Goggles should be used to prevent eye splashes. In cases of spills or splashes, the wet clothing should be immediately removed and the involved skin area thoroughly cleaned. Clean clothing should be issued to workers on a daily basis and showers taken before changing to street clothes.

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MISCELLANEOUS ORGANIC CHEMICALS

BETA-PROPIOLACTONE

DESCRIPTION

OCH_2CH_2CO, beta-propiolactone, is a colorless liquid which slowly hydrolyzes to hydracrylic acid and must be cooled to remain stable.

SYNONYMS

2-Oxetanone, propiolactone, BPL, 3-hydroxy-beta-lactone-propanoic acid.

POTENTIAL OCCUPATIONAL EXPOSURES

beta-Propiolactone is used as a chemical intermediate in synthesis of acrylate plastics and as a vapor sterilizing agent, disinfectant, and a viricidal agent.

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

Acrylate plastic makers	Plastic makers
Chemists	Resin makers
Disinfectant workers	Viricidal agent makers

PERMISSIBLE EXPOSURE LIMITS

beta-Propiolactone is included in the Federal standards for carcinogens; all contact with it should be avoided.

ROUTES OF ENTRY

Inhalation of vapor and percutaneous absorption.

HARMFUL EFFECTS

Local—

Repeated or prolonged contact with liquid may cause erythema, vesication of the skin, and, as reported in animals, hair loss and scarring. In rodents, beta-propiolactone has also produced skin papilloma and sarcoma by skin painting, subcutaneous injection, and oral administration. Tumors of the connective tissue are also suspected. Direct eye contact with concentrated liquid may result in permanent corneal opacification. Skin cancer has not been reported in man.

Systemic—

The systemic effect of beta-propiolactone in humans is unknown due to lack of reported cases. Acute exposure in animals has caused liver necrosis and renal tubular damage. Death has occurred following rapid development of spasms, dyspnea, convulsions, and collapse at relatively low levels (less than 5 ml/kg.) Beta propiolactone has been implicated as a carcinogen by a number of animal studies which produced a variety of skin tumors, stomach tumors, and hepatoma depending on the route of administration.

MEDICAL SURVEILLANCE

Based on its high toxicity and carcinogenic effects in animals, preplacement and periodic examinations should include a history of exposure to other carcinogens, alcohol and smoking habits, medication and family history. The skin, eye, lung, liver, and kidney should be evaluated. Sputum cytology, may be helpful in evaluating the presence or absence of carcinogenic effects.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to pre-

vent all contact with skin or respiratory tract. Full body protective clothing and gloves should be provided as well as fullface supplied air respirators of continuous flow or pressure demand type. Employees should remove and leave protective clothing and equipment at the point of exit, to be placed in impervious containers at the end of work shift for decontamination or disposal. Showers should be taken before dressing in street clothes.

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TRICRESYL PHOSPHATES

DESCRIPTION

Tricresyl phosphates are available as the ortho-isomer (TOCP), the meta-isomer (TMCP), and the para-isomer (TPCP). The ortho-isomer is the most toxic of the three; the meta- and para-isomers are relatively inactive. The commercial product may contain the ortho-isomer as a contaminant unless special precautions are taken during manufacture. Pure tri-para-cresylphosphate is a solid, and ortho- and meta- are colorless, oily, odorless liquids.

SYNONYMS

Tritolyl phosphate, TCP.

POTENTIAL OCCUPATIONAL EXPOSURES

Tricresyl phosphate is used as a plasticizer for chlorinated rubber, vinyl plastics, polystyrene, polyacrylic, and polymethacrylic esters, as an adjuvant in milling of pigment pastes, as a solvent and as a binder in nitrocellulose and various natural resins, and as an additive to synthetic lubricants and gasoline. It is also used as hydraulic fluid, fire retardant and in the recovery of phenol in coke-oven waste waters.

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

- Gasoline additive makers
- Gasoline blenders
- Hydraulic fluid workers
- Lead scavenger makers
- Nitrocellulose workers
- Plasticizer workers
- Polystyrene makers
- Polyvinyl chloride makers
- Solvent workers
- Surgical instrument sterilizers
- Waterproofing makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for tri-ortho-cresyl phosphate is 0.1 mg/m^3; there is no standard for the meta- and para-isomers.

ROUTES OF ENTRY

Inhalation of ortho-isomer vapor or mist, especially when heated;

ingestion and percutaneous absorption of liquids. The widespread epidemics of poisoning that have occurred have been due to ingested ortho-isomer as a contaminant of foodstuff. There have been relatively few reports of neurological symptoms in workers handling these substances. Experimental human studies with labeled phosphorus derivatives show only 0.4% of the applied dose was absorbed.

HARMFUL EFFECTS

Local—

None reported.

Systemic—

The major effects from inhaling, swallowing, or absorbing tricresyl phosphate through the skin are on the spinal cord and peripheral nervous system; the poison attacking the anterior horn cells and pyramidal tract as well as the peripheral nerves. Gastrointestinal symptoms on acute exposure (nausea, vomiting, diarrhea, and abdominal pain) are followed by a latent period of 3 to 30 days with the progressive development of muscle soreness and numbness of fingers, calf muscles, and toes, with foot and wrist drop. In chronic intoxication, the g.i. symptoms pass unnoticed, and after a long latent period, flaccid paralysis of limb and leg muscles appear. There are minor sensory changes and no loss of sphincter control.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should include evaluation of spinal cord and neuromuscular function, especially in the extremities, and a history of exposure to other organo-phosphate esters, pesticides, or neurotoxic agents. Periodic cholinesterase determination may relate to exposure, but not necessarily to neuromuscular effect.

SPECIAL TESTS

None used except for determination of serum or red cell choline or acetylcholine esterases.

PERSONAL PROTECTIVE METHODS

Protective clothing should be worn to prevent skin absorption and, where dust or vapor concentrates, masks should be supplied to employees.

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CARBON DISULFIDE

DESCRIPTION

CS_2, carbon disulfide, is a highly refractive, flammable liquid which in pure form has a sweet odor and in commercial and reagent grades has a foul smell. It can be detected by odor at about 1 ppm but the sense of smell fatigues rapidly and, therefore, odor does not serve as a good warning property. It is slightly soluble in water, but more soluble in organic solvents.

SYNONYMS

Carbon bisulfide, dithiocarbonic anhydride.

POTENTIAL OCCUPATIONAL EXPOSURES

Carbon disulfide is used in the manufacture of viscose rayon, ammonium salt, carbon tetrachloride, carbanilide, xanthogenates, flotation agents, soil disinfectants, dyes, electronic vacuum tubes, optical glass, paints, enamels, paint removers, varnishes, varnish removers, tallow, textiles, explosives, rocket fuel, putty, preservatives, and rubber cement; as a solvent for phosphorus, sulfur, selenium, bromine, iodine, alkali cellulose, fats, waxes, lacquers, camphor, resins, and cold vulcanized rubber. It is also used in degreasing, chemical analysis, electroplating, grain fumigation, oil extraction, and drycleaning.

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

- Ammonium salt makers
- Bromine processors
- Carbon tetrachloride makers
- Degreasers
- Drycleaners
- Electroplaters
- Fat processors
- Flotation agent makers
- Iodine processors
- Oil processors
- Paint workers
- Preservative makers
- Putty makers
- Rayon makers
- Resin makers
- Rocket fuel makers
- Rubber cement makers
- Rubber workers
- Sulfur processors
- Tallow makers
- Textile makers
- Vacuum tube makers
- Varnish makers
- Wax processors

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 20 ppm (60 mg/m^3) determined as an 8-hour TWA. The acceptable ceiling concentration is 30 ppm (90 mg/m^3) with a maximum peak above this for an 8-hour workshift of 100 ppm (300 mg/m^3) for a maximum duration of 30 minutes.

ROUTES OF ENTRY

Inhalation of vapor which may be compounded by percutaneous absorption of liquid or vapor.

HARMFUL EFFECTS

Local—

Carbon disulfide vapor in sufficient quantities is severely irritating to

eyes, skin, and mucous membranes. Contact with liquid may cause blistering with second and third degree burns. Skin sensitization may occur. Skin absorption may result in localized degeneration of peripheral nerves which is most often noted in the hands. Respiratory irritation may result in bronchitis and emphysema, though these effects may be overshadowed by systemic effects.

Systemic—

Intoxication from carbon disulfide is primarily manifested by psychological, neurological, and cardiovascular disorders. Recent evidence indicates that once biochemical alterations are initiated they may remain latent; clinical signs and symptoms then occur following subsequent exposure.

Following repeated carbon disulfide exposure, subjective psychological as well as behavioral disorders have been observed. Acute exposures may result in extreme irritability, uncontrollable anger, suicidal tendencies, and a toxic manic depressive psychosis. Chronic exposures have resulted in insomnia, nightmares, defective memory, and impotency. Less dramatic changes include headache, dizziness, and diminished mental and motor ability, with staggering gait and loss of coordination.

Neurological changes result in polyneuritis. Animal experimentation has revealed pyramidal and extrapyramidal tract lesions and generalized degeneration of the myelin sheaths of peripheral nerves. Chronic exposure signs and symptoms include retrobulbar and optic neuritis, loss of sense of smell, tremors, paresthesias, weakness, and, most typically, loss of lower extremity reflexes.

Atherosclerosis and coronary heart disease have been significantly linked to exposure to carbon disulfide. Atherosclerosis develops most notably in the blood vessels of the brain, glomeruli, and myocardium. Abnormal electroencephalograms and retinal hypertension typically occur before renal involvement is noted. Any of the above three areas may be affected by chronic exposure, but most often only one aspect can be observed. A significant increase in coronary heart disease mortality has been observed in carbon disulfide workers. Studies also reveal higher frequency of angina pectoris and hypertension. Abnormal electrocardiograms may also occur and are also suggestive of carbon disulfide's role in the etiology of coronary disease.

Other specific effects include chronic gastritis with the possible development of gastric and duodenal ulcers; impairment of endocrine activity, specifically adrenal and testicular; abnormal erythrocytic development with hypochromic anemia; and possible liver dysfunction with abnormal serum cholesterol. Also in women, chronic menstrual disorders may occur. These effects usually occur following chronic exposure and are subordinate to the other symptoms.

Recently human experience and animal experimentation have indicated several possible biochemical changes. Carbon disulfide and its metabolites (i.e., dithiocarbamic acids and isothiocyanates) show amino acid interference, cerebral monoamine oxidase inhibition, endocrine dis-

orders, lipoprotein metabolism interference, blood protein, and zinc level abnormalities, and inorganic metabolism interference due to chelating of polyvalent ions. The direct relationship between these biochemical changes and clinical manifestations is only suggestive.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should be concerned especially with skin, eyes, central and peripheral nervous system, cardiovascular disease, as well as liver and kidney function. Electrocardiograms should be taken.

SPECIAL TESTS

CS_2 can be determined in expired air, blood, and urine. The iodine-azide test is most useful although non-specific, and it may indicate other sulfur compounds.

PERSONAL PROTECTIVE METHODS

Local exhaust, general ventilation, and personal protective equipment should be utilized. In modern manufacture, CS_2 fumes are generally controlled by closed operations. Where fumes are present in unacceptable concentrations, vapor gas mask with fullface or used air respirators should be used. In all areas where there is likelihood of spill or splash on any skin area, protection should be afforded by protective clothing, goggles, face shields, aprons, and coats.

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DIMETHYL SULFATE

DESCRIPTION

$(CH_3)_2SO_4$, dimethyl sulfate, is an oily, colorless liquid slightly soluble in water, but more soluble in organic solvents.

SYNONYMS

Sulfuric acid dimethyl ester.

