CRITERIA GUIDE TO BE USED FOR REPORTING OCCUPATIONAL DISEASES

# Maine Occupational Disease Surveillance Program

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# INTRODUCTION

This document has been developed to support 22 MRSA Chapter 259-A Subsection 1491-1495, which mandates that any physician or hospital diagnosing specified occupational disease are required to report them to the Department of Human Services within 30 days. The purpose of this criteria guide is to aid you in determining when these conditions are reportable. Diagnosed or suspected diagnoses are both acceptable and a place has been provided on the reporting form to specify which one is applicable.

Other services are available through this system as well. They include the following:

- Follow-up of selected agricultural injuries by agricultural safety and health nurses
- Access to general and specific information on chemicals.
- Referral to other state programs for information or follow-up services (including industrial hygiene consultation)
- Statistical analyses and reports completed on certain topics.

To access any of the above services or if you have questions, write to:

Occupational Health Program
Bureau of Health
State House Station 11
Augusta, ME 04333
OR

call: (207) 287-5378

This document was orginally compiled by Steve Shannon, DO, MPH and has been updated to include agriculturally related injuries as a reportable condition. The following individuals reviewed and wrote portions of this updated document: John Bielecki, MD, Workplace Health Services, and Maine Institute for Occupational Health Education; Carol Eckert, MD, Sheepscot Valley Health Center and Maine Institute for Occupational Health Education; Allison Hawkes, MD, MS, Occupational Health Program, Bureau of Health; Lebelle Hicks, DABT, Maine Board of Pesticides Control; Anthony Tomassoni, MD, MS, Maine Poison Center.

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# AGRICULTURAL INJURIES TO CHILDREN and ADULTS (also includes forestry and fishing)

## *Criteria for Reporting*

Reportable injuries include any injury associated with agricultural work which requires care by a hospital, physician, physician extender, or chiropractor. Since persons live on farms, as well as, work on farms, injuries that occur to persons living on a farm are reportable (this includes children).

In addition, agricultural work includes not only farmwork, but also, jobs in nurseries, logging, and fishing. Some examples of agricultural occupations are listed below.

**As with farmwork, children may assist their parents with logging or fishing. This "work" may be paid or unpaid. If children are injured while assisting their parents or other adults in this manner, the injuries are reportable.**

- Production Agriculture (either crop or livestock) - occupations include farmers; managers and operators of farms; employees on farms; supervisors; graders, sorters, and inspectors of agricultural products; and nursery employees.

  Common crops in Maine include blueberries, potatoes, a variety of vegetables, cranberries, flax, apples, strawberries, raspberries, and maple syrup. Examples of common animals raised in Maine are sheep, cows, alpacas, chicken, goats, llamas, ostriches and horses. Livestock products include poultry, eggs, milk, beef, beefalo, venison, wool (sheep and alpaca), and cheese.

- Forestry - timber cutting and logging occupations, other forestry workers, and supervisors of forestry and logging workers.

- Fishing - Fishermen, captains and other officers or personnel on fishing vessels, marine life cultivation workers (aquaculture), urchin divers.

# ASBESTOSIS

## *Description of Disease*

Asbestosis refers to the accumulation of asbestos dust (fibers) in the lungs and the tissue reaction to its presence. Strictly speaking, it refers to a fibrotic reaction in the lung parenchyma. Usually, at least 10 years of exposure are required before the small irregular opacities suggestive of asbestosis appear in the lower lung zones on the chest radiograph. Asbestosis may cause impairment of lung volumes and gas exchange and premature mortality. In some cases, symptoms (e.g., decreased diffusing capacity) may occur before chest radiograph changes are visible. Typical lung tissue pathology includes asbestos bodies with diffuse fibrosis (peribronchiolar, interstitial, or honeycomb).

## *Major Occupations at Risk*

Examples of occupations where there may be asbestos exposure include:

- Working in a primary or secondary asbestos industry (e.g., manufacture of asbestos textiles, cleaning of machinery or plants where asbestos is used).
- Mining.
- Construction and demolition trades (insulation workers, pipefitters).
- Shipbuilding and repair (asbestos lagging).
- Pipefitters and millwrights working in older facilities
- Asbestos abatement workers

## *Criteria for Reporting*

To meet the definition for reporting both A & B must be present.

A. History of exposure to airborne asbestos dust.

B. Chest radiograph consistent with asbestosis and/or lung tissue pathology consistent with asbestos exposure.

# BYSSINOSIS

## *Description of Disease*

Byssinosis is a pneumonopathy due to the inhalation of cotton dust. The worker usually develops chest tightness and wheezing on return to the mill on Monday morning (also called "Monday morning asthma"). In the early course of the disease, the symptoms disappear by Tuesday or Wednesday. After several years of exposure, chronic cough and sputum production (i.e., chronic bronchitis) are present. Finally, it can cause permanent obstruction of the small airways resulting in pulmonary impairment.

## *Major Occupations at Risk*

- Cotton workers.
- Cotton processing from gin to finished goods.

## *Criteria for Reporting*

To meet the definition for reporting, A, B, and C must be present.

A. Typical history of chest tightness or shortness of breath particularly on the first day of the work week.

B. Drop in $FEV_1$ (i.e. change of more than 5% preferably 10% during the workday or work week).

C. History of exposure to cotton dust.

# CARPAL TUNNEL SYNDROME

## *Description of Disease*

Manifestations of carpal tunnel syndrome (CTS) include pain, numbness and weakness in the median nerve distribution of the hand as a result of compression or irritation of the median nerve as it passes through the carpal tunnel in the wrist. Without intervention, CTS can lead to marked discomfort, impaired hand function, and disability. Workers who perform repetitive tasks are at risk for CTS.

## *Major Occupations at Risk*

- Garment workers
- Grocery checkers
- Electronic assembly workers
- Packers
- Millwrights
- Carpenters
- Butchers
- Typists/word processors
- Musicians
- Housekeepers/cooks
- Data processors
- Pipefitters

## *Criteria for Reporting*

To meet the definition for reporting, A, B, and C must be present.

A. One or more of the following symptoms suggestive of CTS is present: paresthesias, hypoesthesia, pain or numbness affecting at least part of the median nerve distribution of the hands(s). Symptoms should have lasted at least 1 week or, if intermittent, have occurred on multiple occasions. Other causes of hand numbness or parethesias, such as cervical radiculopathy, thoracic outlet syndrome, and pronator teres syndrome, should be excluded by appropriate clinical evaluation.

B. Objective findings consistent with CTS are present in the affected hand(s) and Wrist(s). These include:

1. Physical examination findings (which can include any of the following): Tinel's sign present; positive Phalen's Test; diminished/absent sensation to pin prick in the median nerve distribution of the hand; evidence of motor dysfunction; thenar wasting; fasciculations.

OR

2. Electrodiagnostic findings indicative of median nerve dysfunction across the carpal tunnel

C. Evidence of work-relatedness suggested by a history of a job involving one or more of the following activities before the development of symptoms. A temporal relationship of symptoms to work or an association with cases of CTS in co-workers performing similar tasks is also evidence of work-relatedness.

1. Frequent repetitive use of the same or similar movements of the hand or wrist on the affected side(s).
2. Regular tasks requiring the generation of high force by the hand on the affected side(s).
3. Regular or sustained tasks requiring awkward hand positions on the affected side(s). Awkward hand positions predisposing to CTS include the use of a pinch grip (as when holding a pencil), extreme flexion, extension, or ulnar deviation of the wrist, and use of the fingers with the wrist flexed.
4. Regular use of vibrating hand-held tools.
5. Frequent or prolonged pressure over the wrist or base of the palm on the affected side(s).

# HEAVY METAL POISONING

## *Criteria for Reporting*

To meet the definition for reporting, both A and B must be present.

### I. ARSENIC

A. History of significant exposure to arsenic in occupations such as battery, bearing, cable and glass manufacture; tanning and taxidermy; manufacture, mixing and application of arsenical pesticides; and smelting of arsenical ores.

B. Urine arsenic (preferably 24 hour) greater than 50 ug/l.

### II. CADMIUM

A. History of significant exposure to cadmium in industries such as electroplating of metals, manufacturing alkali storage batteries, and plastics.

B. Blood cadmium levels greater than 1 ug/dl. Urine cadmium levels greater than 5 ug/l.

### III. LEAD

A. History of significant exposure to airborne lead in industries such as lead smelters and storage battery factories, bridge workers, painters, sandblasters (removing paint), radiator shops, soldering, mechanics.

B. Blood lead levels greater than 25 ug/dl. Urine lead levels greater than 80 ug/l.

### IV. MERCURY

A. History of significant exposure to mercury in industries such as chloralkali plants, mining and refining of mercury, manufacture of mercury containing instruments and occupations such as histology and dental technicians.

B. Blood mercury levels greater than 0.5 ug/dl. Urine mercury levels greater than 20 ug/l.

# HYPERSENSITIVITY PNEUMONITIS

## *Description of Disease*

Human lungs and airways can respond in a variety of ways to a large number of organic and inorganic agents that could be present in an occupational environment. There is an overlapping of asthma with bronchitis, and asthma with the fibrotic disease of the lung, such as hypersensitivity pneumonitis. The clinical presentation of hypersensitivity pneumonitis depends on the immunologic response of the host, the antigenicity and size of the agent, and the intensity and frequency of exposure. In addition, such a respiratory response can occur in an acute or chronic form.