POTENTIAL OCCUPATIONAL EXPOSURES

Industrial use of dimethyl sulfate is based upon its methylating properties. It is used in the manufacture of methyl esters, ethers and amines, in dyes, drugs, perfume, phenol derivatives, and other organic chemicals. It is also used as a solvent in the separation of mineral oils.

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

- Amine makers
- Drug makers
- Dye makers
- Methylation workers
- Organic chemical synthesizers
- Perfume makers
- Phenol derivative makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1 ppm (5 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapor; percutaneous absorption of liquid.

HARMFUL EFFECTS

Local—

Liquid is highly irritating and causes skin vesiculation and analgesia. Lesions are typically slow-healing and may result in scar tissue while analgesia may last several months. Liquid and vapor are irritating to the mucous membranes, and exposure produces lacrimation, rhinitis, edema of the mucosa of the mouth and throat, dysphagia, sore throat, and hoarseness. Irritation of the skin and mucous membranes may be delayed in appearance. Eye irritation may result in conjunctivitis, keratitis, and photophobia. In severe cases corneal opacities, perforation of the nasal septum and permanent or persistent visual disorders have been reported.

Systemic—

The toxicity of dimethyl sulfate is based upon its alkylating properties and its hydrolysis to sulfuric acid and methyl alcohol. Acute exposure may cause respiratory dysfunctions such as pulmonary edema, bronchitis, and pneumonitis following a latent period of 6 to 24 hours. Cerebral edema and other central nervous system effects such as drowsiness, temporary blindness, tachycardia or bradycardia may be linked to dimethyl sulfate's effect on nerve endings. Secondary pulmonary effects such as susceptibility to infection, as well as, more pronounced effects in those persons with preexisting respiratory disorders, are also noteworthy. Chronic poisoning occurs only rarely and is usually limited to ocular and respiratory disabilities. It has been reported to be carcinogenic in rats, but this has not been verified in man.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should give special consideration to the skin, eyes, central nervous system, lung. Chest X-rays should be taken and lung, liver, and kidney functions evaluated.

Sputum and urinary cytology may be useful in detecting the presence or absence of carcinogenic effects.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

These are designed to supplement engineering controls and to reduce skin, eye, or respiratory contact to a negligible level. The liquid and the vapor of dimethyl sulfate are extremely irritating so that the skin, eyes, as well as the respiratory tract should be protected at all times. Protective clothing, gloves, goggles, face shields, aprons, and boots should be used in areas where there is danger of splash or spill. Fullface vapor masks or supplied air respirators may be necessary in areas of vapor build up or leaks. Attention should be given to personal hygiene with a change of work clothes daily and shower before change to street clothes.

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MERCAPTANS

DESCRIPTION

Methyl mercaptan: CH_3SH; ethyl mercaptan: CH_3CH_2SH; n-butyl mercaptan: $CH_3(CH_2)_2CHSH$; and perchloromethyl mercaptan: CCl_3-SCl.

These compounds are typically flammable liquids except methyl mercaptan which is a gas. Perchloromethyl mercaptan is yellow; the rest are colorless. A strong unpleasant odor is the most characteristic property of mercaptans and may be detected at very low levels, i.e. less than 0.5 ppm. Perchloromethyl mercaptan is insoluble in water, but others are slightly soluble.

SYNONYMS

Methyl mercaptans: Methanethiol, mercaptomethane, thiomethyl alcohol, methyl sulfhydrate.

Ethyl mercaptan: Ethanethiol, mercaptoethane, ethyl sulfhydrate, thioethyl alcohol.

n-Butyl mercaptan: 1-Butanethiol, n-butyl thioalcohol, thiobutyl alcohol.

POTENTIAL OCCUPATIONAL EXPOSURES

In general, mercaptans find use as intermediates in the manufacture of pesticides, fumigants, dyes, pharmaceuticals, and other chemicals, and as gas odorants, i.e., to serve as a warning property for hazardous odor-

less gases. Particular usages for methyl mercaptan include the synthesis of methionine and the manufacture of fungicides and jet fuels. Ethyl mercaptan is used as an adhesive stabilizer and butyl mercaptan may be used as a solvent.

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

- Drug makers
- Dye makers
- Fumigant makers
- Fumigators
- Jet fuel blenders
- Methionine makers
- Organic chemical synthesizers
- Pesticide makers
- Warning agent workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for each mercaptan is:

Methyl mercaptan	10 ppm	20 mg/m^3
Ethyl mercaptan	10 ppm	25 mg/m^3
Butyl mercaptan	10 ppm	35 mg/m^3
Perchloromethyl mercaptan	0.1 ppm	0.8 mg/m^3

The above standards are determined as TWAs except ethyl and methyl mercaptan which are ceiling values. ACGIH has lowered the TLVs of all but perchloromethyl mercaptan: Methyl mercaptan, 0.5 ppm (1 mg/m^3); ethyl mercaptan, 0.5 ppm (1.0 mg/m^3); butyl mercaptan, 0.5 ppm (1.5 mg/m^3); all TWAs.

ROUTE OF ENTRY

Inhalation of gas or vapor.

HARMFUL EFFECTS

Local—

Mercaptans have an intensely disagreeable odor and are irritating to skin, eyes, and mucous membranes of the upper respiratory tract. Liquid may cause contact dermatitis and vapor may cause irritation to nose and throat. Perchloromethyl mercaptan is stronger in its irritant ability than the other mercaptans which cause only slight to moderate irritation.

Systemic—

Methyl mercaptan acts toxicologically like hydrogen sulfide and may depress the central nervous system resulting in respiratory paralysis and death. Victims who survive severe exposures may suffer from headache, dizziness, staggering gait, nausea, and vomiting. Respiratory tract irritation may lead to pulmonary edema and possibly renal and hepatic damage. The above effects are based primarily on animal experimentation. In a recent case of acute methyl mercaptan exposure, a worker developed acute anemia and methemoglobinemia 24 hours following coma.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should consider skin, eyes, lung and central nervous system as well as liver and kidney functions. Blood studies may be helpful in following acute intoxication from methyl mercaptan.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

In areas where liquid mercaptan is likely to be spilled or splashed on the skin, impervious clothing, gloves, gauntlets, aprons, and boots should be supplied. Otherwise protective methods are as for sulfur dioxide. (See Sulfur Dioxide.)

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TETRAMETHYLTHIURAM DISULFIDE

DESCRIPTION

$C_6H_{12}N_2S_4$, tetramethylthiuram disulfide, is a white or yellow crystal insoluble in water, but soluble in organic solvents.

SYNONYMS

Thiram, bis-(dimethylthiocarbamyl) disulfide, TMTD, thirad, thiuram.

POTENTIAL OCCUPATIONAL EXPOSURES

Tetramethylthiuram disulfide is used as a rubber accelerator and vulcanizer; a seed, nut, fruit, and mushroom disinfectant; a bacteriostat for edible oils and fats; and as an ingredient in sun-tan and antiseptic sprays and soaps. It is also used as a fungicide, rodent repellent, wood preservative, and may be used in the blending of lubricant oils.

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

- Food disinfectant makers
- Fungicide workers
- Lubricating oil blenders
- Rat repellent makers
- Rubber makers
- Soap makers
- Wood preservative makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for thiram (tetramethylthiuram disulfide) is 5 mg/m³.

ROUTE OF ENTRY

Inhalation of dust, spray, or mist.

HARMFUL EFFECTS

Local—

Irritation of mucous membranes conjunctivitis, rhinitis, sneezing, and cough may result from excessive exposures. Skin irritation with erythema and urticaria may also occur. Allergic contact dermatitis has been reported in workers who wore rubber gloves containing tetramethylthiuram disulfide.

Systemic—

Systemic effects have not been reported in the U.S. literature. Bronchitis was mentioned in one European report in workers exposed to thiram or other products during synthesis. Intolerance to alcohol has been observed in workers exposed to thiram, manifested by flushing of face, palpitation, rapid pulse, dizziness, and hypotension. These effects are thought to be due to the blocking of the oxidation of acetaldehyde. It should be noted in this connection that the diethyl homologue of this compound, tetraethylthiuram disulfide, is marketed as the drug "Antabuse" and that severe and disagreeable symptoms ensue immediately in subjects who ingest the smallest amount of ethyl alcohol after they have been "premedicated" with the drug.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should give special attention to history of skin allergy, eye irritation, and significant respiratory, liver, or kidney disease. Workers should be aware of the potentiating action of alcoholic beverages when working with tetramethylthiuram disulfide.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Skin and eye protection should be provided by protective clothing, gloves, and goggles. Employees should be encouraged to shower following each shift and to change to clean work clothes at the start of each shift. In areas where dust, spray, or mist are excessive, respiratory protection by dust masks or gas mask respirators with proper canister or supplied air respirators should be provided.

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HALOGENS

BROMINE/HYDROGEN BROMIDE

DESCRIPTION

Br, bromide, is a dark reddish-brown, fuming, volatile liquid with a suffocating odor. Bromine is soluble in water and alcohol. HBr, hydrogen bromide, is a corrosive colorless gas.

SYNONYMS

Bromine: none.

Hydrogen bromide: anhydrous hydrobromic acid.

POTENTIAL OCCUPATIONAL EXPOSURES

Bromine is primarily used in the manufacture of gasoline antiknock compounds (1,2-dibromoethane). Other uses are for gold extraction, in brominating hydrocarbons, in bleaching fibers and silk, in the manufacture of pharmaceuticals, military gas, dyestuffs, and as an oxidizing agent.

Hydrogen bromide and its aqueous solutions are used in the manufacture of organic and inorganic bromides, as a reducing agent and catalyst in controlled oxidations, in the alkylation of aromatic compounds, and in the isomerization of conjugated diolefins.

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

Drug makers	Organic chemical synthesizers
Dye makers	Petroleum refinery workers
Gasoline additive makers	Photographic chemical makers
Gold extractors	Silk and fiber bleachers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are: bromine 0.1 ppm (0.7 mg/m^3); and hydrogen bromide 3 ppm (10 mg/m^3).

ROUTES OF ENTRY

Inhalation of vapor or gas. Bromine may be absorbed through the skin.

HARMFUL EFFECTS

Local—

Bromine and hydrogen bromide and its aqueous solutions are extremely irritating to eyes, skin, and mucous membranes of the upper respiratory tract. Severe burns of the eye may result from liquid or concentrated vapor exposure. Liquid bromine splashed on skin may cause vesicles, blisters, and slow healing ulcers. Continued exposure to low concentrations may result in acne-like skin lesions. These are more common in the oral use of sodium bromide as a sedation.

Systemic—

Inhalation of bromine is corrosive to the mucous membranes of

the nasopharynx and upper respiratory tract, producing brownish discoloration of tongue and buccal mucosa, a characteristic odor of the breath, edema and spasm of the glottis, asthmatic bronchitis, and possibly pulmonary edema which may be delayed until several hours following exposure. A measles-like skin rash may occur. Exposure to high concentrations of bromine can lead to rapid death due to choking caused by edema of the glottis and pulmonary edema.

Bromine has cumulative properties and is deposited in tissues as bromides, displacing other halogens. Exposures to low concentrations result in cough, copious mucous secretions, nose bleeds, respiratory difficulty, vertigo, and headache. Usually these symptoms are followed by nausea, diarrhea, abdominal distress, hoarseness, and asthmatic type respiratory difficulty.

Other effects from chronic exposure have been reported in Soviet literature, e.g., loss of corneal reflexes, joint pains, vegetative disorders, thyroid dysfunction, and depression of the bone marrow. These have not been noted in the U.S. literature.

Hydrogen bromide (hydrobromic acid) is less toxic than bromine, but is an irritant to the mucous membranes of the upper respiratory tract. Long term exposures can cause chronic nasal and bronchial discharge and dyspepsia. Skin contact may cause burns.

MEDICAL SURVEILLANCE

The skin, eyes, and respiratory tract should be given special emphasis during preplacement and periodic examinations. Chest X-rays as well as general health, blood, liver, and kidney function should be considered. Exposure to other irritants or bromine compounds in medications may be important.