Acute Hypersensitivity Pneumonitis. The symptoms usually occur four to six hours after exposure and consist of chills, fever to 104°, malaise, cough and shortness of breath. Symptoms persist for 12-18 hours with spontaneous recovery. There is reoccurrence of episodes with exposure to the offending agent. Laboratory studies reveal leukocytosis, and significant eosinophilia (up to 10%) may occur in some patients. Chest x-ray may be normal if episodes are widely spaced, or fine nodules may be seen with a general coalescence of bronchovascular markings. Pulmonary function studies are often abnormal.

Chronic Hypersensitivity Pnemonitis. Some individuals exposed to a particular antigen for a prolonged period may develop irreversible pulmonary damage which is mainly clinically manifested by chronic shortness of breath, weakness, weight loss and shows a restrictive ventilatory defect on pulmonary function studies.

## *Major Occupations at Risk*

The agents listed below are not to be considered a complete list of all agents. There are probably other agents which are associated with hypersensitivity pnemonitis in Maine occupations.

- Insulation Workers (Fiberglass)
- Wood Workers (Fungi in wood dust)
- Farmers (Spores in moldy compost)
- Bird breeders (Animal proteins-avian)

## *Criteria for Reporting*

To meet definition for reporting, both A and B must be present.

A. History of significant exposure to an airborne antigen known to be associated with hypersensitivity pnemonitis.

B. Signs and symptoms characteristic of the condition as described, and if antigen is available, serologic studies utilizing sera and antigens to detect antibodies in the affected individual.

# MESOTHELIOMA

## *Description of Disease*

Malignant mesothelioma of the pleura and peritoneum is an extremely rare tumor in the general population, but it has been observed to account for up to 10 percent of deaths in several asbestos-exposed cohorts. The absence of other recognized etiologies for this malignancy makes the diagnosis quite specific for a past history of asbestos exposure. After diagnosis, few individuals with malignant mesothelioma survive beyond one year, regardless of therapy. Mesothelioma can occur from low-level, transient, and indirect exposure. It shares with asbestos-induced lung cancer a long latency period (decades), but differs from it by exhibiting no association with cigarette smoking.

## *Major Occupations at Risk*

Examples of occupations where there may be asbestos exposure include:

- Working in a primary or secondary asbestos industry (e.g., manufacture of asbestos textiles, cleaning of machinery or plants where asbestos is used).
- Mining.
- Construction and demolition trades (insulation workers, pipefitters).
- Shipbuilding and repair (asbestos lagging).

## *Criteria for Reporting*

Tumor tissue pathology consistent with mesothelioma.

# OCCUPATIONAL ASTHMA

## *Description of Disease*

Occupational asthma is asthma caused by a workplace exposure. More specifically, occupational asthma is reversible generalized airway obstruction that results from acquired bronchial hyperresponsiveness to some substance(s) or process present in the workplace.

## *Occupations at Risk*

Exposure may occur in many different occupations. A few examples of agents known to cause occupational asthma are shown in the following list :

- Wood dust
- Insect debris
- Grain dusts & grain products
- Fish meal and emulsions
- Persulphates
- Formaldehyde
- Flour
- Other animal antigens
- Natural resins
- Animal fat, oil and products
- Nickel salts
- Toluene

## *Criteria for Reporting*

Diagnosis of occupational asthma requires establishing that a patient has asthma (see A below) and that the asthma is associated with some substance(s) or process in the workplace (see B, C or D below).

Substances can produce immediate, late or "dual" bronchospastic responses with each episode of exposure. As a result, the patterns of association between work and airflow obstruction are varied and not always apparent.

A. The following are recommended criteria for evidence of "significant" variability in airways obstruction (only 1 of the criteria need be met):
   1) improvement of at least 10% in $FEV_1$ with bronchodilator;
   2) decrease of $FEV_1$ by more than 10% from baseline either spontaneously or in response to workplace exposure;
   3) At least 20% variability in serial peak expiratory flow rate (PEFR) measurements in a 24 hour period;
   4) Positive inhalation challenge testing with methacholine or histamine (20% fall in $FEV_1$ produced by 5 inhalations of 8 mg/ml or less).

B. Any of the following symptom patterns should suggest an occupational etiology.
   1) symptoms occur at work only;
   2) worsen Monday morning (or first day back to work);
   3) improve on weekends or vacations;
   4) occur in evening on workdays only;
   5) worsen during the course of each work week;
   6) resolve after change in work environment.

C. The following are recommended criteria for significant work-related changes in airways obstruction:
   1) greater than 10% decrease in $FEV_1$ across a workshift;
   2) greater than 10% improvement after removal from exposure (over weekend, during vacation or after removal of suspect agent from workplace);
   3) greater than 20% variation of PEFR in relation to work (either decreasing across workshift or each evening of workdays or improvement during days away from work).

D. A decline of greater than 15% in $FEV_1$ after inhalation challenge with substance present in workplace at same or lower concentration than encountered at work is considered a positive response.

# OUTBREAKS

Any outbreak of an illness involving two or more persons which appears to have resulted from either acute or chronic exposures to chemical, biological, or physical agents in the same occupational setting. Examples would be burns from electromagnetic radiation or dermatitis from a new chemical agent or process.

# PESTICIDE POISONING

Tradenames used below are not intended to be an exhaustive list nor does their use constitute an endorsement by the State of Maine. Pesticides used in Maine are listed in the index by tradename and active ingredient.

## *Description of Disease*

There are several major categories of pesticides. Pesticides are products designed to kill, repel or otherwise mitigate pests. As a group, "pesticides" includes a wide variety of chemical classes with different mechanisms of action. Pesticide products are composed of the active ingredients (active against the target pest) and inactive ingredients (solvents, carriers, surface wetting agents etc.).

The toxicity of a pesticide product will depend on the following:

1. Type of pesticide: insecticides, herbicides, fungicides, or rodenticides etc.,
2. The chemical class of the active ingredient: bipyridyls, organophosphates, carbamates, pyrethroids, insect growth regulators etc.,
3. The individual active ingredient: parathion vs malathion, paraquat vs diquat etc.
4. Presence of inactive ingredients: solvents, carriers, emulsifying agents etc.

The exposure to the pesticide will depend on:

1. Formulations: dusts, ready-to-use sprays, or emulsifiable concentrates,
2. The type of application equipment,
3. The type and condition of personal protective clothing worn, and
4. Frequency and duration of the exposure.

**RISK (HEALTH EFFECT) = TOXICITY X EXPOSURE**

The health of the individual is another important factor in pesticide poisonings. For example, persons with underlying neurological, renal, or hepatic disease may experience adverse health effects from pesticides at lower doses. Genetic make-up, e.g., polymorphisms, may also influence susceptibility.

### *Where to obtain information about pesticide toxicity*

Information regarding a specific product is best obtained from the manufacturer, product label or Material Safety Data Sheets (MSDS).

Information about pesticide toxicity is also available from The Maine Poison Control Center (Tel. 1-800-442-6305) and the Maine Board of Pesticides Control (Tel. 207- 287-2731).

## *Criteria for Reporting*

To meet the definition for reporting, both A and B must be present.

A. History of employment in the manufacture, mixing, handling or application of pesticides. This includes agricultural workers defined as handlers under the Federal Worker Protection Standard 40 CFR 170 (WPS).

B. Signs and symptoms of pesticide exposure with laboratory confirmation if possible.

Listed below and on the next several pages, are examples of the most widely used pesticide products in Maine. Included in the description of these products are their mechanisms of action in the target species (the species the product works on) and nontarget species (humans, pests, beneficial insects etc.,); the signs and symptoms characteristic of exposure, and laboratory confirmation techniques.

**I. INSECTICIDES**

***A. ORGANOPHOSPHATES***

Organophosphate insecticides (selected tradenames) used in Maine include: disulfoton (Disyston), azinphos-methyl (Guthion, Sniper), fonofos (Dyfonate), methamidophos (Monitor), phosmet (Imidan), ethoprop (Mocap), chlorpyrifos (Dursban, Lorsban), diazinon (Diazinon), acephate (Orthene).

Mechanism of action on both target and nontarget species: Irreversible cholinesterase inhibition of acetyl cholinesterase (AchE) in Red Blood Cells and Brain, and in Plasma cholinesterase resulting in a decrease of cholinesterase activity in the central, parasympathetic and sympathetic nervous systems.

2) Signs and symptoms: Tightness in the chest, wheezing, increased salivation and lacrimation, increased sweating, nausea, vomiting, abnormal cramps, diarrhea, tenesmus and involuntary defecation, involuntary urination, and constriction of the pupils. Muscular effects include weakness, involuntary twitching, and cramps. Central nervous system effects include anxiety, restlessness, insomia, headache, emotional instability, neurosis, excessive dreaming and nightmares, apathy and confusion. Slurred speech, tremor, ataxia, depression of circulatory centers, and coma are other CNS effects. Death results from respiratory failure. Onset of symptoms occurs within 4 to 12 hours, usually within 4 hrs.