SPECIAL TESTS

None commonly used. Blood bromides can be determined but are probably not helpful in following exposures.

PERSONAL PROTECTIVE METHODS

Respiratory protection with gas masks with acceptable canister or supplied air respirators is essential in areas of excessive vapor concentration. Where aqueous solutions or liquids are used, or high vapor concentrations are present, skin and eyes should be protected against spills or splashes by impervious clothing, gloves, aprons, and face shields or goggles.

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CHLORINATED LIME

DESCRIPTION

Chlorinated lime is a white or grayish-white hygroscopic powder with a chlorine odor. It is a relatively unstable chlorine carrier in solid form and is a complex compound of indefinite composition. Chemically, it consists of varying proportions of calcium hypochlorite, calcium chlorite, calcium oxychloride, calcium chloride, free calcium hydroxides, and water. The commercial product generally contains 24-37% available chlorine. On exposure to moisture, chlorine is released.

SYNONYMS

Chloride of lime, bleaching powder.

POTENTIAL OCCUPATIONAL EXPOSURES

Chlorinated lime is a bleaching agent, i.e., it has the ability to chemically remove dyes or pigments from materials. It is used in the bleaching of wood pulp, linen, cotton, straw, oils, and soaps, and in laundering, as an oxidizer in calico printing, a chlorinating agent, a disinfectant, particularly for drinking water and sewage, a decontaminant for mustard gas, and as a pesticide for caterpillars.

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

- Disinfectant makers
- Dyers
- Laundry workers
- Oil bleachers
- Sewage treaters
- Soap bleachers
- Straw bleachers
- Textile bleachers
- Textile printers
- Water treaters
- Wood pulp bleachers

PERMISSIBLE EXPOSURE LIMITS

There is no Federal standard for chlorinated lime. (See Chlorine.)

ROUTES OF ENTRY

Inhalation of dust. Inhalation of vapor and ingestion.

HARMFUL EFFECTS

Local—

The toxic effects of chlorinated lime are due to its chlorine content. The powder and its solutions have corrosive action on skin, eyes, and mucous membranes, can produce conjunctivitis, blepharitis, corneal ulceration, gingivitis, contact dermatitis, and may damage the teeth.

Systemic—

The dust is irritating to the respiratory tract and can produce laryn-

gitis and pulmonary edema. Chlorinated lime is extremely hygroscopic and with the addition of water evolves free chlorine. Inhalation of the vapor is extremely irritating and toxic. (See Chlorine.) Ingestion of chlorinated lime causes severe oral, esophageal, and gastric irritation.

MEDICAL SURVEILLANCE

Consider possible effects on skin, teeth, eyes, or respiratory tract. There are no specific diagnostic tests.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

In dusty areas, the worker should be protected by appropriate respirators. Simple dust masks should not be used since the moisture present in expired air will release the chlorine. Skin effects can be minimized with protective clothing. Most important is the fact that free chlorine is liberated when chlorinated lime comes in contact with water. All precautions should be followed to protect the worker under these circumstances. (See Chlorine.)

CHLORINE

DESCRIPTION

Cl, chlorine, is a greenish-yellow gas with a pungent odor. It is slightly soluble in water and is soluble in alkalis. It is the commonest of the four halogens which are among the most chemically reactive of all the elements.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Gaseous chlorine is a bleaching agent in the paper and pulp and textile industries for bleaching cellulose for artificial fibers. It is used in the manufacture of chlorinated lime, inorganic and organic compounds such as metallic chlorides, chlorinated solvents, refrigerants, pesticides, and polymers, e.g. synthetic rubber and plastics; it is used as a disinfectant, particularly for water and refuse, and in detinning and dezincing iron.

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

- Aerosol propellant makers
- Bleachers
- Chlorinated solvent makers
- Disinfectant makers
- Dye makers
- Flour bleachers
- Iron workers
- Laundry workers
- Paper bleachers
- Pesticide makers
- Plastic makers
- Rayon makers
- Refrigerant makers
- Silver extractors
- Swimming pool maintenance workers
- Tin recovery workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 1 ppm (3 mg/m³). NIOSH has recommended a ceiling limit of 0.5 ppm for a 15-minute sampling period.

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

Chlorine reacts with body moisture to form acids. It is itself extremely irritating to skin, eyes, and mucous membranes, and it may cause corrosion of teeth. Prolonged exposure to low concentrations may produce chloracne.

Systemic—

Chlorine in high concentrations acts as an asphyxiant by causing cramps in the muscles of the larynx (choking), swelling of the mucous membranes, nausea, vomiting, anxiety, and syncope. Acute respiratory distress including cough, hemoptysis, chest pain, dyspnea, and cyanosis develop, and later tracheobronchitis, pulmonary edema, and pneumonia may supervene.

MEDICAL SURVEILLANCE

Special emphasis should be given to the skin, eye, teeth, cardiovascular status in placement and periodic examinations. Chest X-rays should be taken and pulmonary function followed.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Whenever there is likelihood of excessive gas levels, workers should use respiratory protection in the form of fullface gas mask with proper canister or supplied air respirator. The skin effects of chlorine can generally be controlled by good personal hygiene practices. Where very high gas concentrations or liquid chlorine may be present, full protective clothing, gloves, and eye protection should be used. Changing work clothes daily and showering following each shift where exposures exist are recommended.

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FLUORINE and COMPOUNDS

DESCRIPTION

F, elemental fluorine, is a yellow gas. Sulfuric acid reacts with fluorspar producing hydrofluoric acid (HF) which is starting material for synthesis of most fluorine compounds. Fluorine forms fluorides but not fluorates or perfluorates.

SYNONYMS

Fluorine: none.

Hydrogen fluoride: hydrofluoric acid gas, fluohydric acid gas, anhydrous hydrofluoric acid.

Fluorides: none.

POTENTIAL OCCUPATIONAL EXPOSURES

Elemental fluorine is used in the conversion of uranium tetrafluoride to uranium hexafluoride, in the synthesis of organic and inorganic fluorine compounds, and as an oxidizer in rocket fuel.

Hydrogen fluoride, its aqueous solution hydrofluoric acid, and its salts are used in production of organic and inorganic fluorine compounds such as fluorides and plastics; as a catalyst, particularly in paraffin alkylation in the petroleum industry; as an insecticide; and to arrest the fermentation in brewing. It is utilized in the fluorination processes, especially in the aluminum industry, in separating uranium isotopes, in cleaning cast iron, copper, and brass, in removing effloresence from brick and stone, in removing sand from metallic castings, in frosting and etching glass and enamel, in polishing crystal, in decomposing cellulose, in enameling and galvanizing iron, in working silk, in dye and analytical chemistry, and to increase the porosity of ceramics.

Fluorides are used as an electrolyte in aluminum manufacture, a flux in smelting nickel, copper, gold, and silver, as a catalyst for organic reactions, a wood preservative, a fluoridation agent for drinking water, a bleaching agent for cane seats, in pesticides, rodenticides, and as a fermentation inhibitor. They are utilized in the manufacture of steel, iron, glass, ceramics, pottery, enamels, in the coagulation of latex, in coatings for welding rods, and in cleaning graphite, metals, windows, and glassware. Exposure to fluorides may also occur during preparation of fertilizer from phosphate rock by addition of sulfuric acid.

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

- Aluminum fluoride makers
- Aluminum makers
- Bleachers
- Brass cleaners
- Casting cleaners
- Ceramic workers
- Copper cleaners
- Crystal glass polishers
- Fermentation workers
- Fluorochemical workers
- Glass etchers
- Incandescent lamp frosters
- Insecticide makers
- Ore dissolvers
- Stone cleaners
- Uranium refiners
- Yeast makers

PERMISSIBLE EXPOSURE LIMITS

The applicable Federal standards are: fluorine 0.1 ppm (0.2 mg/m^3), fluoride as dust (2.5 mg/m^3), hydrogen fluoride 3 ppm, ceiling 5 ppm, and peak 10 ppm for 30 minutes. For hydrogen fluoride NIOSH has recommended 2.5 mg/m^3 (fluoride ion) TWA with a ceiling of 5 mg/m^3 (fluoride ion) for a 15-minute sampling period.

ROUTES OF ENTRY

Inhalation of gas, mist, dust, or fume; ingestion of dust.

HARMFUL EFFECTS

Local—

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

Systemic—

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

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

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

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

Residential districts which adjoin manufacturing areas can be subjected to continual exposures at minimal levels, or to heavy exposure in the event of accident or plant failure, as in the case of the Meuse Valley disaster.

MEDICAL SURVEILLANCE

Preemployment and periodic examinations should consider possible effects on the skin, eyes, teeth, respiratory tract, and kidneys. Chest X-rays and pulmonary function should be followed. Kidney function should be evaluated. If exposures have been heavy and skeletal fluorosis is suspected, pelvic X-rays may be helpful. Intake of fluoride from natural sources in food or water should be known.

SPECIAL TESTS

In the case of exposure to fluoride dusts, periodic urinary fluoride excretion levels have been very useful in evaluating industrial exposures and environmental dietary sources.

PERSONAL PROTECTIVE METHODS

In areas with execssive gas or dust levels for any type of fluorine, worker protection should be provided. Respiratory protection by dust masks or gas masks with an appropriate canister or supplied air respirator should be provided. Goggles or fullface masks should be used. In areas where there is likelihood of splash or spill, acid resistant clothing including gloves, gauntlets, aprons, boots, and goggles or face shield should be provided to the worker. Personal hygiene should be encouraged, with showering following each shift and before change to street clothes. Work clothes should be changed following each shift, especially in dusty areas. Attention should be given promptly to any burns from fluorine compounds due to absorption of the fluorine at the burn site and the possibility of developing systemic symptoms from absorption from burn sites.

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HYDROGEN CHLORIDE

DESCRIPTION

HCl, hydrogen chloride, is a colorless, nonflammable gas, soluble in water. The aqueous solution is known as hydrochloric acid or muriatic acid and may contain as much as 38% HCl.

SYNONYMS

Anhydrous hydrochloric acid, chlorohydric acid.

POTENTIAL OCCUPATIONAL EXPOSURES

Hydrogen chloride itself is used in the manufacture of pharmaceutical hydrochlorides, chlorine, vinyl chloride from acetylene, alkyl chlorides from olefins, arsenic trichloride from arsenic trioxide; in the chlorination of rubber; as a gaseous flux for babbitting operations; and in organic synthesis involving isomerization, polymerization, alkylation, and nitration reactions.

The acid is used in the production of fertilizers, dyes, dyestuffs, artificial silk, and paint pigments; in refining edible oils and fats; in electroplating, leather tanning, ore refining, soap refining, petroleum extraction, pickling of metals, and in the photographic, textile, and rubber industries.

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

- Battery makers
- Bleachers
- Chemical synthesizers
- Dye makers
- Electroplaters
- Fertilizer makers
- Food processors
- Galvanizers
- Glue makers
- Metal cleaners
- Oil well workers
- Organic chemical synthesizers
- Photoengravers
- Plastic workers
- Rubber makers
- Soap makers
- Tannery workers
- Textile workers
- Tantalum ore refiners
- Tin ore refiners
- Wire annealers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for hydrogen chloride is 5 ppm (7mg/m^3) as a ceiling value.

ROUTE OF ENTRY

Inhalation of gas or mist.

HARMFUL EFFECTS

Local—

Hydrochloric acid and high concentrations of hydrogen chloride gas are highly corrosive to eyes, skin, and mucous membranes. The acid may produce burns, ulceration, and scarring on skin and mucous membranes, and it may produce dermatitis on repeated exposure. Eye con-

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
- Aluminum workers
- Ammunition makers
- Fireworks makers
- Foundry workers
- Petroleum refinery workers
- Plastic 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/m³; total dust, 50 mppcf or 15 mg/m³.

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 (As_2O_x), 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^3 of air as As. NIOSH has recommended 0.002 mg/m^3 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

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 drossing 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^3).

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

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for antimony and its compounds is 0.5 mg/m^3, 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-antimoniosis." 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 ($BaSO_4$) and witherite ($BaCO_3$). 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, mangnesium, 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, glassmaking, 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^3.

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$\mu g/m^3$ as an 8-hour TWA with an acceptable ceiling con-

centration of 5 $\mu g/m^3$. The acceptable maximum peak is 25 $\mu g/m^3$ for a maximum duration of 30 minutes. The standard recommended in the NIOSH Criteria Document is 2 μg Be/m^3 as an 8-hour TWA with a peak value of 25 μg Be/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_1), and measurement of body weight. Beryllium workers should receive a periodic health evaluation that includes: spirometry (FVC and FEV_1), medical history questionaire 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 bismuttite 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, $Na_2B_4O_7.5H_2O$, 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 condensors 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 SiO_2 and alkalies.

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 mg/m^3) as a ceiling value; and Boron oxide 15 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 erythymatous 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
- Gasoline additive makers
- Gasoline makers
- Organic chemical synthesizers
- Plastic makers
- Rocket fuel makers
- Rubber makers

PERMISSIBLE EXPOSURE LIMITS

The applicable Federal standards are: Diborane 0.1 ppm (0.1 mg/m³); Pentaborane 0.005 ppm (0.01 mg/m³); Decaborane 0.05 (0.03 mg/m³) 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
- Braziers
- Bronzers
- Core makers
- Galvanizers
- Junk metal refiners
- Welders
- Zinc founders
- Zinc smelters

PERMISSIBLE EXPOSURE LIMITS

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

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

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for cadmium fume is 0.1 mg/m^3 (as Cd) as an 8-hour TWA with an acceptable ceiling of 3 mg/m^3. For cadmium dust, the standard is 0.2 mg/m^3 (Cd) as an 8-hour TWA with an acceptable maximum ceiling of 0.6 mg/m^3. NIOSH has recommended a TWA limit of 40 μg/m^3 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: $HCo(CO)_4$. Below −26.2 C, exists as light yellow solid. At room temperature, a gas. It begins to decompose in air above −26 C.

Cobalt nitrosocarbonyl: $Co(CO)_3(NO)$. Cherry red liquid.

Iron tetracarbonyl: $(Fe(CO)_4)_3$. Dark green lustrous crystals.

Iron pentacarbonyl: $Fe(CO)_5$. Viscous yellow liquid.

Iron nonacarbonyl: $Fe_2(CO)_5$. Yellow to orange crystals.

Iron carbonyl hydride: $H_2Fe(CO)_4$. A gas. Begins to decompose at −10 C.

Iron nitrosyl carbonyl: $Fe(NO)_2(CO)_2$. Dark red crystals.

Molybdenum hexacarbonyl: $Mo(CO)_6$. White crystals.

Nickel carbonyl: $Ni(CO)_4$. Colorless liquid.

Osmium carbonyl chloride: $Os(CO)_2Cl_3$. Dark brown. Deliquescent.

Ruthenium pentacarbonyl: $Ru(CO)_5$. Colorless liquid. Very volatile.

Tungsten carbonyl: $W(CO)_6$. 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. $Fe(CO)_5$ 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 $Fe(CO)_5$; slowly flowing water or gas in an iron pipe generating $Fe(CO)_5$; and in the Fischer-Tropsch process for the liquefaction of coal.

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

Acetylene welders
Blast furnace workers
Metal refiners
Mond process workers
Nickel refiners
Organic chemical synthesizers
Petroleum refinery 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. $Ni(CO)_4$ 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 (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
- Copper etchers
- Electroplaters
- Glass workers
- Lithographers
- Metal workers
- Oil purifiers
- Photoengravers
- Photographers
- Process engravers
- Stainless steel workers
- Textile workers
- Welders

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for chromic acid and chromates is 0.1 mg/m^3 as a ceiling concentration. The NIOSH Criteria for a Recommended Standard would set work place limits for chromic acid of 0.05 mg/m^3 as chromium trioxide as a TWA with a ceiling concentration of 0.1 mg/m^3 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}Co) is used in the treatment of cancer.

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

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

PERMISSIBLE EXPOSURE LIMITS

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

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 mg/m^3, and for copper dusts and mists, 1 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
- Dental alloy makers
- Electroplaters
- Glass makers
- Phosphor makers
- Rectifier makers
- Semiconductor makers
- Transistor makers
- Vacuum tube 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^3).

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 Fe_2O_3) and brown hematite (brown iron ore, mainly limonite, a hydrated sesquioxide of iron). Magnetic iron oxide, Fe_3O_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
- Bessemer operators
- Electric arc welders
- Flame cutters
- Friction saw operators
- Metalizers
- Seam welders
- Stainless steel makers
- Steel foundry workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for iron oxide fume is 10 mg/m³. 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 $Ni(CO)_4$ 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
- Brass founders
- Ceramic makers
- Enamel workers
- Glass makers
- Imitation pearl makers
- Insecticide workers
- Lubricant makers
- Match makers
- Painters
- Plumbers
- Solderers

PERMISSIBLE EXPOSURE LIMITS

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

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 coproporphyric 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^3 and for tetramethyl lead 0.07 mg Pb/m^3.

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

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for magnesium oxide fume is 15 mg/m^3.

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

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for mercury is 1 mg/10m^3 as a ceiling value. The recommended standard is 0.05 mg Hg/m^3 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 - $CH_3HgNHC(:NH)NHCN$. Soluble in water.

Ethyl mercury compounds: ethylmercuric chloride: C_2H_5HgCl. Insoluble in water. Ethylmercuric phosphate: $(C_2H_5Hg)PO$. Soluble in water. N-(Ethylmercuric)-p-toluenesulphonanilide: $C_6H_5N(HgC_2H_5)$-$SO_2C_6H_4CH_3$. 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/m³ as an 8-hour TWA with an acceptable ceiling of 0.04 mg/m³.

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. MoO_3 sublimes 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^3. Molybdenum: insoluble compounds, 15 mg/m^3.

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

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for nickel metal and its soluble compounds is I mg/m^3 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

$Ni(CO)_4$, 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
Gas platers
Mond process workers
Organic chemical synthesizers
Petroleum refinery workers

PERMISSIBLE EXPOSURE LIMITS

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

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 (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
- Histology technicians
- Organic chemical synthesizers
- Synthetic ammonia makers
- Platinum hardeners

PERMISSIBLE EXPOSURE LIMITS

There is presently no Federal standard for osmium itself; the standard for osmium tetroxide is 0.002 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 preplacement 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_3, 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
- Cement workers
- Firemen
- Grain fumigators
- Metal refiners
- Metal slag workers
- Metallic phosphate workers
- Organic chemical synthesizers
- Rustproofers
- Welders

PERMISSIBLE EXPOSURE LIMITS

0.3 ppm (0.4 mg/m^3) 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 lacrimation, 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. Phorphorus 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
Bronze alloy makers
Chlorinated compound makers
Electroluminescent coating makers
Fertilizer makers
Fireworks makers
Hydraulic fluid makers
Incendiary makers
Metal refiners
Metallic phosphide makers
Munitions workers
Pesticide workers
Rat poison workers
Semiconductor makers
Smoke bomb makers

PERMISSIBLE EXPOSURE LIMITS

Federal standards are: Phosphorus (yellow) 0.1 mg/m³, phosphoric acid 1.0 mg/m³, phosphorus trichloride 3.0 mg/m³, phosphorus pentachloride 1.0 mg/m³, phosphorus pentasulfide 1.0 mg/m³.

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 Na_2PtCl_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, H_2PtCl_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
- Catalyst workers
- Ceramic workers
- Dental alloy makers
- Drug makers
- Electronic equipment makers
- Electroplaters
- Gasoline additive makers
- Indelible ink makers
- Jewelry makers
- Laboratory ware makers
- Mirror makers
- Spark plug makers
- Zinc etchers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for soluble salts of platinum is 0.002 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
- Copper smelters
- Electric rectifier makers
- Glass makers
- Organic chemical synthesizers
- Pesticide makers
- Photographic chemical makers
- Pigment makers
- Plastic workers
- Pyrite roasters
- Rubber makers
- Semiconductor makers
- Sulfuric acid makers
- Textile workers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are: Selenium compounds (as Se): 0.2 mg/m³. Selenium hexafluoride: 0.05 ppm, 0.4 mg/m³. Hydrogen selenide: 0.05 ppm, 0.2 mg/m³.

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^3).

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

PERMISSIBLE EXPOSURE LIMITS

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

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 pigmentated.

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, toe nails, 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
- Ceramic makers
- Copper refinery workers
- Electronic workers
- Enamel makers
- Foundry workers
- Glass makers
- Lead refinery workers
- Porcelain makers
- Rubber workers
- Semiconductor makers
- Silverware makers
- Stainless steel makers
- Thermoelectric device makers

PERMISSIBLE EXPOSURE LIMITS

The applicable Federal standards are: Tellurium: 0.1 mg/m³. Tellurium hexafluoride: 0.02 ppm (mg/m³).

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

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for thallium (soluble compounds) is 0.1 mg Tl/m³.

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 thorinite, 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 $1X10^{-6}$ μCi/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 (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 mg/m^3 and for inorganic compounds excluding the oxides it is 2.0 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 pneumoconosis 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 radioopaque, 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_2, 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^3. 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^3; and uranium, insoluble compounds, 0.25 mg/m^3.

ROUTES OF ENTRY

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

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

Uranyl fluoride, UO_2F_2.

Uranium pentachloride, UCl_5.

Uranium trioxide (uranyl oxide), UO_3.
Sodium diuranate (sodium uranate (VI), $Na_2U_2O_7.H_2O$).
Ammonium diuranate (ammonium uranate (VI) $(NH_4)_2U_2O_7$).
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^3, and V_2O_5 fume, 0.1 mg/m^3, 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 conjuctiva. 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

$ZnCl_2$, 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,
- Dental cement makers
- Deodorant makers
- Disinfectant makers
- Dry cell battery makers
- Dye makers
- Embalmers
- Military personnel
- Paper makers
- Petroleum refinery workers
- Rubber workers
- Solderers
- Taxidermists
- Textile finishers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for zinc chloride fume is 1 mg/m^3.

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^3, there were complaints of irritation of the nose, throat, and chest after 2 minutes. With exposure to 80 mg/m^3 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
- Brass foundry workers
- Ceramic makers
- Chemical synthesizers
- Cosmetic makers
- Electroplaters
- Galvanizers
- Lacquer makers
- Paint makers
- Pigment makers
- Plastic makers
- Rubber workers
- Welders

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for zinc oxide fume is 5 mg/m^3.

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

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for zirconium compounds is 5 mg/m³ 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

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

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for ammonia is an 8-hour time weighted average of 50 ppm (35 mg/m^3). NIOSH has recommended 50 ppm expressed as a ceiling and determined by a 5-minute sampling period.

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

Contact with anhydrous liquid ammonia or with aqueous solutions is intensely irritating to the mucous membranes, eyes, and skin. Eye symptoms range from lacrimation, blepharospasm, and palpebral edema to a rise of intraocular pressure, and other signs resembling acute-angle closure glaucoma, corneal ulceration, and blindness. There may be corrosive burns of skin or blister formation. Ammonia gas is also irritating to the eyes and to moist skin.

Systemic—

Mild to moderate exposure to the gas can produce headache, salivation, burning of throat, anosmia, perspiration, nausea, vomiting, and substernal pain. Irritation of ammonia gas in eyes and nose may be sufficiently intense to compel workers to leave the area. If escape is not possible, there may be severe irritation of the respiratory tract with the production of cough, glottal edema, bronchospasm, pulmonary edema, or respiratory arrest. Bronchitis or pneumonia may follow a severe exposure if patient survives. Urticaria is a rare allergic manifestation from inhalation of the gas.