3) Laboratory confirmation: A 25% depression below normal values of plasma and/or RBC cholinesterase enzyme as measured by any of the generally available methods. When using this method, the individual's post-exposure cholinesterase results should be compared with his/her own pre-exposure baseline, obtained from the same lab, using the same technique. Pre-exposure baselines should be obtained pre-season. Identification of metabolites in the urine is another confirmatory test.

### *B. CARBAMATES*

Carbamates insecticides (selected tradenames) used in Maine: oxamyl (Vydate), formentanate HCl (Carzol), carbaryl (Sevin), bendiocarb (Ficam), methomyl (Lannate) and propoxur (Baygon).

1) Mechanism of action on both target and nontarget species: Reversible cholinesterase inhibition, of acetyl cholinesterase (AchE) in Red Blood Cells and Brain, and in Plasma cholinesterase resulting in a decrease of cholinesterase activity in the central, parasympathetic and sympathetic nervous systems.

2) Signs and symptoms: Tightness in the chest, wheezing, increased salivation and lacrimation, increased sweating, nausea, vomiting, abnormal cramps, diarrhea, tenesmus and involuntary defecation, involuntary urination, and constriction of the pupils. Muscular effects include weakness, involuntary twitching, and cramps. Central nervous system effects include anxiety, restlessness, insomia, headache, emotional instability, neurosis, excessive dreaming and nightmares, apathy and confusion. Slurred speech, tremor, ataxia, depression of circulatory centers, and coma are other CNS effects. CNS effects of carbamates are not as severe as organophosphates. Death results from respiratory failure. Onset of symptoms is usually between 15 and 30 minutes, sometimes more rapid. Non-fatal carbamate poisoning is self limiting and symptoms usually fade within 5 to 6 hrs with complete recovery occuring in 24 hrs.

3) Laboratory confirmation: Due to the rapid reversibility (in vivo and in vitro) of the carbamylation of the cholinesterase enzyme, measuring cholinesterase activity levels are not reliable indicators of carbamate poisoning. Confirmation of the poisoning can be accomplished by measuring unique metabolites of the carbamates in the urine.

### *C. ORGANOCHLORINE PESTICIDES*

Organochlorine insecticides (tradenames) used in Maine: endosulfan (Thiodan), lindane (Lindane), methoxychlor (Marlate), dicofol (Kelthane).

1) Mechanism of action on target and nontarget species: Interference with fluxes of cations (Sodium, Potassium, and Calcium) across the nerve cell membranes.

2) Signs and symptoms: Apprehension, excitability, headache, nausea, vomiting, dizziness, incoordination, tremors, disorientation, weakness, paresthesia, and convulsions. Convulsions in the absence of other symptoms may occur in cyclodiene and toxaphene poisoning. Seizures may be followed by coma and respiratory depression. Death can result from interference with pulmonary gas exchange and severe acidosis from the convulsions.

3) Laboratory confirmation**:** Pesticide and/or metabolites can usually be identified in blood or urine by gas-liquid chromatographic examination of samples taken within 72 hours of poisoning.

### *D. PYRETHRINS AND SYNTHETIC PYRETHROIDS*

Pyrethrins and pyrethroids (tradenames) used in Maine: fenvalerate (Asana, Pydrin), cyfluthrin (Baythroid), permethrin (Ambush, Pounce), cypermethrin (Demon), resmethrin (Resmethrin).

1) Mechanism of action on both target and nontarget species: Disruption of open sodium channels in neuronal membranes.

2) Signs and symptoms: Pyrethrins and pyrethroids are low in mammalian toxicity, however overexposure can lead to hypersensitivity reactions such as stuffy/runny nose, asthmatic wheezing, sudden bronchospasm, swelling of oral and mucous membranes and shock. Delayed appearance of dyspnea, cough and fever with patchy lung infiltrates suggest hypersensitivity pneumonitis. Nervous irritability, tremors, and ataxia have occurred rarely in individuals who have had massive inhalation exposure. Pyrethrins, especially crude preparations, contain dermal and respiratory allergens.

## II. HERBICIDES

### *A. CHLOROPHENOXY HERBICIDES*

Chlorophenoxy herbicides (tradenames) used in Maine: 2,4-dichlorophenoxyacetic acid, salts and esters (2,4-D, Weedone, Formula 40, Weedar); 2-methyl-4-chlorophenoxy acetic acid, salts, or esters (MCPA, Weedone), dicamba (Banvel).

A common mixture of 2,4-D, MCPA and Dicamba is call Trimec and is often found in commercial Weed and Feed lawn care products. Triclopyr (Garlon) is also used in Maine. It is a structural analog of the chlorophenoxys, a chloropyridinyl herbicide.