MEDICAL SURVEILLANCE

Preemployment physical examinations for workers in ammonia exposure areas should be directed toward significant changes in the skin, eyes, and respiratory system. Persons with corneal disease, and glaucoma, or chronic respiratory diseases may suffer increased risk. Periodic examinations should include evaluation of skin, eyes, and respiratory system, and pulmonary function tests to compare with baselines established at preemployment examination.

SPECIAL TESTS

None.

PERSONAL PROTECTIVE METHODS

Where ammonia hazards exist in concentrations above the standard, respiratory, eye, and skin protection should be provided. Fullface gas masks with ammonia canister or supplied air respirators, both with full facepieces, afford good protection. In areas where exposure to liquid ammonia occurs, goggles or face shields, as well as protective clothing impervious to ammonia and including gloves, aprons, and boots should be required. Where ammonia gas or concentrated ammonia solution is

splashed in eyes, immediate flooding of the eyes with large quantities of water for 15 minutes or longer is advised, followed at once by medical examination. In heavy concentrations of ammonia gas, workers should be outfitted with complete self-contained protective suits impervious to ammonia, with supplied air source, and full headpiece and facepiece. Work clothes wetted with concentrated ammonia solutions should be changed immediately, and the exposed area of the body washed thoroughly with water.

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CALCIUM OXIDE

DESCRIPTION

CaO, calcium oxide, occurs as white or grayish-white lumps or granular powder. The presence of iron gives it a yellowish or brownish tint. It is soluble in water and acids.

SYNONYMS

Lime, burnt lime, quicklime, calx, fluxing lime.

POTENTIAL OCCUPATIONAL EXPOSURES

Calcium oxide is used as a refractory material, a binding agent in bricks, plaster, mortar, stucco and other building materials, a dehydrating agent, a flux in steel manufacturing, and a laboratory agent to absorb CO_2; in the manufacture of aluminum, magnesium, glass, pulp and paper, sodium carbonate, calcium hydroxide, chlorinated lime, calcium salts, and other chemicals; in the flotation of nonferrous ores, water and sewage treatment, soil treatment in agriculture, dehairing hides, the clarification of cane and beet sugar juice, and in fungicides, insecticides, drilling fluids, and lubricants.

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

- Brick masons
- Fertilizer makers
- Fungicide workers
- Glass makers
- Insecticide makers
- Metal smelters
- Mortar workers
- Paper makers
- Plaster makers
- Steel workers
- Sugar refiners
- Tannery workers
- Water treaters

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for calcium oxide is 5 mg/m³.

ROUTE OF ENTRY

Inhalation of dust.

HARMFUL EFFECTS

Local—

The irritant action of calcium oxide is due primarily to its alkalinity and exothermic reaction with water. It is irritating and may be caustic to the skin, conjunctiva, cornea, and mucous membranes of upper respiratory tract, may produce burns or dermatitis with desquamation and vesicular rash, lacrimation, spasmodic blinking, ulceration, and ocular perforation, ulceration and inflammation of the respiratory passages, ulceration of nasal and buccal mucosa, and perforation of nasal septum.

Systemic—

Bronchitis and pneumonia have been reported from inhalation of dust. The lower respiratory tract is generally not affected because irritation of upper respiratory passages is so severe that workers are forced to leave the area.

MEDICAL SURVEILLANCE

Preemployment physical examinations should be directed to significant problems of the eyes, skin, and the upper respiratory tract. Periodic examinations should evaluate the skin, changes in the eyes, especially the cornea and conjunctiva, muscosal ulcerations of the nose, mouth, and nasal septum, and any pulmonary symptoms. Smoking history should be known.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

In areas where workers are exposed to calcium oxide levels above the standard, protection to the skin, eyes, and respiratory tract should be provided. Skin protection can be provided by protective clothing and gloves. All dusty area workers should be provided with goggles and dust masks with proper cartridges. Personal hygiene is to be encouraged, with frequent change of work clothes and showering after each shift before change to street clothes.

CARBON DIOXIDE

DESCRIPTION

CO_2, carbon dioxide, is a colorless, odorless, non-combustible gas, soluble in water. It is commonly sold in the compressed liquid form, and the solid form (dry ice).

SYNONYMS

Carbonic acid gas, carbonic anhydride.

POTENTIAL OCCUPATIONAL EXPOSURES

Gaseous carbon dioxide is used to carbonate beverages, as a weak acid in the textile, leather, and chemical industries, in water treatment,

and in the manufacture of aspirin and white lead, for hardening molds in foundries, in food preservation, in purging tanks and pipelines, as a fire extinguisher, in foams, and in welding. Because it is relatively inert, it is utilized as a pressure medium. It is also used as a propellant in aerosols, to promote plant growth in green houses; it is used medically as a respiratory stimulant, in the manufacture of carbonates, and to produce an inert atmosphere when an explosive or flammable hazard exists. The liquid is used in fire extinguishing equipment, in cylinders for inflating life rafts, in the manufacturing of dry ice, and as a refrigerant. Dry ice is used primarily as a refrigerant.

Occupational exposure to carbon dioxide may also occur in any place where fermentation processes may deplete oxygen with the formation of carbon dioxide, e.g., in mines, silos, wells, vats, ships' holds, etc.

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

- Aerosol packagers
- Beverage carbonators
- Blast furnace workers
- Brewery workers
- Carbonic acid makers
- Charcoal burners
- Chemical synthesizers
- Explosive makers
- Fire extinguisher makers
- Firemen
- Foundry workers
- Grain elevator workers
- Inert atmosphere welders
- Insecticide makers
- Miners
- Refrigerating car workers
- Refrigerating plant workers
- Soda makers
- Tannery workers
- Textile workers
- Vatmen
- Well cleaners

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5,000 ppm (9,000 mg/m^3).

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

Frostbite may result from contact with dry ice or gas at low temperature.

Systemic—

Carbon dioxide is a simple asphyxiant. Concentrations of 10% (100,000 ppm) can produce unconsciousness and death from oxygen deficiency. A concentration of 5% may produce shortness of breath and headache. Continuous exposure to 1.5% CO_2 may cause changes in some physiological processes. The concentration of carbon dioxide in the blood affects the rate of breathing.

MEDICAL SURVEILLANCE

No special considerations are necessary although persons with cardiovascular or pulmonary disease may be at increased risk.

SPECIAL TESTS

None in common use.

PERSONAL PROTECTIVE METHODS

Carbon dioxide is a heavy gas and accumulates at low levels in depressions and along the floor. Generally, adequate ventilation will provide sufficient protection for the worker. Where concentrations are of a high order, supplied air respirators are recommended.

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CARBON MONOXIDE

DESCRIPTION

CO, carbon monoxide, is a colorless, odorless, tasteless gas, partially soluble in water, but one which decomposes.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Carbon monoxide is used in metallurgy as a reducing agent, particularly in the Mond process for nickel; in organic synthesis, especially in the Fischer-Tropsch process for petroleum products and in the oxo reaction; and in the manufacture of metal carbonyls.

It is usually encountered in industry as a waste product of incomplete combustion of carbonaceous material (complete combustion produces CO_2). The major source of CO emission in the atmosphere is the gasoline-powered internal combustion engine. Specific industrial processes which contribute significantly to CO emission are iron foundries particularly the cupola; fluid catalytic crackers, fluid coking, and moving-bed catalytic crackers in petroleum refining; lime kilns and Kraft recovery furnaces in Kraft paper mills; furnace, channel, and thermal operations in carbon black plants; beehive coke ovens, basic oxygen furnaces, sintering of blast furnace feed in steel mills; and formaldehyde manufacture. There are numerous other operations in which a flame touches a surface that is cooler than the ignition temperature of the gaseous part of the flame where exposure to CO may occur, e.g., arc welding, automobile repair, traffic control, tunnel construction, fire fighting, mines, use of explosives, etc.

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

Acetylene workers	Metal oxide reducers
Blast furnace workers	Miners
Boiler room workers	Mond process workers
Brewery workers	Organic chemical synthesizers
Carbon black makers	Petroleum refinery workers
Coke oven workers	Pulp and paper workers
Diesel engine operators	Steel workers
Garage mechanics	Water gas workers

PERMISSIBLE EXPOSURE LIMITS

The present Federal standard is 50 ppm (55 mg/m^3). The standard recommended by NIOSH is 35 ppm with a ceiling value of 200 ppm. This latter value is to limit carboxyhemoglobin formation to 5% in a nonsmoker engaged in sedentary activity at normal altitude.

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

None.

Systemic—

Carbon monoxide combines with hemoglobin to form carboxyhemoglobin which interferes with the oxygen carrying capacity of blood, resulting in a state of tissue hypoxia. The typical signs and symptoms of acute CO poisoning are headache, dizziness, drowsiness, nausea, vomiting, collapse, coma, and death. Initially the victim is pale; later the skin and mucous membranes may be cherry-red in color. Loss of consciousness occurs at about the 50% carboxyhemoglobin level. The amount of carboxyhemoglobin formed is dependent on concentration and duration of CO exposure, ambient temperature, health, and metabolism of the individual. The formation of carboxyhemoglobin is a reversible process. Recovery from acute poisoning usually occurs without sequelae unless tissue hypoxia was severe enough to result in brain cell degeneration.

Carbon monoxide at low levels may initiate or enhance deleterious myocardial alterations in individuals with restricted coronary artery blood flow and decreased myocardial lactate production.

Severe carbon monoxide poisoning has been reported to permanently damage the extrapyramidal system, including the basal ganglia.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should give special attention to significant cardiovascular disease and any medical conditions which could be exacerbated by exposure to CO. Heavy smokers may be at greater risk. Methylene chloride exposure may also cause an

increase of carboxyhemoglobin. Smokers usually have higher levels of carboxyhemoglobin than nonsmokers (often 5 - 10% or more).

SPECIAL TESTS

Carboxyhemoglobin levels are reliable indicators of exposure and hazard.

PERSONAL PROTECTIVE METHODS

Under certain circumstances where carbon monoxide levels are not exceedingly high, gas masks with proper canisters can be used for short periods but are not recommended. In areas with high concentrations, self-contained air apparatus is recommended.

GRAPHITE

DESCRIPTION

Graphite is crystallized carbon and usually appears as soft, black scales. There are two types of graphite, natural and artificial.

SYNONYMS

Plumbago, black lead, mineral carbon.

POTENTIAL OCCUPATIONAL EXPOSURES

Natural graphite is used in foundry facings, steelmaking, lubricants, refractories, crucibles, pencil "lead," paints, pigments, and stove polish. Artificial graphite may be substituted for these uses with the exception of clay crucibles; other types of crucibles may be produced from artificial graphite. Additionally, it may be used as a high temperature lubricant or for electrodes. It is utilized in the electrical industry in electrodes, brushes, contacts, and electronic tube rectifier elements; as a constituent in lubricating oils and greases; to treat friction elements, such as brake linings; to prevent molds from sticking together; and in moderators in nuclear reactors.

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

- Brake lining makers
- Cathode ray tube makers
- Commutator brush makers
- Crucible makers
- Electrode makers
- Explosive makers
- Foundry workers
- Lubricant makers
- Match makers
- Nuclear reactor workers
- Paint makers
- Pencil lead makers
- Pigment makers
- Refractory material makers
- Steel makers
- Stove polish makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for natural graphite is 15 mppcf.

ROUTE OF ENTRY

Inhalation of dust.

HARMFUL EFFECTS

Local—

None.

Systemic—

Exposure to natural graphite may produce a progressive and disabling pneumoconiosis similar to anthracosilicosis. Symptoms include headache, coughing, depression, decreased appetite, dyspnea, and the production of black sputum. Some individuals may be asymptomatic for many years then suddenly become disabled. It has not yet been determined whether the free crystalline silica in graphite is solely responsible for development of the disease. There is evidence that artificial graphite may be capable of producing a pneumoconiosis.

MEDICAL SURVEILLANCE

Preemployment and periodic examinations should be directed toward detecting significant respiratory disease, through chest X-rays and pulmonary function tests.

SPECIAL TESTS

None.

PERSONAL PROTECTIVE METHODS

Workers in exposed areas should be provided with dust masks with proper cartridges and should be instructed in their maintenance.

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HYDROGEN PEROXIDE

DESCRIPTION

H_2O_2, anhydrous hydrogen peroxide, is a colorless rather unstable liquid with a bitter taste. Hydrogen peroxide is completely miscible with water and is commercially sold in concentrations of 3, 35, 50, 70, and 90 percent solutions.

SYNONYMS

Peroxide, hydrogen dioxide, hydroperoxide.

POTENTIAL OCCUPATIONAL EXPOSURES

Hydrogen peroxide is used in the manufacture of acetone, antichlor, antiseptics, benzol peroxide, buttons, disinfectants, pharmaceuticals, felt hats, plastic foam, rocket fuel, and sponge rubber. It is also used in

bleaching bone, feathers, flour, fruit, fur, gelatin, glue, hair, ivory, silk, soap, straw, textiles, wax, and wood pulp, and as an oxygen source in respiratory protective equipment. Other specific occupations with potential exposure include liquor and wine agers, dyers, electroplaters, fat refiners, photographic film developers, wool printers, veterinarians, and water treaters.

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

- Acetone makers
- Alcoholic beverage agers
- Antichlor makers
- Antiseptic makers
- Benzol peroxide makers
- Button makers
- Disinfectant makers
- Drug makers
- Dyers
- Electroplaters
- Fat refiners
- Hide disinfectors
- Metal cleaners
- Photographic film developers
- Plastic foam makers
- Rocket fuel workers
- Sponge rubber makers
- Veterinarians
- Water treaters
- Wool printers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for hydrogen peroxide (90 percent) is 1 ppm (1.4 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor or mist.

HARMFUL EFFECTS

Local—

The skin, eyes, and mucous membranes may be irritated by concentrated vapor or mist. Bleaching and a burning sensation may occur at lower levels, while high concentrations may result in blistering and severe eye injury, which may be delayed in appearance.

Systemic—

Inhalation of vapor or mist may produce pulmonary irritation ranging from mild bronchitis to pulmonary edema. No chronic systemic effects have been observed.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should be directed to evaluation of the general health with particular reference to the skin, eyes, mucous membranes, and respiratory tract.

SPECIAL TESTS

None.

PERSONAL PROTECTIVE METHODS

In areas where concentrated hydrogen peroxide is being used, if there is danger of spill or splash, skin protection should be provided by protective clothing, gloves, goggles, and boots. Where fumes or vapor are excessive, workers should be provided with gas masks with full face pieces and proper canisters or supplied air respirators. Additional health hazards may occur from the decomposition of hydrogen peroxide. Oxygen, possibly at high pressure, may form, which may create an explosion hazard. Hydrogen peroxide is generally handled in a closed system to prevent contamination.

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HYDROGEN SULFIDE

DESCRIPTION

H_2S, hydrogen sulfide, is a flammable, colorless gas with a characteristic rotten-egg odor and is soluble in water.

SYNONYMS

Sulfuretted hydrogen, hydrosulfuric acid, stink damp.

POTENTIAL OCCUPATIONAL EXPOSURES

Hydrogen sulfide is used in the synthesis of inorganic sulfides, sulfuric acid, and organic sulfur compounds, as an analytical reagent, as a disinfectant in agriculture, and in metallurgy. It is generated in many industrial processes as a by-product and also during the decomposition of sulfur-containing organic matter, so potential for exposure exists in a variety of situations. Hydrogen sulfide is found in natural gas, volcanic gas, and in certain natural spring waters. It may also be encountered in the manufacture of barium carbonate, barium salt, cellophane, depilatories, dyes and pigments, felt, fertilizer, adhesives, viscose rayon, lithopone, synthetic petroleum products; in the processing of sugar beets; in mining, particularly where sulfide ores are present; in sewers and sewage treatment plants; during excavation of swampy or filled ground for tunnels, wells, and caissons; during drilling of oil and gas wells; in purification of hydrochloric acid and phosphates; during the low temperature carbonization of coal; in tanneries, breweries, slaughterhouses; in fat rendering; and in lithography and photoengraving.

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

Barium carbonate makers
Brewery workers
Caisson workers
Cellophane makers
Coke oven workers
Depilatory makers
Dye makers
Fat renderers
Felt makers
Lithographers
Miners
Natural gas makers
Paper pulp makers
Photoengravers
Rayon makers
Sewage treatment plant workers
Sewer workers
Silk makers
Slaughterhouse workers
Soap makers
Sugar beet processors
Sulfuric acid purifiers
Sulfur makers
Synthetic fiber makers
Tannery workers
Tunnel workers
Well diggers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is a ceiling value of 20 ppm (30 mg/m^3) with a maximum peak above this value for an 8-hour shift of 50 ppm (75 mg/m^3) for a maximum duration of 10 minutes once only if no other measurable exposure occurs.

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

Palpebral edema, bulbar conjunctivitis, keratoconjunctivitis, and ocular lesions may occur when hydrogen sulfide comes in contact with the eyes. Photophobia and lacrimation may also develop. Direct irritation of the respiratory tract may cause rhinitis, pharyngitis, bronchitis, and pneumonia. Hydrogen sulfide may penetrate deep into the lungs and cause hemorrhagic pulmonary edema. Hydrogen sulfide's irritative effects are due to the formation of alkali sulfide when the gas comes in contact with moist tissues.

Systemic—

Acute exposure may cause immediate coma which may occur with or without convulsions. Death may result with extreme rapidity from respiratory failure. Post-mortem signs include a typical greenish cyanosis of the chest and face with green casts found in viscera and blood. The toxic action of hydrogen sulfide is thought to be due to inhibition of cytochrome oxidase by binding iron which is essential for cellular respiration. Subacute exposure results in headache, dizziness, staggering gait, and excitement suggestive of neurological damage, and nausea and diarrhea suggestive of gastritis. Recovery is usually complete although rarely polyneuritis may develop as a result of vestibular and extrapyra-

midal tract damage. Tremors, weakness, and numbness of extremities may also occur. Physicians may observe a "rotten-egg" breath and abnormal electrocardiograms in victims. Systemic effects from chronic exposure to hydrogen sulfide have not been established.

MEDICAL SURVEILLANCE

Preplacement medical examinations should evaluate any preexisting neurological, eye, and respiratory conditions and any history of fainting seizures.

SPECIAL TESTS

None in common use for surveillance purposes.

PERSONAL PROTECTIVE METHODS

Hydrogen sulfide's strong odor, noticeable at low concentrations, is a poor warning sign as it may cause olfactory paralysis, and some persons are congenitally unable to smell H_2S.

Accidental exposure may occur when workers enter sewage tanks and other confined areas in which hydrogen sulfide is formed by decomposition. In a number of cases workers enter unsuspectingly and collapse almost immediately. Workers, therefore, should not enter enclosed spaces without proper precautions.

All Federal standard and other safety precautions must be observed when tanks or other confined spaces are to be entered. In areas where the exposure to hydrogen sulfide exceeds the standards, workers should be provided with fullface canister gas masks or preferably supplied air respirators.

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NITROGEN OXIDES

DESCRIPTION

Nitrogen oxides include:
Nitrous oxide: N_2O
Nitric oxide: NO
Nitrogen dioxide: NO_2

Nitrogen trioxide: N_2O_3
Nitrogen tetroxide: N_2O_4
Nitrogen pentoxide: N_2O_5
Nitric acid: HNO_3
Nitrous acid: HNO_2

Nitrous oxide, N_2O, is a colorless, noncombustible gas, sweet-tasting, and slightly soluble in water. Nitric oxide is a colorless gas slightly soluble in water. Nitric oxide combines with oxygen to form nitrogen dioxide which is a reddish-brown gas with a characteristic odor. Nitrogen dioxide exists in equilibrium with nitrogen tetroxide, and these two compounds and oxygen are in equilibrium with the crystalline nitrogen pentoxide. However, nitrogen dioxide and nitric oxide are the dissociation products of nitrogen trioxide. When nitrogen dioxide comes in contact with water, nitrous acid and nitric acid are formed. Nitric acid is a colorless liquid when pure, but on exposure to light, the liquid may turn yellowish-brown as a result of nitrogen dioxide formation. Nitric acid mist almost always contains nitrogen oxide gases and is, therefore, included in this group. Nitrogen dioxide decomposes in water, Nitrogen pentoxide is slightly soluble in water and nitric acid (70% aqueous solution) is soluble in water.

SYNONYMS

Nitrous oxide: Nitrogen monoxide
Nitric oxide: Mononitrogen monoxide
Nitrogen dioxide: None
Nitrogen trioxide: Dinitrogen trioxide, nitrous anhydride
Nitrogen tetroxide: Dinitrogen tetroxide
Nitrogen pentoxide: Nitric anhydride
Nitrous acid: None
Nitric acid: Aqua fortis, azotic acid, hydrogen nitrate

POTENTIAL OCCUPATIONAL EXPOSURES

Exposure to nitrogen oxides is typically a mixed exposure to "nitrous fumes" which may evolve from various manufacturing processes and in many other industrial situations. Exposure to nitrogen oxides may occur during the manufacture of nitric and sulfuric acid, oxidized cellulose compounds, explosives, rocket propellants, fertilizers, dyes and dyestuffs, pharmaceuticals, and various other organic and inorganic chemicals such as nitrites, nitrates, and other nitro compounds, aqua regia, arsenic acid, oxalic acid, nitrous acid, phthalic acid, and phosphoric acid. Exposure may also occur during jewelry manufacturing, etching, brazing, lithographing, metal cleaning, textile (rayon) and food bleaching, glass blowing, electroplating, gas and electric arc welding, and during the nitration of chloroform. Nitrogen oxides also occur in garages from automobile exhaust, in silos from organic material decomposition, in tunnels following blasting, and when nitric acid comes in contact with organic materials.

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

Braziers	Medical technicians
Dentists	Metal cleaners
Dye makers	Nurses
Fertilizer makers	Organic chemical synthesizers
Food and textile bleachers	Photoengravers
Garage workers	Physicians
Gas and electric arc welders	Silo fillers
Jewelry makers	Sulfuric acid makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standards are: nitric oxide — 25 ppm (30 mg/m^3), nitrogen dioxide — 5 ppm (9 mg/m^3), and nitric acid — 2 ppm (5 mg/m^3); determined as a TWA. Currently there are no standards for the other listed compounds. NIOSH has recommended a ceiling level of 1 ppm for nitrogen dioxide.

ROUTES OF ENTRY

Inhalation of gas in the case of nitrogen oxide gases; inhalation of the mist or vapor, in the case of nitric and nitrous acids.

HARMFUL EFFECTS

Local—

Nitrogen oxide gases may produce irritation of the eyes and mucous membranes. Prolonged low level exposure may produce yellowish or brownish staining of the skin and teeth; however, this sign usually indicates nitric acid exposure. Nitric acid and nitrogen tetroxide are extremely corrosive liquids and may cause severe burns, ulcers, and necrosis of the skin, mucous membranes, and eye tissues.

Systemic—

Exposure to high concentrations of nitrogen oxides may result in severe pulmonary irritation and methemoglobinemia. The former is believed to be caused by the nitrogen dioxide portion, while the latter is mainly caused by nitric oxide. It is postulated that nitric oxide is nonirritating but the distinction is of questionable importance since nitric oxide exposure generally includes other nitrogen oxides; moreover, nitric oxide at even moderate concentrations oxidizes rapidly and spontaneously in the presence of atmospheric oxygen.