1) Mechanism of action:

a. *in broadleaf plants*: Mimics the plant growth hormone auxin causing growth stimulation

b. *in mammals*: Poorly understood

2) Signs and symptoms: Irritation of the skin (dermal exposure) and GI tract for oral exposures. Inhalation may result in burning sensation in nasopharynx and chest. Oral exposure leads to vomiting, pain in chest and abdomen, and diarrhea may ensue. Headache, mental confusion, and bizarre behavior may be seen. Unconsciousness and myotonia are other signs. Convulsions occur rarely.

3) Laboratory confirmation: Measure the chlorophenoxy in the blood or urine using Gas Liquid Chromatography.

### *B. DIPYRIDILS*

Dipyridils (tradenames) used in Maine: diquat (diquat) and paraquat (Gramoxone).

1) Mechanism of action in target and nontarget species:

After undergoing NADPH-dependent one electron reduction, free radicals are formed and lipid peroxidation occurs.

2) Signs and symptoms: Tissue damage: blistering and ulceration on skin, nose bleeds with inhalation, and severe eye irritation and even corneal opacity on contact. Sufficient absorption can occur from skin for a systemic response.

When ingested, Phase I toxicity includes: intense nausea, vomiting, diarrhea, distension of the GI tract, gastrointestinal ulceration, hyperexcitability and convulsions.

Damage to the liver and kidney, and myocardial and skeletal muscles occur in phase II.

The third phase of paraquat toxicity involves deposition in the lungs with subsequent damage to the lungs regardless of the route of exposure. Pulmonary edema occurs within 2 - 14 days of exposure. Lung effects, as seen in paraquat poisoning, are not produced by diquat. In addition there are severe CNS problems not seen in paraquat poisoning.

3) Laboratory confirmation: Qualitative and quantitative methods are available at some toxicology laboratories. Call Zeneca Ag Products at 1-800-327- 8633 for toxicology and management consultation 24 hrs/day. They will provide Bentonite clay and paraquat analysis.

### *C. MISCELLANEOUS HERBICIDES*

Herbicides (tradenames) used in Maine: maleic hydrazide (Royal MH-30, Sprout stop); glyphosate (Roundup, Rodeo, and Accord); Triazines--atrazine (Aatrex), hexazinone (Velpar, Pronone), metribuzin (Sencor), simazine (Princep, Aquazine) and cyanazine (Bladex); Carbamate herbicides (weak if any, inhibitors of cholinesterase)--EPTC (Eptam), pendimethalin (Prowl, Stomp), napropamide (Naproguard), and linuron (Lorax).

1) Mechanism of action: These herbicides have mechanisms of action specific to plants, and therefore, have low acute toxicity in mammals. However if acute over exposure occurs they can be irritating to the eyes, skin and mucous membranes. Chronic effects may occur.

## III. FUNGICIDES

Fungicides are commonly used in Maine. Repeated reports of irritation or dermatitis with a history of fungicide use may help identify individuals who need personal protective equipment in order to avoid continued exposure and the chronic problems associated with occupational exposure to fungicides. Fungicides used (> 5,000 lbs sold in 1992) in Maine can be separated into two groups:

### *A. ORGANIC CHEMICALS*

Ethylene Bis Dithio carbonates (EBDC) Fungicides: Largest volume of sale on a lbs active ingredient basis for 1992. Commercial products of this group include: maneb (Dithane M-22, Manex), zineb and macozeb (Dithane M-45).

Captan (Captec), chlorothalonil (Bravo), benomyl (Benlate), dodecylguanidine (Dodine), metalaxyl (Ridomil) are different chemical classes of fungicides which were sold in amounts of < 5.000 lbs active ingredient in 1992.

Signs and symptoms: These compounds have low acute oral toxicity, may cause irritation and/or contact dermatitis, and may also have chronic toxicity problems.

### *B. INORGANIC COPPER COMPOUNDS*

**Copper sulfate -** irritating to the skin, eyes, and GI tract; corrosive to the cornea. Likely to cause systemic effects on ingestion.

**Copper oxychloride** - irritating but less likely to cause the systemic effects.

Signs and symptoms : GI irritation, headache, sweating, weakness, in some cases shock, increased liver size and jaundice, hemolysis and methemoglobin formation, albuminuria, hemoglobinuria and acute renal failure in some cases.

***References:***


Farm Chemicals Handbook (1994) Meister Publishing Company Willoughby, OH

Klaassen, C. (1996) Cassarett and Doull's Toxicology the Basic Science of Poisons McGraw-Hill NY.

Morgan D. (1989) Recognition and Management of Pesticide Poisoning USEPA 540/9-88-001.

Hayes, W. J. and Lawes, E. R. (1991) Handbook of Pesticide Toxicology Academic Press San Diego.

# SILICOSIS

## *Description of Disease*

Silicosis refers to a spectrum of pulmonary diseases attributed to the inhalation of the various forms of free crystalline silicon dioxide or silica and the tissue reaction to its presence. The extent of silicosis is described by the degree of radiographic involvement. In mild cases of classic silicosis, respiratory impairment due to silica exposure is not present. The major health concerns associated with this type are a predisposition to mycobacterial infections and to disabling progressive massive fibrosis. Pulmonary fibrosis typically does cause respiratory impairment (restrictive disease). Early detection of simple silicosis and removal from dust exposure is recommended to reduce the likelihood of developing progressive massive fibrosis. The rate at which silicosis develops varies, and the clinical presentation (acute, chronic or accelerated) is based on the time course necessary to develop the disease.

Acute Silicosis is a massive consolidation of lung tissue due to pulmonary alveolar proteinosis resulting from very heavy silica exposure, especially in an enclosed space. This form of the disease frequently results in death.

Chronic (classic) Silicosis is a slowly progressing pneumoconiosis characterized by silicotic nodules in the lungs and rounded opacities visible on chest X-Ray. Duration of exposure before this type of silicosis is first diagnosed is usually more than 20 years. Accelerated silicosis results from exposure to higher concentrations of silica over 5 to 10 years.

For chronic and accelerated silicosis, classical lung tissue pathology is: (a) collagenous nodules, with concentric "onion-skinned" arrangement of collagen fiber, central hyalinization, and a cellular peripheral capsule; and (b) lightly birefringent particles (alpha-quartz) on examination with polarized light. Depending on the relative concentration of crystalline silica compared to other minerals in the dust, the fibrotic reaction may have an irregular, haphazard pattern or be interstitial in distribution.

## *Major Occupations at Risk*

Examples of industries in Maine where there may be silica exposure include:

- Construction
- Agriculture
- Electronics
- Ceramics, clay and pottery
- Stone cutting
- Railroad (setting and laying track)
- Sand blasting
- Shipyards
- Slate and Flint Quarrying

## *Criteria for Reporting*

To meet definition for reporting, both A & B must be present.

A. History of significant exposure to airborne silica dust.

B. A chest radiograph consistent with silicosis and without other clinical explanation and/or lung tissue pathology consistent with silica exposure.

### *Reference*

Balaan MR and Banks DE. Silicosis. In: Rom WN, editor. *Environmental and Occupational Medicine*. 2nd edition. Boston: Little, Brown and Company; 1992: 345-358.

# SOLVENT TOXICITY

## *Description of the Disease*

Most organic solvents including hydrocarbons, chlorinated hydrocarbons, alcohols, ethers, esters, and ketones have the potential upon acute high level exposure to cause narcosis and death. Acute exposure can result in disorientation, euphoria, giddiness and confusion, which can progress to paralysis, convulsions and death from respiratory or cardiovasular arrest. The liver, bone marrow, kidney and nervous system have all been shown to be susceptible to severe direct effects from lower, subacute levels of some solvents. For example, carbon tetrachloride has been shown to cause both acute and chronic liver damage. Benzene has been linked to bone marrow suppression with resulting aplastic anemia. Renal failure has been caused by organic solvents. Carbon disulfide had been shown to cause both peripheral neuropathy and central nervous system toxicity.

## *Occupations at Risk*

- Painters
- Autobody Technicians
- Boat Builders
- Fiberglass workers (in industrial tank/piping manufacturers)

  Specific examples of exposures:

  - Benzene in printing, lithography and dry cleaning.
  - Carbon tetrachloride in dry cleaning.

Other very common solvents in Maine are trichloroethylene, trichloroethane and toluene.

## *Criteria for Reporting*

To meet the criteria for reporting solvent toxicity, A, B and C must be present.

A. History of occupational exposure to a specific agent. The specific substance believed to cause the injury should be named in the report.

B. Signs and symptoms of solvent toxicity. Depends on the specific organ system affected and the exposure agent.

C. Laboratory confirmation of damage. This includes laboratory evidence of damage known to be characteristic of exposure to the particular substance. For example, abnormal liver function studies consistent with acute or chronic damage resulting from benzene exposure, or abnormal nerve conduction studies consistent with a neuropathy.

# TOXIC GAS POISONING

## I. AMMONIA

### *Description of Disease*

Physical Form - A colorless gas.

Signs and symptoms - Ammonia is a severe irritant of the eyes, respiratory tract and skin. Liquid anhydrous ammonia exposure causes severe corneal irritation, dypsnea, bronchospasm, chest pain, and pulmonary edema. Chronic airways hypereactivity and asthma following massive ammonia exposure have been documented.