Nitrogen dioxide at high concentrations has also been shown to cause methemoglobinemia in the dog. Typically, acute exposure may produce immediate malaise, cyanosis, cough, dyspnea, chills, fever, headache, nausea, and vomiting. Collapse and death may occur if exposure is sufficiently high. When lower concentrations are encountered, there may be only mild signs of bronchial irritation followed by a five- to twelve-hour symptom-free period. Subsequently, the onset of signs and symptoms of acute pulmonary edema occur suddenly, which unfortunately may take place away from prompt medical aid.

Nitrogen oxides may be formed from green silage in amounts which,

when restrained to the confines of a silo, may constitute a serious health hazard. "Silo-filler's disease" is the name used to designate the syndrome culminating in bronchiolitis fibrosa obliterans, caused by exposure to nitrogen oxides evolved in this way.

If the acute episode is survived, bronchiolitis fibrosa obliterans may develop usually within a few days but may be latent for as long as six weeks. Victims may develop severe and increasing dyspnea which is often accompanied by fever and cyanosis. Chest roentgenogram may reveal a diffuse, reticular, and fine nodular infiltration or numerous, uniform, scattered nodular densities ranging in size from 1 to 5 mm in diameter.

Chronic exposure may result in pulmonary dysfunction with decreased vital capacity, maximum breathing capacity and lung compliance, and increased residual volume. The most common complaint is of dyspnea upon exertion. Signs include moist rales and wheezes, sporadic cough with mucopurulent expectoration, a decrease in blood pH and serum proteins, and an increase in urinary hydroxyproline and acid mucopolysaccharides. These findings are suggestive of emphysema, although they are as yet inconclusive.

The development of methemoglobinemia is typically mild and transient. In rare cases individuals may have a preexisting constantly high methemoglobin level due to a genetic defect. Such individuals are more susceptible to toxic methemoglobinemia.

MEDICAL SURVEILLANCE

Preplacement and periodic examinations should be concerned particularly with the skin, eyes, and with significant pulmonary and heart diseases. Periodic chest X-rays and pulmonary function tests may be useful. Smoking history should be known. Methemoglobin studies may be of interest if exposure to nitric oxide is present. In the case of nitric acid vapor mist exposure, dental effects may be present.

SPECIAL TESTS

None.

PERSONAL PROTECTIVE METHODS

Workers should not enter confined areas where nitrogen oxides may accumulate (for example, silos) without appropriate eye and respiratory protection.

Individuals should be equipped with supplied air respirators with full face piece or chemical goggles, and enclosed areas should be properly ventilated before entering. An observer equipped with appropriate respiratory protection should be outside the area and standing by to supply any aid needed.

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OZONE

DESCRIPTION

O_3, ozone, is a bluish gas with a characteristic pungent odor, slightly soluble in water. Ozone is found naturally in the atmosphere as a result of the action of solar radiation and electrical storms. It is also formed around electrical sources such as X-ray or ultraviolet generators, electric arcs, mercury vapor lamps, linear accelerators, and electrical discharges.

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Ozone is used as an oxidizing agent in the organic chemical industry (e.g., production of azelaic acid); as a disinfectant for food in cold storage rooms and for water (e.g., public water supplies, swimming pools, sewage treatment); for bleaching textiles, waxes, flour, mineral oils and their derivatives, paper pulp, starch, and sugar; for aging liquor and wood; for processing certain perfumes, vanillin, and camphor; in treating industrial wastes; in the rapid drying of varnishes and printing inks; and in the deodorizing of feathers.

Industrial exposure often occurs around ozone generating sources, particularly during inert-gas shielded arc welding.

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

- Air treaters
- Arc welders
- Cold storage food preservers
- Industrial waste treaters
- Liquor agers
- Odor controllers
- Oil bleachers
- Organic chemical synthesizers
- Sewage treaters
- Textile bleachers
- Water treaters
- Wax bleachers
- Wood agers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 0.1 ppm (0.2 mg/m^3).

ROUTES OF ENTRY

Inhalation of the gas.

HARMFUL EFFECTS

Local—

Ozone is irritating to the eyes and all mucous membranes. In human exposures, the respiratory signs and symptoms in order of increasing ozone concentrations are: dryness of upper respiratory passages; irritation of mucous membranes of nose and throat; choking, coughing, and severe fatigue; bronchial irritation, substernal soreness, and cough. Pulmonary edema may occur, sometimes several hours after exposure has ceased. In severe cases, the pulmonary edema may be fatal.

Animal experiments demonstrate that ozone causes inflammation and congestion of respiratory tract and, in acute exposure, pulmonary edema, hemorrhage, and death.

Chronic exposure of laboratory animals resulted in chronic bronchitis, bronchiolitis, emphysematous and fibrotic changes in pulmonary parenchyma.

Systemic—

Symptoms and signs of subacute exposure include headache, malaise, shortness of breath, drowsiness, reduced ability to concentrate, slowing of heart and respiration rate, visual changes, and decreased desaturation of oxyhemoglobin in capillaries. Animal experiments with chronic exposure showed aging effects and acceleration of lung tumorigenesis in lung-tumor susceptible mice.

Animal experiments further demonstrated that tolerance to acute pulmonary effects of ozone is developed and that this provided cross tolerance to other edemagenic agents. Antagonism and synergism with other chemicals also occur.

Ozone also has radiomimetic characteristics, probably related to its free-radical structure. Experimentally produced chromosomal aberrations have been observed.

MEDICAL SURVEILLANCE

Preemployment and periodic physical examinations should be concerned especially with significant respiratory diseases. Eye irritation may also be important. Chest X-rays and periodic pulmonary function tests are advisable.

SPECIAL TESTS

None.

PERSONAL PROTECTIVE METHODS

In areas of excessive concentration, gas masks with proper canister and full face piece or goggles or the use of supplied air respirators is recommended.

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PHOSGENE

DESCRIPTION

$COCl_2$, phosgene, is a colorless, noncombustible gas with a sweet, not pleasant odor in low concentrations. In higher concentrations, it is irritating and pungent. It decomposes in water but is soluble in organic solvents.

SYNONYMS

Carbonyl chloride, carbon oxychloride, carbonic acid dichloride, chloroformyl chloride, combat gas.

POTENTIAL OCCUPATIONAL EXPOSURES

Phosgene is used in the manufacture of dyestuffs based on triphenylmethane, coal tar, and urea. It is also used in the organic synthesis of isocyanates and their derivatives, carbonic acid esters (polycarbonates), and acid chlorides. Occasional applications include its utilization in metallurgy, and in the manufacture of some insecticides and pharmaceuticals.

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

- Chlorinated compound makers
- Drug makers
- Dye makers
- Firemen
- Insecticide makers
- Metallurgists
- Organic chemical synthesizers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for concentrations of phosgene in air is 0.1 ppm (0.4 mg/m^3).

ROUTE OF ENTRY

Inhalation of gas.

HARMFUL EFFECTS

Local—

Conjunctivitis, lacrimation, and upper respiratory tract irritation may develop from gas. Liquid may cause severe burns.

Systemic—

Acute exposure to phosgene may produce pulmonary edema frequently preceded by a latent period of 5-6 hours but seldom longer than 12 hours. The symptoms are dizziness, chills, discomfort, thirst, increasingly tormenting cough, and viscous sputum. Sputum may then be-

come thin and foamy, and dyspnea, a feeling of suffocation, tracheal rhonci, and grey-blue cyanosis may follow. Death may result from respiratory or cardiac failure. The hazard of phosgene is increased because at low levels (205 mg/m³) it is lacking in warning symptoms.

Chronic exposure to phosgene may result in some tolerance to acute edemagenic doses, but may cause irreversible pulmonary changes of emphysema and fibrosis. Animal experimentation has shown an increased incidence of chronic pneumonitis and acute and fibrinous pneumonia from exposure to this agent.

MEDICAL SURVEILLANCE

Preemployment medical examinations should include chest X-rays and baseline pulmonary function tests. The eyes and skin should be examined. Smoking history should be known. Periodic pulmonary function studies should be done. Workers who are known to have inhaled phosgene should remain under medical observation for at least 24 hours to insure that delayed symptoms do not occur.

SPECIAL TESTS

None.

PERSONAL PROTECTIVE METHODS

Where liquid phosgene is encountered, protective clothing should be supplied which is impervious to phosgene. Where gas is encountered above safe limits, fullface gas masks with phosgene canister or supplied air respirators should be used.

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PORTLAND CEMENT

DESCRIPTION

Portland cement is a class of hydraulic cements whose two essential constituents are tricalcium silicate and dicalcium silicate with varying amounts of alumina, tricalcium aluminate, and iron oxide. The quartz content of most is below one percent. The average composition of regular Portland cement is as follows:

CaO: 64%
SiO_2: 21%
Al_2O_3: 5.8%
FeO_3: 2.9%
MgO: 2.5%
Alkali Oxides: 1.4%
SO_3: 1.7%

SYNONYMS

None.

POTENTIAL OCCUPATIONAL EXPOSURES

Cement is used as a binding agent in mortar and concrete (a mixture of cement, gravel, and sand). Potentially hazardous exposure may occur during both the manufacture and use of cement.

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

- Asbestos cement workers
- Brick masons
- Bridge builders
- Building construction workers
- Burial vault builders
- Cement workers
- Concrete workers
- Drain tile makers
- Heat insulation makers
- Oil well builders
- Silo builders
- Storage tank builders
- Tunnel builders
- Water pipe makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for Portland cement is 50 mppcf.

ROUTE OF ENTRY

Inhalation of dust.

HARMFUL EFFECTS

Local—

Exposure may produce cement dermatitis which is usually due to primary irritation from the alkaline, hygroscopic, and abrasive properties of cement. Chronic irritation of the eyes and nose may occur. In some cases, cement workers have developed an allergic sensitivity to constituents of cement such as hexavalent chromate. It is not unusual for cement dermatitis to be prolonged and to involve covered areas of the body.

Systemic—

No documented cases of pneumoconiosis or other systemic manifestations attributed to finished Portland cement exposure have been reported. Conflicting reports of pneumoconiosis from cement dust appear related to exposures that occurred in mining, quarrying, or crushing silica-containing raw materials.

MEDICAL SURVEILLANCE

Preemployment and periodic medical examinations should stress significant respiratory problems, chest X-ray, pulmonary function tests, smoking history, and allergic skin sensitivities, especially to chromates. The eyes should be examined.

SPECIAL TESTS

Patch test studies may be useful in dermatitis cases.

PERSONAL PROTECTIVE METHODS

In areas exceeding safe dust levels, masks with proper cartridges

should be provided. Gloves, barrier creams, and protective clothing (long sleeved shirts, etc.) will help protect workers subject to dermatitis. Personal hygiene is very important, and all cement workers should be encouraged to shower following each shift before changing to street clothes. Freshly laundered work clothing should be supplied on a daily basis.

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SODIUM HYDROXIDE/POTASSIUM HYDROXIDE

DESCRIPTION

NaOH, sodium hydroxide, is a white, deliquescent material sold as pellets, flakes, lumps, or sticks. It is soluble in water, alcohol, and glycerine. Aqueous solutions are known as soda lye.

KOH, potassium hydroxide, exists as white or slightly yellow deliquescent lumps, rods, or pellets. It is soluble in water. Aqueous solutions are known as lye.

SYNONYMS

Sodium hydroxide: caustic soda, caustic alkali, caustic flake, sodium hydrate.

Potassium hydroxide: potassium hydrate, caustic potash, potassa, caustic alkali.