### *Major Occupations at Risk*

- Aluminum makers
- Annealers
- Chemical workers
- Dye makers
- Fertilizer workers
- Refrigeration workers
- Tannery workers
- Metal powder processors
- Mirror silverers
- Pesticide makers
- Pulp and paper workers
- Rayon makers
- Water treaters
- Metal Extractors

### *Criteria for Reporting*

To meet the definition for reporting, both A and B must be present

A. History of occupational exposure.

B. Presentation of symptom(s) listed above.

## II. CARBON MONOXIDE POISONING

### *Description of Disease*

Physical Form: A colorless, odorless, tasteless gas.

Signs and Symptoms: Carbon monoxide is usually encountered in industry as a waste product of incomplete combustion. It is classed toxicologically as a chemical asphyxiant, exerting its toxic action by combining with hemoglobin from performing its normal function of carrying oxygen to the tissues. The basal ganglia and subthalamus of the brain are the areas most sensitive to carbon monoxide hypoxia. Signs and symptoms of carbon monoxide poisoning include headache, weakness, chest pain, nausea, dizziness, dimness of vision, fainting, coma and convulsions. Recovery from acute exposure usually occurs without sequelae. However, severe carbon monoxide poisoning may result in pemanent brain damage.

### *Major Occupations at Risk*

- Acetylene workers
- Blast furnace workers
- Boiler room workers
- Mond process workers
- Organic chemical synthesis
- Black Carbon makers
- Diesel engine operators
- Water gas workers
- Metal oxide reducers
- Miners
- Coke oven workers
- Brewery workers
- Petroleum refinery workers
- Pulp and paper workers
- Steel workers
- Garage mechanics

### *Criteria for Reporting*

To meet the definition for reporting, both A and B must be present.

A. History of occupational exposure
B. Five percent or greater carboxyhemoglobin in blood (nonsmokers) at time of exposure

## III. CHLORINE

### *Description of Disease*

Physical Form: A greenish-yellow gas with an irritating odor.

Signs and symptoms: Chlorine is a potent irritant of the eyes, mucous membranes, and skin. Inhalation causes pulmonary edema and chemical pneumonitis. Other symptoms of exposure include burning of the eyes, nose and mouth, sternal pain, nausea, vomiting, headache, dizziness.

### *Major Occupations at Risk*

- Aerosol propellant makers
- Chlorinated solvent makers
- Refigerant makers
- Silver extractors
- Pool maintenance workers
- Tin recovery workers
- Water treatment workers
- Laundry workers
- Pesticide makers
- Plastic makers
- Rayon makers
- Disinfectant makers
- Dye makers
- Flour bleachers
- Iron workers
- Pulp bleachers

### *Criteria for Reporting*

To meet the definition for reporting both A and B must be present.

A. History of occupational exposure.
B. Presentation of symptoms listed above.

## IV. HYDROGEN SULFIDE

### *Description of Disease*

Physical Form : a colorless gas having an offensive rotten egg odor (*The offensive odor is unreliable as a warning signal because olfactory fatigue occurs).

Signs and Symptoms: Inhalation of the gas causes irritation of the eyes and respiratory tract at low concentrations. Exposure to high concentrations causes respiratory paralysis, asphyxia and possibly coma and death. Low levels may cause neurological effects, such as nervousness, and delerium. Other symptoms may include nausea, abdominal cramps, vomiting and severe diarrhea (may occur in subacute intoxication). Low levels can also produce acute conjunctivitis, lacrimation, photophobia, rhinitis, pharyngitis, bronchitis, and pneumonitis.

Chronic effects are less well established.

### *Major Occupations at Risk*

- Barium carbonate
- Photoengravers
- Caisson workers
- Cellophane makers
- Coke oven workers
- Depilatory makers
- Dye makers
- Fat renderers
- Felt makers
- Lithographers
- Miners
- Natural gas makers
- Rayon makers
- Sewage treatment workers
- Sewer workers
- Slaughterhouse workers
- Soap makers
- Sugar beet processors
- Sulfuric acid purifiers
- Synthetic fiber makers
- Tannery workers
- Tunnel workers
- Well diggers
- Pulp and paper workers

### *Criteria for Reporting*

To meet the definition for reporting, both A and B must be present.

A. History of occupational exposure.
B. Presentation of symptoms listed above.

## V. MISCELLANEOUS

There are other gases with the potential for toxic exposure in Maine workplaces. The more common ones with examples of occupations at risk include:

a. Nitrogen Oxides: Dentists, Garage workers, Welders

b. Freon: Brazier, Refrigeration workers, Air conditioning workers, Degreaser foam blowers

c. Ozone: Arc welders

d. Sulfur Dioxide: Water treatment, Pulp mill workers, Firemen, boiler house workers

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