POTENTIAL OCCUPATIONAL EXPOSURES

Sodium hydroxide is utilized to neutralize acids and make sodium salts in petroleum refining, viscose rayon, cellophane, and plastic production, and in the reclamation of rubber. It hydrolyzes fats to form soaps, and it precipitates alkaloids and most metals from aqueous solutions of their salts. It is used in the manufacture of mercerized cotton, paper, explosives, and dyestuffs, in metal cleaning, electrolytic extraction of zinc, tin plating, oxide coating, laundering, bleaching, and dishwashing, and it is used in the chemical industries.

Potassium hydroxide is used in the manufacture of liquid soap, as a mordant for wood, as a carbon dioxide absorber, in mercerizing cotton, in electroplating, photoengraving, and lithography, in printing inks, in paint and varnish removers, and in analytical chemistry, organic synthesis, and the production of other potassium compounds.

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

Bleachers	Paint removers
Bleach makers	Paper makers
Cellophane makers	Photoengravers
Chemical laboratory workers	Printers
Dye makers	Printing ink makers
Electroplaters	Rayon makers
Etchers	Rubber reclaimers
Explosive makers	Soap makers
Laundry workers	Textile bleachers
Lithographers	Tin platers
Mercerizers	Varnish removers
Organic chemical synthesizers	Zinc extractors

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for sodium hydroxide is 2 mg/m^3. There is no standard for potassium hydroxide.

ROUTE OF ENTRY

Inhalation of dust or mist.

HARMFUL EFFECTS

Local—

Both compounds are extremely alkaline in nature and are very corrosive to body tissues. Dermatitis may result from repeated exposure to dilute solutions in the form of liquids, dusts, or mists.

Systemic—

Systemic effects are due entirely to local tissue injury. Extreme pulmonary irritation may result from inhalation of dust or mist.

MEDICAL SURVEILLANCE

The skin, eyes, and respiratory tract should receive special attention in any placement or periodic examination.

SPECIAL TESTS

None.

PERSONAL PROTECTIVE METHODS

Protection should be provided by impervious protective clothing, rubber boots, face and eye shields, and dust respirators. All skin area burns, especially of the eyes, demand immediate care by flooding with large quantities of water for 15 minutes or longer and specialized medical care.

SULFUR CHLORIDE

DESCRIPTION

S_2Cl_2, sulfur chloride, is a fuming, oily liquid with a yellowish-red

to amber color and a suffocating odor. It has an added hazard since it oxidizes and hydrolyzes to sulfur dioxide and hydrogen chloride.

SYNONYMS

Sulfur monochloride, sulfur subchloride, disulfur dichloride.

POTENTIAL OCCUPATIONAL EXPOSURES

Sulfur chloride finds use as a chlorinating agent and an intermediate in the manufacture of organic chemicals, e.g., carbon tetrachloride, and sulfur dyes, insecticides, synthetic rubber, and pharmaceuticals. Exposure may also occur during the extraction of gold, purification of sugar juice, finishing and dyeing textiles, processing vegetable oils, hardening wood, and vulcanization of rubber.

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

- Carbon tetrachloride makers
- Drug makers
- Gold extractors
- Insecticide makers
- Rubber workers
- Sugar juice purifiers
- Sulfur dye makers
- Synthetic rubber makers
- Textile dye makers and finishers
- Vegetable oil processors
- Wood hardeners

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for sulfur chloride (sulfur monochloride) is 1 ppm (6 mg/m^3).

ROUTE OF ENTRY

Inhalation of vapor.

HARMFUL EFFECTS

Local—

Fumes, in sufficient quantity, may cause severe irritation to eyes, skin, and mucous membranes of the upper respiratory tract.

Systemic—

Although this compound is capable of producing severe pulmonary irritation, very few serious cases of industrial exposure have been reported. This is probably because the pronounced irritant effects of sulfur chloride serve as an immediate warning signal when concentration of the gas approaches a hazardous level.

MEDICAL SURVEILLANCE

Preemployment and periodic examinations should give special emphasis to the skin, eyes, and respiratory system. Pulmonary function tests may be useful. Exposures may also include sulfur dioxide and hydrochloric acid. (See these compounds).

SPECIAL TESTS

None are known to be useful.

PERSONAL PROTECTIVE METHODS

In areas where vapor levels are excessive, workers should be supplied with fullface gas masks with proper canister or supplied air respirators with full face piece. Skin protection can usually be afforded by work clothes and barrier creams, but under certain instances (spills, etc.), full impervious protective suits may be necessary.

SULFUR DIOXIDE

DESCRIPTION

SO_2, sulfur dioxide, is a colorless gas at ambient temperatures with a characteristic strong suffocating odor. It is soluble in water and organic solvents.

SYNONYMS

Sulfurous anhydride, sulfurous oxide.

POTENTIAL OCCUPATIONAL EXPOSURES

Sulfur dioxide is used in the manufacture of sodium sulfite, sulfuric acid, sulfuryl chloride, thionyl chloride, organic sulfonate, disinfectants, fumigants, glass, wine, ice, industrial and edible protein, and vapor pressure thermometers. It is also used in the bleaching of beet sugar, flour, fruit, gelatin, glue, grain, oil, straw, textiles, wicker ware, wood pulp, and wool; in the tanning of leather; in brewing and preserving; and in the refrigeration industry. Exposure may also occur in various other industrial processes as it is a by-product of ore smelting, coal and fuel oil combustion, paper manufacturing, and petroleum refining.

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

- Beet sugar bleachers
- Boiler water treaters
- Brewery workers
- Disinfectant makers
- Diesel engine operators and repairmen
- Firemen
- Fumigant makers
- Furnace operators
- Gelatin bleachers
- Glass makers
- Ice makers
- Ore smelter workers
- Paper makers
- Petroleum refinery workers
- Protein makers
- Refrigeration workers
- Sodium sulfite makers
- Sulfuric acid makers
- Tannery workers
- Thermometer makers (vapor)
- Wine makers
- Wood bleachers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard is 5 ppm (13 mg/m^3). NIOSH has recommended lowering this standard to 2 ppm as a TWA.

ROUTE OF ENTRY

Inhalation of gas. Direct contact of gas or liquid phase on skin and mucous membranes.

HARMFUL EFFECTS

Local—

Gaseous sulfur dioxide is particularly irritating to mucous membranes of the upper respiratory tract. Chronic effects include rhinitis, dryness of the throat, and cough. Conjunctivitis, corneal burns, and corneal opacity may occur following direct contact with liquid.

Systemic—

Acute over-exposure may result in death from asphyxia. Survivors may later develop chemical bronchopneumonia with bronchiolitis obliterans. Bronchoconstriction with increased pulmonary resistance, high-pitched rales, and a tendency to prolongation of the expiratory phase may result from moderate exposure, though bronchoconstriction may be asymptomatic. The effects on pulmonary function are increased in the presence of respirable particles.

Chronic exposure may result in nasopharyngitis, fatigue, altered sense of smell, and chronic bronchitis symptoms such as dyspnea on exertion, cough, and increased mucous excretion. Transient stimulation of erythropoietic activity of the bone marrow has been reported. Slight tolerance, at least to the odor threshold, and general acclimatization are common. Sensitization in a few individuals, particularly young adults, may also develop following repeated exposures. There is some evidence that some individuals may be innately hypersusceptible to SO_2. Animal experimentation has also indicated that sulfur dioxide may be a possible co-carcinogenic agent.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should be concerned especially with the skin, eye, and respiratory tract. Pulmonary function should be evaluated, as well as smoking habits, and exposure to other pulmonary irritants.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

In areas where levels of sulfur dioxide gas are excessive, the worker should be supplied with fullface piece cartridge or canister respirator or with supplied air respirators. Goggles, protective clothing, and gloves should be worn if splashes with liquid are likely. Work clothing should be changed at least twice a week to freshly laundered work clothes. Showering following each work shift should be encouraged. In areas of splash or spill, impervious clothing should be supplied, but if work clothes are wetted by sulfur dioxide, they should be promptly removed and the skin area thoroughly washed.

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SULFURIC ACID

DESCRIPTION

H_2SO_4, concentrated sulfuric acid, is a colorless, odorless, oily liquid which is commercially sold at 93 to 98% H_2SO_4, the remainder being water. Fuming sulfuric acid (oleum) gives off free sulfur trioxide and is a colorless or slightly colored, viscous liquid. Sulfuric acid is soluble in water and alcohol.

SYNONYMS

Oil of vitriol, spirit of vitriol, spirit of sulfur, hydrogen sulfate.

POTENTIAL OCCUPATIONAL EXPOSURES

Sulfuric acid is used as a chemical feedstock in the manufacture of acetic acid, hydrochloric acid, citric acid, phosphoric acid, aluminum sulfate, ammonium sulfate, barium sulfate, copper sulfate, phenol, superphosphates, titanium dioxide, as well as synthetic fertilizers, nitrate explosives, artificial fibers, dyes, pharmaceuticals, detergents, glue, paint, and paper. It finds use as a dehydrating agent for esters and ethers due to its high affinity for water, as an electrolyte in storage batteries, for the hydrolysis of cellulose to obtain glucose, in the refining of mineral and vegetable oil, and in the leather industry. Other uses include fur and food processing, carbonization of wool fabrics, gas drying, uranium extraction from pitchblende, and laboratory analysis.

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

- Aluminum sulfate makers
- Battery makers
- Cellulose workers
- Chemical synthesizers
- Copper sulfate makers
- Detergent makers
- Dye makers
- Explosive makers
- Food processors
- Glue makers
- Jewelers
- Leather workers
- Metal cleaners
- Paint makers
- Paper makers
- Phenol makers

PERMISSIBLE EXPOSURE LIMITS

The Federal standard for sulfuric acid is 1 mg/m^3.

ROUTE OF ENTRY

Inhalation of mist.

HARMFUL EFFECTS

Local—

Burning and charring of the skin are a result of the great affinity for, and strong exothermic reaction with, water. Concentrated sulfuric acid

will effectively remove the elements of water from many organic materials with which it comes in contact. It is even more rapidly injurious to mucous membranes and exceedingly dangerous to the eyes. Ingestion causes serious burns of the mouth or perforation of the esophagus or stomach. Dilute sulfuric acid does not possess this property, but is an irritant to skin and mucous membranes due to its acidity and may cause irreparable corneal damage and blindness as well as scarring of the eyelids and face.

Systemic—

Sulfuric acid mist exposure causes irritation of the mucous membranes, including the eye, but principally the respiratory tract epithelium. The mist also causes etching of the dental enamel followed by erosion of the enamel and dentine with loss of tooth substance. Central and lateral incisors are mainly affected. Breathing high concentrations of sulfuric acid causes tickling in the nose and throat, sneezing, and coughing. At lower levels sulfuric acid causes a reflex increase in respiratory rate and diminution of depth, with reflex bronchoconstriction resulting in increased pulmonary air flow resistance. A single overexposure may lead to laryngeal, tracheobronchial, and pulmonary edema. Repeated excessive exposures over long periods have resulted in bronchitic symptoms, and rhinorrhea, lacrimation, and epistaxis. Long exposures are claimed to result in conjunctivitis, frequent respiratory infections, emphysema, and digestive disturbances.

MEDICAL SURVEILLANCE

Preplacement and periodic medical examinations should give special consideration to possible effects on the skin, eyes, teeth, and respiratory tract. Pulmonary function tests should be performed.

SPECIAL TESTS

None commonly used.

PERSONAL PROTECTIVE METHODS

In all areas where liquid sulfuric acid is handled, impervious clothing should be provided, including gloves, goggles or face mask, rubber suits, and rubber shoes. Any work clothing wetted by sulfuric acid should be immediately changed and the skin area thoroughly washed and flooded with water. In areas where mist or gas is excessive, gas masks with appropriate canister or supplied air respirators should be provided. In either instance the worker should be supplied with full face protection.

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